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Effects of toxic chemicals in the marine environment: predictions of impacts from laboratory studies

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The degree to which toxicity testing can lead to predictions of long-term environmental consequences of contaminant exposure has been widely debated. Laboratory approaches designed to address both chemical concerns of contaminant bioavailability and persistence in addition to biological concerns of sublethal effects on marine organisms would be most useful in providing the linkage between laboratory and field evaluations. Examples of bioenergetic, developmental, and reproductive abnormalities observed with exposure to lipophilic organic contaminants are discussed in reference to consequences at higher levels of biological organization. Alterations in bioenergetics linked with observations of reduced fecundity and viability of larvae, abnormalities in gamete and embryological development, and reduced reproductive effort provide a strong empirical basis for examination of population responses. Such empirical data can be incorporated into population models to assess the effects of energetic, reproductive and developmental aberrations on population success and provide the basis for further examining the predictive value of toxicity testing.

Key words: Toxicity; Chemical pollution; Marine environment

INTRODUCTION

The toxic effects of chemical contaminants on marine organisms are dependent on bioavailability and persistence, the ability of organisms to accumulate and metabolize contaminants, and the interference of contaminants with specific metabolic or ecological processes. A wide range of toxicity tests has been developed

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in recent years to provide rapid and accurate screening of chemicals for toxic effects. Acute toxicity tests are useful in defining guidelines for maximum allowable concentrations of toxicants in effluents and other wastes and in comparing relative sensitivities of organisms to contaminants and predicting the environmental consequences of contaminant inputs. Although the conceptual framework of toxicity testing has changed little since it was first used in assessing water quality (see review by Buikema et al., 1982), modifications in design have expanded its utility in biomonitoring (Hansen, 1985; Gentile and Schimmel, 1985; Long and Chapman, 1985; Stebbing, 1980). Further development of toxicity testing procedures has occurred recently for the application of such methods to specific waste management problems, such as ocean dumping, ocean incineration, disposal of dredged materials, and characterization of complex industrial mixtures.

The degree to which acute toxicity testing can lead to predictions of long-term environmental consequences of contaminant exposure has been widely debated. After comparing margins of uncertainty for survival in acute single species toxicity tests, chronic exposure, and multispecies tests, Sloof et al. (1986) concluded that acute toxicity tests were useful in predicting both the chronic responses of single species and multispecies/ecosystem responses. They further suggested that research effort would be better directed toward understanding of underlying mechanisms of acute response coupled with estimates of chemical transformations and bioavailability of contaminants to allow site-specific estimates of ecological impact to be made. Woltering (1985), however, argued that single species tests based on survival could not adequately predict the indirect consequences of contaminant exposure, specifically on population processes, alterations in life history patterns, and species interactions.

Gentile and Schimmel (1985) suggested that in order for the results of toxicity testing to be used effectively in assessing environmental consequences, the design of such tests must: (1) couple laboratory derived evaluations of toxicity with environmentally realistic estimates of contaminant exposure; (2) lead to predictions of field consequences; and (3) when combined with monitoring approaches, serve as a feedback mechanism to verify or compare field observations with predictions. Harwell et al. (1986) discussed the uncertainties associated with extrapolation of the results of toxicity testing to predictions of ecological response and recommended that environmental decision-making should include a comprehensive retrospective analysis of laboratory predictions and field responses to strengthen our predictions of environmental impact. Often lacking in laboratory and field assessments of environmental impacts are the predictive linkages among various testing procedures. Koojiman and Metz (1984) suggested that the sublethal effects of contaminant exposure should be interpreted in light of the survival probabilities and reproductive success of populations, thus bridging the gap between individual and population responses. Laboratory approaches designed to address both chemical concerns of contaminant bioavailability and persistence in addition to biological concerns of

sublethal effects on marine organisms would be most useful in providing the linkage between laboratory and field evaluations.

BIOLOGICAL EFFECTS

The responses of organisms to toxic chemicals can be manifested at four levels of biological organization: (1) biochemical and cellular; (2) organismal, including the integration of physiological, biochemical and behavioral responses; (3) population, including alterations in population dynamics; and (4) community, resulting in community structure and dynamics (Table I). Biological effects can be manifested at biochemical, cellular, and organismal levels of organization before disturbances

TABLE I

Response levels of marine organisms to chemical contaminants.

