
A Guide to Bivalve Diseases



**for Aquaculturists
in the
Northeastern U.S.**

*Bruce J. Barber
School of Marine Sciences
University of Maine*

Guide to Bivalve Diseases for Aquaculturists in the Northeastern U.S.

Bruce J. Barber
School of Marine Sciences
University of Maine
Orono, ME 04469

With funding from the Maine Aquaculture Innovation Center and the
Maine/New Hampshire Sea Grant Program

Guide to Bivalve Diseases for Aquaculturists in the Northeastern U.S. and Atlantic Canada

Table of Contents	Page
• Purpose of this document	
✓ What is disease?.....	2
✓ Preventing Disease outbreak.....	3
• Overview of Important Diseases	
✓ Oysters	
□ MSX Disease (<i>Haplosporidium nelsoni</i>).....	4
□ Dermo Disease (<i>Perkinsus marinus</i>).....	5
□ Juvenile Oyster Disease.....	6
□ Bonamiasis (<i>Bonamia ostreae</i>).....	7
✓ Clams	
□ Hemic Neoplasia.....	8
□ Gonadal Neoplasia.....	8
□ QPX	9
• Further Information	
✓ Literature.....	10
✓ World Wide Web.....	10
• Contacts	
✓ Shellfish Pathologists.....	11
✓ Extension Agents.....	11

Cover photo:

Editorial assistance provided by Susan White and Dana Morse

Technical comments provided by Susan Ford

Photo credits: B. J. Barber and C. V. Davis

- **Purpose of this document**

Aquaculture, by definition, is the large scale husbandry or rearing of aquatic organisms. Like other forms of agriculture, aquaculture operations from time to time experience the negative impacts of disease. To minimize the occurrence of disease, health management (preventing and managing outbreaks of disease) should be an integral part of the overall management scheme.



Figure 1. Oyster nursery lease on the Damariscotta River, Maine

The goal of this document is to inform culturists in the northeastern United States of the common diseases of marine bivalves and available means for preventing or minimizing their impacts. Hopefully this will lead to increased productivity and profit. **Note that these diseases affect bivalves only, and have no impacts on humans who consume them.**

What is disease?

Disease is defined as any impairment of normal structure or function. From a practical standpoint, diseases result in reduced growth, condition, reproductive potential, and mortality. Three components are needed for disease to occur. First, the species you are culturing serve as hosts. Susceptibility of hosts to diseases varies with species (genetics), season, and age. Second, a causative agent, which can be either an obligate or an opportunistic pathogen, could be a virus, bacterium, fungus, protozoan (single-celled organism), or metazoan (multicelled organism). Abiotic factors, such as chemical pollution, may also cause disease. Agents vary in their distribution and virulence; several will be discussed on the following pages. The third component required for disease to occur is the proper environment (salinity, temperature, food supply, dissolved oxygen, etc.) which either favors the agent or compromises the host. Knowledge of these three factors and their inter-relationships are crucial for effective health management.

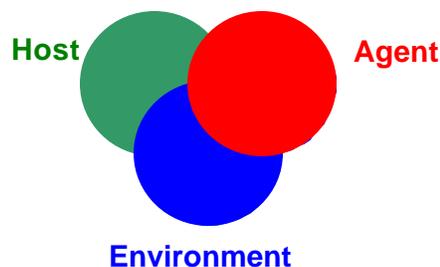


Figure 2. The Disease Triad

Preventing Disease Outbreak

For obligate pathogens, the optimal approach would be to use “disease free” seed and plant it in a non-disease endemic area for grow out to market size. To minimize the transfer of disease agents from one area to another, most states have strict regulations regarding the inter- and intra-state movement of live, marine organisms. For opportunistic or facultative pathogens, good husbandry practices are crucial for preventing disease outbreaks.

If one grower brings animals harboring disease agents into a region, all growers will potentially be impacted.

