



Point of view

Disease-translocation across geographic boundaries must be recognized as a risk even in the absence of disease identification: the case with Australian *Sardinops*

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Abstract

In 1995 and 1998/99 single species mass mortalities of sardine/pilchard *Sardinops sagax* (Clupeidae) spread rapidly throughout this species' range in Australia from the central coast of South Australia, dramatically decreasing the population size and representing the two most extensive mass mortalities recorded for marine organisms. The behavior of each epizootic indicated that an exotic pathogen was responsible, with the fatal agent shown to be a previously unknown herpesvirus. The focal origin of both events from a limited region within the extensive Australian range of *S. sagax* was not random. Tuna (*Thunnus maccoyii*) feedlots located in the same region as the epizootics' origins are responsible for delivering to the marine environment the largest quantities of *S. sagax* imported into Australia, which provides qualitative evidence of a link between the two events and imported *S. sagax*. This link provides an example of the need to undertake a review of the current international standards for import risk analysis (IRA) that requires a disease to be identified before it can be considered a risk. Regardless of the identity of the disease agents responsible, case histories of mass mortalities need to be given due consideration in both deciding whether to instigate an IRA and also form part of the IRA. Identification of a hazard should thus not be restricted to dealing only with identifiable diseases, but should also embrace case histories of epizootic events linked to (a) previously unidentified pathogens and (b) translocation of particular species between geographically separate populations.

The problem

Transport of untreated, frozen fish products, as with virtually all animals and plants, across geographic boundaries carries a risk of spreading disease (Office International des Epizooties (OIE), 2001a). Increases in the incidence of epidemics and in the number of “new” diseases in marine organisms is a global trend (Harvell et al., 1999) and one of three major causes for the trend in emerging infectious diseases can be ascribed to host or parasite translocations (Daszak et al., 2000). For several decades man has been recognized as the primary means of translocating fish parasites, both within and between continents (Hoffman, 1970; Arthur, 1995; Hill, 1996). The need for recognition at an international level of the risk of translocation of parasites, but without the concurrent need for detailed knowledge of any particular parasites forms the basis of this point of view.

Although Harvell et al. (1999) and Daszak et al. (2000) have demonstrated that there is a problem with man moving parasites, including infectious diseases, around the world, there are still mechanisms in place that do not allow sufficient consideration of their conclusions. For example, guidelines developed to prevent the release of animals carrying exotic diseases are currently underused (Daszak et al., 2000). Legislation that attempts to deal with the problem of limiting disease transfer during international trade in animal and plant commodities is ineffective, or at least lends itself to an interpretation(s) that allows ineffective management of the risk of disease transfer. In recognition of the continuing demands for increased global trade along with the associated political lobbying to decrease barriers to trade, it is becoming even more imperative that the risk of translocation of disease be managed and the most likely acceptable framework to do this is through import risk analysis (IRA). The risk of translocation of parasites, and the associated problem of poor disease information for the majority of aquatic species, is not a new concern for the trade in biological commodities. Arthur (1995) noted that legislation in some areas has previously taken the positive step of “listing” certain pathogens but, was inadequate in dealing with unlisted pathogens. Thus, although Arthur (1995) highlighted the problem of attempting to exclude disease pathogens by referring only to known pathogens, this still remains a central problem to controlling the international spread of disease (Jones, 2000).

The OIE provides the international standard for IRA, particularly for World Trade Organization (WTO) members. Hazard identification, which OIE views as a crucial preliminary step that must be undertaken in order for an IRA to proceed (OIE, 2001a, Article 1.4.2.1, and see below), is currently restricted to dealing only with identifiable diseases. Thus, a pathogenic agent must be identified in a commodity from the exporting country but not be present in the importing country in order for an IRA to proceed. However, the ability to ascertain the disease status of an aquatic commodity (i.e., to identify hazards) can be extremely difficult and this represents a flaw in OIE approach to IRA (Jones, 2000). For example, a disease can essentially remain hidden if it exists in a latent state at the time researchers examine the disease status of a particular population. Jones (2000) provides the example of pearl oyster (*Pinctada maxima*) in northern Australia, whose population is known to harbor a haplosporidian that was not, however, detected in an exhaustive disease survey of 4,500 individual oysters. Importantly, Jones (2000) points out that part of the problem of poor information on disease status of a commodity is caused by the low prevalence and patchy occurrence of a disease when at equilibrium with its host population, which makes it difficult to obtain data on disease even from directed surveys of diseases. For example, although whirling disease (*Myxosoma cerebralis*) spread throughout much of the trout-farming industry in Europe following World War II and most likely came from wild fish, it was not commonly detected in wild salmonids (Hoffman, 1970). Contrasting levels of knowledge can affect outcomes when dealing with disease and translocation of aquatic organisms (Arthur, 1995). Despite the fact that highly controlled quarantine programs were implemented prior to the entry of Atlantic salmon (*Salmo salar*) and bay scallop (*Argopecten irradians*) into Canada, the latter still resulted in the introduction of two protozoan pathogens (Arthur, 1995). Whereas the disease history of Atlantic salmon was well documented, that of bay scallop was less so. Arthur (1995, p. 13) attributed this relatively poor understanding of disease-knowledge as a major factor in the bay scallop pathogens being “missed” despite rigorous disease-inspections over four generations during the quarantine period. Arthur (1995) concluded that, “Even the most competent diagnostician may overlook infections by previously unreported or undescribed pathogens.” Although this example referred to diseases associated with living organisms, it is also applicable to patho-

