Limnol. Oceanogr., 44(3, part 2), 1999, 903–909

The effects of lipophilic organic contaminants on reproductive physiology and disease processes in marine bivalve molluscs

Judith E. McDowell, Bruce A. Lancaster, Dale F. Leavitt, Pirjo Rantamaki,[1] *and Bonnie Ripley*
Woods Hole Oceanographic Institution, Mail Stop 2, Woods Hole, Massachusetts 02543

Abstract

Marine bivalve molluscs such as the blue mussel (*Mytilus edulis*) and the soft shell clam (*Mya arenaria*) have been used as sentinel organisms of contaminant bioavailability and the biological consequences of contaminant exposure. Biological responses that may contribute to the impairment of reproductive and developmental processes include responses that can be categorized as interfering with bioenergetic processes such as feeding and nutrient allocation; biosynthetic processes, such as the synthesis of energy stores; and morphogenic processes, such as those involved in structural development. Case studies within New Bedford and Boston Harbors (Massachusetts) are used to examine the relationship between contaminant uptake and effects on the reproductive cycle and bioenergetics of mussels and soft shell clams. The results observed illustrate that disruption in bioenergetics in bivalve molluscs exposed to chemical contaminants can result in loss of reproductive output and increased susceptibility to disease. Differences in the extent of reproductive impairment may be linked specifically to energetic strategies of individual species.

Natural and managed ecosystems are exposed continually to natural and anthropogenic perturbations. Physical destruction of habitats, input of toxic chemicals, and nutrient enrichment leading to eutrophication are among the stressors that result in alterations in the sustainability of coastal ecosystems and a reduction in biodiversity and harvestable yields. Ecological consequences of such stressors in coastal marine ecosystems include changes in parasitic infections (Lafferty and Kuris 1999; Lenihan et al. 1999), species distributions and abundance, degradation of habitats, and changes in energy flow and biogeochemical cycles. Inputs of toxic chemical contaminants can lead to impairment of feeding, growth, development, and recruitment of fish and shellfish populations that may result in alterations in reproductive and developmental success and changes in population structure and dynamics (Capuzzo 1987). Although contaminant exposure is often viewed as a single stressor, it is only one of many stressors that all populations of marine animals experience during their life cycles. The interactive effects of contaminant exposure and natural stressors, however, are not well understood, making it difficult to establish the relationship between responses of organisms to contaminated habitats and large-scale alterations in the functioning of marine ecosystems as well as large-scale contamination of fish and shellfish resources.

Although general trends in the distribution of lipophilic organic contaminants in coastal ecosystems have been defined (e.g., NOAA 1989, 1991), critical information on the biological effects of contaminants, specifically on population processes, is lacking. Recent studies of the incidence of tumors and other histopathological disorders in bottom-dwelling fish and shellfish from contaminated coastal areas have suggested a possible link between levels of lipophilic organic contaminants and the increased incidence of histopathological conditions (Sindermann 1996; Capuzzo et al. 1988). Sublethal toxic effects of contaminants in marine organisms may also include impairment of physiological processes that may alter the energy available for growth and reproduction (Capuzzo et al. 1988) and other effects on reproductive and developmental processes including direct genetic damage (Nacci and Jackim 1989).

Marine bivalve molluscs such as the blue mussel (*Mytilus edulis*) and the soft shell clam (*Mya arenaria*) have been used as sentinel organisms of contaminant bioavailability and the biological consequences of contaminant exposure. Biological responses that may contribute to the impairment of reproductive and developmental processes include responses that can be categorized as interfering with bioenergetic processes such as feeding and nutrient allocation, biosynthetic processes such as the synthesis of energy stores, and morphogenic processes such as those involved in structural development. Populations of bivalve molluscs comprise an important ecological and economic resource in coastal and estuarine habitats. Variations in reproductive output and population dynamics of bivalve molluscs are highly sensitive to environmental perturbations (Lowe and Pipe 1987; Kluytmans et al. 1988; Weinberg et al. 1997). Understanding the interactive effects of different life history strategies, natural stressors such as disease or predation, and anthropogenic stressors such as habitat alteration or contaminant exposure may lead to new insights on the adaptive mechanisms employed by coastal shellfish populations in coping with multiple stressors in their environment. Case studies within New Bedford and Boston Harbors (Massachusetts) are used to examine such relationships, specifically by examining changes in the reproductive cycle and bioenergetics of mussels and soft shell clams and the distribution of contaminants in bivalve tissues. Detailed methodologies of chemical and

---

[1] Present address: Agricultural Research Center of Finland, Food Research Institute, 31600 Jokioinen, Finland.