In spite of our best efforts, however, outbreaks of known diseases can and do occur. Also, new disease problems develop over time, especially as more species are cultured. Under these circumstances, researchers and growers work together to determine causes

and management options. The species being cultured, the particular disease, and the location of the farm, will determine the management options available to minimize the impact of the disease.

The following list is intended as an introduction to what is known about the more common bivalve diseases and their management. For updated information, contact one of the resources listed at the end of this guide.

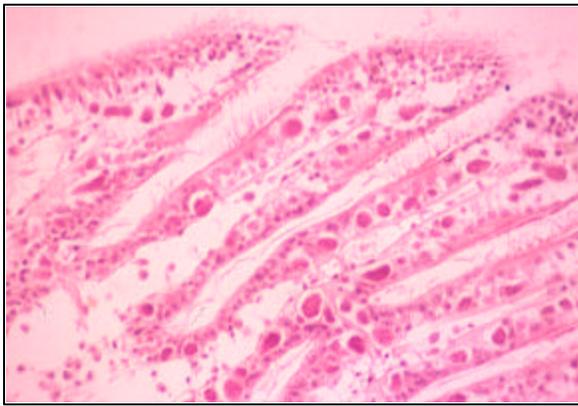
- **Overview of Important Diseases**

Disease outbreaks in bivalve hatcheries are, for the most part, caused by viruses, bacteria, and fungi, and are readily controlled with proper management practices. These will not be discussed here. Once animals are moved into the natural environment, however, they are exposed to local (endemic) disease agents and environments.

✓ Oysters



□ **MSX disease** is caused by the protozoan, *Haplosporidium nelsoni*. It first appeared in 1957 in Delaware Bay, where it caused massive mortalities of eastern oysters, *Crassostrea virginica*. It was first given the name MSX (Multi-nucleated Sphere X) because scientists had not seen it before. Today, it ranges from Maine to Florida. Effects of the disease include decreased condition (meat quality), reduced reproductive capacity, and mortality, which occurs primarily in the late summer and fall. An outbreak of MSX occurred in the Piscataqua River in 1995 in association with a drought. More recently, prevalence of MSX in Long Island Sound has increased.



The presence of MSX can only be confirmed by microscopic examination of oyster tissue by a professional pathologist (Fig. 3). *H. nelsoni* cells usually appear first in gill tissue, indicating that the infective stage is water borne. The mode of transmission of this pathogen, however, is not known. Definition of its life cycle is complicated by the fact that direct transmission between oysters has not been demonstrated.

Figure 3. Cells of *H. nelsoni* visible in oyster gill tissue; 200 X

Management of MSX disease is aided by the fact that *H. nelsoni* cannot tolerate salinity below 10 ppt and only causes severe mortality above 20 ppt. Thus sites that regularly experience salinity below 20 ppt will be less impacted by MSX than other areas. *H. nelsoni* can actually be purged from oysters by moving them to salinity of 10 ppt or less. Research has also shown that resistance to MSX mortality can be reduced through selective breeding. Contact your local extension agent for information on the availability of MSX resistant seed.

□ **Dermo disease** of eastern oysters, *C. virginica*, was first noted in Louisiana in the 1940s. Initially thought to be a fungus, it was later recognized as a protozoan, *Perkinsus marinus*. Today *P. marinus* is prevalent throughout the warm waters of the Gulf of Mexico and southeastern U. S. In recent years its range has extended as far north as southern Maine, in conjunction with high winter temperatures.

P. marinus is highly infectious, capable of spreading rapidly throughout a growing area. The disease is transmitted directly from infected to uninfected oysters. A decrease in growth is usually noted prior to mortality, which is typically between 40% and 90% after two to three years of exposure. Even though *P. marinus* is most lethal at temperatures above 20 °C and salinity above 15 ppt, it can persist at much lower temperatures and salinities.

