We have placed disease interactions with exotic organisms into four categories: (1) effects of exotic diseases on local organisms, (2) effects of local disease on exotic organisms, (3) increased susceptibility to disease of exotic organisms, cultured in unsuitable or marginal environmental conditions, and (4) predisposition of the environment to pathogen problems. A few examples in each category are considered in this paper, without attempting to describe all such interactions.

**EFFECTS OF EXOTIC DISEASES ON LOCAL ORGANISMS**

**Extinction**

**Diadema Mass Mortalities**

In 1983-1984, 95-99% of the black long-spined sea urchins, *Diadema antillarum* Phillippi, in the Western North Atlantic died (Lessios 1988) (Fig. 1). A waterborne disease agent, possibly a virus, has been suggested as the cause (Williams & Williams 1987). Lessios (1988) suggested that this event was the largest mass mortality of an invertebrate ever recorded, one that could bring the species close to extinction. Williams et al. (1991) found repeated recurrences of mortalities. If mortalities continue, this urchin could become extinct. Studies of the recurrences are in progress (Williams et al., unpubl. data).

**Figure 1.** Fibropapillomas in a green turtle from the Caribbean.

**Turtle Tumor Outbreaks**

Fibropapillomas of sea turtles (Fig. 2) have increased to alarmingly high levels in Hawaii and Florida in the last 10 years, and in the Caribbean in the last 4-5 years (Williams et al., unpublished manuscript). This condition was originally described in the Florida Keys in 1938. Twenty years later, it was noted in the Indo-Pacific. Pacific cases may have been the result of introductions from the Atlantic. We have found some early cases of this tumor in Hawaiian aquaria and are attempting to determine if

**Figure 2.** Stages in the death of black longspined sea urchins during recent mass mortalities.
these were local in origin or introduced (Williams et al., unpubl. data). The disease could contribute to the extinction of the green sea turtle, which is already threatened or endangered in many parts of its range.

Like many introduced parasites, *Anquillacola crassus* will continue to spread by natural movements of hosts, but principally by human transport for stocking aquaculture ponds and for market — within and across national boundaries.

The rapid spread of this introduced parasite through the native eel populations of Europe provides an excellent illustration of the reality that once an introduced pathogen is established in a marine/catadromous/anadromous fish population, restricting or controlling its further spread in natural waters is difficult, if not impossible. Control in aquaculture facilities is, however, feasible.

**Giant Clam Mortalities**

*Tridacna* sp. have been brought into the Caribbean in at least 3 cases and in many cases to South Florida (Williams and Bunkley-Williams 1990). A *Perkinus* sp. (protozoan) caused mass mortalities of these clams in Australia. This parasite does not seem to be very host specific — an alarming quality in any introduced parasite. The exact geographic range, how easily and predictably this parasite can be detected, and its effect on Atlantic mollusks remain unknown. Cooperation was suggested among those holding and culturing the clams to assure proper disease screening (Williams and Bunkley-Williams 1990).

The only remaining culture project in the Caribbean (that we are aware of) seems to be maintaining reasonable precautions. The demonstrations at "Walt Disney World" in Florida, the "Biosphere" in Arizona, and most of the smaller units in south Florida have failed, but the promotion and sale of these clams as popular aquarium animals broadens the potential for transmission of disease agents.

**Abalone Disease**

The introduction of abalone, *Haliotis* sp., from California to Puerto Rico and other Caribbean areas for culture has been proposed. Since 1987, a "shrunken foot disease" has caused mass mortalities and declines in the populations of black abalone and other species. Until this condition is thoroughly understood, it would seem unwise to attempt culture of these organisms outside their normal ranges.

**THE EFFECT OF LOCAL DISEASES ON EXOTIC ORGANISMS**

**Acute Mortalities Due to Lack of Resistance**

**Mass Mortalities of Tilapia**

The smothering gillworm, *Neobenedenia melleni* (MacCallum), has been known for the last 60 years to damage and kill aquarium-held fishes. Apparently, tilapi-
as have even less resistance than local fishes, as caged tilapia suffer incredibly dense infestations and rapid death. *Tilapia aurea* and *T. mossambica* perished in marine cage culture experiments in Puerto Rico during 1972-1989 (Williams & Bunkley-Williams, unpubl. data), and red tilapias have suffered from this parasitic disease in commercial culture facilities in the Bahamas, Jamaica and Puerto Rico.

While various chemical treatments and salinity or cage depth changes are at least temporarily effective, they may not be economically feasible. The susceptibility of tilapias to this ectoparasite may severely limit their culture. A new Caribbean *Neobenedenia paragueraensis*, which may be involved in some of the mortalities, has been described recently (Dyer, Williams and Bunkley-Williams, in press) (Fig. 4).

