

A Review of the Effects on Fishery Stocks of Pathogens Released by Aquaculture

**By Dr Rob Day
and Dr Jeremy Prince.**

April 2007

A Report to the Abalone Council of Australia

Executive summary.

There has been a growing realisation of the environmental impacts of aquaculture, which include water pollution; habitat destruction; depletion of wild fish stocks for fish meal and fish oils to feed to aquaculture fish and as a source of wild seedstock; genetic dilution of wild stocks from farm escapes of fish or eggs; introduction of exotic pest species; and introduction and/or maintenance of diseases and parasites in wild stocks. This review focuses on the introduction of diseases as a result of aquaculture, but other effects should also temper the enthusiasm with which governments embrace aquaculture development.

A long history of research into diseases of humans and livestock has shown that most of the very damaging diseases are a result of host species “jumps”. That is, the pathogen occurs in another host species, in which it often causes little damage, but when it infects a new host, it causes much more damage – it is virulent. The danger of disease spread from wild animals to intensive animal husbandry on land has been well known for decades. It is well understood that the same issues pertain to animals held in high density in aquaculture. When animals are cultured at high density, diseases can be quickly and easily transmitted between animals. High densities of hosts, often in a stressed condition that reduces their immune function, provide a tinderbox for diseases to catch hold. Further, in intensive animal husbandry (such as aquaculture), one expects disease organisms to evolve to be more virulent, if the disease is not rapidly eliminated.

There have been many examples of diseases that have affected wild stocks as a result of aquaculture, often with devastating long-term results; and these examples were recognised when the Australian national guidelines for translocations in aquaculture were produced in 1999. For almost every important marine species in aquaculture, including abalone, ample evidence of the dangers of infection of wild stocks by diseases from farms existed prior to 1999.

Three types of effects have been identified:

- The introduction of new pathogens to a stock through the importation of exotic animals for culture
- The transfer of pathogens between areas due to the movement of cultured animals
- Amplification of pathogens that already exist in an area, and their transmission from cultured to wild populations.

In-sea aquaculture poses especially high risks of disease transfer if wild stocks are nearby.

The controls applicable on land such as culling, vaccination and quarantine are not available once pathogens have entered marine systems, our knowledge base is poor and predictive tools have not been developed for marine diseases. Further, marine epidemics can spread far faster than most land diseases, perhaps due to the lack of barriers and the ability of pathogens to survive in water between hosts. Thus we would argue that the only good defense for wild stocks is to ensure that farm animals are not within contact range of wild stocks. The range should be measured in kilometers.

TABLE OF CONTENTS

Executive Summary.....2

1. The context: Aquaculture, wild stocks and diseases.....4

2. Pathogens that have affected wild stocks as a result of aquaculture.....6

 Salmon aquaculture6

 Tuna aquaculture.....7

 Trout and Carp aquaculture.....7

 Shrimp aquaculture.....7

 Oyster aquaculture.....8

 Abalone aquaculture.....8

3. How disease virulence may change in aquaculture and wild stocks.....10

4. Minimizing introductions of pathogens into wild stocks.....11

References.....12

1. The context: Aquaculture, wild stocks and diseases.

Worldwide fisheries production is declining and most wild capture fisheries are over-fished or at least fully exploited, so that governments and seafood industry marketers look to aquaculture to meet rapidly increasing demand. As a result, statements such as: “Aquaculture, not the internet, represents the most promising investment opportunity of the 21st century” (Peter Drucker, Economist and Nobel laureate) are believed and used to capture the interest and support of investors and also politicians in setting up Aquaculture. The US Department of Commerce, for example, has called for the development of a \$5billion aquaculture industry in the USA by 2025¹. Thus aquaculture has strong backing and there is an expectation that governments will allow it to develop rapidly. In fact worldwide aquaculture production is growing at about 9% annually¹; much of this being carp production in China. In developed countries, aquaculture is focused on high value species that supply relatively low volume but high price markets², with the expectation that the product value will remain high. This has meant that the best prospects for profits, such as prawns, salmon pearl oyster and Southern Bluefin Tuna farming, have been developed first. These species thus provide a false picture of the profit potential of future aquaculture.

In fact the history of farming these high prices species suggest that successful cultivation moves them towards being a lower price commodity; and unlike fisheries, aquaculture industries can move to the countries providing the best package of labor cost, power costs, biosecurity costs, access to markets, and production risks.

There has been a growing realisation of the environmental impacts of aquaculture, which include water pollution; habitat destruction; depletion of wild fish stocks for fish meal and fish oils to feed to carnivorous aquaculture fish and as a source of wild seedstock; genetic dilution of wild stocks from farm escapes of fish or eggs; introduction of exotic pest species; and introduction and/or maintenance of diseases and parasites in wild stocks^{1,3}. Thus one of the recommendations of the US Marine Aquaculture Taskforce in 2007¹ was that operators of aquaculture facilities should be liable for damage caused by their activities. While this review focuses on the introduction of diseases as a result of aquaculture, the other effects are also reasons to temper the enthusiasm with which governments have embraced the development of aquaculture. In particular, in-sea aquaculture may have many deleterious effects, as discussed for salmon aquaculture below. Of course, these impacts also affect aquaculture operations, which may face ruin from epidemic diseases or tarnishing of their products’ “clean, green” image.

