Disease Risks Associated with Importation of Nonindigenous Marine Animals

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Introduction

Considerations of introduced marine organisms usually involve concerns that may be categorized as:

1) Those related to possible ecological changes (especially changes in habitat, competition, and predation);

2) Those related to possible genetic influences on native species; or

3) Those related to the introduction of pathogens not endemic to the receiving area.

Of these concerns, much attention has been directed to the third—disease and its implications in introductions. This is entirely logical, since disease may have profound effects on populations, especially those of economic value, and since some marine disease problems have proven to be remarkably intractable for extended periods.

In this paper I consider several case histories that illustrate emerging concepts about introduced diseases and then offer recommendations to reduce the risks of disease when marine animals are inserted into new environments by human acts. The case histories discussed are: 1) Viral diseases of shrimp, 2) viral diseases of salmon, 3) rickettsial disease of coho salmon, 4) oyster diseases, 5) *Perkinsus* (protozoan) disease of bay scallops, and 6) eel nematodes.

Viral Diseases of Shrimp

Growing worldwide interest in penaeid shrimp culture has led to extensive transfers and introductions of species with desirable culture characteristics. Six lethal viral pathogens have been recognized. Of these, IHHNV (infectious hypodermal and hematopoietic necrosis virus) is one of the most serious. This highly lethal pathogen poses a serious threat to shrimp culture (Lightner et al., 1983a; Bell and Lightner, 1983, 1984). Found in cultured *Penaeus stylirostris, P. monodon, P. japonicus,* and *P. vannamei,* and in wild-caught *P. monodon* brood stock in Southeast Asia (Lightner et al., 1989), it has been introduced into aquaculture facilities in Hawaii, Florida, Texas, Tahiti, Philippines, Guam, and elsewhere. *Penaeus stylirostris* is especially susceptible, and extensive mortalities from IHHNV infections have been reported. *Penaeus vannamei* harbors subacute infections that may cause stunting (Kalagayan et al., 1991). Experimental infections with IHHNV have been achieved in *P. setiferus, P. aztecus,* and *P. duorarum.*

IHHNV disease was first recognized in 1981 in Hawaiian shrimp culture facilities rearing *P. stylirostris* introduced from Panama (Lightner et al., 1983b). Soon after, the role of *P. vannamei* as a carrier of the virus was recognized. The virus can be observed in juveniles and adults, where its effects are found, but not in larvae or postlarvae. Of major concern is the possibility of infection of native populations by the introduced pathogen, even though there is yet no definitive evidence demonstrating that this has happened. Strict quarantine of imports is extremely important, as is care in selection of IHHNV-free sources of brood stock.

The most recent event in the continuing shrimp virus story is the spread, in 1989 and 1990, of IHHNV to the developing shrimp aquaculture industry of northwest Mexico, with consequent major production losses (Lightner et al., 1992).

Some appreciation for the extent of the viral problem in shrimp can be seen by examining the movement of stocks during the past two decades from centers of research and commercial development in Hawaii, Tahiti, Panama, Japan, and elsewhere. Such extensive movements create a transfer network, often operating without adequate disease inspection. IHHNV and other viruses have already entered this network

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ABSTRACT—Transfers and introductions of marine species have occurred and are occurring on a worldwide basis, largely in response to perceived needs of expanding aquaculture industries. Greatest interest is in salmon (cage rearing and ocean ranching), shrimp, and bivalve mollusks, although other organisms are being considered. Such movements of animals carry an associated risk of moving pathogens into areas where they did not occur previously, possibly resulting in infections in native species. Many case histories of the effects of introduced pathogens and parasites now exist—enough to suggest that national and international action is necessary. Viral pathogens of shrimp and salmon, as well as protozoan parasites of mollusks and nematode parasites of eels, have entered complex "transfer networks" developed by humans, and have been transported globally with their hosts in several well-documented instances. Examining the records of transfers and introductions of marine species, incomplete as they are, permits the statement of emerging principles—foremost of which is that severe disease outbreaks can result from inadequately controlled or uncontrolled movements of marine animals.

and are being introduced into farms far from their original geographic range (Fig. 1).

This concept of a transfer network is extremely important. Humans are busy creating these complex transfer networks with many cultured marine species, but especially with shrimp, oysters, salmon, and eels. Severe pathogens are entering these networks and moving along them, killing substantial numbers of introduced and native stocks. As pointed out by Lightner (1990), "It is apparent that if an unrecognized pathogen entered any facility in the transfer network, the mechanism exists for it to be rapidly transferred to several other facilities. Further, if the pathogen remained undetected, it could be easily introduced into all of the facilities in the transfer network."

Viral Diseases of Salmon

Sporadic attempts to introduce Pacific salmon, *Oncorhynchus* spp., to Atlantic waters have been made for more than a century (Solomon, 1979, 1980; Harache, 1992). Long-term establishment of runs and reproductive populations have been unsuccessful to date—in the sense that continued existence of runs depends on annual importation of eggs from Pacific sources.