Acknowledgments

This research was supported by grants to J. McDowell from the Pew Charitable Trust, the Donaldson Charitable Trust, NOAA (NA 260A0462-01), NOAA Sea Grant Program (NA46RG0470), and a Cooperative Agreement between the U.S. Environmental Protection Agency and the Massachusetts Bays Program (CE001534-01).

Table 1. Selected PCBs and PAHs in sediments, clams, and mussels from New Bedford Harbor.

Contaminant	Sediment*	*Mya arenaria*† (mg g⁻¹ dry wt)	*Mytilus edulis*†
PCB (IUPAC No.)			
2,4,4′ (28)	—	2,020	1,970
2,2′,5,5′ (52)	1,300	1,060	915
2,2′,3,5′,6 (95)	—	2,030	2,490
2,2′,4,5,5′ (101)	1,800	1,190	1,220
2,3′,4,4′,5 (118)	2,200	1.560	—
2,2′,3,4,4′5′ (138)	1,300	870	665
2,2′,4,4′,5,5′ (153)	1,500	1,020	670
2,2′,3,4,4′,5,5′ (180)	260	46	25
Total PCBs (as Aroclor 1254)	30,000	20,100	9,350
PAH			
Phenanthrene	1,200	225	635
Fluoranthene	2,500	900	970
Pyrene	2,800	840	675
Chrysene	1,400	1,055	145
Benz[a]anthracene	1,200	2,500	635
Benzo[g,h,i]perylene	1,100	260	20
Total PAHs	26,000	10,090	1,940

* Pruell et al. 1990.
† Farrington et al. unpubl. data. Each point is the mean of three replicates with a C.V. ≤15%.

biological analyses have been reported by Capuzzo et al. (1989), Leavitt et al. (1990*b*), and McDowell Capuzzo and Shea (unpubl. rep.).

Case study: New Bedford Harbor

Ongoing studies in New Bedford Harbor and adjacent Buzzards Bay provide an excellent opportunity to investigate the bioavailability, bioconcentration, and biological effects of lipophilic organic contaminants in marine bivalve molluscs. The history of contaminant inputs to New Bedford Harbor is a carefully documented series of disturbances. Inputs of PCBs, PAHs, and heavy metals are well described. The fates of these contaminants have been examined in regards to chemical modifications, distributions, biogeochemical cycling, and physical transport of contaminants within the Acushnet River estuary and in the outer regions of Buzzards Bay (Farrington et al. 1986; Pruell et al. 1990).

Uptake of both polycyclic aromatic hydrocarbons (PAHs) and polychlorinated biphenyls (PCBs) have been documented in native populations of bivalve molluscs such as *M. arenaria* and transplanted *M. edulis* (Table 1). Capuzzo et al. (1989) examined the distribution of chlorobiphenyl congeners in *M. edulis* L. collected from a reference station in Sandwich, Massachusetts, and transferred to two stations in Buzzards Bay (Cleveland Ledge and New Bedford Harbor) and one station in Nantucket Sound, Massachusetts. Mussels were sampled bi-weekly and analyzed for concentrations of 20 chlorobiphenyl congeners or pairs of congeners, in addition to changes in bioenergetics and reproductive cycle.

Table 2. Pre-spawning condition indices (CI), scope for growth values (SG), maximum lipid content (LC), and reproductive effort (RE) of *Mytilus edulis* transplanted to New Bedford Harbor and reference sites.

Station	CI*	SG†	LC‡	RE§
Nantucket Sound	335 ± 30	1.8 ± 0.3	36.2 ± 0.8	0.88
Cleveland Ledge	340 ± 20	0.4 ± 0.1‖	32.9 ± 0.8	0.71
New Bedford Harbor	230 ± 15‖	0.2 ± 0.1‖	20.7 ± 0.4‖	0.34

* mg dry wt (shell vol.)$^{-1}$, mean ± 1 SE.
† kJ d^{-1}, mean values from eight individual animals at each site.
‡ mg triglycerides g^{-1} dry wt, mean values from eight individual animals at each site.
§ Calculated as the proportion of energy allocated to reproduction through the collection of released gametes in relation to the total amount of energy assimilated and partitioned to growth and respiratory demands; determined from mean values from eight individual animals at each site; C.V. ≤15%.
‖ $P < 0.01$.

Individual stations represented a gradient of chemical contamination; temperature profiles, chlorophyll *a* concentrations, and total suspended matter concentrations varied seasonally at all stations but were comparable at each of the stations during a specific sampling period. The sampling period extended over a complete annual cycle.