Diseases (pathogens) are parasites that may cause damage to host species. They may persist in another less affected host species, in which they can survive and re-infect the target host. Such resistant hosts are called reservoirs. Diseases in marine organisms are of particular recent concern. There has been an increasing incidence of diseases, some a result of human activity, reported from a wide variety of marine species over the last 30 years; the spread of some epidemics in the sea appear to be faster than in terrestrial epidemics; and major changes in marine populations and communities have been noted⁴. *Yet the controls applied on land such as culling, vaccination and quarantine are not available once pathogens have entered marine systems*; microbiological and molecular tools to identify marine pathogens are underdeveloped; knowledge of the origins and reservoirs of pathogens and the longevity and host range of infectious stages is poor; and epidemiological models still need to be adapted to analyse and predict marine diseases^{4,5}.

Marine epidemics can spread far faster than most land diseases, at rates of up to 3-10,000 km/yr, perhaps due to the lack of barriers and the ability of pathogens to survive in water between hosts⁶. Parasites are known to comprise a very large proportion of the species on the planet, and every wild species is expected to have many parasite species. The fact that marine systems are much more diverse – there are more species of both hosts and parasites – makes the development of all the required knowledge a very daunting task.

A long history of research into diseases of humans and livestock has shown that most of the very damaging diseases are a result of host species “jumps”. That is, the pathogen occurs in another host species, in which it often causes little damage, but when it infects a new host, it causes much more damage – it is virulent. There is no consensus on whether the damage a pathogen causes a host should be called disease severity, pathogenicity or virulence, and the damage will depend on host genes, host condition and the environment as well as the pathogen genes. I will use virulence to mean the characteristics of a pathogen that influence the damage a host suffers. The original host may act as a reservoir or source of new infections. For example wild birds act as a reservoir for bird flu, which affects chickens in Asia. But usually pathogens that “jump” are not transmitted from one new host to another very effectively, especially if the new host is very different from the reservoir host. Thus bird flu is not easily transmitted from one human to another. Obviously in hosts that are stressed and at high density (as in Asian chicken farms) the disease may jump and then spread more easily.

The danger of disease spread from wild animals to intensive animal husbandry on land has been well known for decades. When a disease “jumps” or is introduced from other farms, high densities of hosts, often in a stressed condition that reduces their immune function, provide a tinderbox for diseases to catch hold. When animals are at high density, the disease can be quickly and easily transmitted between animals, and each new animal is likely to be infected by a high dose (many particles) of the pathogen, so their immune defenses are more likely to be overwhelmed. Further, theoretical models predict that these conditions in intensive animal husbandry will lead to an increase in the virulence of epidemic diseases during the outbreak⁷. ***This is explained further in the section on pathogen evolution below. It is well understood that the same issues pertain to animals held in high density in aquaculture***¹.

It has also become well-known that diseases may spill over from farm animals into wildlife⁸. Examples include the introduction of rinderpest to African grazers⁹ – it took decades for the Serengeti wildlife herds to recover; and the effect of bovine TB on gazelles, buffalo and lions in South Africa’s Kruger National Park¹⁰. ***There have also been many examples of diseases that have affected wild stocks as a result of aquaculture, often with devastating long-term results (see below); and these examples were recognized when the Australian national guidelines for translocations in aquaculture were produced in 1999***¹¹. This, together with the annual export value and capital value of the abalone wild fishery makes it particularly inexplicable that a review of environmental monitoring requirements for Victorian aquaculture¹², in assessing the hazard of disease transfer to wild stock from abalone aquaculture in 2002, classified the likelihood as “The event may occur at some time, say once in 10 years”, but the consequence as “moderate harm with mid-term recovery” and thus the Risk as “Low: no major concern”. Obviously, wild stocks are managed for sustainable production over decades, there are NO known remedies once diseases take hold in the sea, and recoveries

of wild fish stocks from serious disease mortality are likely to take several decades. Thus the consequence and risk appear to have been wildly underrated.

2. Pathogens that have affected wild stocks as a result of aquaculture

Most aquaculture has developed on species that fetch a high price, and most research has also been done on such species, although the diseases of species that have been in aquaculture for longer periods, such as oysters, are also fairly well known. There also tends to be better knowledge of the diseases (and immune defenses) of fish than of invertebrates, because methods to work on vertebrates are better understood.

Three types of effects have been identified¹³ :

- *The introduction of new pathogens to a stock through the importation of exotic animals for culture*
- *The transfer of pathogens between areas due to the movement of cultured animals*
- *Amplification of pathogens that already exist in an area, and their transmission from cultured to wild populations.*

I would argue that there is also an obvious danger of the introduction of pathogens from one local host to another, and its subsequent adaptation to the new host in aquaculture, or even hybridization of resident pathogens, especially where new strains or hybrid animals are cultured. This form of effect is obviously more difficult to demonstrate, and this may be why it has not yet been detected.

Salmon aquaculture

Probably the best understood effects of aquaculture on wild stocks are related to salmon culture. Salmon culture developed first as restocking programs in rivers, then came to involve huge sea-pens in Norway and Canada. This technology is now used worldwide, e.g. in Maine, Chile, Tasmania. A number of pathogens have been spread to wild stocks. Infectious salmon anaemia has spread from farmed to wild populations, and from Norway to Canada and Maine over several years¹⁴. Furunculosis spread to 20 Norwegian river salmon populations after the introduction of allegedly infected Atlantic salmon for aquaculture from Scotland¹⁵. The best-studied example involves sea lice, which attach to and feed on salmon. The young wild salmon that move down their native rivers now become infected as they reach the sea by sea lice from the farms located near river entrances, and this leads to much lower survival of these fish in some areas^{16,17}, although salmon survival may be high in some years, perhaps because good rainfall reduces the lice numbers^{18,19}. This problem has been particularly well documented in British Columbia, where infection was estimated at 70 times the normal levels near farms¹⁷, but it occurs in many other countries with wild salmon runs. ***The problem stems from the high densities of fish farmed in the sea, in close proximity to wild stocks.***

