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The developing framework of marine ecotoxicology: Pollutants as a variable in marine ecosystems?

Samuel N. Luoma

Mail Stop 465 US Geological Survey 345 Middlefield Road, Menlo Park, CA 94025, USA

Abstract

Marine ecosystems include a subset in which at least some interrelated geochemical, biochemical, physiological, population and community characteristics are changed by pollutants. Moderate contamination is relatively widespread in coastal and estuarine ecosystems, so the subset of ecosystems with at least some processes affected could be relatively large. Pollutant influences have changed and will probably continue to change on time scales of decades. Biological exposures and dose in such ecosystems are species-specific and determined by how the species is exposed to different environmental media and the geochemistry of individual pollutants within those media. Bioaccumulation models offer significant promise for interpreting such exposures. Biological responses to pollutants need to be more directly linked to exposure and dose. At the level of the individual this might be improved by better understanding relationships between tissue concentrations of pollutants and responses to pollutants. Multi-discipline field and laboratory studies combined with advanced understanding of some basic processes have reduced the ambiguities in interpreting a few physiological/organismic responses to pollutants in nature. Recognition of pollutant-induced patterns in population responses could lead to similar advances. A rational framework for ecotoxicology is developing, but its further advance is dependent upon better integration of ecotoxicology with basic marine ecology and biology.

1. Introduction

1.1. Ecotoxicology needs a 'coherent set of ideas to guide the study of pollutant effects', Bayne, 1988.

Ecotoxicology 'integrates the ecological and toxicological effects of chemical pollutants on populations, communities and ecosystems with the fate (transport, transformation and breakdown) of such pollutants in the environment', (Forbes and Forbes, 1994). The need for a coherent framework in this new field was recognized by (Bayne et al., 1988). Depledge (1994) also described the field as only recently being

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more than a 'collection of procedures for protecting the environment through management and monitoring of pollutant discharges'. In this review, I suggest that growing knowledge is beginning to produce the desired framework. The components include recognition of the spatial extent and temporal trends of contamination in marine ecosystems and the growing understanding of how ecosystem processes affect pollutant concentrations, fate and distributions. Improved recognition of the complex routes by which marine organisms are exposed to pollutants contributes to the framework. The framework also includes recognition of effects of pollutants as a continuum of responses that occur across all levels of biological organization. Multi-disciplinary strategies to successfully define pollutant influences are developing, as are strategies for relating lower level effects on individuals to higher level effects on populations. Advances in understanding effects at the community level of organization are also occurring, although these will not be discussed here.

An important part of the new ecotoxicologic framework is recognition that standardized, 'simple', ecotoxicologic approaches lack power (i.e. are insensitive) in explaining implications of pollutants in complicated circumstances (Cairns, 1992; Luoma, 1995). Without more sophisticated understanding, ecosystem managers will continue to have difficulties using science to determine when changes in guidelines or regulations are justified. A more coherent framework might view pollutants as just one of the several influential physical, chemical or biological variables in many modern marine ecosystems. The goal of pollutant management then becomes understanding the ecological and biological circumstances where pollutants are influential, as a substitute for searching for the best tool to demonstrate 'damage' from pollutants.

Finally, the framework includes a growing appreciation of the need for better mechanistic understanding, especially of biological and ecosystem processes. Inadequate communication between ecotoxicologists and more traditionally trained ecologists, biologists and geochemists is a long-standing cause for concern (Cairns, 1992; Luoma and Carter, 1991; Iannuzzi et al., 1995). There is a need for greater appreciation of ecosystem processes and basic biology in studies of pollutant effects. Greater consideration of pollutants as an influential variable also would benefit basic ecosystem research and speed the effective flow of concepts and techniques between basic research and pollution research communities (Azam, 1995).

Much of the research that establishes the expanding framework of ecotoxicology is discussed in discipline-specific reviews (a few recent examples with relevant marine information include Baudo et al., 1990; Newman et al., 1991; Coull and Chandler, 1992; Huggett et al., 1992; Dalinger and Rainbow, 1993; Phillips and Rainbow, 1993; Malins et al., 1994). The present discussion will be limited to describing the components of the framework suggested above. How widespread is contamination in marine ecosystems? Are pollutant influences changing on decadal time scales? How are marine organisms exposed to pollutants? Do such exposures affect the (Darwinian) fitness of individuals over large spatial scales (i.e. outside of extreme hotspots)? Can pollutant-induced changes in fitness be related to changes in populations of marine organisms? Specific discussions of pollutant effects will be constrained to synthetic organic compounds (e.g. organochlorine pesticides), natural products that can be toxic (e.g. PAH's), and trace elements (e.g. heavy metals like mercury, copper), although it is recognized that types of pollutants vary widely.

2. How widespread is contamination in marine ecosystems?

Generalizations that describe distributions of pollutants in marine ecosystems are increasingly possible, as a result of a growing body of work deriving from analytical advances and improved approaches to characterization studies. Most trace chemical contamination is presently restricted to coastal waters and estuaries, although pollutant enrichment attributable to human activities can be detected in some open ocean waters (Sanudo-Wilhelmy and Flegal, 1992) or even off the continental shelf near areas of sludge dumping (Bothner et al., 1994). Contamination is most intense nearest human activities (Serricano et al., 1990; Hanson et al., 1993; Daskalakis and O'Connor, 1995), but the details of distributions can be modified by physical processes that dilute, concentrate or redistribute the pollutants. Concentrations are also most variable in the most contaminated ecosystems (Luoma, 1990).

Dissolved pollutants tend to be well mixed and distributions may be more homogenous than distributions in sediments, if properly analyzed and interpreted (e.g. Flegal et al., 1991). Biological events, such as phytoplankton blooms and episodic or seasonal changes in contaminated inflows can affect distributions of dissolved contaminants. Ecosystem-scale contamination in sediments or the tissues of sessile biota usually includes superimposed 'hot spots' that reflect, sometimes numerous, local sources of pollutant input and their zones of dilution (O'Connor and Huggett, 1988; Luoma and Phillips, 1988). Characterization and monitoring of pollutant concentrations and trends at relevant spatial scales could play an increasing role in advancing ecotoxicologic knowledge, although effective and efficient designs for monitoring pose a challenge that is often under-appreciated.

The frequency distribution of contaminant concentrations in coastal ecosystems (e.g. in sediments) is typically log-normal (Grimalt et al., 1992). Only a small proportion of locations are heavily contaminated, but moderate contamination appears to be widespread. Within a sediment database of 13 500 sediment samples from coastal US waters, 70% of localities had concentrations of at least one pollutant at a level 1 standard deviation higher than the overall geometric mean concentration of all samples; 15% had concentrations of at least one chemical 5 times higher than the geometric mean (Daskalakis and O'Connor, 1995). The geometric mean is, of course, higher than background concentrations because of the influence of high values. Hanson et al. (1993) defined 'geologic background' in US coastal sediments by correlation with aluminium among sites presumed to be undisturbed by human activities. Fig. 1 illustrates the frequency of contamination by copper and lead as deviations from that baseline.