Mussel transplants in New Bedford Harbor showed uptake of chlorobiphenyl congeners to exceedingly high concentrations (10^{-6} g g^{-1} dry wt range). Fluctuation in the concentration of some chlorobiphenyl congeners (IUPAC No. 28/31 and 95) was apparent during late spring and early summer with a marked decline during autumn; this pattern was correlated with gametogenesis and spawning activity (Capuzzo et al. 1989). Relative redistribution and release of individual chlorobiphenyl congeners associated with spawning is not consistent, suggesting differential partitioning of specific congeners in different tissues or different lipid pools. Similar seasonal patterns in the fluctuation of individual chlorobiphenyl congeners were observed among mussels at other stations, although concentrations at each of these stations were at least 1–2 orders of magnitude lower than those observed at New Bedford Harbor. Differential uptake and loss of lighter chlorobiphenyls (di- and tri-chlorobiphenyls) and heavier chlorinated biphenyls (hexa- and hepta-chlorinated biphenyls) have also been noted for the green-lipped mussel *Perna viridis* (Tanabe et al. 1987).

The relative distribution of specific chlorobiphenyls in mussels transplanted to New Bedford Harbor is markedly different from the distributions observed in mussels from other sites. For example, IUPAC 28/31 (a pair of trichlorobiphenyls) and IUPAC 95 (a pentachlorobiphenyl) are present in greater relative abundance than other chlorobiphenyls in mussels transplanted to New Bedford Harbor, whereas these compounds are at relatively low to intermediate concentrations at the other two sites. Although IUPAC 28/31 have lower K_{OW} than other chlorobiphenyls measured, their relatively high concentrations in harbor waters influence their rate of uptake.

Measurements of condition index and lipid content in mussels at New Bedford Harbor are significantly lower than values for mussels at other stations during the pre-spawning period (Table 2). Highest values for scope for growth during the pre-spawning period were measured for mussels from the Nantucket Sound site and these are significantly higher than measurements made at either the Cleveland Ledge or New Bedford Harbor sites. The low values for scope for growth measurements in mussels at the Cleveland Ledge site coincide with a small oil spill that occurred during the transplant experiment 60 d before the mid-April spawning event. Other bioenergetic measurements (condition index and lipid content) were not significantly different from the Nantucket Sound mussels and reproductive effort was only slightly reduced.

Accumulation of lipids (primarily triglycerides) is correlated with seasonal changes in the reproductive cycle and maximum levels are detected just prior to spawning. Maximum lipid accumulation in mussels at New Bedford Harbor is only 57.2% of values observed in mussels at Nantucket Sound and Cleveland Ledge. As spawning proceeds, the condition index and lipid content decrease in all populations as lipid is incorporated in the production of new gametes.

Bioenergetic estimates of reproductive effort (measured as the proportion of energy allocated to reproduction through the collection of released gametes in relation to the total amount of energy assimilated and partitioned to growth and respiratory demands) was calculated for the entire spawning period (April–October). Reproductive effort (RE) was lowest for mussels from the New Bedford Harbor station. Detailed analysis of the stereology of gonadal tissues in mussels transplanted to New Bedford Harbor confirms the bioenergetic estimates of reduced reproductive effort. The percentage of developing follicles in female mussels from New Bedford Harbor transplants is significantly reduced in comparison with control populations. Mussel populations from Nantucket Sound and Cleveland Ledge had gonadal indices of 0.75 in both males and females just prior to spawning, indicating that 75% of the mantle volume was occupied by developing follicles. New Bedford Harbor mussels had a maximum gonadal index of 0.33 for females and 0.70 for males. The reduction in developing oocytes in mussels from New Bedford Harbor is correlated with the reduction in the lipid content and total mass of mantle-gonadal tissues. No aberrations in nutritive cells (adipogranular cells) or connective tissue (vesicular connective tissue cells) was noted. In addition to the reduction in developing oocytes observed, female mussels at New Bedford Harbor also showed a high degree of oocyte atresia or resorption prior to spawning.

Several other investigators have reported contaminant effects in bivalve molluscs from New Bedford Harbor. These include DNA strand breakage in gill tissues (Nacci et al. 1992) and alterations in gill function (Nelson and Gutjahr-Gobell 1990) in mussels, and high levels of hematopoietic neoplasia in *M. arenaria* (Leavitt et al. 1990*b*). Both Reinisch et al. (1984) and Leavitt et al. (1990*b*) reported a high prevalence of hematopoietic neoplasia in *M. arenaria* populations from a site in New Bedford Harbor in comparison with uncontaminated sites in Buzzards Bay. Leavitt et al. (1990*b*) observed an overall prevalence of 39.3%. Unlike populations of *Mytilus,* there were no differences in gonadal development, condition indices, or lipid content between clam populations from New Bedford Harbor and a reference site (Little Buttermilk Bay, Massachusetts), except with the most advanced stage of hematopoietic neoplasia. Clams with advanced stages of the disease showed a reduction in physiological condition (Leavitt et al. 1990*b*) and fecundity (Potts 1993). The degree to which contaminant exposure influences the prevalence of hematopoietic neoplasia in clam populations is not fully understood but may be a function of increased susceptibility to the disease when animals are in poor physiological condition or are already energetically compromised.