Level	Types of responses	Effects at next level
Biochemical-cellular	Toxication	Toxic metabolites
	Metabolic impairment	Disruption in energetics and cellular processes
	Cellular damage	
	Detoxication	Adaptation
Organismal	Physiological changes	Reduction in population performance
	Behavioral changes	
	Susceptibility to disease	
	Reproductive effort	
	Larval viability	
	Adjustment in rate functions	Regulation and adaptation of populations
	Immune responses	
Population	Age/Size structure	Effects on species productivity and coexisting species and community
	Recruitment	
	Mortality	
	Biomass	
	Adjustment of reproductive output and other demographic characteristics	Adaptation of population
Community	Species abundance	Replacement by more adaptive competitors
	Species distribution	
	Biomass	Reduced secondary production
	Trophic interactions	
	Ecosystem adaptation	No change in community structure and function

at the population level develop (Capuzzo, 1981). All responses are not disruptive in nature and do not necessarily result in degeneration at the next level of organization. Only when the compensatory or adaptive mechanisms at one level begin to fail do deleterious effects become apparent at the next level. An important aspect of comparing responses at various levels of biological organization is ascertaining the degree to which adaptive responses at each of the four levels can persist with increasing concentrations of contaminants. The initial responses in each case are the triggering of mechanisms to reduce or resist the impact of the toxicant; these mechanisms may include the induction of toxicant-metabolizing processes (at the biochemical level) or the selection of toxicant resistant forms (at the population level). Adaptive processes are capable of countering disruptive processes until the system reaches a threshold for the toxicant, at which point the adaptive potential is completely overridden by the degeneration imposed on the system by disruptive effects.

For predictive purposes, it is important to understand the early warning signs of stress at each level of organization before compensatory mechanisms are surpassed. From the biochemical level to the community level, the degree of system complexity, the number of compensatory mechanisms available and the lag time to measure a response increase dramatically, thereby increasing the predictive difficulties at each level. Chronic exposure to chemical contaminants can result in alterations in reproductive and developmental potential of populations of marine organisms, resulting in possible changes in population structure and dynamics. It is difficult to ascertain, however, the relationship between chronic responses of organisms to contaminants and large-scale alterations in the functioning of marine ecosystems or the sustainable yield of harvestable species. Cairns (1983) argued that our ability to detect toxic effects at higher levels of biological organization is limited by the lack of reliable predictive tests at population, community, and ecosystem levels. He further suggested that much effort is needed in these areas before we can adequately address environmental hazards as a result of contaminant inputs.

Although a wide range of sublethal stress indices have been proposed for evaluation of chronic responses of organisms to contaminants, few have been linked to the survival potential of the individual organism or the reproductive potential of the population. Experimental studies directed at determining effects on energy metabolism or effects that influence growth and reproduction would be most appropriate for linking effects at higher levels or organization.

Lipophilic organic contaminants such as PAHs, PCBs and other synthetic compounds, are highly resistant to degradation and such compounds or their metabolites may accumulate to high levels in animal tissues and interfere with normal metabolic processes that affect growth, development, and reproduction. The bioavailability, bioaccumulation, and toxic effects of lipophilic contaminants are related to their pharmacological and toxicological properties (Widdows et al., 1987; Donkin and Widdows, 1986; Capuzzo, 1984, 1987). In a review of the effects of

petroleum hydrocarbons on marine organisms, Capuzzo (1987) concluded that the differential sensitivity of different phylogenetic groups, various life history stages, and species from different biogeographical regions were related to hydrocarbon bioavailability, capacity for hydrocarbon biotransformation, and the metabolic consequences of hydrocarbon exposure. The increased sensitivity of early developmental stages and the seasonal differences in the responses of adult animals may be related to stage-specific or seasonal dependency on particular metabolic processes (e.g., storage and mobilization of energy reserves, hormonal processes), with the result of altering developmental and reproductive success.

An understanding of reproductive and developmental processes provides the

TABLE II

Bioenergetic, reproductive, and developmental abnormalities of marine animals exposed to lipophilic organic contaminants.