3. Are pollutant concentrations changing?

Through the last century, the history of contamination is one of continual change, as indicated by concentrations of particle reactive contaminants in dated sediment cores from North America and Europe (Valette-Silver, 1993). In San Francisco Bay, for example, combustion products (PAH's) and Hg (the latter probably from mining activities) dominated the chemical mixture in sediments between 1900 and 1950 (van Geen et al., unpub.)(Fig. 2). Enrichment of a complex mixture of pollutants increased

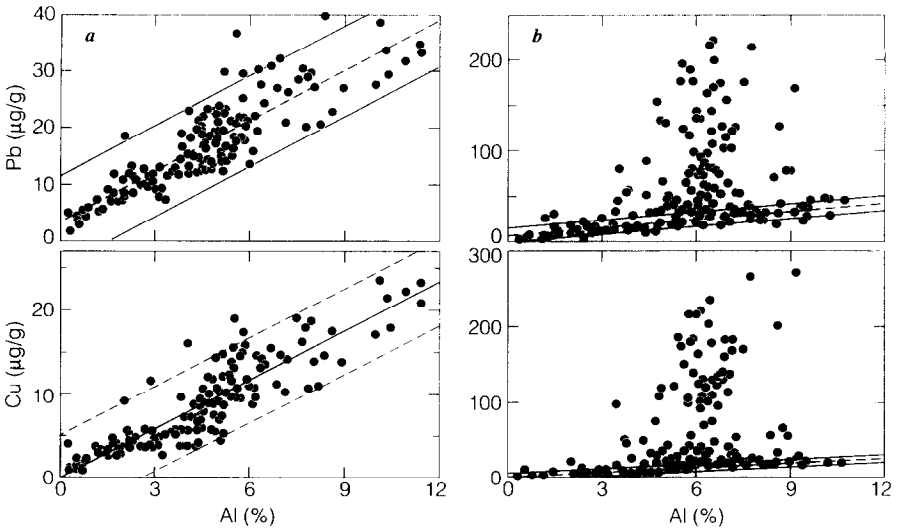

Fig. 1. Concentrations of lead and copper in marine sediments from the coastal zone of the U.S. as collected by National Status and Trends monitoring. (a) Reference conditions are established by correlation of metal concentrations with aluminum from stations not expected to be enriched by human activities. (b) The frequency of contaminated sites is evidenced by positive deviations from the reference relationship. Copper and lead contamination do not necessarily co-occur. (Re-drawn from Hanson et al., 1993).

after World War II, when economic and population growth were rapid and waste treatment to remove trace pollutants was of minimal concern. The highest concentrations of PCB's, DDT's, PAH's, Se, Cd, Hg, Ag, Cu, Zn and Pb occurred in about the 1960's–1980's. Societies in North America and Western Europe invested heavily in advanced waste treatment, beginning in the late 1960's and controls were imposed on use of some chemicals (some forms of methylated mercury, organochlorine pesticides; PCB's; lead). Two decades later, modern sediments retain the imprint of human activities but concentrations of at least some individual pollutants have declined in organisms and sediments (Fig. 3). The mixture of pollutants to which organisms are exposed today is simpler than that present before the investment (Fig. 2).

The trends in sediment contamination were accompanied by some documented biological trends (and probably changes that were not documented). Pollutants caused the reproductive failures and population declines in seed eating birds and piscivorous birds, first recognized during the late 1970's. Pollutants could also have contributed to some of the declines in marine mammals and fishes that occurred during that period and later (Kitchell, 1995). Assuming toxicity is additive among pollutants, the mixture of contaminants in San Francisco Bay in the 1970's would have caused acute toxicity in sediment bioassays (to species such as amphipods), had such tests been available (extrapolating from data assembled by Long et al., 1995). Recoveries of brown pelican, peregrine falcon and bald eagle appear directly related to declining loads of the pollutants; recoveries of native species of large fish (lake trout, burbot, native ciscoe,

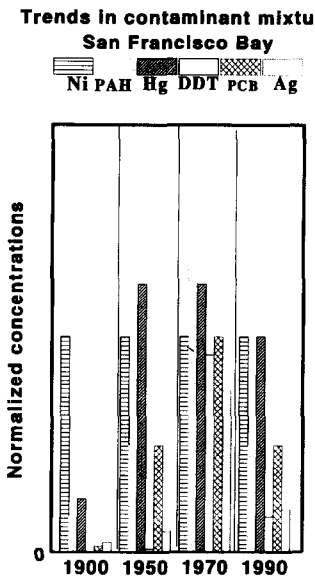

Fig. 2. Changes in the mixture of contaminants through time as observed in sediment cores from San Francisco Bay (van Geen et al., unpublished). Concentrations are normalized to the same scale. Dates, obtained by analysis of ^{137}Cs and ^{210}Pb, are approximate because the sediments are mixed 20–30 cm in some areas. The complexity of the contaminant mixture increased from near the turn of the century until the 1960–1980 period. The contaminant mixture was simpler in surface sediments in 1990 than when maximum concentrations were observed, but more complex than in sediments deposited before industrialization accelerated. Nickel concentrations are naturally high in the area, due to Ni-rich ultramafic rocks on the watershed.

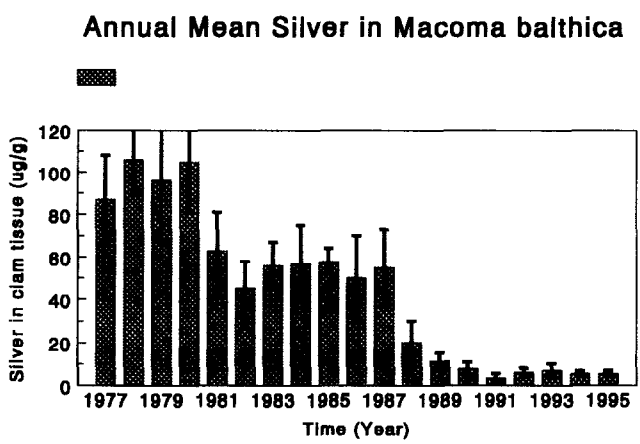

Fig. 3. Annual mean concentrations of silver in the soft tissues of the clam *Macoma balthica* collected at near monthly intervals between 1977 and 1995 at a mudflat near a treatment works in South San Francisco Bay. Declining trends in contamination are evident with the increased investment in advanced waste treatment since 1980.

walleye) and perhaps even mayflies in ecosystems like the Great Lake may include a pollutant component (Kitchell, 1995).

Although some of the chemicals traditionally associated with industrial wastes are declining in concentration, new chemicals and new combinations of chemicals are released to marine ecosystems faster than analytical procedures can be developed. Effects of tributyl tins (TBT) may be reaching proportions analogous to that of the most potent industrial contamination in the 1970's (Huet et al., 1996). A new generation of pesticides is mobilized in complex ways and may manifest their effects differently than those of the pesticides that were banned. Moreover, the chemical effects of industrialization in the ecosystems affected by the majority of the world's population have not been moderated by advanced waste treatment and/or regulation. Implicit and explicit societal decisions about investments in waste treatment determine the effects of pollutant-generating activities. The consequences for ecosystems lag those decisions by a decade or two. As societal attitudes and economic growth change, pollutant concentrations and their influences in marine ecosystems will continue to change with time.