Case study: Boston Harbor and Massachusetts Bay

Sediments and biota from Boston Harbor are highly contaminated with a variety of lipophilic organic contaminants including petroleum hydrocarbons (both low molecular weight and high molecular weight hydrocarbons such as PAHs), chlorinated pesticides (total DDT, lindane, dieldrin, and chlordane), and PCBs. Concentrations of total PAHs in tissues of *M. edulis* are in the upper 15% of the most contaminated sites from the U.S. coastline surveyed in the National Status and Trends Program (MacDonald 1991). Other contaminants that show elevated levels in mussels collected from Boston Harbor include total DDT, total PCBs, lindane, dieldrin, and total chlordane. Although general trends in contaminant distributions in Boston Harbor-Massachusetts Bays have been defined (e.g., higher concentrations of total PAHs in the inner harbor of Boston, lesser concentrations with distance from the inner harbor), critical information on biological effects of chemical contaminants, specifically on population processes is lacking.

Gardner and Pruell (1988) found significant histopathological lesions in *M. arenaria* at selected contaminated sites in Quincy Bay including gill inflammation, atypical cell hyperplasia in gill and kidney, hyperparasitism with rickettsia in digestive ducts-tubules, and general parasitism. In addition, reproductive development and spawning among male and female clams appeared to be asynchronous. Kimball (1994) also observed asynchrony in reproductive development among three populations of *M. edulis* in Massachusetts and Cape Cod Bays, but reductions in reproductive effort among female mussels could not be attributed to the effects of contaminants alone. Recent studies conducted by Moore et al. (unpubl. rep.) in Massachusetts and Cape Cod Bays revealed a suite of histopathological conditions associated with chemical contaminant exposure in fish and shellfish. Populations of *M. arenaria* and *M. edulis* collected along a gradient of PAH contamination showed evidence of a wide range of pathologies including gill hyperplasia and carcinomas, hematopoietic neoplasia, gonadal inflammation, parasitic infections in connective tissues and kidney, and kidney hyperplasia. Discriminant analysis indicated that the prevalence of these pathologies was strongly correlated with high levels of PAH contamination.

To examine the effects of lipophilic contaminants on population processes of marine organisms in the Boston Harbor-Massachusetts Bays ecosystem, we examined the bioenergetics, reproductive cycle, and population growth of *M. arenaria* L. collected along a gradient of PAH contamination. Clams collected at each of the sites in Massachusetts

Table 3. Selected PCBs and PAHs in sediments and clams (both in mg g^{-1} dry wt) from Boston Harbor.

Contaminant	Sediment*			*Mya arenaria**		
	Neponset	Saugus	Ft. Pt. Ch.	Neponset	Saugus	Ft. Pt. Ch.
PCB (IUPAC No.)						
2,4,4′ (28)	<0.10	1.74	<0.10	5.48	3.74	2.86
2,2′,5,5′ (52)	<0.10	1.78	<0.10	7.13	4.60	1.91
2,2′,4,5,5′ (101)	0.39	0.55	1.79	17.73	5.05	4.82
2,3′,4,4′,5 (118)	0.73	0.77	1.35	14.52	9.46	7.77
2,2′3,4,4′5′ (138)	0.75	1.99	6.24	16.21	13.80	8.18
2,2′,3,4,4′,5,5′ (180)	0.30	1.39	6.71	0.00	5.73	0.72
Total PCBs	5.39	12.81	34.80	130.49	91.08	56.71
PAH						
Phenanthrene	65	945	5,331	70	245	274
Fluoranthene	166	1,308	5,179	266	924	1,136
Pyrene	170	1,080	4,332	239	825	1,065
Chrysene	51	474	1,670	216	609	885
Benz[a]anthracene	38	655	1,931	81	314	484
Benzo[g,h,i]perylene	36	1,574	2,223	230	348	730
Total PAHs	1,450	18,342	66,121	1,904	5,113	7,374

* Range in concentrations at three upper Massachusetts Bays stations (Fort Point Channel, Saugus River, and Neponset River) (McDowell Capuzzo and Shea unpubl. rep.). Sediment sample is a composite sample and clam sample is a composite of five clams; both composite samples were taken in March 1995.