**Figure 4. Neobenedenia paragueraensis** a serious parasite of Caribbean fishes.

Rickettsial disease of Coho Salmon

During the past decade, *Oncorhyncus kisutch*, have been introduced to coastal waters of Chile for net pen culture. Beginning in winter 1989, mass mortalities of up to 90% of stocks occurred in some locations in southern Chile (Bravo and Campos 1989) and an epizootic of a rickettsial organism has been identified as the causative agent (Fryer et al. 1990, Cvitanich et al. 1990, Branson and Diaz-Munoz 1991).

Although infectious agents from North America, such as the bacterium *Renibacterium salmoninarum*, which causes kidney disease, were introduced with imports to Chile from the northern hemisphere, the rickettsial disease was believed to have its source in native aquatic species of Chile (Fryer et al. 1990). Interesting aspects of the microorganism were that it was not reported in fish held in fresh water, and was first observed 6-12 weeks after transfer of fish to saltwater rearing pens. Horizontal transmission of the pathogen was achieved experimentally in both fresh water and sea water (Cvitanich et al. 1991).

Fryer et al. (1990), who isolated the organism from Chilean fish and studied it in Chile and Oregon, made the following comment:

*The potential pathogenicity of the organism, its apparent virulence, and the fact that it is not known to occur outside of Chile dictate that these studies be conducted in the area where the disease is endemic. For these reasons, the agent will be returned to Chile for infection experiments in coho salmon.*

This very logical and matter-of-fact statement by Fryer et al. (1990) illustrates a broader admonition about the potential for introduction of disease agents – that, unless absolutely fail-safe quarantine facilities exist, studies of a pathogen should be conducted in the area where a disease is endemic; the pathogen should not be transported in any way to non-endemic areas for in vivo studies, simply because of the availability of expertise and facilities in those areas.

This guideline is based on the likelihood that a non-endemic pathogen may somehow be disseminated outside the laboratory or experimental facility if it is transported for study beyond the area where it occurs. Such dissemination could involve accidental infection of commercially valuable native fish stocks, with resulting mass mortalities.

In the case history described – rickettsial infection in coho salmon – the stricture against experimentation with the pathogen outside its endemic area does not seem to have been followed. Other investigators (Cvitanich et al. 1991) reported infection experiments in seawater aquaria apparently conducted in Oregon, although the actual site of the study was not specified in their report – except that it was a “non-fish rearing quarantine facility.”

The conditions defining quarantine have become just too variable in many marine infection studies to justify confidence in absolute exclusion of non-endemic pathogens from natural waters of recipient countries. 1

**Chronic Losses and/or Diminished Quality of Product Pacific Coast Protozoans**

Some tissue-invading protozoan (myxosporean) parasites have been found recently to invade introduced species. Several examples have been reported in cage culture of salmonids on the west coast of the United States. One is the transfer of the histozoic myxosporean, *Paracaudula* sp., from Pacific cod, *Gadus macrocephalus*, to cage-reared salmonids, and the transfer of another histozoic myxosporean, *Kudoa thyrsites*, from Pacific whiting, *Merluccius productus*, to Atlantic salmon being reared in Pacific wa-
ters (Kabata and Whitaker 1985, Schiewe et al. 1988).

INCREASED SUSCEPTIBILITY TO DISEASE OF EXOTIC ORGANISMS CULTURED IN UNSUITABLE OR MARGINAL ENVIRONMENTAL CONDITIONS

Acute Mortalities

Bacterial Diseases

*Tilapia aurea* and red tilapia, *Tilapia* sp., are reared in seawater because of 1) increased growth in sea water, 2) lack of freshwater resources in many countries, and 3) product identification with the marine environment in areas where undesirable freshwater tilapia exist. Although *Tilapia mossambica* has become an established marine exotic off some Pacific islands, the cultured tilapias have not become established in salt water (though not through any lack of opportunity). If water conditions and diet are not very carefully maintained, these animals suffer devastating outbreaks of external lesions and systemic infections with local facultative marine bacteria. *Vibrio* sp., *Pseudomonas* sp., and oddly *Aeromonas* sp. have been isolated from these mass mortalities. This essentially physiological problem may prove to be a severe limitation to this promising culture practice (Bunkley-Williams and Williams, unpubl. data).

Chronic Losses

Pacific oysters, *Crassostrea gigas*, have been imported to the west coast of North America as seed from Japan since the 1920s, and now form the basis for a significant food-producing industry. Production now equals that of the Gulf of Mexico and exceeds that of the Middle Atlantic States. Periodically, though, production has been affected in some grow-out areas by so-called “summer mortalities,” resulting in deaths of adult oysters in some parts of Willapa Bay and southern Puget Sound in particular. Similar mortalities have been observed in native stocks in Matsushima Bay, Japan (Imai et al. 1965, 1968, Kanno 1965, Tamate et al. 1965, Koganazawa 1975), and were attributed to effects of physiological stress, particularly high temperatures, eutrophication of bays and over-ripening of gonads.