4. How are organisms exposed to pollutants?

Assessing ecosystem risks from pollutant enrichment requires understanding the exposures of organisms, food webs and ecosystems to pollutants. Exposure is determined by concentration and bioavailability at the scale proximate to an organism and by processes that control the fate of the chemical on the ecosystem scale.

4.1. Biological availability

Dose is the concentration of a pollutant (or its metabolites) that appears in an organism's tissues and is the starting point for adverse effects (all toxicity is derived from dose-response relationships). Bioavailability determines dose; it is the relationship between pollutant concentrations in the environment and pollutant bioaccumulation (uptake into tissues from all sources). Bioaccumulation is not related to total pollutant concentrations (Sunda and Guillard, 1976; MacKay, 1982; DiToro et al., 1990); nor is it possible to measure one chemical fraction that is universally and exclusively the bioavailable fraction for any chemical.

Wide differences occur in bioaccumulation among organic chemicals; those that are more hydrophobic and more lipophilic are, in general, concentrated more efficiently by biota. In the broadest sense, concentrations in each environmental medium (including tissues) are defined at equilibrium by the fugacity of the chemical: the concentration times a proportionality constant (MacKay and Diamond, 1989). The constants are determined by the properties of the chemical and critical characteristics of the medium (e.g. relationships with octanol–water partitioning, normalization to lipid content of organisms and normalization to organic content of sediments). At equilibrium, all environmental media should be at equifugacity, theoretically allowing prediction of distributions among media, including organisms (Clark et al., 1990).

Fugacity has not been particularly useful for predicting distributions or bioavailability

of trace elements. Bioaccumulation of metals cannot be explained by a single proportionality constant linked to a single principle like hydrophobicity. This is probably because of the myriad of reactions that occur within organisms, within solution and on the surface of particles. The bioavailability of dissolved Cu, Cd, Pb or Zn correlates with the activity of the free ion (or the chemical potential of the metal) not total metal (Sunda and Guillard, 1976; Sunda et al., 1978; Anderson and Morel, 1978; Campbell and Tessier, 1989). The dominance of free ion activity as a control on bioavailability of dissolved trace elements is not universal, however. Occurrence of the chloro-complex of dissolved Ag in marine waters correlates with uptake, suggesting transport pathways exist for this form, in addition to that for the free Ag ion (Engel and Fowler, 1979). Methylation of some trace elements (e.g. Hg and Sn) enhances bioavailability by facilitating additional modes of transport and sequestration. Oxidation state controls the bioavailability of elements that behave as anions (Se, As, Cr, V).

Bioaccumulation of pollutants is not fully predictable from the above principles alone. Bioavailability from sediments, for example, has been assessed by chemical extractions (Luoma, 1989), normalization to geochemical characteristics of sediments (such as iron oxides–Luoma and Bryan, 1978; Langston, 1980, 1982; or acid volatile sulfides–DiToro et al., 1990), or equilibrium models that predict pore water concentrations (DiToro et al., 1991; Tessier et al., 1993; Amyot et al., 1994). Each of these advances provided some of the ingredients needed to explain when, where and how metals in sediments become bioavailable, but none has yet proven to be a universally acceptable approach.

In nature, concentration ratios between fish tissues and water consistently exceed those predicted from fugacity alone for organic chemicals (Oliver and Niimi, 1988; Swackhamer and Hites, 1988; Bremle et al., 1995). In some circumstances trace element bioaccumulation can be determined from models that predict chemical potential in solution alone (as in studies of Cd bioaccumulation by freshwater mussels in Quebec, Tessier et al., 1993). But, in the same ecosystems, complexities such as seasonal variability in bioaccumulation (Amyot et al., 1994) or species-specific differences in bioaccumulation (Hare et al., 1994) are not predictable. Luoma et al. (1992) showed that neither total dissolved Se concentrations nor knowledge of Se speciation in solution was, alone, sufficient to predict bioaccumulation of this element by clams in San Francisco Bay.

A number of factors contribute to the difficulties of predicting pollutant bioavailability in nature. Poorly known reactions with natural organic materials made it difficult to determine metal activities in solution until recently (Hare and Tessier, 1996). Reactions of trace elements with particle surfaces are difficult to model in marine sediments where particles are complex multi-ligand aggregates (Luoma and Davis, 1983). Scale is a difficult problem, in that sediments and pore waters are usually characterized geochemically on scales much broader than the micro-habitat scales experienced by benthic organisms. The geochemical and ecological influences of the living component of sediments (diatoms, meiofauna, bacteria) are difficult to separate and their geochemical or biological role is poorly known (Decho and Luoma, 1996).

The most important factor, however, is that the dose an organism receives in nature is not a function of just the geochemistry of one medium (Clark et al., 1990; Luoma et al., 1992; Landrum et al., 1992; Luoma and Fisher, in press). An organism's exposure to the

chemicals in its environment is integrated among media. Pollutants are distributed among solution, suspended particles, sediments, pore waters and specific (living and non-living) food sources within all of these. Each species' exposure to those pollutants is detemined by how that species 'samples' this complex milieu and by the accessibility of pollutants within each compartment of the milieu. In biological terms, consideration of singular environmental media is artificial and inhibits understanding bioavailability processes.

Acceptance that pollutant exposures occur via multiple pathways was slowed by controversy about the role of dietary pollutant uptake. Pollutant bioaccumulation or toxicity can be related to dissolved uptake alone under some experimental conditions (Kemp and Swartz, 1988). But it has also been demonstrated repeatedly that organic and inorganic pollutants can be taken up by organisms from at least some ingested materials (Luoma, 1989; James and Kleinow, 1994). Although they superficially appear contradictory, such results, in fact, suggest neither pathway can be excluded from consideration. Pathway contributions depend upon specific circumstances. Assimilation of ingested trace elements differs with food type and availability from solution differs with geochemical conditions (Renfro et al., 1975; Luoma and Jenne, 1977; Reinfelder and Fisher, 1991; Decho and Luoma, 1994). In addition, where experiments have separated pathways of bioaccumulation, and pollutants were bioavailable from food, the pathways were additive (Luoma and Jenne, 1977; Weston, 1990; Chandler et al., 1993). Thus, experimentally, all pathways must be resolved to avoid underestimating full exposures. Conceptual and quantitative bioaccumulation models that predict tissue burdens of organisms under a variety of circumstances also should include a dietary term and a dissolved term that are additive. Bioaccumulation models were first developed for marine ecosystems by radioecologists (Pentreath, 1973). Additive pathways have been successfully incorporated into fugacity-driven bioaccumulation models, improving explanations of organic chemical concentrations in fishes (Thomann, 1989; Clark et al., 1990). Kidd et al. (1995) used stable isotopes of N and C to trace trophic interactions of fishes in several lakes contaminated by organochlorines. They found large differences in organochlorine bioaccumulation by predators among the lakes, and related the differences to differences in food web structure (more bioaccumulation with more complex food webs). Additive pathway models were more recently applied to explain trace element bioaccumulation by molluscs in San Francisco Bay and Long Island Sound (Luoma et al., 1992; Thomann et al., 1995; Wang et al., 1996), as realistic coefficients were experimentally developed (e.g. Fisher et al., 1991). The models used concentrations or particle/water distributions of elements to predict tissue burdens similar to those determined in the ecosystem.