Despite this record of failure, attempts still continue, adding new approaches such as cage culture in coastal waters and private ocean ranching dependent on hatchery production of young fish. Coho salmon, *O. kisutch,* have been the focus of cage culture efforts; pink salmon, *O. gorbuscha*, and chum salmon, *O. keta*, are species of choice for ocean ranching, principally because they may be released to open waters at an early age. France has had an active program importing coho for sea cage culture (Fig. 2) (Harache, 1992).

Several large-scale introductions of Pacific salmon have been made in Atlantic waters (Fig. 3). The Soviet Union transferred large numbers (up to 35 million per year) of pink salmon eggs from the Kamchatka Peninsula and Sakhalin Island to the Kola Peninsula near northern Norway during 1957–77 (Grinyuk et al., 1978). Some self-sustaining populations appear to have developed, although there is doubt about their long-term persistence in the absence of continued importation of eggs from the Pacific. Some straying to northern Norway occurred (Bjerknes and Vaag, 1980).

The State of New Hampshire had a long-term (1960–90) program of importation of coho salmon eggs. There was limited evidence for some natural spawning, with low survival (Stolte, 1974) but permanent runs have not been established.

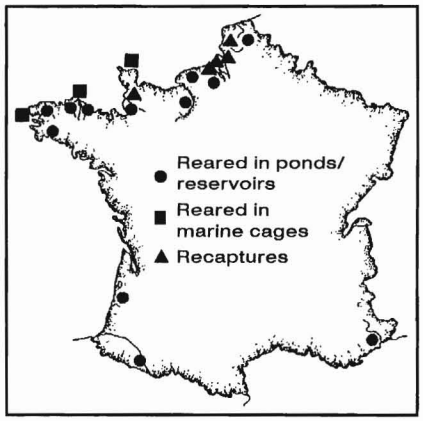

Figure 2.— Locations in France where nonindigenous coho salmon were reported during 1970–80 (modified from Euzenat and Fournel, 1981).

In all of these Pacific salmon introductions, primary concerns have been competition with native Atlantic salmon, *Salmo salar*, and the possible importation of diseases. Diseases of greatest recent concern are infectious hematopoietic necrosis (IHN) and viral hemorrhagic septicemia (VHS)—both lethal viral diseases:

1) IHN now occurs in Italy and France (Bovo et al., 1987; Hattenberger-Baudouy and de Kinkelin, 1988), probably introduced with rainbow trout, *O. mykiss*, eggs

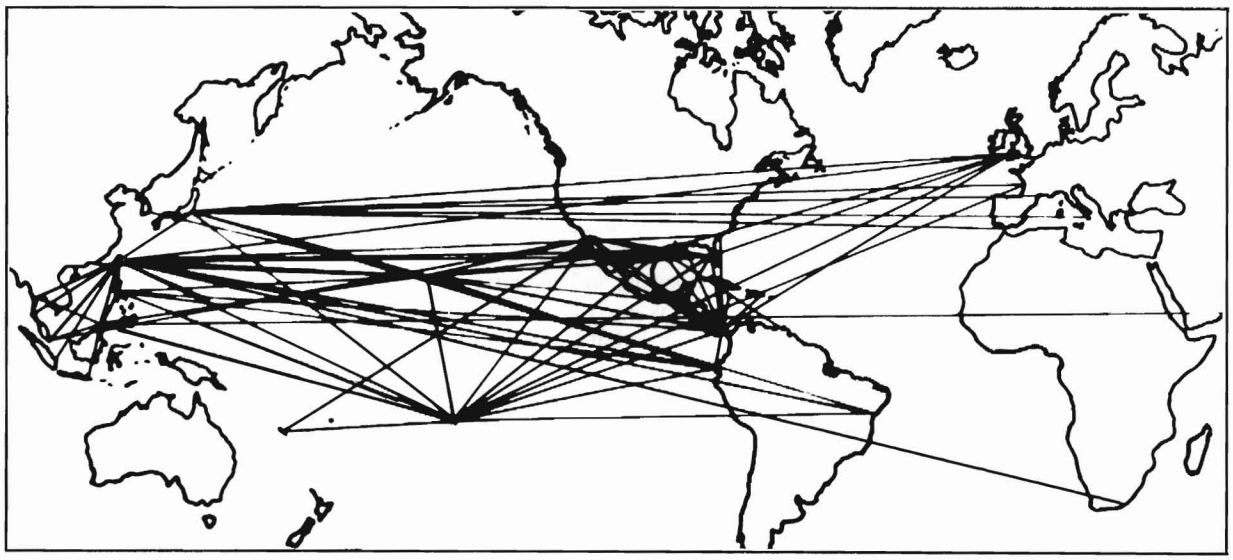

Figure 1.— A "shrimp transfer network" prepared by and courtesy of D. V. Lightner (1990) from published and unpublished reports of shrimp stock movements.