Pathologists usually determine the presence of *P. marinus* by microscopic examination of oyster tissue cultured in thioglycollate medium. Fixed, stained tissue will also confirm the presence of this parasite, but is much less sensitive in detecting early infections (Fig. 4). Because it is often seen in gut tissue, its primary mode of infection may be via ingested material. *P. marinus*, or closely related species, have been found in bivalves other than *C. virginica*. Thus, any bivalve from an area where Dermo is endemic is a potential carrier of this disease.

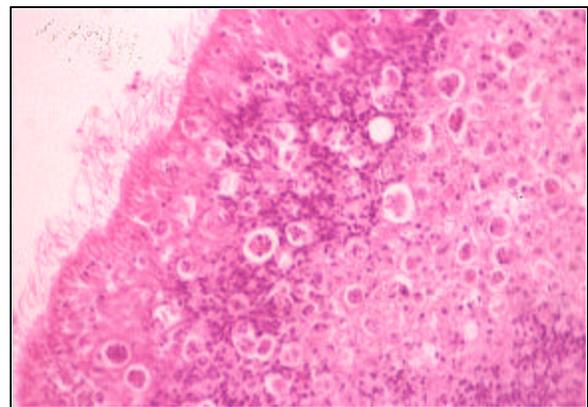


Figure 4. Cells of *P. marinus* in oyster gut epithelium; 200X

To date, no effective management for Dermo has been developed. Improving survival through selective breeding is one option currently being investigated. For culturists in areas where Dermo disease is endemic, achieving growth to market size in two growing seasons will minimize disease losses. Growth can be improved by utilizing selected lines (seed available through commercial hatcheries) and by the use of suspended culture techniques.

□ **Juvenile Oyster Disease (JOD)** was first noted in 1988 in hatchery-produced eastern oyster seed (*C. virginica*) held at nursery sites in Maine, New York, and Massachusetts. It has been responsible for mortalities of over 90% in certain locations since then. Initial signs of the disease are reduced growth accompanied by uneven shell margins (Fig. 5). Mortality ensues and JOD is confirmed by the presence of conchiolin deposits (“brown rings”) on the inner shell surfaces (Fig. 6). JOD follows a seasonal pattern, with most mortality occurring in mid- to late summer in conjunction with maximal water temperatures. It does not affect other bivalve species. The disease can be transmitted from oyster to oyster, but the causative agent is yet to be positively identified.



Figure 5. Juvenile oysters (10 mm) showing uneven shell growth, typical of JOD; actual size

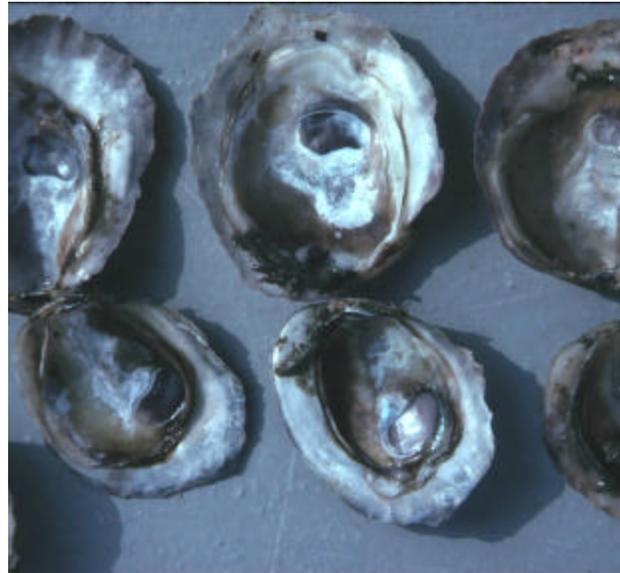


Figure 6. Juvenile oyster shells (10 mm) with “brown rings” caused by JOD; actual size

In most areas, effective management can be achieved in various ways. First, sites where JOD occurs can be avoided entirely or until a shell height of 25 mm is reached. Second, if a 25 mm size is reached before June, losses will be minimal. This can be attained with the use of seed which has been selected for fast growth and produced early in the year. Third, there is evidence that survivors of exposure to JOD have a genetically based resistance to the disease. Again, consult with your marine extension agent for information on local availability of seed.