The possibility grows of resolving some long-perplexing questions as bioaccumulation models become more sophisticated and integrated exposures are better understood. Large toxicity databases based on purely water-borne exposures were developed to support regulatory criteria for pollutants but these toxicity studies probably have underestimated the exposures of many organisms in many circumstances (e.g. Luoma, 1995). It is now necessary to resolve such insensitivities. Knowledge of the contribution of food and water pathways of bioaccumulation can aid choices of what environmental media provide the most critical information for monitoring or regulation. Recent studies

suggest that trace element trophic transfer differs with intracellular metal partitioning between cytosolic and particulate forms in prey (Nott and Nicolaidou, 1990; Reinfelder and Fisher, 1991) or with digestive processing strategies by the feeding organism (Decho and Luoma, 1991, 1994; Wallace and Lopez, in press). Better knowledge of interspecific differences in food selection (e.g. of different living or non-living materials), feeding rates, digestion, species-specific dissolved uptake coefficients and other aspects of ecophysiology is critical to determine how different species experience the chemicals in their environment.

4.2. Ecosystem view of pollutant exposure

Exposures depend just as much on processes that control the fate of pollutants as on bioavailabililty in proximate terms. A few determinations of pollutant concentrations in water, sediments or biotic tissues are not sufficient to characterize pollutant exposures at the ecosystem scale. Total concentrations of pollutants can be affected by major events within the ecosystem (Fig. 4). Dynamic physical processes can redistribute concentrations, permanently deposit them or transport them to other ecosystems. Interacting geochemical reactions and biological processes can change pollutant form and bioavailability as ecosystem conditions change in time and space. An appreciation of processes at the ecosystem scale is probably essential to avoiding naive conclusions about pollutant sources (Diamond, 1995) or effects.

Fate, distribution and dynamics of pollutants cannot be resolved by studying only pollutants, nor is it only a geochemical problem. For example, long-term ecosystem studies show that South San Francisco Bay has a recurrent spring phytoplankton bloom that is phased with the tides and therefore somewhat predictable. Although it occurs only once per year, the bloom is an ecological event that rapidly transforms some elements in directions unpredicted from thermodynamics. Metal concentrations and forms, for example, rapidly changed during the bloom in 1994 (Luoma, van Geen, Lee and Cloern, unpubl.). Concentrations of dissolved Cd and Zn were rapidly depleted (Cu was not) and particulate Cd concentrations increased. Trophic transfer could also have been changed by the bloom. A bioaccumulation model predicted that Cd exposures in the clam *Macoma balthica* increased slightly if the animals consumed the primarily non-diatom particles, but greatly increased if the animals selectively switched to a diatom diet during the bloom (experiments show a high assimilation of Cd from diatoms, e.g. Decho and Luoma, 1996). A mass equivalent to 50%–60% of the total annual input of Cd, Ni and Zn from local waste treatment plants was removed from solution by the phytoplankton during the bloom.

4.3. Linking bioavailability and toxicity.

Exposure is presently linked to effects of pollutants either through observation (correlation between environmental concentration and effect) or through experiments (bioassays) that determine the relationship between a constant pollutant concentration and an effect. It is difficult to experimentally simulate the integrated exposures that occur in complex, dynamic ecosystems and relate those to toxicity (for example, many

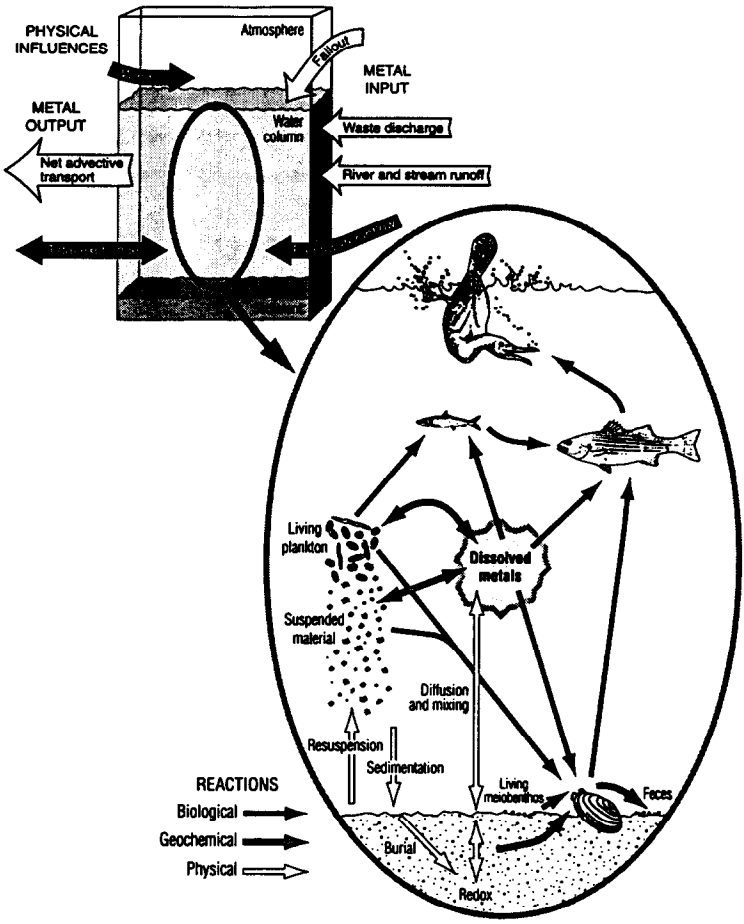

Fig. 4. A simplified overview of the interacting physical, chemical and biological processes that affect the fate and bioavailability of metals (and thus exposure of organisms to those pollutants) in South San Francisco Bay.

experimental exposures consider only water or abiotic sediments or extend for time periods that are short relative to the generation time of the organism). Bioassays, which can take many forms, are valuable experimental tools to study exposure and response but the appropriate uses of bioassay data in identifying pollutant concentrations that cause toxicity remains a subject of intense discussion (e.g. Chapman, 1995a,b; Luoma, 1995). It is increasingly accepted that substantial unresolved uncertainties lie in extrapolating to nature the prediction of toxic concentrations from simplified single species bioassays (Cairns, 1983, 1986; Connolly, 1985; Depledge, 1989; Iannuzzi et al., 1995; Luoma, 1995). Both insensitivities (Luoma, 1995) and over-sensitivities (Iannuzzi et al., 1995) are a concern.

The bioassay is an indirect surrogate for the more fundamental dose–response relationship, because dose is the chemical absorbed by the organism (Connolly, 1985).