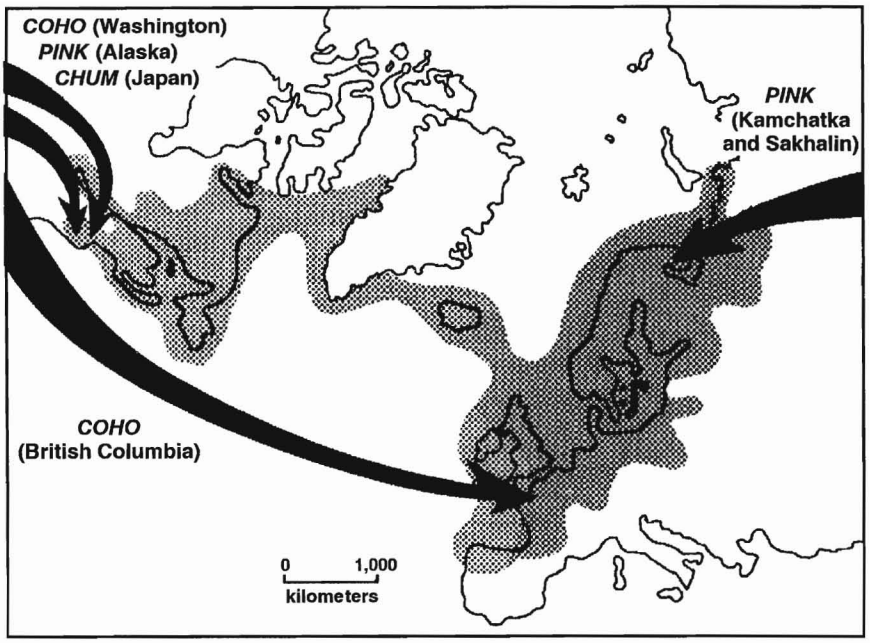

Figure 3.— Distribution of Atlantic salmon, with arrows indicating principal locations of recent Pacific salmon introductions (modified from MacCrimmon and Gots, 1979).

from the west coast of United States, where the pathogen is common.

2) IHNV was introduced into Hokkaido, Japan, probably with the transfer of sockeye salmon, *O. nerka*, eggs from Alaska in 1968. The virus spread rapidly to Honshu island where it has become enzootic. IHNV has also been isolated from rainbow trout mortalities in Taiwan, Korea, and the People's Republic of China, probably as a result of the importation of rainbow trout or eggs from Japan (Chen et al., 1985).

3) VHS—common in Europe, especially in rainbow trout—was reported in salmonids from the Pacific coast of North America in 1988 (Brunson et al., 1989; Hopper, 1989; Winton et al., 1989). Initial concerns were that the virus had been imported from Europe as a consequence of aquaculture activities. However, recent studies have disclosed genetic and virulence differences in VHS virus isolates from Pacific salmon as compared with European isolates (Bernard et al., 1991; Oshima et al., 1991; Winton et al., 1991), and VHS virus was isolated in 1990 and 1991 from Pacific cod caught in Alaska—possibly suggesting a reservoir for North American strains of the virus in marine species (Meyers et al., 1991).

The possible introduction of severe pathogens with movements of salmon leads directly to another concept—that all introductions of pathogens or parasites are "accidental," even though the introduction of host animals can be deliberate. This fact is often overlooked in discussions of "intentional" vs. "unintentional" introductions, and even in drafting legislation concerning introduced species. Emphasis must therefore be on control and exclusion of diseased animals and infected resistant carriers—through programs similar to those that have evolved to control disease dissemination in terrestrial animals.

Rickettsial Disease of Coho Salmon

During the past decade, a number of salmonids, including the coho salmon, 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, *Piscirickettsia salmonis*, has been identified as the causative agent (Cvitanich et al., 1990; Fryer et al., 1990; Branson and Diaz-Munoz, 1991; Fryer et al., 1992; Garcés et al., 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 is believed to have its source in native aquatic species of Chile (Fryer et al., 1990). Interesting aspects of the microorganism are 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 and salt water (Cvitanich et al., 1991). In addition to coho salmon, other salmonid species, notably Atlantic salmon and chinook salmon, *O. tshawytscha*, and rainbow trout, have been found to be susceptible to the rickettsial infection.

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

"In vivo studies are planned to test this hypothesis [that the rickettsia isolated is the causative agent of the coho salmon disease]. 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 statement by Fryer et al. is in my opinion the embodiment of another important concept—albeit one difficult to implement in practice:

Studies of a pathogen should be conducted, insofar as possible, in the area where the disease it causes is enzootic; the pathogen should not be transported to nonenzootic areas for in vivo stud-

ies, simply because of the availability of expertise and facilities in those areas. Reality suggests that some exceptions might be considered. Isolation wet-laboratories, designed for the express purpose of studying exotic aquaculture pathogens in their natural or experimental hosts, could be used when studies are not feasible in areas where the pathogen in question is enzootic. Establishment of several regional and/or international isolation wet-labs (with funding for construction and operation) would help to reduce risks from introduced pathogens.