□ **Bonamiasis** of European oysters, *Ostrea edulis*, is caused by the “microcell” parasite, *Bonamia ostreae*. The history of this disease illustrates how movement of live oysters from one area to another can have deadly consequences. First noticed in oysters brought into the United States from Europe, *B. ostreae* was apparently moved to California and subsequently transported with seed to Brittany, France, where it led to the demise of the flat oyster industry throughout Europe. It now also occurs in Washington state and in Maine, where oysters were introduced between 1949 and the 1970’s, before regulations were enacted to prevent such occurrences.

B. ostreae enters, but is not destroyed, by host blood cells. It thus multiplies within blood cells, eventually spreading to all tissues, interfering with defense and other physiological processes. Mortality can approach 100%, occurring throughout the year, at temperatures from 12° to 20 °C. It may be limited by low temperature, however, as the parasite has not been detected in oysters from Atlantic Canada nor northern Europe. In Maine, *B. ostreae* is found consistently at low prevalences in some (but not all) populations of *O. edulis*. In temperate climates, the disease is highly infectious, and is transmitted directly from oyster to oyster.

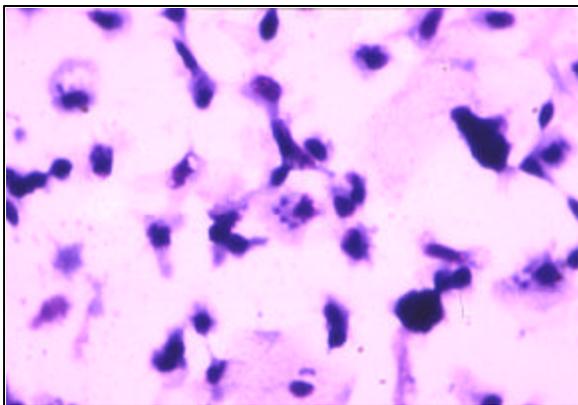


Figure 7. Cells of *B. ostreae* in oyster blood cells: 500X

Early stages of bonamiasis are indicated by shell gaping, an inability to close completely. Definitive diagnosis of *B. ostreae* requires microscopic examination of oyster blood or tissue by a professional pathologist (Fig. 7).

The Dutch have attempted, with minimal success, to eradicate *B. ostreae* by removing all oysters from growing areas for three years. Short of that drastic measure, effects of this disease may be lessened by reducing oyster density or utilizing

suspended culture techniques. In addition, it appears that resistance to the disease has developed in previously exposed stocks.

✓ Clams



There are two proliferative diseases of potential consequence to the production of the soft-shell clam, *Mya arenaria*. Contrary to popular belief, there is no obvious correlation between levels of pollution and the prevalence of these diseases.

□ The first of these is **hemic neoplasia**, or an uncontrolled multiplication of blood cells. The nuclei of these abnormal cells are much larger than those in normal cells. They multiply rapidly, as evidenced by numerous actively dividing cells (Fig. 8). As the disease advances, the number of abnormal cells increases, ultimately destroying physiological function. Mortality occurs in >90% of affected clams

within 60 days or less. There is evidence that this disease is caused by a virus and is therefore infectious. It occurs throughout the range of the clam, from Labrador to South Carolina, but prevalence is generally low.

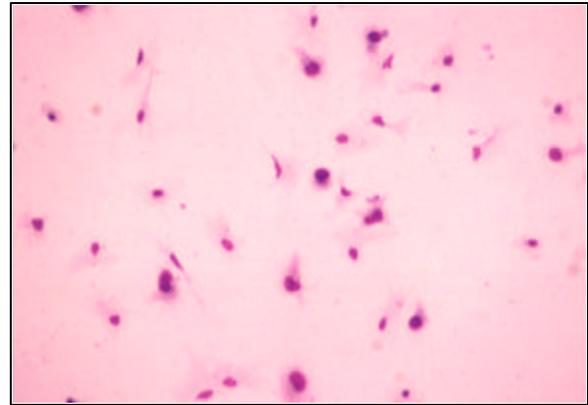


Figure 8. Neoplastic blood cells from a softshell clam; note enlarged nuclei; 200X