Tissue concentrations could be a more direct measure of exposure. If tissue concentrations (tissue residues) relate to biochemical, cellular or organismic response, then biologically effective exposures could be determined from direct measurements (or model predictions) of those concentrations, whatever the complexity of the exposure. Metabolism of at least some organic compounds (e.g. PAH in fish) and storage of detoxified forms of chemicals (e.g. metals in some types of inclusions) undoubtedly add to the complexity of linking residues and response in nature. Probably because of the anticipated complexities, such relationships have not been a direct focus of very many studies in ecotoxicology, although in a few instances promising relationships have been found in nature (Widdows and Donkin, 1989; Collier and Varanasi, 1991). A recent workshop on metals criteria for aquatic life in the U.S. recommended departing from the purely observational, operational bioassay approach to estimating toxicity (Bergman et al., in prep). They recommended that emphasis in regulatory science shift to building mechanistic knowledge of how whole body pollutant residues, target organ residues or concentrations in biochemical fractions of tissues or organs (as direct indicators of exposure or bioavailability) relate to pollution effects in key species of fish and invertebrates. Bioassays (i.e. toxicity tests) might also become more predictive if appropriate bioaccumulation principles and models were incorporated and integrated as a matter of course.

5. Do pollutants affect fitness of individuals in ecosystems?

Studies at all levels of biological organization demonstrate that pollutants can cause adverse effects, at high enough concentrations. Biological responses to pollutants in nature can be much more complex than the first order responses observed in the laboratory. Subtle changes in biological processes can affect the Darwinian fitness of an organism (Depledge et al., 1995) but pervasive environmental stochasticity (amplified by inadequate understanding of undisturbed processes) and confounding or interacting factors can make it difficult to unambiguously identify why a change in a process occurs. Below, some of the ways that the pollutant variable can interact biochemically through whole animal processes in nature, are discussed. Appreciation of interactions between pollutants and at least some biological processes is becoming increasingly sophisticated. The resultant understanding is leading to at least some unambiguous interpretations of pollutant effects. The growth of such knowledge has been slow, probably because the greatest successes have required a combination of interdisciplinary or multi-investigator studies, persistent field documentation of responses in different environments and experimental studies with at least some mechanistic value. Continuation of multi-faceted science should continue to expand the framework defining the 'real' responses of organisms to the pollutant variable. A methodical and systematic expansion of such knowledge is far preferable to increased reliance on simplistic tests and investigator's judgement suggested as an alternative by some (Chapman, 1991, 1995a).

Attributing a biological change to the specific influences of pollutants first requires understanding what processes are sensitive to pollutants. Adverse effects may be manifested in pollutant-specific ways at the biochemical level in organisms, but

aggregated biochemical effects can appear at higher levels of organization as general stress responses in individuals, depauperate populations or the absence of species. Many factors can cause the general responses. The contribution of pollutants to changes in these processes is determined from a linkage to exposure, separation of a response from background fluctuations and elimination of the effects of confounding variables. Linkage to exposure might be improved by better knowledge of tissue-residue based responses, as described above. 'Background fluctuations' are the natural variations of the process; thus better separation of responses could be improved by better understanding of basic biological processes. Confounding variables are natural factors in the ecosystem, so understanding their effects will be improved by better understanding of ecosystem processes.

Responses to contaminant exposure are manifested as a continuum of biochemical, physiological, population and community changes (Bayne, 1985; Moriarity, 1988). As pollutant exposures increase, a progressive deterioration in health is, theoretically, followed by overt disease and eventual mortality (Depledge et al., 1995). Although a simple progression of symptoms can sometimes be induced experimentally (Sanders et al., 1991), it is not necessarily how responses are detected in nature (e.g. Couillard et al., 1995).

The most basic effects of pollutants occur at the molecular level, when critical biochemical functions (e.g. metabolic, transport, hormonal, immune or genetic systems) are disrupted (Vernberg et al., 1982). 'Biomarkers' are measurable signals of the changes in cellular or biochemical processes, structures or functions that are induced by pollutant exposure (Huggett et al., 1992). Some of the measurable biochemical responses are indicators of compensation (Depledge et al., 1995). For example, metal exposure induces metallothioneins and proliferation of subcellular inclusions such as granules, lysosomes and vesicles. These are detoxification mechanisms. Their expression does not mean that toxic responses significant to the individual are occurring; although such a relationship is not excluded either. Exposure to organic contaminants can induce production of xenobiotic metabolizing enzymes (e.g. mixed function oxidase enzymes such as cytochrome P450, now known as CYP2K1), another detoxification mechanism.

Pollutants may also cause responses indicating that biochemical processes are on countering stress, such as induction of antioxidant enzymes (e.g. glutathione peroxidase) (Stebbings et al., 1992) or stress proteins (Sanders et al., 1991). Some pollutant-induced biochemical changes are directly indicative of subcellular damage. These include damage to lysosomes (Moore, 1985), disruption of Na or Ca transport (Wood, 1992), presence of DNA-adducts (Varanasi et al., 1989) or atrophy of cells (Moore, 1988). Shifts in metal associations among cytosolic protein fractions or among metallothionein isomorphs (Roesijadi, 1994; Ringwood and Brouwer, 1995) are signals that compensatory responses to metal exposures are being overwhelmed.

A sufficient level or duration of pollutant exposure should induce physiological impairment. Relating biomarker responses with physiological costs to fitness of the individual is an important goal of many pollutant studies; although, at low doses the two need not necessarily co-occur. Physiological responses to pollutants, of course, differ among species and among pollutants. Reproductive processes can be directly effected (sexual maturation, follicle or gamete development) (Kluytmans et al., 1988), as can

development (embryogenesis and larval development are especially sensitive in some species, Martin et al., 1981; Sundelin, 1983). Pollutant exposure can inhibit mechanisms that resist disease (Roszell and Anderson, 1994), initiate cell leakage that results in increased hemolymph protein levels, disrupt Ca regulation or cause histopathological damage indicative of disease (Malins et al., 1994). Pollutants can affect osmoregulation. They can disrupt either the 'supply' side of energetics by affecting feeding rate (Capuzzo and Sasner, 1977) or the 'demand' side by affecting basal metabolism, respiration, heart rate or protein utilization (Calabrese et al., 1977; Bayne et al., 1981; Widdows, 1985; Carr et al., 1985). Effects on energetics are manifested in the individual by reduced growth efficiency or growth potential (scope for growth) (Widdows et al., 1990), poor condition or reproductive failure. Where responses reduce Darwinian fitness, the probability of survival at the population level is affected.

Physiological compensation adds to the complexity of responses to pollutants and to the complexity of relating physiological and biochemical responses. For example, Cd inhibits follicle formation in mussels (*Mytilus edulis*) but in the early stages of inhibition, spawning frequency is increased and little net effect on gamete production is observed (Kluytmans et al., 1988). Inherent differences in sensitivity to pollutants among individuals may be a form of compensation. Depledge et al. (1995) identified four 'physiotypes' of crabs (*Carcinus maenus*) by the range of hemolymph protein concentrations in unexposed animals. They then showed that the individuals with initially lower protein concentrations were the most vulnerable to Cu exposure.