This concept is based on the likelihood that an introduced pathogen may be accidentally disseminated outside the laboratory or experimental facilities if it is transported for in vivo studies in experimental fish populations outside the area where it currently occurs. Such dissemination could involve accidental infection of commercially valuable native fish stocks with resulting mass mortalities.

Unfortunately, in the case history described in this section—rickettsial infection in coho salmon—the stricture against in vivo experimentation with the pathogen outside its enzootic area does not seem to have been followed. 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 introduced pathogens from the natural waters of recipient countries. Exceptions to this admittedly severe restriction might be made only for experiments in facilities where absolute control of pathogens and of experimental animals can be guaranteed—where containment is equivalent to that of facilities involved in experimental studies using pathogens of public health significance.

(Note: The principle elaborated here may have some merit, but the example used—the Chilean rickettsia—shows signs of collapsing. Recent information (Hoskins[1]) 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.)

Oyster Diseases

Oysters have long been known to be subject to mass mortalities, but the past three decades have been especially troublesome. Major and widespread mortalities have occurred and in some cases are still occurring in the United States, Europe, and Japan. Specific pathogens, often viruses or protozoans, have been identified as causative organisms in most instances.

Oysters have also been moved from place to place probably more frequently than any other marine animal group, providing excellent vehicles for dissemination of pathogens and parasites. Transfer networks comparable to those known for shrimp have been created—especially for the Pacific oyster, *Crassostrea gigas* (Chew, 1990), and important pathogens have been transported, sometimes by complex pathways (Elston et al., 1986).

One of the largest experiments in the introduction of marine species was the importation to the coast of France of the Pacific oyster, *C. gigas*, during 1966–77. Introduced as seed and as adults from Japan and British Columbia, *C. gigas* replaced declining populations of the so-called "Portuguese oyster," *C. angulata*, which had been the industry mainstay for decades. The Pacific oyster prospered; reproduction was successful in some parts of the French coast, and production now exceeds 150,000 t (Grizel and Héral, 1991).

What have we learned from this massive French experiment from a disease perspective? A remarkable series of epizootics in the two native species of oysters occurred simultaneously with the Pacific oyster introductions. Beginning in 1966, the native oyster *C. angulata* died in large numbers from a viral gill disease (Comps and Duthoit, 1976; Comps et al., 1976). By 1973, when the epizootic subsided, populations of *C. angulata* had been largely destroyed, and the introduced Pacific oyster, *C. gigas*, had replaced them in most grow-out areas. During the same period, populations of the European flat oyster, *Ostrea edulis*, were also affected severely by epizootic disease (Comps, 1970; Comps et al., 1980). Beginning in 1968, the protistan parasite *Marteilia refringens* affected oyster growing areas. As that epizootic waned in the mid-1970's, *Bonamia ostreae*, another protistan parasite, increased to epizootic proportions and further reduced populations of *O. edulis*. This epizootic continues at present, and, as a result, *O. edulis* culture is at a standstill. There is some evidence that the pathogen *Bonamia* was introduced to France with imports of *O. edulis* seed from a California hatchery that had been rearing offspring of a stock that originated decades earlier in the Netherlands, and was first introduced into Connecticut in the 1950's (Elston et al., 1986).

The occurrence within a single decade of three major oyster epizootics, each with accompanying mass mortalities, is unique in the long and well-documented history of oyster culture. Also unique is the scale of importation of a replacement species (*C. gigas*) during this same period. Despite extensive research by European pathologists, the role that this massive introduction of a nonindigenous species may have played in disease outbreaks in the native species is unknown, and a direct relationship has not been demonstrated, although suggestions have been made and some associations proposed—like the one describing the history of *Bonamia*.

The worldwide experience with introduced *C. gigas* has indicated that at least three categories of disease risks exist.

1) One risk is from the known pathogens of the species, which may or may not be transferred to the native species. For *C. gigas*, five pathogens are known:

[1]Hoskins, G. 1992. Salmonid rickettsial agent identified in British Columbia. Pacific N.W. Fish Health Prot. Comm., Vancouver (December 1991), Meet. Highlights, Newsl. March 1992, p. 1.

a) An iridovirus that causes "larval velar disease" (and which is similar to the virus that killed *C. angulata* in France);

b) A bacterium, *Nocardia* sp., which causes fatal inflammatory bacteremia;

c) An ascetosporan protozoan parasite, *Marteilioides chungmuensis*, which affects ova;

d) A presumptive ascetosporan protozoan, *Mikrocytos mackini*, which causes "Denman Island disease" in *C. gigas* introduced on the west coast of North America, but yet unreported from native stocks in Japan (Farley et al., 1988); and

e) A parasitic copepod, *Mytilicola orientalis,* found in the gut.

2) Another risk is from other organisms with unknown pathogenicity to *C. gigas*, but possibly pathogenic to other species of oysters (an example of this category would be the reported occurrence of a haplosporidan parasite, similar to *Haplosporidium nelsoni*, a severe pathogen of American oysters, in *C. gigas* from Korea, but not known to be pathogenic to *C. gigas*).