and Cape Cod Bays reflect differences in body burdens of lipophilic organic contaminants indicative of sediment contamination at each site (McDowell Capuzzo and Shea unpubl. rep.; Table 3). Bioavailability of specific compounds (especially pyrogenically derived PAH), however, varies from site to site and is dependent on the relative partitioning to porewater. Changes in condition index and the digestive gland-gonad index for each clam population at the various sampling periods reflect seasonal differences in the reproductive cycle with the highest values being detected prior to spawning. There is a significant interactive effect of sampling season and site, and all stations in upper Massachusetts Bay (Saugus River, Neponset River, and Fort Point Channel) have significantly lower ($P < 0.01$) values at pre-spawning (Table 4). Total lipid content (primarily triglycerides) of the digestive gland-gonad complex was not significantly different among the five populations of clams, but the total mass of the digestive gland-gonad complex was significantly reduced at the contaminated sites. There was no significant difference in length or live weight of clam populations from any site during any sampling period. Thus, differences in estimates in physiological condition are not related to size differences but to specific differences in energy and lipid storage.

Table 4. Pre-spawning digestive gland-gonad index and maximum lipid content of *Mya arenaria* collected from five sites in Massachusetts Bay.

Station	Digestive gland-gonad index*	Lipid content†
Barnstable Harbor	30.6 ± 1.5	105.5 ± 6.4
Wellfleet Harbor	34.1 ± 1.2	116.9 ± 5.3
Neponset River	19.8 ± 0.7‡	105.2 ± 5.9
Saugus River	22.1 ± 0.7‡	84.0 ± 3.7
Fort Point Channel	19.9 ± 1.0‡	101.5 ± 6.0

* Wet wt of the digestive gland-gonad index/ wet wt of all soft tissue × 100; mean of 25 replicates ± 1 SE.
† Total lipid content (mg g^{-1} dry wt) of digestive gland-gonad complex; mean of 25 replicates ± 1 SE.
‡ $P < 0.01$.

The reproductive cycle of clam populations from the five sites varied with respect to the timing and extent of the spawning season but not with respect to the number of developing oocytes during a spawning event. Both female and male clams from the reference sites had advanced stages of gamete development during late spring and spawning continued through early fall. The large relative size of the digestive gland-gonad complex and accumulated lipid provided sufficient energy for this extended reproductive season. Populations from the upper Massachusetts Bay sites (Fort Point Channel, Saugus River, and Neponset River) did not spawn until midsummer and spawning occurred for only a short period of time. Asynchrony in gamete development between males and females was not observed at any of the five sites. In addition to an abbreviated spawning season, clam populations from the contaminated sites also showed a high prevalence of gonadal inflammation (cell proliferation) that was significantly different ($P < 0.001$) from reference populations especially during late fall to early winter (September–December). The highest values for hematopoietic neoplasia were detected at the Fort Point Channel site during the December sampling (100%).

Discussion

Bivalve molluscs have been used extensively during the past two decades as sentinel monitors of chemical contamination (Butler 1973; Natl. Res. Counc. 1980; Farrington et al. 1983) and more recently as organisms in biological effects monitoring (Bayne et al. 1988). As the relationships between levels of chemical contaminants and biological responses in bivalve molluscs continue to be explored, insight of the toxic action of specific compounds and groups of compounds have been elucidated. However, our knowledge of cause and effect relationships between tissue burdens of many contaminants and biological consequences in most species is still incomplete.

The most important physiological changes associated with exposure to lipophilic organic contaminants are those responses that may affect an organism's growth and survival and thus its potential to contribute to the population gene pool. Alterations in growth potential may take place as a result of changes in feeding behavior, respiratory metabolism, or digestive efficiencies. Reductions in physiological measurements (e.g., respiration rates, carbon turnover, and scope for growth) have correlated with reduced growth rates measured for bivalve populations from oil-contaminated habitats (Gilfillan et al. 1976; Gilfillan and Vandermeulen 1978). Alterations in bioenergetics and growth of bivalve molluscs appear to be related to tissue burdens of aromatic hydrocarbons (Gilfillan et al. 1977; Widdows et al. 1982, 1987; Donkin et al. 1990). Widdows et al. (1982) demonstrated a negative correlation between cellular and physiological stress indices (lysosomal properties and scope for growth) and tissue concentrations of aromatic hydrocarbons with long-term exposure of *M. edulis* to low concentrations of North Sea crude oil. Recovery of mussels following long-term exposure to low concentrations of diesel oil coincided with depuration of aromatic hydrocarbons (Widdows et al. 1987). Donkin et al. (1990) suggested that reductions in scope for growth in *M. edulis* were related to the accumulation of 2- and 3-ring aromatic hydrocarbons, as these compounds induced a narcotizing effect on ciliary feeding mechanisms.