Compensation can (perhaps, usually) occur at a cost, adding complexity to the compensation response. Protein concentrations declined with Cu exposures in the surviving crab physiotypes described by Depledge et al. (1995). In nature, pre-exposed, but surviving crabs could became more vulnerable to additional stress because of their lower hemolymph protein. A pre-existing stress may also affect vulnerability to a pollutant. *M. edulis* is more sensitive to Cu when energy reserves are depleted after spawning, than before spawning (Akberali and Trueman, 1985). The effects of chemicals on filtration rate, digestion or metabolism in starved mussels are greater than when mussels are adequately fed (Kooijman et al., 1989). It is also possible that contaminated organisms are more vulnerable to external stresses than organisms carrying a lesser body burden of pollutants (Viarengo et al., 1995). Alternatively, an exceptional tolerance to crude oil and PAH was found in the meiofaunal communities from contaminated Louisiana mudflats and was attributed to the long history of exposure of these communities to hydrocarbon and PAH contamination (Fleeger and Chandler, 1983; Carman et al., 1995). The relative sensitivities of species to pollutants is poorly known overall, but especially with regard to such complexities.

Response of a process to pollutants in the laboratory does not necessarily mean that responses will be detectable in nature. The species that exhibit whole organism, physiological or biochemical responses in the field are those that survived. Organismal responses cannot be studied in populations that were eliminated by the pollutant. It can be critical what species are employed to study pollutant effects. For example, some organisms are suitable, for practical reasons, for determination of scope for growth (e.g. mussels); some are not (e.g. many gastropods) (Widdows, 1985; Lowe, 1988). Variability of responses are also a consideration. For example, condition index which changes in

response to energetic stress, also varies with the reproductive cycle (e.g. in molluscs). Only if condition index is studied by repeated sampling, so that seasonality associated with reproduction is known, can subtle disruption of patterns be identified. In San Francisco Bay, clams with high Cd tissue burdens show less seasonal increase in condition index compared to organisms in locations with lower Cd burdens, indicating that animals experiencing contamination may be less able to add gametic tissue and energy reserves during reproduction (Brown and Luoma, unpubl.). The causes of variability in some potential measures of pollutant stress are not yet sufficiently well known to use to advantage in the field, however. For example, stress protein induction and antioxidant enzyme activities can be highly sensitive to many stresses and highly variable among individuals. Tri-butyl tin (TBT) exposure induces stress proteins in mussels in the laboratory, but so do elevated temperature, salinity stress, oxidative stress or ultraviolet light (Sanders, 1993). Lundebye et al. (in review) were unable to correlate stress protein induction with TBT across a strong concentration gradient among Danish Harbors because of unexplained inter-individual variability.

Because of confounding factors and compensatory complexities, observation of a response in a contaminated ecosystem or even correlation with a gradient in pollutant concentrations may not be sufficient to prove pollutants are the cause of an impairment. Martin et al. (1984) found statistically significant changes in energetics (scope for growth), changes in condition index and reproductive impairment in mussels deployed along a pollutant gradient in San Francisco Bay. The responses correlated with tissue burdens of PAH and some metals, as well as very high levels of suspended particulate materials. Because energetic responses to general environmental stresses like high SPM were not well understood, the effects of suspended materials could not be convincingly separated from the effects of pollutants.

Simple linear correlations between a pollutant concentration and a response may be the exception rather than the rule in complex field situations (Fig. 5 and Fig. 6). High variance in a response may be typical at low pollutant concentrations. Many levels of performance, driven by factors other than pollutant exposure, are possible when the pollutant is not a controlling factor in the process. However, if pollutants affect performance, that effect may be manifested as a 'cap' on the response with declining performance as pollutant dose increases. In such a case, both the median and the variance of the response might decline as pollutant concentrations increase (as observed in the data field in Fig. 5 and Fig. 6). The declining maxima in the data field might reflect the maximum physiological response possible under the influence of a given pollutant exposure; the cap, rather than the regression line, being the equivalent of the dose response. The traditional linear responses between pollutant exposure and response is an expectation derived from controlled experiments; such data may not be applicable to circumstances where important variables are not controlled.

Although the caveats for providing absolute proof are formidable, signs of stress consistent with pollutant effects have been demonstrated in contaminated marine ecosystems (Roesijadi et al., 1984; Stebbings et al., 1992; Roesijadi, 1994). Three examples provide models that demonstrate how ambiguities in demonstrating pollutant effects can be reduced. Elevated tissue residues, sediment concentrations of pollutants and biochemical signs of organic chemical exposure have been linked to neoplasms

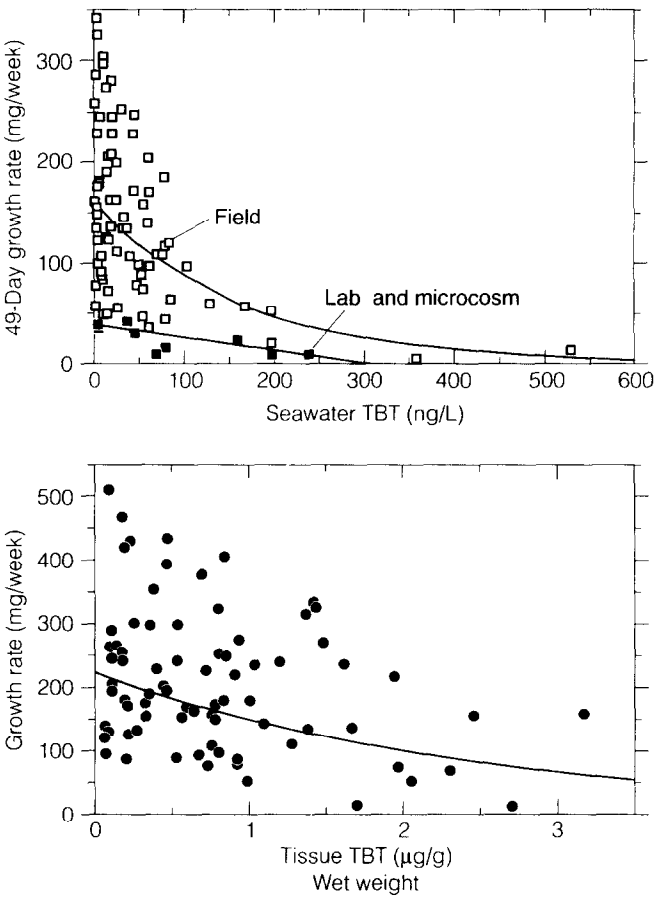

Fig. 5. Growth rates in translocated juvenile mussels as a function of tributyltin (TBT) concentrations in water or in tissues in San Diego Bay. The response in the field is not linear as observed in controlled experiments. A variety of growth rates can occur at low TBT concentrations (in water or tissues), because a variety of factors can influence growth (TBT is not the only influential variable). At higher TBT exposures higher growth rates are not observed; TBT appears to progressively cap growth in the mussels. (Redrawn from Salazar and Salazar, 1990)

(cancers) and other histopathological anomalies in fish repeatedly in different ecosystems (although the early demonstrations were in Puget Sound, Malins et al., 1994; Collier and Varanasi, 1991). These responses were linked to changes in fitness as indicated by reduced reproductive success and increased likelihood to produce abnormal larvae (Spies et al., 1988; Casillas et al., 1991). Mechanistic studies demonstrated how a progression of pollutant-induced biochemical changes induces the lesions (Moore, 1992). Absolute proof that tissue lesions are induced by pollutant exposure remains challenging for any individual study (Vethaak, 1992), but the weight of the existing literature makes it clear that when high rates of neoplasm or pre-neoplastic pathologies are observed in marine