In addition, salmon farming is usually done in leases in the sea, and it is noteworthy that this form of aquaculture has now been found to have caused significant losses of benthic biodiversity, changes to sediments, increased nutrients and harmful algal blooms, increases in scavenging birds, and reduction of native fish due to fish escapes^{20,21}. ***Thus in-sea aquaculture, besides posing increased risks of disease transfer if wild stocks are nearby, may impose many other deleterious effects on the marine environment and resources.***

Tuna aquaculture

Another example involves Australia. Pilchards (also called sardines) along the coasts of southern Australia contracted a herpes virus, which caused two separate epidemics of pilchard deaths, in March-May 1995²² and 1998/9²³ that killed perhaps 75% of the pilchard stocks. The first disease epidemic spread as a visible front of dead adult pilchards along the coast (at 11,000 km/yr in 1995 and at 5,480 km/yr in 1998), and extended over the entire distribution of the pilchards, from north of Fremantle to Queensland within 3 months²². The pilchard deaths in turn caused mass starvation and lowered breeding success in little terns in Victoria²⁴ and of little penguins, which are a major tourist industry for Victoria²⁵. Furthermore the pilchards are an important food source for juvenile wild Southern Bluefin Tuna in the Great Australian Bight²⁶, so the epidemics may well have negatively affected the wild tuna stocks too. This is on top of the significant socio-economic impacts on the West Australian pilchard fishery²³.

Both epidemics started close to the area that Southern Bluefin Tuna are cultured in South Australia. Although there appears to be no definitive evidence to link this farming to the epidemic deaths, as the virus has not been matched to viruses elsewhere in the world, the tuna were fed using frozen pilchards imported into Australia (10-16,000 tons in 1995), so that a pathogen imported in the feed remains the only reasonable hypothesis^{27,28}. It is known that the infective agent can be transported in frozen fish, as infected stocks of pilchards were fished in Western Australia and transported frozen to New Zealand as fish food, and subsequently a similar epidemic began in New Zealand²⁹. Its rate of spread through the wild pilchard population can be modeled in terms of the transmission of an infectious virus between pilchards³⁰. For these reasons *the likely origin of this disease was a pathogen imported in feed, and placed into cages in the sea close to wild stocks.*

Trout and Carp aquaculture

In other fish species that have been brought into aquaculture for some time, the same pattern is evident. Both trout and carp are known to have introduced translocated diseases into wild stocks. Whirling disease spread from fish culture and stocking operations to wild populations throughout North America^{1,31}. The koi herpesvirus in carp was first detected in the USA³² and has spread to numerous countries worldwide, including Japan^{33,34,35}. The virus is excreted in feces and thus probably transmitted through the water³⁶. It remains infective in water for very long periods³⁷.

Shrimp aquaculture

Another widely used type of aquaculture is shrimp farming. Whitespot and Yellowhead viruses first caused huge losses in farms in Asia, then appeared in both farmed and wild shrimp populations in the United States in the 1990s³. The whitespot virus is thought to have been introduced into the US by release into coastal waters of untreated wastes from plants processing imported Asian tiger shrimp, and by shipping of contaminated white shrimp larvae throughout the Americas. It has now been reported in several countries in Central and South America^{3,38}. There are now over twenty different shrimp viruses recorded, and many of these have spread through farms into many different shrimp species³⁸.

Oyster aquaculture

Oyster farming has a long history, and led to an early epidemic in wild oyster stocks in the 1950s. As a result of new DNA analyses, we now have evidence that MSX disease was introduced into California and subsequently the East coast of the USA from Asia, in introduced Pacific Oysters^{1,39}. It killed huge numbers of the US native Eastern oysters and devastated the US wild oyster harvesting industry⁴⁰. The transfer of oysters around the world by aquaculture entrepreneurs has also resulted in the introduction of drilling gastropods, that are predators of oysters and of other bivalves. The American oyster drill was introduced into the UK⁴¹ and more recently a Japanese oyster drill into France⁴². Both have also been introduced into the Pacific coast of the USA⁴³.

The pearl oyster industry began as a wild fishery, but has evolved into a combination of the collection of oysters and their cultivation to produce pearls. There are few publications on diseases of pearl oysters, but this may be a result of the fact that much of the research in this industry is confidential. In Australia they seem to have been protected by the distance between the wild stocks and the culture areas, although diseases have been identified in aquaculture, and a recent episode of mortalities (named '*Oyster Oedema Disease*' by WA Fish Health)⁴⁴ may be a viral disease, although it is not yet certain that it is caused by an infectious agent.

Abalone aquaculture

Abalone farming is relatively recent, except in Japan, where local animals have been bred in hatcheries to restock local populations since the mid- seventies. But the development of full-scale grow-out farming of abalone in California quickly led to two disastrous pathogen problems. The first, best-documented case is the South African sabellid worm. This is a very small worm that forms thousands of tiny, almost invisible burrows in the shell of abalone and other gastropod mollusks on the coast of South Africa⁴⁵. It remained unknown until a Californian abalone farm imported a number of species of abalone from other countries in the 1980s, apparently to determine which species grew best in their farm. They imported the South African abalone species *Haliotis midae*, and subsequent examination of the shells from these imported abalone showed that they harboured the parasite⁴⁶.

The parasite was first reported at the International Abalone symposium in Hobart in 1994⁴⁷. It was discovered in 1993, because it had crossed into the big red Californian abalone *Haliotis rufescens* in farms; and this species has no resistance. The worm broods eggs in its tubes, and then tiny larvae creep down the shell to burrow between the animal and the shell lip⁴⁵. The abalone is then manipulated to stop normal shell growth and to surround the larval worms with pearly nacre, thus forming a tube for them⁴⁸. The changed shell growth distorts the shell, and in heavily infested abalone growth is halted, and the animals are in poor condition⁴⁷.