Many contaminants do not occur in isolation, thus, organisms are often exposed to complex environmental mixtures of several lipophilic organic contaminants and trace metals. Examination of responses to multiple classes of contaminants and the interaction of environmental factors, such as seasonal variability in response, can add new insight to the additive, synergistic, or antagonistic effects of multiple contaminants. Responses of *M. edulis* collected along a contaminant gradient in Langesundsfjord, Norway, showed alterations in lipid distribution, reductions in scope for growth, and increased occurrence of histopathological responses coincident with accumulation of PAHs and PCBs (Moore 1988; Capuzzo and Leavitt 1988; Widdows and Johnson 1988; Lowe 1988). Widdows et al. (1990) examined changes in scope for growth in the turkey wing mussel (*Arca zebra*) collected along contaminant gradients in the waters surrounding Bermuda. Reduction in scope for growth as a result of both reductions in feeding rates and increases in metabolic expenditures correlated with significant accumulation of lead, tri- and di-butyltin, petroleum hydrocarbons and their polar oxygenated derivatives, and PCBs. Decreases in feeding rates could be attributed to non-specific narcosis associated with the accumulation of low molecular weight hydrocarbons (Donkin et al. 1990), whereas the increases in energy expenditures through increased metabolic rates could be related to the accumulation of TBT through effects on oxidative phosphorylation (Widdows et al. 1990). Bivalves collected along the same gradient showed changes in biochemical composition, especially in the ratio of neutral to polar lipids and carbohydrate content (Leavitt et al. 1990*a*).

The relationship between disease processes and exposure to lipophilic organic contaminants in bivalve molluscs has not been elucidated. Mix (1988) suggested that our limited understanding of the distribution of toxic chemicals in the environment, their effects on cellular and physiological processes, and mechanisms of biotransformation hinder our ability to explore the relationship between contaminant exposure and disease progression. Recent reports by Gardner et al. (1991*a,b*) have provided strong empirical evidence of causal relationships between contaminant exposure and the prevalence of neoplastic conditions in bivalve molluscs. Seasonal variation in both disease processes and the disposition of contaminants in bivalve molluscs can influence the ultimate expression of disease in populations from contaminated habitats (Leavitt et al. 1990*b*; Elston et al. 1992).

In both *M. edulis* populations from New Bedford Harbor and *M. arenaria* populations from Boston Harbor, differences in condition index and lipid reserves between contaminated and uncontaminated sites were greatest during the prespawning period, consistent with the accumulation and utilization of lipid reserves for reproductive development. Differences in the levels of reproductive impairment between the two species may be directly related to the energetics of the reproductive cycle. Populations of *M. edulis* incorporated most of the accumulated energy to gonadal reserves, whereas populations of *M. arenaria* had sufficient somatic reserves in addition to gonadal reserves. Lowe and Pipe (1987) suggested that the reallocation of energy reserves from resorbed oocytes in *M. edulis* following exposure to petroleum hydrocarbons may serve as a resistance strategy to survive the effects of hydrocarbon exposure. Other investigators have observed that a reduction in egg numbers in *M. edulis* with cadmium exposure was compensated by an increase in spawning frequency (Kluytmans et al. 1988).

The results observed in the two case histories reported here illustrate that disruption in bioenergetics in bivalve molluscs exposed to chemical contaminants can result in loss of reproductive output and increased susceptibility to disease. From these observations, effects on reproductive output appear to be largely determined by effects on energy storage and utilization. Differences in the extent of reproductive impairment may be linked specifically to energetic strategies during the life history of individual species. The combined stressors of energetic demand of reproduction, disease, and contaminant exposure warrant that the evaluation of contaminant effects be conducted during the entire reproductive cycle to assess the full extent of reproductive impairment.

Establishing a causal relationship between a specific contaminant and observed responses in populations of bivalve molluscs from any contaminated habitat is problematic. Several classes of contaminants occur simultaneously, the responses observed might occur with exposure to different contaminants, and synergistic or antagonistic effects of several classes of contaminants may occur. An understanding of the specific mechanisms of toxic effects on gamete and larval viability related to different life history strategies may lead to the development of biomarkers for reproductive impairment in bivalve molluscs. Alterations in bioenergetics linked with observations of reduced fecundity and viability of larvae, abnormalities in gamete and embryological development, and reduced reproductive success provide a strong empirical basis for examination of population responses. Incorporation of these responses in demographic models may lead to new insights on adaptations of specific life history strategies to contaminant perturbations.

References

BAYNE, B. L., AND OTHERS. 1988. An overview of the GEEP workshop. Mar. Ecol. Prog. Ser. **46:** 235–243.

BUTLER, P. A. 1973. Residue in fish, wildlife and estuaries. Organochlorine residues in estuarine mollusks, 1965–72. Pest. Monit. J. **6:** 238–246.

CAPUZZO, J. M. 1987. Biological effects of petroleum hydrocarbons: Assessments from experimental results, p. 343–410. *In* D. Boesch and N. N. Rabalais [eds.], Long-term environmental effects of offshore oil and gas development. Elsevier.