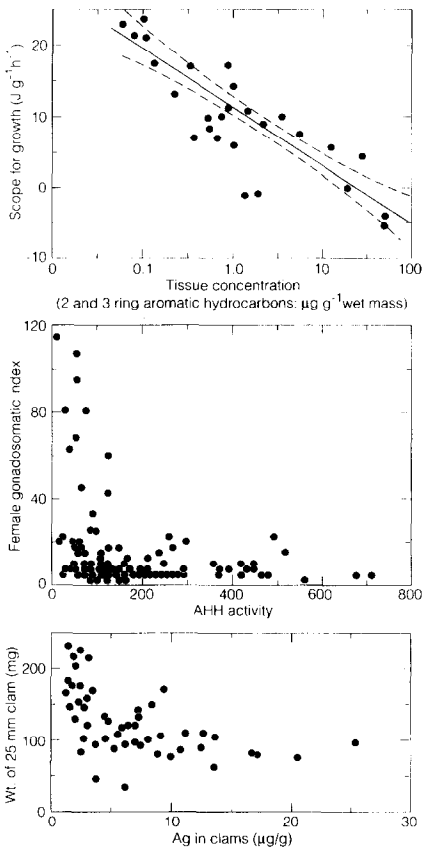

Fig. 6. Three instances of relationships between tissue residues or biochemical indicators of exposure and physiological responses. (a) Relationship between scope for growth and hydrocarbon concentrations in mussels, *Mytilus edulis*, found in two studies in the North Sea. (Data from Widdows and Donkin, 1989). (b) Relationship between hepatic aryl hydrocarbon hydroxylase activity (AHH is an exposure biomarker) and female gonadosomatic index in flatfish (*Platichthys stellatus*) in San Francisco Bay. A variety of responses occur at low AHH, perhaps because many factors other than AHH can affect reproduction. However, high indices of reproduction are not observed at higher AHH activities (Data from Spies et al., 1988). (c) Relationship between condition index and Ag concentrations in clam tissues (*Macoma balthica*) from samples collected near monthly between 1989 and 1994 from a mudflat in South San Francisco Bay. A progressively declining ceiling on the maximum condition index is observed as silver concentrations in tissues increase; at low Ag concentrations a wide variety of indices are observed, perhaps because factors other than Ag affect growth. (Data from Luoma et al., unpub.)

organisms, pollutants should be considered as a first order cause and significant effects on populations are likely.

Where enough processes are studied, signs of stress are also more likely to be detected. Because knowledge of what to expect is incomplete, some approaches will yield positive responses and some will be ambiguous. For example, an abundant flatfish, dab (*Limanda limanda*), was chosen in order to study the effects of a large scale

contamination gradient in the German Bight, employing a variety of biochemical and histopathological methodologies (as well as bioassays and benthic community analysis) (Stebbings et al., 1992). Results from studies of antioxidant enzymes and metallothionein induction were ambiguous. However, hepatic mixed function oxidase was a reliable indicator of organic contaminant exposure; cholinesterase inhibition was observed along the same gradient as was lysosomal damage. Indications of liver degeneration were also observed in the fish, as were liver neoplasia. Not all tests for pollutant effects were positive, but enough evidence was assembled in this intensive effort to eliminate ambiguities about the presence of a pollutant influence.

Expanding the physiological and ecological breadth of studies beyond immediate consideration of only pollutants may be as valuable as studying multiple responses to pollutants. Studies of energetics have long been used to assess the response of mussels (*Mytilus edulis*) to their environment (Bayne et al., 1981; Hawkins and Bayne, 1991). Widdows et al. (1995) built on this basic knowledge to establish the energetic cost of different body burdens of pollutants (PAH or TBT) to growth potential (scope for growth) in mussels, both in experiments and in relatively simple field circumstances. More recently they studied Venice Lagoon, which is subjected to the complex influences more common in many marine ecosystems (Widdows et al., in press). Scope for growth in mussels from Venice Lagoon was correlated with both pollutant burdens and particulate organic material concentrations (POM), but physiological performance was impaired as expected at the most contaminated sites. However, the inhibitory effects of the toxicants were masked in a site subject to nutrient addition. There, the expected pollutant effects on scope for growth were at least partly offset by the positive effects of very high food availability. Inabilities to decipher direct pollutant effects are probably often confounded by such interactions, but advanced understanding of energetics helped resolve ambiguities.

When conducted alone, simplistic toxicity tests, a single biomarker, whole organism analysis or studies that exclude variables other than pollutants will probably be insensitive to all but the most extreme influences of the pollutant variable. Preponderance of evidence approaches will be similarly insensitive if designs are too simplistic (e.g. Chapman et al., 1987). A combination of persistent and intensive study of exposure and response in the field, study of critical ecosystem-specific and organism-specific processes, as well as iteration with experimental studies, are useful (and perhaps necessary) strategies to discern if pollutants are affecting fitness in complicated ecosystems. As knowledge of processes involved in neoplasm induction or changes in scope for growth has grown more sophisticated, pollutant effects have become understandable in more complicated circumstances. Similar persistence could lead to a similar degree of understanding for other key responses.

6. Can effects on fitness be related to changes in populations?

Evidence of population effects can be decisive in attributing changes in marine ecosystems to pollutants. The evidence that chlorinated hydrocarbons (DDT and other pesticides) were contributing to the near extinction of species of piscivorous marine

birds contributed to the ban on that class of pollutants in the 1970's. The links between pollutants and the effects were made by a combined understanding of exposures (high concentrations in tissues and eggs), identification of sensitive physiological processes (egg-shell thinning), observations of reduced recruitment (lack of success in hatching) and observations of declining abundance. The recovery of the threatened populations that occurred after the ban proved that the lines of conjecture were correct. In the late 1980's, a similar sequential series of discoveries conclusively linked organotins (TBT) to decimation of mollusc populations. Exposures were linked to widespread effects on development and reproduction of several species (snails such the predator *Nucella*, the clam *Scrobicularia plana* and oysters, *Crassostrea gigas*) (Bryan and Gibbs, 1991). Declines in wild populations or reduced production of cultured populations were demonstrated and this anti-foulant was banned for uses on small boats. The most serious effects of TBT and chlorinated hydrocarbons were disruptions of hormonal systems manifested as specific, easily identifiable physiological or morphological responses (imposex; egg shell thinning). The specificity of the response aided linking lower order and higher order effects but the observations of population disruption was critical in defining significance.

Despite its significance, population biology is insufficiently exploited in ecotoxicologic studies, possibly because population responses to stresses are complex and the causes of population change can be difficult to differentiate. Lower order adverse effects on survival, growth or reproduction not only have direct consequences for populations but the costs of surviving pollutant exposure via physiological compensation may also translate into impairment of processes important to populations (e.g. reduced growth rate or reductions in fecundity) (Sibly and Calow, 1989). Indirect effects on changes in food availability or quality are another source of population disturbance.