An alternative explanation for the cause of mortalities was a presumed bacterial disease of *C. gigas* from Japan and the state of Washington, reported by Sindermann and Rosenfield (1967) and described as “focal necrosis” (Fig. 5). The condition was similar to that described as “multiple abscesses” by Japanese workers (Numachi and Oinumi 1965). Recent reexamination of the condition has led to identification of an actinomycete, *Nocardia* sp., as the etiological agent of the fatal inflammatory disease (Elston et al. 1987, Friedman and Hedrick 1991). A significant observation made during summer mortalities of *C. gigas* on the west coast of North America was that the mortalities seemed to be associated with certain warm (> 20°C) eutrophic embayments in Washington and British Columbia, and to occur especially among oysters undergoing excessive gonad maturation (Glude 1975, Perdue 1983, Beattie et al. 1988).

Reporting on a recent study of oyster mortalities, Friedman and Hedrick (1991) suggested that extreme environmental conditions present in summer may act as stressors, decreasing the oysters' resistance to bacterial infection—in particular to opportunistic pathogens such as *Nocardia* sp. Summer mortalities have occurred frequently—even annually—in some of the embayments in which the introduced oyster is grown, indicating a probable marginal environment for that species in those areas.

PREDISPOSITION OF ENVIRONMENT TO PATHOGEN PROBLEMS

Introduction of Invertebrate Intermediate Hosts for Potentially Damaging Parasites

The complexity and specificity of intermediate hosts in the life cycle of many devastating parasites of marine organisms act as barriers to their dispersion. The greater the diversity of exotics, the more likelihood exists of establishing suitable intermediate host mixes. Many marine invertebrates are being transferred to ports around the world in or on ships. We should be cautious in disseminating the invertebrate populations, since a parasite with suitable intermediate host requirements could be spread throughout the world with such a network.

Trematode Introduction to North America

An excellent and detailed example of the effects of an introduced parasite on the recipient ecosystem can be seen in the introduction, more than a century ago, of the heterophyid trematode *Cryptocotyle lingua* to the north-

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**Figure 5.** Histopathology associated with bacterial disease (nocardiosis) in *C. gigas.*
east coast of the United States. The worm entered from northern Europe probably with its snail intermediate host, the periwinkle Littorina littorea, on the hulls of sailing ships. The snail multiplied profusely and now dominates rocky intertidal areas of the New England coast— even serving as the base for a minor food production industry in Maine.

Other hosts in the life cycle of C. lingua are juvenile Atlantic herring, Clupea harengus, and other inshore fishes, and (as definitive host) the sea gull, Larus argentatus (Fig. 6). Each host in the life cycle is affected, sometimes severely, by the presence of the trematode:

1. Trematode infections obliterate the digestive gland and gonad of the snail first intermediate host, leading to cessation of egg and sperm production and increased mortality. Furthermore, snails infected by C. lingua do not participate in normal vertical intertidal migrations, and many remain behind in the harsh winter high tide zone in New England.

2. Cercariae of C. lingua invade and encyst in the fins and integument of herring, cunner, Tautogolabrus adspersus, and a number of other inshore western Atlantic species, causing the formation of conspicuous cysts or "black spots" (Fig. 7). It has been demonstrated experimentally (Sindermann and Rosenfield 1954) that massive cercarial invasion will blind and kill immature herring (Fig. 8), and it has been postulated that invasions of comparable magnitude are possible in the inshore habitat of the fish. A similar conclusion about mortality in wild populations of O-group plaice, Pleuronectes platessa, in the British Isles as a consequence of invasion by larval C. lingua was reported by Steele (1966) and MacKenzie (1968), and was supported by findings of experimentally induced mortality when plaice were exposed to 100-1000 cercariae per fish (MacKenzie 1971).

3. Adult C. lingua inhabit the digestive tract of adult
3. Increased susceptibility of introduced species to disease, when they are cultured in unsuitable or marginal environmental conditions; and
4. Predisposition of the environment to pathogen problems.

The range of examples provided in this report seems broad enough to permit the conclusion that disease interactions associated with introductions can be severe, sometimes leading to drastic ecosystem dislocations.

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LITERATURE CITED

Glude, J. B. 1975. A summary report of Pacific oyster


1 (Note: The principle elaborated here may have some merit, but the coho/rickettsia example used shows signs of collapsing. Recent information (Hoskins informal report 1992) suggests that a rickettsial organism, serologically the same as the Chilean isolate, may have been seen as early as 1970 in several salmonid species from British Columbia, but not noted there as a virulent pathogen.)