3) A third risk arises from still other organisms, rare or unrecognized in populations of *C. gigas*, that may be pathogenic to related species of oysters (examples suggested, without supporting evidence, are *Marteilia* and *Bonamia* in flat oysters, *O. edulis*).

The uncertainties present in this listing are reflections of the relative youth of marine pathology as a scientific discipline. Many diseases of oysters and other species are unknown or poorly understood, so they would not form part of normal inspection protocols.

Closer to home, the native east coast American oyster, *C. virginica*, has been hard hit in the Middle Atlantic states by epizootics of two protozoan pathogens that began in the late 1950's and still persist. Oysters are frequently shipped from state to state, and documentation is good for the transfer of one of the diseases ("MSX" disease, caused by the protozoan *Haplosporidium nelsoni*) to states where it had been previously unknown (Massachusetts, South Carolina).

The transfer network for some cultured species can be extraordinarily complex, and some pathways can be quickly obscured or never revealed. An excellent example is seen in the attempt by Elston et al. (1986) to trace the origin of the flat oyster pathogen, *Bonamia ostreae*, that has destroyed most European production of that species beginning in 1979. Export of infected seed oysters to France from a hatchery on the California coast was proposed as the immediate source of the disease. That hatchery had received brood stock (presumably infected) from the Milford Laboratory in Connecticut which, in turn, had received the original introductions from the Netherlands in the 1950's. Somewhere in the early part of this chain of events, possibly at Milford, *Bonamia* infected the introduced species. The severe consequences of bonamiasis for European culture of flat oysters, *Ostrea edulis*, have been described in detail by Balouet et al. (1983), Figueras (1991), Hudson and Hill (1991), McArdle et al. (1991), Stewart (1991), and Van Banning (1991).

This and other examples illustrate the reality that unless early scientific attention is directed to the occurrence of a new parasite or microbial pathogen, the history of its introduction and subsequent dissemination may be quickly lost. Thus, the entire episode will become forever a matter of speculation and conjecture (examples include the origin of *H. nelsoni* (MSX) disease in oysters of Delaware Bay and the origins of recent oyster disease outbreaks in France). As Stewart (1991) has stated, ". . . although many diseases are suspected to have been introduced in one manner or another, irrefutable evidence is usually lacking."

Perkinsus (Protozoan) Disease of Bay Scallops

The previous section on oyster diseases illustrates some of the complexities and uncertainties of disease transmission through transfer networks. One melancholy fact not yet pointed out, however, is that pathological examination of candidate species for introduction may not always disclose the presence of known disease agents and is unlikely to identify unknown agents except by chance. Known disease agents may have cryptic stages or may be extremely rare in the samples examined, and they may elude recognition. Pathological effects of undescribed pathogens may be labeled "idiopathic lesions" or "nonspecific granulomas" and thus escape deserved attention.

An excellent case history of just such an event can be found in the introduction into Canadian waters of a parasitic protozoan—a species of *Perkinsus*, with its host, the bay scallop, *Argopecten irradians*. Histopathological examination of the original imports from United States in 1979–80 disclosed only chlamydia-like and rickettsial infections and nonspecific granulomas (Morrison and Shum, 1982, 1983). The scallops were reared in quarantine for three generations before field release. Then in 1989, after open-water culture had begun, hatchery brood stocks were examined and a *Perkinsus* agent was recognized in the formerly described "granulomas," and subsequently described as *P. karlssoni* by McGladdery et al. (1991). A similar or identical organism had been seen much earlier in bay scallops from U.S. waters (Ray and Chandler, 1955), and more recently by Karlsson (1990). This episode illustrates very well the principle that disease risks from introductions are never zero, even when adequate regulatory and inspection systems exist. Canada has been diligent in developing regulations and a regionally based infrastructure to control introductions of nonindigenous species; yet despite this concern and action, the pathogen, *P. karlssoni*, was introduced into Canadian waters and is now present in Canadian bay scallop populations. The pathogenicity of *P. karlssoni* to bay scallops has yet to be clearly established, however, and interspecies transmission to other molluscs has not been observed (McGladdery et al., 1991).

Eel Nematodes

A recent and expanding disease problem in Europe is the spread of nematode worms, *Anguillicola crassus*,

in native European eel, *Anguilla anguilla*, populations. The worms were introduced with shipments of live Japanese eels, *A. japonica*, from Asia. They are large bloodsucking organisms that occlude the swim bladder (Fig. 4), cause emaciation, result in mortality in holding pens, and interfere with spawning migrations (Koie, 1991).

Infection of native eels was first noticed in Germany in 1982, probably as a consequence of release of infected eels shipped from Taiwan in 1980 (Koops and Hartmann, 1989). The parasites now occur in most of the countries of Europe (Netherlands, Denmark, Poland, England, Spain, Greece, etc.), often with high prevalences and intensities. Infections can be acquired as early as the elver stage, in which an acute inflammatory reaction occurs in the swim bladder; small crustaceans serve as intermediate hosts.