□ The second is **gonadal neoplasia**, or the presence of primary germ cells in reproductive tissue that proliferate rather than develop into gametes (eggs or sperm). Eventually these tumors invade other tissues, most likely causing death. The disease appears to progress slowly, however, and the level of mortality is unknown. The primary impact of gonadal neoplasia is a loss of reproductive output. The cause of this

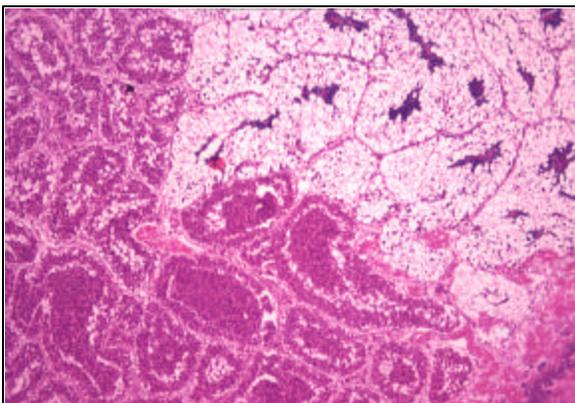


Figure 9. Gonadal follicles of male clam showing proliferation of primary germ cells; 50X

disease is unknown, and for *M. arenaria*, its range is quite limited, only from Maine to Atlantic Canada. Therefore its potential impact on culture of this species is low. A similar disorder occurs in the quahog, *Mercenaria mercenaria*, in the southeastern U.S.

Clams with advanced cases of gonadal neoplasia have a visually mottled visceral mass (gonad). Definitive diagnosis requires microscopic examination of fixed clam tissue by a trained pathologist (Fig. 9).

❑ Cultured hard clams (northern quahog), *Mercenaria mercenaria*, experienced unusual mortalities on several farms in Massachusetts in 1995. Dead and dying clams were found to be infected with a protozoan given the name **QPX** (Quahog Parasite X). In 1959, a similar parasite was identified in wild clams in New Brunswick, Canada. QPX has also been found in farmed clams as far south as Virginia. It is thought that QPX is an opportunistic pathogen, occurring naturally in clam growing areas, but causing acute disease outbreaks only under conditions of “stress.”

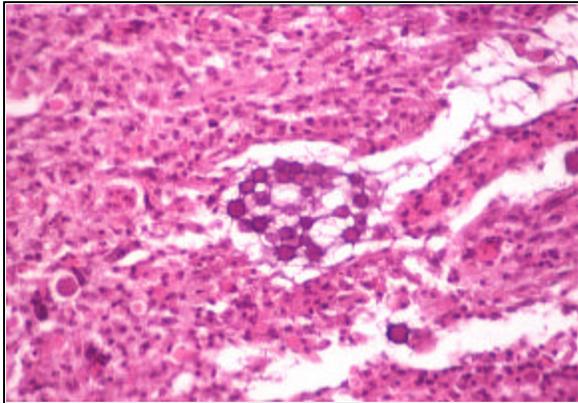


Figure 10. QPX parasites with characteristic mucofilamentous net surrounded by blood cells; 200X

Prior to dying, clams experience reduced growth; swollen, retracted mantle edges; and occasional 2-5 mm round, yellow-tan nodules in the mantle tissue. Microscopically, the parasite is most abundant in mantle and gill tissue (Fig. 10). Consult with a trained pathologist for a definitive determination.

To minimize disease outbreaks, clams should be planted at reasonable densities (50-75/ ft.² for 15 mm seed) in areas known to support good growth. It is preferable to utilize local seed stocks. Do not transfer

clams to your lease from outside areas without proper disease inspection. Wet storage of non-native hard clams near cultured stocks is also discouraged.