———, J. W. FARRINGTON, AND OTHERS. 1989. The relationship between lipid composition and seasonal differences in the distribution of PCBs in *Mytilus edulis* L. Mar. Environ. Res. **28:** 259–264.

———, AND D. F. LEAVITT. 1988. Lipid class composition of the digestive glands of *Mytilus edulis* and *Carcinus maenas* in response to contaminant gradients. Mar. Ecol. Prog. Ser. **46:** 139–145.

———, M. N. MOORE, AND J. WIDDOWS. 1988. Effects of toxic chemicals in the marine environment: Predictions of impacts from laboratory studies. Aquat. Toxicol. **11:** 303–311.

DONKIN, P., J. WIDDOWS, S. V. EVANS, C. M. WORRALL, AND M. CARR. 1990. Quantitative structure-activity relationships for the effect of hydrophobic chemicals on rate of feeding by mussels (*Mytilus edulis*). Aquat. Toxicol. **14:** 277–294.

ELSTON, R. A., J. D. MOORE, AND K. BROOKS. 1992. Disseminated neoplasia of bivalve molluscs. Rev. Aquat. Sci. **6:** 405–466.

FARRINGTON, J. W., A. C. DAVIS, AND OTHERS. 1986. Some aspects of the biogeochemistry of polychlorinated biphenyls in the Acushnet River estuary, Massachusetts, U.S.A., p. 174–197. *In* Estuarine and marine chemistry: Organic geochemistry. Am. Chem. Soc. Symp. Ser. 305.

———, E. D. GOLDBERG, R. W. RISEBROUGH, J. H. MARTIN, AND V. T. BOWEN. 1983. U.S. "Mussel Watch" 1976–1978: An overview of the trace metal, DDE, PCB, hydrocarbon and artificial radionuclide data. Environ. Sci. Technol. **17:** 490–496.

GARDNER, G. R., AND R. J. PRUELL. 1988. A histopathological and chemical assessment of winter flounder, lobster and soft-shelled clam indigenous to Quincy Bay, Boston Harbor and an in situ evaluation of oysters including sediment (surface and cores) chemistry. U.S. EPA. Environ. Res. Lab., Narragansett.

———, P. P. YEVICH, J. C. HARSHBARGER, AND A. R. MALCOLM. 1991*a*. Carcinogenicity of Black Rock Harbor sediment to the eastern oyster and trophic transfer of Black Rock Harbor carcinogens from the blue mussel to the winter flounder. Environ. Health Perspect. **90:** 53–66.

———, ———, AND OTHERS. 1991*b*. Germinomas and teratoid siphon anomalies in softshell clams, *Mya arenaria,* environmentally exposed to herbicides. Environ. Health Perspect. **90:** 43–51.

GILFILLAN, E. S., D. MAYO, S. HANSON, D. DONOVAN, AND L. C. JIANG. 1976. Reduction in carbon flux in *Mya arenaria* caused by a spill of No. 6 fuel oil. Mar. Biol. **37:** 115–123.

———, ——— D. S. PAGS, D. DONOVAN, AND S. HANSON. 1977. Effects of varying concentrations of petroleum hydrocarbons in sediments on carbon flux in *Mya arenaria,* p. 299–314. *In* F. J. Vernberg et al. [eds.], Physiological responses of marine biota to pollutants. Academic.

———, AND J. H. VANDERMEULEN. 1978. Alterations in growth and physiology of soft shell clams, *Mya arenaria*: Chronically oiled with Bunker C from Chedabucto Bay, Nova Scotia, 1970–76. J. Fish. Res. Bd Can. **35:** 630–636.

KIMBALL, D. M. 1994. The reproductive cycle in three populations of the blue mussel, *Mytilus edulis,* from Boston Harbor and Cape Cod Bay. Ph.D. thesis, Univ. Massachusetts-Boston.

KLUYTMANS, J. H., F. BRANDS, AND D. I. ZANDEE. 1988. Interactions of cadmium with the reproductive cycle of *Mytilus edulis* L. Mar. Environ. Res. **24:** 189–192.

LAFFERTY, K. D., AND A. M. KURIS. 1999. How environmental stress affects the impacts of parasites. Limnol. Oceanogr. **44:** 925–931.

LEAVITT, D. F., B. A. LANCASTER, A. S. LANCASTER, AND J. MCDOWELL CAPUZZO. 1990*a*. Changes in the biochemical composition of a subtropical bivalve, *Arca zebra,* in response to contaminant gradients in Bermuda. J. Exp. Mar. Biol. Ecol. **138:** 85–98.