By 1994, the disease had spread to all 18 abalone farms in California due to the exchange of seed juveniles between farms⁴⁹, and subsequently it was reported from farms in Mexico and Chile⁵⁰. Furthermore, it had become established in various wild gastropods on a rocky shore in front of at least one farm. This is probably a result of the fact that the parasite remains alive in dead shell pieces, and the water from the farm was pumped directly onto the rocky shore⁴⁹. Subsequently, US government agencies removed all gastropod mollusks from an extensive area of the rocky shore, and subsequent annual

surveys suggest that the disease has been eliminated from this area⁴⁹. There are unconfirmed reports that infected stock was sold to conservation groups to be used to seed into abalone habitat in California, to “enhance” the local stocks, but as the local stocks of abalone were subsequently devastated by another disease, the withering syndrome, there have been no reports of sabellid infested wild stocks.

The Withering Syndrome (WS) was discovered when it began causing mass mortalities in wild stocks of the intertidal black abalone (*H. cracherodii*) in California in 1985⁵¹. The foot withers and the abalone lose their hold on the rocks. Fishing and pollution had previously reduced mainland populations⁵², but large stocks remained on the Californian channel islands, and these populations virtually disappeared (95-100% mortality by 1992), due to WS⁵³, and the disease reportedly also devastated stocks in Mexico^{1,54}, then spread northwards along the California coast⁵⁴. The rate of spread was estimated as 20.7 km/yr⁵⁵. The disease was also found in the three major commercial species in California and Mexico: *H. rufescens*, (red abalone) *H. corrugata* (pink abalone) and *H. fulgens* (the blue abalone important in Mexico)^{56,57}, and the endangered deep water white abalone *H. sorenseni*, which is particularly susceptible⁵⁸. There are indications that warm El Niño conditions increases its effects^{59,60}. More recently it has been spread further into Northern California red abalone stocks due to seeding of infected farm abalone onto reefs⁶¹. Further, recent work shows that not only did local recruitment fail once adult stocks declined below 1 per square meter, but also the habitat changed due to the lack of adults, so that it became unsuitable for recruitment by juvenile abalone⁶². This suggests that recoveries from devastating declines due to diseases may take many decades, as the habitat must be gradually recolonised and made suitable by adult abalone.

WS is now known to be caused by a Rickettsia-like parasite (*Candidatus Xenohalotis californiensis*) in the abalone gut wall⁶³. A Rickettsia-like organism has also been identified in the South African *H. midae*, which is apparently unaffected⁶⁴, and a similar disease has been seen in Chinese abalone farms in *Haliotis discus hannai*, the northern Japanese abalone⁶⁵. While the parasite DNA has apparently not yet been compared to the Californian WS agent in either case, it seems plausible that this disease also was introduced to California from one of these sources by the aquaculture farm that imported foreign species and introduced the sabellid worm. In fact a former farm worker (personal communication) stated he saw the disease first in black abalone near the outfall of this farm. More recently, the WS disease has been transported in *Haliotis rufescens* to farms in Chile⁶⁶, where it has been transferred to the Japanese abalone *H. discus hannai*⁶⁷, and also into Iceland, where *H. rufescens* is cultured, and from there to Ireland, where both the European *H. tuberculata* and the Japanese *H. discus discus* are cultured^{68,69}. While there are no local wild abalone stocks in either of these areas, WS has now been transferred in cultured *H. tuberculata* from Ireland to Spain, and apparently also France, where *H. tuberculata* exists in wild stocks; and WS has now been detected in wild adults in Spain, although no pathology was observed⁷⁰. It is not clear what the effect of WS on the Spanish wild stocks will be.

H. tuberculata stocks on the coast of France and the English Channel Islands have already suffered devastating losses (50-90% mortalities) due to another disease in 1998, 2000 and 2005, caused by the bacterium *Vibrio carchariae* (= *V. harveyi*)^{71,72}. The disease destroyed almost all populations in areas with maximum temperatures above 16 degrees along the coast. The bacterium is responsible for a disease in Japanese abalone, but also infects a wide variety of hosts, such as prawns and fish⁷¹, and may well have

arrived in France as a result of aquaculture of one of these species. The capacity for diseases to transfer between animals in intensive aquaculture is illustrated by the fact that *Vibrio vulnificus*, common in Oysters, have infected abalone in barrel culture in aquaculture leases in Chile⁷³

While the WS disease in California and the *Vibrio* disease in France were not reported in the scientific literature as being the result of aquaculture, the sabellid disease was well described, and well-known in Australia in the late 1990s. Thus ***for almost every important marine species in aquaculture, including abalone, ample evidence of the dangers of infection of wild stocks by diseases from farms existed prior to the development of biosecurity and transfer policies for the Australian abalone aquaculture industry.***

3. How disease virulence may change in aquaculture and wild stocks.

While virulence change is seldom measured, there is evidence for the evolution of increased virulence in Mareks disease on chicken farms⁷⁴. This is of particular interest here, as Mareks disease is caused by a virus related to the alphaherpes viruses that seek out nerves, as the ganglioneuritis virus in abalone does. This shows that evolutionary changes in virulence in these viruses is possible over short periods, despite the perception that their genetic structure is very stable. The best experimental evidence for virulence change in pathogens are for myxomatosis in Australian rabbits^{75, 76}, and for the nematode parasites of fig wasps⁷⁷, because these systems are more amenable to study. These studies however, strongly support the theoretical work on the evolution of virulence in parasites that has been developed over the last 20 years⁷⁸, as does the other more limited experimental work on other pathogens. The theory shows that the virulence of a pathogen will evolve to an intermediate level, and this level depends on the density of the host and how the parasite is transmitted – in particular the link between virulence and the chance of transmission between hosts^{7, 75}.