• **Further Information**

✓ **Literature**

Elston, R. A. 1990. Mollusc Diseases- Guide for the Shellfish Farmer. Washington State Sea Grant Program, University of Washington Press, Seattle, 73 pp.

Elston, R. A. 1999. Health management, development and histology of seed oysters. World Aquaculture Society.

Fisher, W. S., ed. 1988. Disease Processes in Marine Bivalve Molluscs. Am. Fisheries Soc. Spec. Publ. 18.

Sindermann, C. J. and D. V. Lightner, eds. 1988. Disease Diagnosis and Control in North American Marine Aquaculture. Elsevier, New York, 431 pp.

Ewart, J. and S. E. Ford. 1993. History and Impact of MSX and Dermo Diseases on Oyster Stocks in the Northeast Region. Northeastern Regional Aquaculture Center, 8 pp.

QPX Advisory Bulletin. 1996. Ocean State Aquaculture Association Special Publication, 4 pp.

✓ **World Wide Web**

Aquaculture Health Page:

www.geocities.com/CapeCanaveral/Lab/7490/index.html

USGS:

www.nfrcg.gov/nas/diseases/oyster.dis.htm

IFREMER Pathology Page:

www.ifremer.fr/gap/patho/anglais/patho.htm

DFO Synopsis of Infectious Diseases:

www.pac.dfo.ca/sealane/aquac/pages/title.htm

• **Contacts**

✓ **Shellfish Pathologists**

Dr. Bruce Barber, School of Marine Sciences, University of Maine, Orono, ME 04469; (207) 581-2783;
bjbarber@maine.edu

Dr. Susan Ford, Haskin Shellfish Laboratory, Rutgers University, Port Norris, NJ 08349; (609) 785-0074;
susan@hsrl.rutgers.edu

Dr. Fred Kern, Southeast Fisheries Science Center, Oxford, MD 21654; (410) 226-5193;
fkern@hatteras.bea.nmfs.gov

Dr. Sharon McGladdery, Dept. of Fisheries and Oceans, Moncton, N.B. E1C 9B6; (506) 851-2018;
mcgladderys@mar.dfo-mpo.gc.ca

Dr. Roxanna Smolowitz, Marine Biological Laboratory, Woods Hole, MA 02543 (508) 289-7663;
rsoml@mbi.edu

✓ **Extension Agents**

Mr. Rollie Barnaby, University of New Hampshire Cooperative Extension, 113 North Road, Brentwood, NH; (603) 679-5616; r_barnaby@unh.edu

Ms. Nancy Balcom, Connecticut Sea Grant, 1084 Shennecossett Rd., Groton, CT 06340; (203) 445-8664;
balcom@uconnvm.edu

Mr. John Ewart, College of Marine Studies, University of Delaware, Lewes, DE 19958; (302) 645-4060;
ewart@udel.edu

Mr. Gef Flimlin, Rutgers Sea Grant Extension, 1623 Whitesville Rd., Toms River, NJ 08755 (908) 349-1210;
flimlin@aesop.rutgers.edu

Mr. Don Meritt, Horn Point Environmental Labs, P.O. Box 775, Cambridge, MD 21613;
meritt@hpel.umd.edu

Mr. Dana Morse, Maine Sea Grant, Darling Marine Center, Walpole, ME; (207) 563-3146 ext. 205;
dana.l.morse@umit.maine.edu

Dr. Dale Leavitt, SEMAC, Massachusetts Maritime Academy, Buzzards Bay, MA 02532;
dleavitt@whoi.edu

Mr. Michael Rice, Aquaculture Center, University of Rhode Island, Kingston, RI 02881; (401) 874-2114;
rice@uriacc.uri.edu

Mr. Gregg Rivara, Cornell Cooperative Extension, Suffolk Co. Marine Environmental Learning Center, Southold, NY 11971; (516) 852-8660; grivera@cce.cornell.edu