It has been difficult to infer quantitative implications for populations from physiological changes (Munkittrick, 1992; Sindermann, 1996). Population models that consider interacting functional relationships in the life cycle provide important theoretical insights but direct applications of these to specific field circumstances are rare (Kooijman et al., 1989). Multi-generation, population level bioassays have also demonstrated the sensitivity of population processes to pollutants. Effects that are difficult to correlate with contamination in the field may cause sensitive populations to disappear when studied in these controlled conditions. For example, amphipods are one of the most sensitive marine benthic invertebrate groups to at least some types of pollutants (Swartz et al., 1982). In a multi-generation experiment, *Pontoporeia affinis* juveniles survived in Cd contaminated sediments, matured to adults, grew normally and reproduced. However, young amphipods were not recruited into the populations (Sundelin, 1983), apparently because of effects on embryo development. The population failed, but only after several generations of reduced recruitment. Similar results were observed in a separate study with *Ampelisca abdita*. Signs of stress included suppressed birth rates, reduced growth rates and delayed time to first reproduction (Scott and Redmond, 1989). Population models showed that the population would disappear due to reduced recruitment. It is conceivable that species are absent or relegated to relict populations in many modern contaminated habitats because of such effects. The frequency of effects or the thresholds at which they are initiated, in nature are not well known. It is also not well known if the

relative sensitivity of different species to pollutants can be related to characteristics of population biology.

Although population level responses to pollutants can be demonstrated in the laboratory or in models, differentiation of responses in nature is challenging. Habitat, food availability, natural disturbances and pollutants can all affect population dynamics. As a result, many populations are characterized by periodic large natural fluctuations and natural differences in abundance at different locations. Statistical sensitivity can be an important constraint (Underwood, 1994) and separation of natural from pollutant-induced trends can be problematic.

Underwood (1994) suggested that the mechanisms that generate resilience and compensate for disturbance are so strong in populations that human disturbances are only rarely meaningful, when they move populations out of their natural range of variability. Compensation mechanisms, indeed, can help at least some populations persist despite effects from pollutants (Marshall and Mellinger, 1980; Munkittrick and Dixon, 1989). In estuaries, populations of opportunistic species can exploit natural disturbances and persist through fluctuations in recruitment and episodes of mortality. For example, complex age structures allowed polychaete populations to persist despite episodic recruitment failures and age-specific mortality caused by dredging (Zajac and Whitlach, 1989). Specific tolerance to pollutants (via physiological adaptation or selection for tolerant genomes)(Luoma, 1977) can allow populations of adult fish (Klerks and Weis, 1987), oligochaetes (Klerks and Levinton, 1989), polychaetes (Bryan and Hummerstone, 1973) or clams (Luoma et al., 1983) to persist in highly contaminated estuarine habitats. Of course the tolerant population may represent a reduced genotype and/or be less able to adapt to other ecosystem changes or stresses (Klerks and Weis, 1987). Where the spatial distribution of contamination is patchy (a common occurrence), the geographic scale of pollutant impact may be smaller than the geographic range of motile species (e.g. fish or zooplankton, Sindermann, 1996) or the geographic distribution of the larvae of invertebrates species. Thus, the local populations of tolerant or stressed adults need not recruit directly, but may persist via immigration or recruitment from less contaminated habitats. If populations are absent from a few polluted habitats the overall effects on abundance might not be measurable if the range of the species is large (Sindermann, 1996). On the other hand, fragmentation of high quality habitat, a problem more typically associated with human effects on terrestrial ecosystems, might be a consideration in marine ecosystems because of contamination.

Of course, not all species have strong compensatory capabilities in their populations. Population characteristics range widely along the r- to k-selected continuum. Natural factors may also compete with pollutant stress for compensation capacity (Marshall and Mellinger, 1980).

Observations of survival or abundance are not sufficient measures of population responses. Populations are maintained by a dynamic balance among mortality, fecundity (including reproduction and recruitment) and migration. Dynamics can be inferred from age structure, mean age, growth rate, condition, age at maturation, fecundity and population size, all of which can be directly or indirectly measured. Typical patterns of response in the variables that drive dynamics may characterize responses when fish populations are affected by different kinds of stress (Munkittrick and Dixon, 1989;

Munkittrick, 1992; Gibbons and Munkittrick, 1994). For example, both exploitation and elimination of older age classes by age-specific toxicity are characterized by reduced mean age, shift toward a younger age structure, higher growth rate and condition (because of less competition for food), younger age at maturation, increased fecundity and decreased population size. Episodic recruitment failure, chronic recruitment failure, redistribution of energy resources and other types of effects on population each have a slightly different, but characteristic, pattern of response at the population level. This diagnostic scheme requires critical selection of the species to study, comparisons with appropriate reference sites (or some comparable sampling strategy, Underwood, 1994) and understanding the expected nature of the pollutant effect.

As patterns of population responses to pollutants are further identified, recognition could grow of the types of circumstances where populations are influenced by the pollutant variable. It is unlikely that such influences will be exclusive. It is also unlikely that they will be identified without study of lower order processes and understanding of higher order processes (community) important to the population. Again, the critical ingredients probably include multi-investigator studies, knowledge at several levels of organization, persistent and systematic field study of the circumstances of interest and iteration between experiment and field observation.

7. Conclusions

A rational framework for ecotoxicology recognizes contaminated marine environments as a subset of ecosystems in which at least some interrelated geochemical, biochemical, physiological, population and community characteristics are changed by pollutants. Moderate contamination is relatively widespread in coastal and estuarine ecosystems so the subset of ecosystems with at least some processes or species affected could be relatively large. Pollutant influences have changed and will probably continue to change on time scales of decades. Ecological studies should recognize the possibility of a continually changing influence from pollutants. Biological exposures to pollutants are complex. Dose is determined by how species are exposed to different environmental media and the geochemistry of individual pollutants within those media. Respect for the complexity of biological responses to pollutants in marine ecosystems is also important: "Pollution contributes to stress in marine animals as an added environmental factor with which they must cope; and of equal importance" (Sindermann, 1996). It is naive to assume that pollutants are the variable dominating living processes in any but the most extreme circumstances (although many approaches to pollutant management seem to make this assumption). On the other hand, it is also naive to ignore this variable in modern ecological studies. The 'pollutant' variable is, thus, one of the several exogenous variables that modern biologists and ecologists should consider when trying to determine controls on processes, from the sub-cellular level to the community. Interdisciplinary synthesis and practical benefit might come from simply expanding our curiosity about pollutant influences on fundamental ecosystem processes. Solutions to the complex problems of ecosystem change will not be found via a singular view of cause and effect (Kitchell, 1995) or a continued quest for universally applicable or simplistic quick

measures of pollutant effects. An appreciation of knowledge from all disciplines in ecotoxicology, not competition between disciplines, (i.e. a definition of interdisciplinary study) is necessary to understand the role of the pollutant variable. Models, monitoring, multi-discipline iteration between experiment and long-term field observation and persistence in pursuing understanding at both basic and applied levels will be the tools that underlie a more coherent framework for ecotoxicology.

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