Most parasites can only be transmitted while the host is alive, or within a short period after the death of the host. For example only live humans transmit flu. Evolution on parasites will act to maximise transmission from one host to the next, as this determines the probability that the parasite will persist and spread^{75, 76}. The theory is developed on the basis that the virulence of a parasite is related to its replication rate - if a parasite rapidly produces copies of itself in its host, this would kill the host rapidly, preventing further transmission. If the chance of transmission to the next host is high, then the parasite should make many copies to infect as many new hosts as possible as fast as possible. But if the chance of getting into another host is low, then the parasite should keep the host alive for much longer, to ensure it has many opportunities to enter another host. This would involve causing less damage to the host by producing copies of itself more slowly.

Thus theoretical papers have argued that in the process of an epidemic among hosts at high density, pathogens will evolve to be more virulent^{79, 80}. ***Thus in intensive animal husbandry (such as aquaculture), one expects disease organisms to evolve to be more virulent, if the disease is not rapidly eliminated.*** In contrast in natural populations, the pathogens should evolve to have a lower level of virulence, especially when the host stocks are at low density and there is no vector that carries the pathogen from one host to another, so that transmission between hosts is limited. The model papers however, have focused on the equilibrium levels of hosts and pathogens, and we are not aware of work

that deals with the rate at which evolution towards lower virulence would occur as a disease epidemic proceeds in a host population. Logic would suggest that evolution towards reduced virulence would be faster if the host stock density is very low, as this would make transmission of the pathogen between hosts less likely. ***Obviously a virulent pathogen will dramatically reduce the host density, and at that stage one would expect strong selection on the pathogen to become less virulent.***

4. Minimizing introductions of pathogens into wild stocks.

One might argue that since it is in the interest of farmers to exclude pathogens from farms, it is sufficient to ensure that all stock brought onto farms are sampled to screen for pathogens. The problem here is that screening is unlikely to reveal pathogens in wild animals brought into a farm. In wild populations, pathogens are likely to be at a low level, so that only exhaustive screening would detect them, and previously unknown pathogens are likely to be missed, as even the most experienced pathologists are likely to miss parasites that have not previously been described.

Interestingly, a survey of biosecurity use in the USA and Canada in recirculating finfish aquaculture (where they are most important to the long-term success of the operation) showed that biosecurity practice was very variable, depending on factors such as the experience of the farm manager and relative costs, and not the availability of knowledge of the best techniques⁸¹. It appears that managers will balance risks and costs of biosecurity according to their own perceptions of risk, whether these are correct or not.

While good biosecurity in aquaculture reduces the risks of epidemics among farm stocks, the literature shows the emergence of new, and therefore unmonitored diseases in some aquaculture facilities can be regarded as inevitable. Thus systems to protect wild stocks from diseases in farms are essential. For in-sea aquaculture the risks of release are especially problematic. ***Thus we would argue that the only good defense for wild stocks is to ensure that farm animals are not within contact range of wild stocks.*** The definition of contact range for pathogens is problematic, but the distances that diseases such as Withering syndrome in the USA have spread between island populations show that ***the range should be measured in kilometers***, and clearly currents and inadvertent human transport must also be considered.

References.

1. Marine Aquaculture Taskforce [USA], (2007). Sustainable marine aquaculture: fulfilling the promise, managing the risks. Report to the US congress. [www.who.edu/sites/marineaquataskforce]
2. Muir JF & Young JA. (1998). Aquaculture and marine fisheries: will capture fisheries remain competitive? *Journal of Northwest Atlantic Fisheries Science* **23**:157-174.
3. Naylor RL, Goldburg RJ, Primavera JH, Kautsky N, Beveridge MCM, Clay J, Folke C, Lubchenco J, Mooney H & Troell M. (2000). Effect of Aquaculture on world fish supplies. *Nature* **205**: 1017-1024.
4. Harvell D, Aronson R, Baron N, Connell J, Dobson A, Ellner S, Gerber L, Kim K, Kuris A, McCallum H, Lafferty K, McKay B, Porter J, Pascual M, Smith G, Sutherland K & Ward J. (2004). The rising tide of ocean diseases: unsolved problems and research priorities. *Frontiers in Ecology and Environment*. **2**:375-382.
5. McCallum HI, Kuris A, Harvell CD, Lafferty KD, Smith GW & Porter J. (2004). Does terrestrial epidemiology apply to marine systems? *Trends in Ecology and Evolution*. **19**: 585-591.
6. McCallum H, Harvell CD & Dobson A. (2003). Rates of spread of marine pathogens. *Ecological Letters* **12**: 1062-1067.
7. Frank SA. (1996). Models of parasite virulence. *Quarterly Review of Biology* **71**: 37-78.
8. Power A & Mitchell C. (2004). Pathogen spillover in disease epidemics. *American Naturalist*. **164**:S79-S89.
9. Dobson AP & May RM. (1986). Disease and conservation. In: Conservation Biology (ed. Soule ME). Sinauer, pp. 345-365.
10. Michel AL. (2002). Implications of tuberculosis in African wildlife and livestock. *Annals of the New York Academy of Sciences* **969**: 251-255.
11. Ministerial Council on Forestry, Fisheries and Aquaculture (1999). National Policy for the Translocation of Live Aquatic Organisms – issues, principles and guidelines. Canberra.
12. Gavine F & McKinnon L. (2002). Environmental monitoring of marine aquaculture in Victorian Coastal Waters: a review of appropriate methods. Final report. Report no. 46, Marine and Freshwater Resources Institute, Department of natural Resources and Environment, Victoria. (See Appendix, hazard assessment).
13. Blazer VS & La Patra SE. (2002). Pathogens of cultured fishes: potential risks to wild fish populations. In: Aquaculture and the environment in the United States. (ed. Tomasso J.) U.S. Aquaculture Society, a chapter of the World Aquaculture Society. Baton Rouge, LA, USA. pp 197-224.
14. Ritchie RJ, Cook M, Melville K, Simard N, Cusack R & Griffiths S. (2001). Identification of infectious salmon anaemia virus in Atlantic salmon from Nova Scotia: evidence for functional strain differences. *Diseases of Aquatic Organisms* **44**: 171-178.
15. Hastein T & Linstad T. (1991). Diseases in wild and cultured salmon: possible interaction. *Aquaculture* **98**: 277-288
16. Morton A, Routledge RD & Williams R. (2005) Mortality rates for juvenile pink, *Oncorhynchus gorbuscha*, and chum, *O. keta*, salmon infested with sea lice, *Lepeophtheirus salmonis*, in the Broughton Archipelago. *Alaska Fishery Research Bulletin*. **11**: 146-152.