———, J. MCDOWELL CAPUZZO, AND OTHERS. 1990*b*. Incidence of hematopoietic neoplasia in *Mya arenaria*: Monthly monitoring of prevalence and indices of physiological condition. Mar. Biol. **105:** 313–321.

LENIHAN, H. S., F. MICHELI, S. W. SHELTON, AND C. H. PETERSON. 1999. The influence of multiple environmental stressors on susceptibility to parasites: An experimental determination with oysters. Limnol. Oceanogr. **44:** 910–924.

LOWE, D. M. 1988. Alterations in cellular structure of *Mytilus edulis* resulting from exposure to environmental contaminants under field and experimental conditions. Mar. Ecol. Prog. Ser. **46:** 91–100.

———, AND R. K. PIPE. 1987. Mortality and quantitative aspects of storage cell utilization in mussels, *Mytilus edulis,* following exposure to diesel oil hydrocarbons. Mar. Environ. Res. **22:** 243–251.

MACDONALD, D. A. 1991. Status and trends in concentrations of selected contaminants in Boston Harbor sediments and biota. NOAA Tech. Memo. NOS OMA 56.

MIX, M. C. 1988. Shellfish diseases in relation to toxic chemicals. Aquat. Toxicol. **11:** 29–42.

MOORE, M. N. 1988. Cytochemical responses of the lysosomal system and NADPH-ferrihemoprotein reductase in molluscan digestive cells to environmental and experimental exposure to xenobiotics. Mar. Ecol. Prog. Ser. **46:** 81–89.

NACCI, D., AND E. JACKIM. 1989. Using the DNA alkaline unwinding assay to detect DNA damage in laboratory and environmentally exposed cells and tissues. Mar. Environ. Res. **28:** 333–337.

———, S. NELSON, W. NELSON, AND E. JACKIM. 1992. Application of the DNA alkaline unwinding assay to detect DNA strand breaks in marine bivalves. Mar. Environ. Res. **33:** 83–100.

NATIONAL RESEARCH COUNCIL. 1980. The international mussel watch. Natl. Acad.

NELSON, W., AND R. GUTJAHR-GOBELL. 1990. New Bedford Harbor Pilot Study progress report: Biological effects in *Mytilus edulis* after pre-operational deployments. U.S. EPA. Environ. Res. Lab., Narragansett.

NOAA. 1989. A summary of data on tissue contamination from the first three years (1986–1988) of the Mussel Watch Project. NOAA Tech. Mem. NOS OMA 49.

———. 1991. Second summary of data on chemical contaminants in sediments from the National Status and Trends Program. NOAA Tech. Memo. NOS OMA 59.

POTTS, M. S. 1993. Effects of hematopietic neoplasia on physiological processes in the soft-shell clam *Mya arenaria* (Linne). Ph.D. thesis, Univ. New Hampshire.

PRUELL, R. J., AND OTHERS. 1990. Geochemical study of sediment contamination in New Bedford Harbor, Massachusetts. Mar. Environ. Res. **29:** 77–101.

REINISCH, C. L., A. M. CHARLES, AND A. M. STONE. 1984. Epizootic neoplasia in soft shell clams collected from New Bedford Harbor. Haz. Waste **1:** 73–81.

SINDERMANN, C. J. 1996. Ocean pollution. Effects on living resources and humans. CRC.

TANABE, S., R. TATSUKAWA, AND D. J. H. PHILLIPS. 1987. Mussels as bioindicators of PCB pollution: A case study on uptake and release of PCB isomers and congeners in green-lipped mussels (*Perna viridis*) in Hong Kong waters. Environ. Pollut. **47:** 41–62.

WEINBERG, J. R., D. F. LEAVITT, B. A. LANCASTER, AND J. MCDOWELL CAPUZZO. 1997. Experimental field studies with *Mya arenaria* (Bivalvia) on the induction and effect of hematopoietic neoplasia. J. Invert. Pathol. **69:** 183–194.

WIDDOWS, J., T. BAKKE, AND OTHERS. 1982. Responses of *Mytilus edulis* on exposure to the water accommodated fraction of North Sea oil. Mar. Biol. **67:** 15–31.

———, K. A. BURNS, N. R. MENON, D. S. PAGE, AND S. SORIA. 1990. Measurement of physiological energetics (scope for growth) and chemical contaminants in mussels (*Arca zebra*) transplanted along a contamination gradient in Bermuda. J. Exp. Mar. Biol. Ecol. **138:** 99–117.

———, P. DONKIN, AND S. V. EVANS. 1987. Physiological responses of *Mytilus edulis* during chronic oil exposure and recovery. Mar. Environ. Res. **23:** 15–32.

———, AND D. JOHNSON. 1988. Physiological energetics of *Mytilus edulis*: Scope for growth. Mar. Ecol. Prog. Ser. **46:** 113–121.