Population expansion following introduction has been rapid. In one river in England (where the worm was first reported in 1987), prevalence levels of 100% and average intensities of 6.7 worms were attained in 1 year (Kennedy and Fitch, 1990). An active network of eel transfers in Europe has undoubtedly favored the rapid expansion of nematode populations throughout the subcontinent. Eel farms have been seriously affected, with reduction in growth rates, emaciation, and mortalities of up to 65% in captive populations.

Like many introduced parasites, *Anguillicola* 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 excellent illustration of at least two more concepts:

1) Once an introduced pathogen is established in a marine/catadromous/anadromous fish population, it is difficult if not impossible to restrict or control its further spread in natural waters. (Control in aquaculture facilities is, however, feasible.)

2) The colonization potential of many parasites in new environments is predictable on theoretical grounds, but not the relative importance of different methods of dissemination (especially human-assisted movements of infected hosts).

Concepts and Principles

The preceding discussion of selected case histories helps to elaborate a sequence of emerging concepts or principles. I have already proposed seven:

1) The development of "transfer networks" of aquaculture species along which pathogens may move;

2) The reality that all introductions of aquatic pathogens have been to date accidental, even though introduction of host animals may have been intentional;

3) Restriction of in vivo experimental studies of fish pathogens to facilities within the zone where the organism is enzootic, unless fail-safe containment facilities exist elsewhere;

4) The need for early scientific attention to the appearance of a new parasite or pathogen;

5) The reality that disease risks from introductions are never zero;

6) The demonstration that once an introduced pathogen is established in a marine population it is difficult if not impossible to control its further spread; and

7) The observation that the colonization potential of many parasites in new environments is predictable.

Examination of additional cases of the relationship of diseases to importation of nonnative species discloses other generalizations:

1) Although evidence is less than robust and associations must often be made by deduction or inference, it is likely that many of the recent outbreaks of disease in marine populations of commercial importance—especially shellfish—are results of introductions of pathogens from other geographic areas. This may not be the case for salmonids, however, in which the most devastating outbreaks of disease are often due to agents already enzootic, and which are either amplified in aquaculture situations or which can infect an introduced species.

2) Introduced pathogens can be considered logically as "biological pollutants" and, as such, should be subject to all regulations governing pollutant discharge control. This perception of introduced agents has received some support, but has not yet, to my knowledge, been incorporated directly into regulatory regimes concerned with ocean pollution.

3) Disease-causing organisms may seem benign and innocuous in adapted host populations, but may become serious pathogens of related species when introduced into other geographic areas (for example, the eel nematode). Assessment of this potential risk can only be

Figure 4.— Nematodes, *Anguillicola crassus*, occluding swim bladder of European eel, *Anguilla anguilla*. Photograph courtesy of P. van Banning.

done in appropriately designed isolation wet-labs where imported and native stocks can be reared and studied together for some reasonable period of time (preferably one complete life cycle).

4) The possible role of infected resistant carriers in transmitting a disease to a susceptible but geographically separate subpopulation of the same species must be considered in decisions about transfers within the total geographic range of the species.

5) Two forms of disease risks exist when animals are relocated outside their normal range:

a) The infection of introduced stocks by an enzootic pathogen in the recipient country, to which native stocks are resistant—with the possible overwhelming of that resistance by increased infection pressure or increased virulence (Fig. 5).

b) The introduction of a pathogen that affects susceptible native species but to which the introduced stock is resistant (Fig. 6).

These dual risks may be further complicated by interbreeding of a resistant introduced stock with remnants of a disease-ravaged native stock.