17. Krkosek M, Lewis MA, Morton A, Frazer LN & Volpe JP. (2006). Epizootics of wild fish induced by farm fish. *Proceedings of the National Academy of Sciences* **103**: 15506-15510.
18. Beamish RJ, Jones S, Neville C-E, Sweeting R, Karreman G, Saksida S, & Gordon E. (2006). Exceptional marine survival of pink salmon that entered the marine environment in 2003 suggests that farmed Atlantic salmon and Pacific salmon can coexist successfully in a marine ecosystem on the Pacific coast of Canada. *ICES Journal of Marine Science*. **63**: 1326-1337.
19. Hilborn R. (2006). Salmon-farming impacts on wild salmon. *Proceedings of the National Academy of Sciences* **103**: 15277.
20. Goldburg R & Naylor R. (2005). Future seascapes, fishing, and fish farming. *Frontiers in Ecology and the Environment*. **3**:21-28.
21. Buschmann AH, Riquelme VA, Hernández-González MC, Varela D, Jiménez JE, Henríquez LA, Vergara PA, Guíñez R & Filún L. (2006) A review of the impacts of salmonid farming on marine coastal ecosystems in the southeast Pacific. *ICES Journal of Marine Science*. **63**: 1338-1345.
22. Fletcher WJ, Jones B, Pearce AF & Hosja W. (1997). Environmental and biological aspects of the mass mortality of pilchards (autumn 1995) in Western Australia. *WA Fisheries Research Report*. No. 106.
23. Gaughan DJ, Mitchell RW & Blight SJ. (2000). Impact of mortality, possibly due to herpes virus, on pilchard *Sardinops sagax* stocks along the south coast of Western Australia in 1998-99. *Marine and Freshwater Research* **51**: 601-612.
24. Taylor, IR & Roe EL. (2004). Feeding ecology of little terns *Sterna albifrons sinensis* in south-eastern Australia and the effects of pilchard mass mortality on breeding success and population size.. *Marine and Freshwater Research* **55**: 799-808.
25. Dann P, Norman FI, Cullen JM, Neira FJ & Chiaradia A. (2000). Mortality and breeding failure in little penguins, *Eudyptula minor*, in Victoria, 1995-96, following a widespread mortality of pilchard, *Sardinops sagax*. *Marine and Freshwater Research* **51**: 355-362.
26. Ward TM, McLeay LJ, Dimmlich WF, Rogers PJ, McClatchie SAM, Matthews R, Kampf J, Van Ruth PD (2006) Pelagic ecology of a northern boundary current system: effects of upwelling on the production and distribution of sardine (*Sardinops sagax*), anchovy (*Engraulis australis*) and southern bluefin tuna (*Thunnus maccoyii*) in the Great Australian Bight *Fisheries Oceanography* **15**: 191-207.
27. Gaughan DJ. (2002). Disease-translocation across geographic boundaries must be recognized as a risk even in the absence of disease identification: the case with Australian *Sardinops*. *Reviews in Fish Biology & Fisheries* **11**: 113-123.
28. Ward TM, Hoedt F, McLeay L, Dimmlich WF, Kinloch M, Jackson G, McGarvey R, Rogers PJ & Jones K. (2001). Effects of the 1995 and 1998 mass mortality events on the spawning biomass of sardine, *Sardinops sagax*, in South Australian waters. *ICES Journal of Marine Science*. **58**: 865-875.
29. Whittington RJ, Jones JB, Hine PM & Hyatt AD. (1997). Epizootic mortality in the pilchard *Sardinops sagax neopilchardicus* in Australia and New Zealand in 1995. I. Pathology and epizootiology. *Diseases of Aquatic Organisms* **28**: 231-239.
30. Murray AG, O'Callaghan M, Jones B. (2003). A model of spatially evolving herpesvirus epidemics causing mass mortality in Australian pilchard *Sardinops sagax* *Diseases of Aquatic Organisms* **54**, (1) 1-14.
31. Nickum D. (1999). Whirling disease in the United States: a summary of progress in research and management. Trout Unlimited, Arlington, VA.