Species	Contaminant	Effect	Reference
Acartia tonsa	Fenvalerate	Altered feeding, respiration, and egg production Reduced viability of early developmental stages Reduced yolk reserves	Capuzzo (unpubl.)
	Fenthion	Altered feeding, respiration, and egg production	Capuzzo (unpubl.)
Centropages hamatus	Crude oil	Reduced feeding and egg viability	Cowles and Remillard (1983)
		Altered food perception	Cowles (1983)
Clupea harengus	Benzene	Delayed embryonic development	Eldridge et al. (1977)
	Crude oil	Morphological abnormalities	Smith and Cameron (1979)
Eurytemora affinis	High aromatic oil	Reduced egg production	Berdugo et al. (1977)
	Naphthalene and methylated derivatives	Reduced feeding Reduced reproductive effort	Ott et al. (1978)
Hypomesus pretiosus	Crude oil	Necrotic neurons in embryos	Hawkes and Stehr (1982)
Homarus americanus	Crude oil	Reduced respiration, delayed molting, and reduced growth Altered lipid storage utilization	Capuzzo et al. (1984)
Mercenaria mercenaria	Phenol	Cytological damage to gills and digestive gland	Fries and Tripp (1977)

TABLE II, continued.

Species	Contaminant	Effect	Reference
Mya arenaria	No. 6 fuel oil	Reduced carbon flux	Gilfillan et al. (1976)
Mytilus edulis	Aromatic hydrocarbons	Lysosomal function and structure	Lowe et al. (1981, (1982) Moore and Clarke (1982) Moore et al. (1987) Pipe and Moore (1985)
		Increased degeneration of oocytes Atrophy of digestive tubule epithelium Reduction in gamete volume	Lowe and Pipe (1985)
	North Sea oil	Reduced scope for growth	Widdows et al. (1982)
	Diesel oil	Reduced scope for growth	Widdows et al. (1985)
Paracentrotus lividus	Benzo[*a*]pyrene	Abnormal embryonic cleavage	Ceas (1974)
Parophrys vetulus	Benzo[*a*]pyrene	Metabolite binding to DNA	Varanasi and Gmur (1981)
Pleuronectes platessa	Crude oil	Suppressed ovarian development	Stott et al. (1983)
Rhithropanopeus harrisii	No. 2 fuel oil	Delayed development	Laughlin et al. (1978)
	Phenanthrene	Developmental abnormalities, delayed development	Laughlin and Neff (1979)
Umbra pygmaea	Benzo[*a*]pyrene	Chromosomal aberrations and increased sister chromatid exchange	Hooftman and Vink (1981)

critical link between responses to contaminants at the organismal and suborganismal levels and population consequences. Examples of bioenergetic, developmental, and reproductive abnormalities observed with exposure to lipophilic organic contaminants are presented in Table II. Alterations in bioenergetics linked with observations of reduced fecundity and viability of larvae, abnormalities in gamete and embryological development, and reduced reproductive effort provide a strong empirical basis for examination of population responses. Critical factors that may contribute to the impairment of reproductive and developmental processes in response to exposure to lipophilic contaminants include: (1) deposition of contaminants in gametes and developing embryos; (2) lysosomal dysfunction associated with oocyte resorption; (3) interference with feeding mechanisms, such that exposure mimics starvation responses; (4) failure to incorporate sufficient yolk in oocytes; (5) morphological abnormalities during embryogenesis resulting from failure of morphological systems to develop properly; (6) limited capacity of developmental stages to metabolize or depurate contaminants; and (7) limited capacity of early developmen-

tal stages and reproducing adults to draw on excess energy reserves. Linkages clearly exist between (1) developmental, reproductive, and energetic abnormalities; (2) the physiological and molecular processes involved in uptake, retention, and loss of lipophilic contaminants; and (3) the toxicity and transformation of lipophilic contaminants. Such empirical data can be incorporated into population models such as those derived by Koojiman and Metz (1985) to assess the effects of energetic, reproductive and developmental aberrations on population success and provide the basis for examining further the predictive value of toxicity testing in evaluating environmental consequences.

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