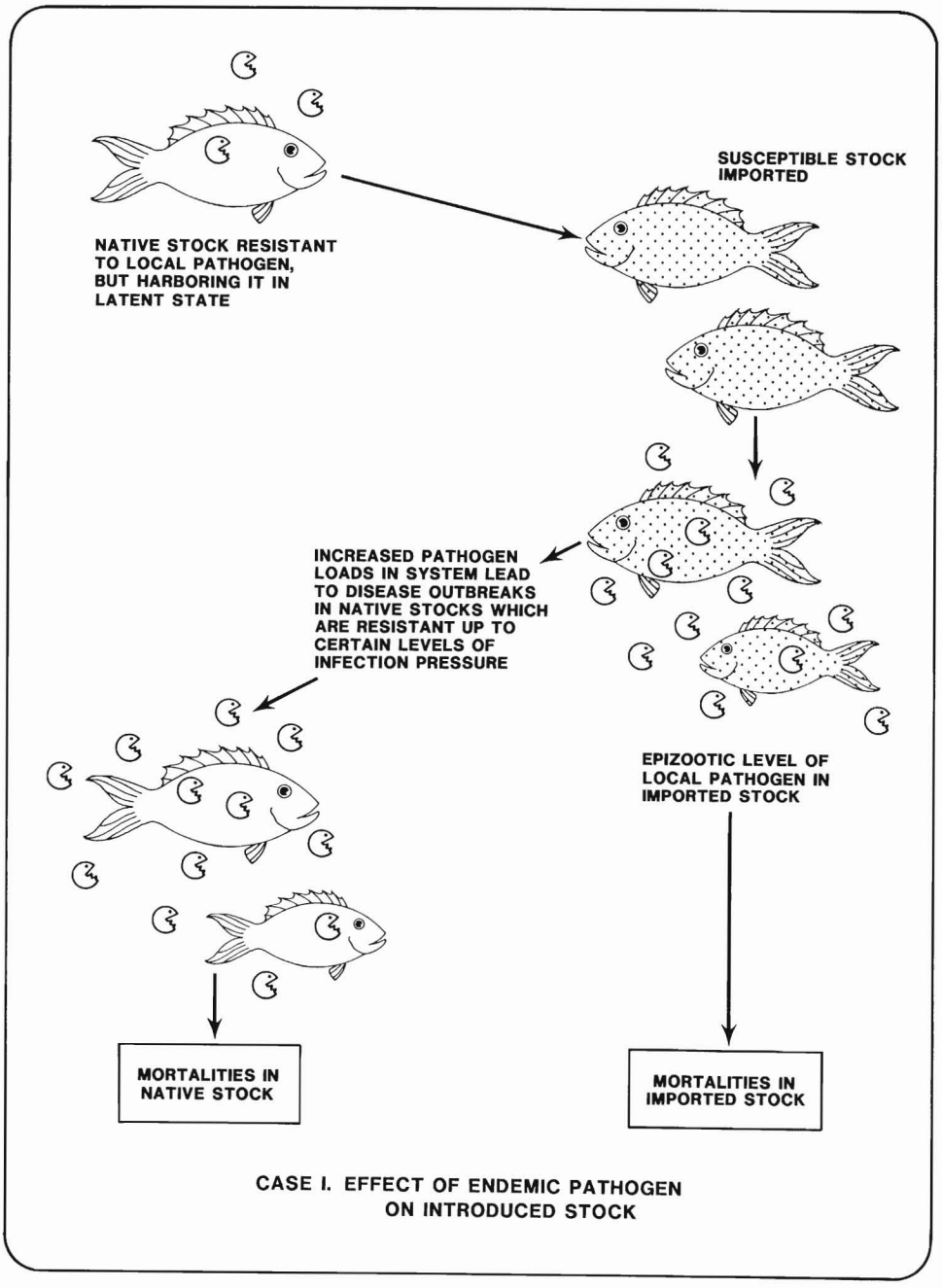

Figure 5. — Effect of an enzootic pathogen on an introduced stock.