32. Hedrick RP, Gilad O, Yun S & Spangenberg JV. (2000). A herpesvirus associated with mass mortality of juvenile and adult koi, a strain of common carp. *Journal of Aquatic Animal Health* **12**: 44-57.
33. Haenen OIM, Way K, Bergmann SM & Ariel E. (2004). The emergence of koi herpesvirus and its significance to European aquaculture. *Bulletin of the European Association of Fish Pathologists* **24**: 293-307.
34. Pokorova D, Vesely T, Piackova V, Reschova S & Hulova J. (2005) Current knowledge on koi herpesvirus (KHV): a review. *Vetinary Medicine* **50**: 139-147.
35. Haramoto E, Kitajima M, Katayama H & Ohgaki S. (2007). Detection of koi herpesvirus in river water in Japan. *Journal of Fish Diseases* **30**: 59-61.
36. Dishon A, Perelberg A, Bishara-Shieban J, Ilouze M, Davidovitch M, Werker S & Kotler M. (2005). Detection of carp interstitial nephritis and gill necrosis virus in fish droppings. *Applied and Environmental Microbiology* **71**: 7285-7291.
37. Perelberg A, Smimov M, Hutoran M, Diamant A, Bejerano Y & Kotler M. (2003). Epidemiological description of a new viral disease afflicting cultured *Cyprinus carpio* in Israel. *Israeli Journal of Aquaculture (Bamidgeh)* **55**: 5-12.
38. McClennan C. (2004). White spot syndrome virus: the economic, environmental and technical implications on the development of Latin American shrimp farming. MA thesis, The Fletcher School, Tufts University.
39. Bureson EM, Stokes NA & Friedman CS. (2000). Increased virulence in an introduced pathogen: *Haplosporidium nelsoni* (MSX) in the eastern oyster *Crassostrea virginica*. *Journal of Aquatic Animal Health* **12**: 1-8.
40. Farley CA. (1992). Mass mortalities and infectious lethal diseases in bivalve molluscs and associations with geographic transfers of populations. *In*: Dispersal of living organisms into aquatic ecosystems (ed. Rosenfield A & Mann R.). Maryland Sea Grant, College Park, MD. pp 139-154.
41. Cole HA. (1942). The American whelk tingle, *Urosalpinx cinerea* (Say) on British oyster beds. *Journal of the Marine Biological Association U.K.* **25**: 477-508.
42. Garcia-Meunier P, Martel C, Pigeot J, Chevalier G, Blanchard G, Gouilletquer P, Robert S & Sauriau P-G. (2002). Recent invasion of the Japanese oyster drill along the French Atlantic coast: identification of specific molecular markers that differentiate Japanese, *Ocenebrellus inornatus*, and European, *Ocenebra erinacea*, oyster drills. *Aquatic Living Resources* **15**: 67-71 (doi: 10.1016/S0990-7440(01)01146-9)
43. Exotics Guide Website: http://www.exoticsguide.org/species_pages.html
44. Jones, B. (2007). Fish Health Issues. Report to Pearling Industry Advisory Committee.
45. Fitzhugh K & Rouse GW. (1999). A remarkable new genus and species of fan worm (Polychaeta: Sabellidae, Sabellinae) associated with marine gastropods. *Invertebrate Biology* **118**: 357-390.
46. Kuris AM & Culver CS. (1999). An introduced sabellid polychaete pest infesting cultured abalones and its potential spread to other Californian gastropods. *Invertebrate Biology* **118**: 391-403.
47. Oakes FR & Fields RC. (1996). Infestation of *Haliotis rufescens* shells by a sabellid polychaete. *Aquaculture* **140**: 139-143.
48. Day RW & Kuris AM. unpublished data.
49. Moore JD, Juhasz C, Robbins TT & Grosholz E. (2006). Sabellid polychaete infestations of farmed California abalone. Abstracts 6th International Abalone Symposium, Puerto Varras, Chile. p 41.

50. Cañete JI, Cárdenas C & Mansilla A. (2006). Monitoring of diseases in the Chilean abalone aquaculture: identification of borer polychaetes. Abstracts 6th International Abalone Symposium, Puerto Varras, Chile. p 146.
51. Haaker PL, Parker DO, Togstad H, Richards D, Davis GE & Friedman CS. (1992). Mass mortality and withering foot syndrome in black abalone, *Haliotis cracherodii*, in California. In: Abalone of the world: biology, fisheries and culture. (ed. Shepherd SA, Tegner MJ & Guzman del Proo SA.) Fishing News books, Cambridge. pp 214-224.
52. Miller AC & Lawrenz-Miller SE. (1993). Long term trends in black abalone, *Haliotis cracherodii* Leach 1814, populations along the Palos Verdes Peninsula, California. *Journal of Shellfish Research* **12**: 195-200.
53. VanBlaricom GR, Ruediger JL, Friedman CS, Woodward DD & Hedrick RP. (1993). Discovery of withering syndrome among black abalone *Haliotis crcherodii* Leach 1814, populations at San Nicholas Island, California. *Journal of Shellfish Research* **12**: 185-188.
54. Altstatt, JM, Ambrose RF, Engle JM, Haaker PL, Lafferty KD & Raimondi PT. (1996). Recent declines of black abalone *Haliotis cracherodii* on the mainland coast of central California. *Marine Ecology Progress Series* **142**: 185-192.
55. Lafferty KD & Kuris AM.(1993). Mass mortality of abalone *Haliotis cracherodii* on the Californian Channel Islands: tests of epidemiological hypotheses. *Marine Ecology Progress Series* **96**: 239-248.
56. Cáceres Martínez J, Álvarez Tinajero C, Guerrero Rentería Y & González Avilés JG. (2000). *Rickettsiales*-like prokaryotes in cultured and natural populations of the red abalone, *Haliotis rufescens*, blue abalone, *Haliotis fulgens*, and the yellow abalone, *Haliotis corrugata*, from Baja California, Mexico. (abstract only) *Journal of Shellfish Research* **19**: 503.
57. Álvarez Tinajero MC, Cáceres Martínez J & González Avilés JG. (2002). Histopathological evaluation of the yellow abalone *Haliotis corrugata* and the blue abalone *Haliotis fulgens* from Baja California, Mexico. *Journal of Shellfish Research* **21**: 825-830.
58. Friedman CS, Scott BB, Streng RE, Wight NA, McCormick TB & Trevelyan G. (2006). Optimization of oxytetracycline treatment in two abalone species, *Haliotis sorenseni* and *H. rufescens*. Abstracts, 6th International Abalone Symposium, Puerto Varras, Chile. p 31.
59. Moore JD, Robbins TT & Friedman CS. (2000). Withering syndrome in farmed red abalone *Haliotis rufescens*: thermal induction and association with a gastrointestinal *Rickettsiales*- like prokaryote. *Journal of Aquatic Animal Health* **12**: 26-34.
60. Raimondi PT, Wilson CM, Ambrose RF, Engle JM & Minchinton TE. (2002). Continued declines of black abalone along the coast of California: are mass mortalities related to El Niño events? *Marine Ecology Progress Series* **242**: 143-152.
61. Friedman CS & Finley CA. (2003). Anthropogenic introduction of the etiological agent of withering syndrome into northern California abalone populations via conservation efforts. *Canadian Journal of Fisheries and aquatic Sciences* **60**: 1424-1431.
62. Miner CM, Altstatt JM, Raimondi PT & Minchininton TE. (2006). Recruitment failure and shifts in community structure following mass mortality limit recovery prospects of black abalone. *Marine Ecology Progress Series* **327**: 107-117.

63. Friedman CS, Andree KB, Beauchamp Ka, Moore JD, Robbins TT, Shields JD & Hendrick RP. (2000). '*Candidatus Xenohaliothis californiensis*' a newly described pathogen of abalone, *Haliotis* spp., along the west coast of North America. *International Journal of Systematic Evolution and Microbiology* **50**: 487-855.
64. Mouton A. (2000). Health management and disease surveillance in abalone, *Haliotis midae*, in South Africa. (abstract only). *Journal of Shellfish Research* **19**: 526.
65. Gao X, Ford SE & Zhang F. (1999). Molluscan aquaculture in China. *Journal of Shellfish Research* **18**: 19-31.
66. Godoy M. & Aedo I. (2006). Withering syndrome of the red abalone (*Haliotis rufescens*) in Chile. Abstracts, 6th International Abalone Symposium, Puerto Varras, Chile. p 52.
67. Cortés C, Lohrmann KB, Needham P, Defranchi Y, Winkler F, Romero A & Enriquez R. (2006). Transmission of *Candidatus Xenohaliothis californiensis* from *Haliotis rufescens* to *H. discus hannai* in Chile. Abstracts, 6th International Abalone Symposium, Puerto Varras, Chile. p 30.
68. Anonymous. (2004). Infection with *Candidatus Xenohaliothis californiensis* in Iceland. *Disease Information* **17**: 160.
69. Bower SM. (2006). Synopsis of infectious diseases and parasites of commercially exploited shellfish: withering syndrome of abalone. **URL:** http://pacrhqis7/shellldis/pages/fswab_e.htm.
70. Balseiro P, Aranguren R, Gestal C, Novoa B & Figueras A. (in press) *Candidatus Xenohaliothis californiensis* and *Haplosporidium montforti* associated with mortalities of abalone *Haliotis tuberculata* cultured in Europe. *Aquaculture*, doi: 10.1016/j.aquaculture.2006.03.046.
71. Nicolas JL, Basuyaux O, Mazurié J & Thébault A. (2002). *Vibrio charchariae*, a pathogen of the abalone *Haliotis tuberculata*. *Diseases of Aquatic Organisms* **50**: 35-43.
72. Travers M-A, Koken M, Huchette S & Paillard C. (2006). *Vibrio harveyi* detection and interaction with *Haliotis tuberculata* haemocytes. Abstracts, 6th International Abalone Symposium, Puerto Varras, Chile. p 151.
73. Aedo I, Godoy M, Fernández M, Flores R & Ramirez R. (2006). Vibriosis by *Vibrio vulnificus* in red abalone seeds (*Haliotis rufescens*). Abstracts, 6th International Abalone Symposium, Puerto Varras, Chile. p 39.
74. Witter RL. (1997) Increased virulence of Marek's disease virus field isolates. *Avian Diseases* **41**: 149-163.
75. Anderson RM & May RM. (1982). Coevolution of hosts and parasites. *Parasitology* **85**: 411-426.
76. May RM & Anderson RM. (1983). Epidemiology and genetics in the coevolution of parasites and hosts. *Proceedings of the Royal Society of London, Series B, Biological Sciences*. **219**: 281-313.
77. Herre EA. (1993). Population structure and the evolution of virulence in nematode parasites of fig wasps. *Science* **259**: 1442-1445.
78. Dwyer G, Levin SA & Buttel L. (1990). A simulation model of the population dynamics and evolution of myxomatosis. *Ecological Monographs* **60**: 423-447.
79. Ewald PW. (1983). Host-parasite relations, vectors, and the evolution of disease severity. *Annual Review of Ecology and Systematics* **14**: 465-485.
80. Day T. (2003). Virulence evolution and the timing of disease life-history events. *Trends in Ecology and Evolution* **18**: 113-118.

81. Delabbio J, Murphy BR, Johnson GR & McMullin SL. (2004). An assessment of biosecurity utilization in the recirculation sector of finfish aquaculture in the United States and Canada. *Aquaculture* **242**: 165-179.