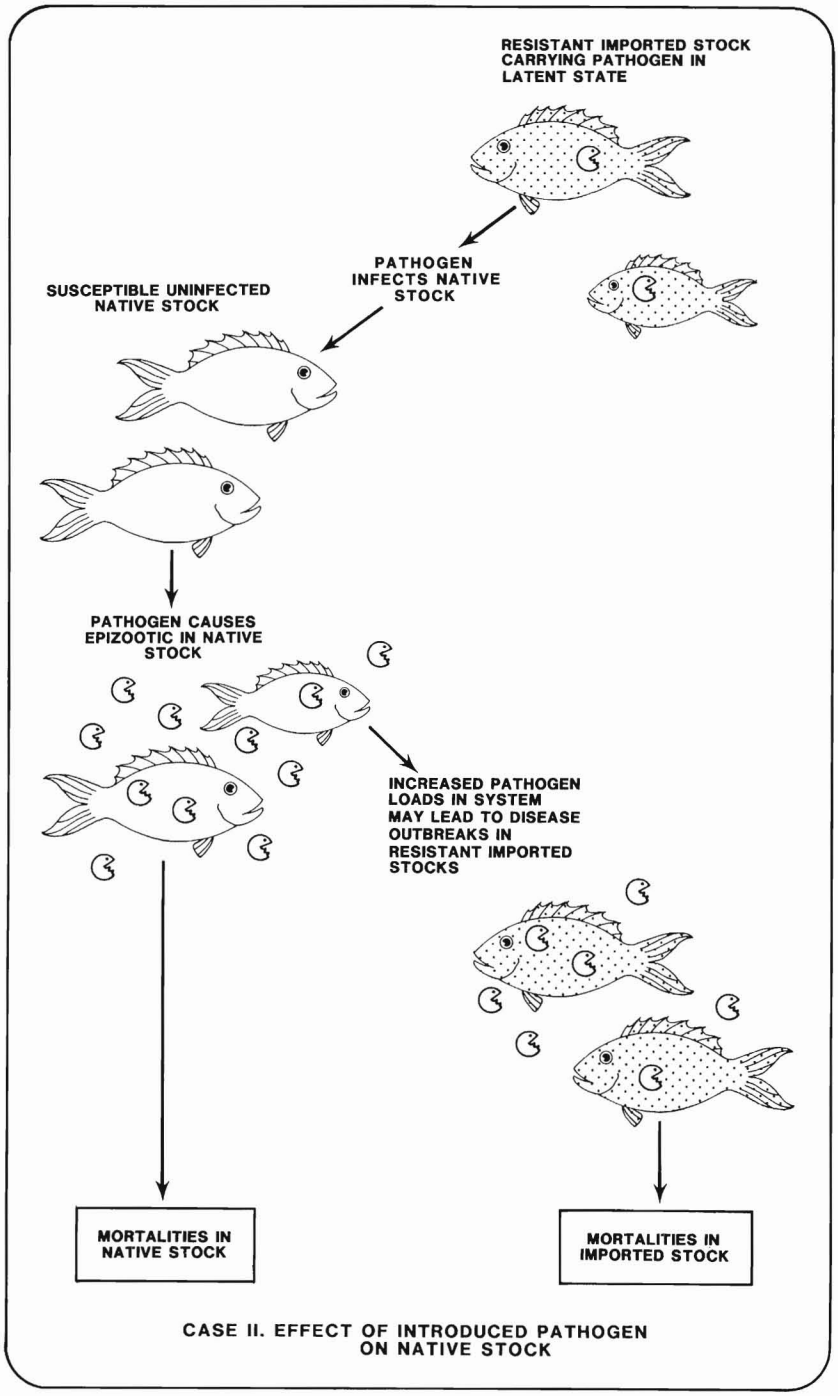

Figure 6. — Effect of an introduced pathogen on native stock.

This may lower the resistance of the new population to the pathogen. Another danger, particularly from introduced viral agents, is the rapid potentiation of virulence in intensive rearing facilities—as has been seen with IHNV in trout farms.

The validity of any of these proposed concepts or principles must, of course, be scrutinized continuously as new information becomes available. The listing here merely represents the author's assessment of our present understanding.

Recommendations

It is becoming increasingly apparent that national and international action is necessary to provide some measure of oversight and control of introductions of aquatic organisms, whether they be deliberate or accidental. Beyond this general recommendation, several proposed actions relate specifically to the problem of introduced diseases:

1) A substantial reduction in the global dissemination of diseases of aquatic organisms could be attained by the development of native species or species stocks, through scientific management and aquaculture practices (including selective breeding and genetic manipulation) as an alternative to introducing nonnative species.

2) A vigorous international program of marine disease research and control should be developed by the Permanent Commission for the Study of Fish Diseases of the International Office of Epizootics (OIE). This intergovernmental veterinary organization, based in Europe, is a logical focus for the kind of coordination that will be required (de Kinkelin et al., 1990). Included would be the development of models for inspection and certification programs, standardized protocols for disease examinations, and the implementation of an effective communication network.

3) Regional maps should be developed and kept current for each host species, showing the presence and abundance of each disease that affects the species. Movement of infected animals from an area where the disease is present to one where it is absent should be prohibited.

4) National and regional disease diagnostic centers, with supporting research capabilities, should be established to develop information about species proposed for introduction, or approved for introduction, or introduced accidentally.

5) Specific pathogen-free stocks of marine fish and shellfish should be identified for aquaculture purposes; these stocks should be used as sources of seed. A program should be developed that is modeled on one developed

principally by the U.S. Fish and Wildlife Service and already in use for salmonid hatcheries in the United States.

These recommendations relate specifically to disease problems; other and broader recommendations could and should be made concerning ecological, genetic, legal, and economic aspects of the importation of nonindigenous marine animals. Certain to be included would be these:

1) A clear national policy on introduced species, whether the introductions are accidental or deliberate, should be developed and stated.

2) A national system of inspection and quarantine, with adequate back-up research capabilities, should be developed and funded.

3) An effective regulatory regime and an enforcement system to ensure that regulations are not circumvented should be developed.

4) Proposed introductions should have clearly stated and demonstrated rational bases. Proposals which are without adequate rationale, poorly planned, or unnecessarily risky, should not be approved.

5) Decisionmakers should be aware of, and sensitive to, the practical, economic, social, and political aspects of introductions, but should evaluate proposals principally on the basis of the available scientific data. Relevant scientific implications and viewpoints include, but are not limited to:

a) Ecological considerations—including competition, predation, and community characteristics of species (diversity, carrying capacity);

b) Genetic considerations—including the potential for hybridization, change in gene frequency (genetic diversity), and change or modification in disease and/or parasite resistance;

c) Behavioral considerations—including interactions between native and exotic species; and

d) Pathological considerations—including the potential for unintentional introduction of diseases and parasites.

6) All proposed introductions should be accompanied by full and adequate procedures and provisions for post-importation (follow-up) monitoring.

Early consideration should also be given to acceptance, nationally and internationally, of a uniform code of practice concerned with movements of nonindigenous marine species. Consideration might also be given, in developing a U.S. policy on introduced aquatic species, to adopting the "precautionary principle" proposed by Germany and accepted at the Second International Conference on the Protection of the North Sea in 1987. That principle "requires action to reduce pollution even in the absence of soundly established scientific proof for cause and effect relationships." The principle could be applied especially to control accidental introductions (including pathogens), which are clearly forms of "biological pollution."

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