gens which survive freezing or remain viable outside of a living host.

Unintentional translocation of known diseases also occurs despite knowledge of both the impacts and epizootiology of some pathogens and their mode of transport between regions. The spread of mollusc diseases between countries in the Northern Hemisphere can clearly be attributed the trade in molluscs for aquaculture purposes (Renault, 1996). For whitespot syndrome virus, a devastating disease of some penaeids, translocation has occurred for two decades. Whitespot syndrome became established in south-east Asia in 1992 (e.g., Kasornchandra et al., 1998; Chou et al., 1998), most likely due to the introduction of east Pacific and South American penaeids (Choo, 2001). The spread of this and other viral diseases through south-east Asia was assisted by the common practice of including minced prawn flesh in the feed for farmed prawns. Prior to this, the disease had spread with the transfer of live penaeids between central America, Mexico, Texas, Florida, Hawaii, Tahiti, Philippines, Guam and Israel (Langdon, 1989; Lightner et al., 1992). Thus, the spread through south-east Asia occurred despite previous documentation of the spread of several prawn viruses in North America and the eastern Pacific that resulted from transfers and introductions of infected penaeids. White spot syndrome now affects the penaeid culture industry in Japan, China, Taiwan, Thailand, Korea, Indonesia, India and Bangladesh (Hossain et al., 2001). Likewise, the spread of Infectious Salmon Anemia through Scotland between May 1998 and November 1999 was facilitated by industry work practices (Stagg et al., 2001); that is, the disease was unintentionally spread by those people making a living, either directly or indirectly, out of salmon farming. The most important method of spreading the disease within Scotland was by movement of infected fish, long recognized as the usual method of disease transfer (Hoffman, 1970). As outlined by Morse (1993), and borrowing his terminology, disease traffic is of primary concern in managing the spread and “emergence” of diseases in new areas. Thus, although the spread in wild Norwegian salmon of both *Gyrodactylus salaris* and furunculosis (Johnsen and Jensen, 1991, 1994) by industry practices is well documented, disease continues to be spread between regions. Even in well studied and economically important aquatic organisms such as penaeids and salmon, disease traffic remains a serious problem. Given this scenario with well studied taxa, it is clear that there can still be considerable risk

of disease translocation even if potentially offensive pathogens have not been identified in an imported commodity and even more so if a disease survey of the population from which the commodity was extracted has not been undertaken.

Import risk analysis

Current international guidelines for IRA as detailed in the *International Aquatic Animal Health Code – 2001* (OIE, 2001a, Section 1.4, Article 1.4.1.1) consist of a three-step model as follows:

Hazard identification → *Risk assessment* ↔ *Risk management*,

with *Risk communication* linked to each of these steps to maintain a fully transparent process. Despite the concerns expressed above (e.g., Arthur, 1995; Jones, 2000) the guidelines, however, still require a pathogenic agent to be identified before the *Risk assessment* can proceed (OIE, 2001a, Article 1.4.2.1), as indicated above. This conflicts with the four subsequent steps that make up the procedure for a *Risk assessment* (OIE, 2001a, Article 1.4.2.4) in that only the latter allow qualitative information to be used. The four *Risk assessment* steps (OIE, 2001a, Articles 1.4.2.4.1–1.4.2.4.4) are: *Release assessment*, *Exposure assessment*, *Consequence assessment*, and *Risk estimation*. *Release assessment*, the first of the risk assessment steps, allows for evaluation of factors, such as the species involved, that may lead to the introduction of a disease. Note that the OIE’s *International Animal Health Code – 2001* (OIE, 2001b, Chapter 1.3.2, Article 1.3.2.4) explicitly explains that for the *Release assessment* step a quantitative or qualitative evaluation is acceptable. By contrast, in the *International Aquatic Animal Health Code – 2001* the ability to use qualitative information is outlined as a “general principle” of *Risk assessment* (OIE, 2001a, Article 1.4.2.3), but is not explicitly mentioned under each of the four steps of risk assessment. However, given that the same principles and methods apply whether the commodities in question are derived from aquatic or terrestrial animals (OIE *International Aquatic Animal Health Code*, Article 1.4.1.1), quantitative or qualitative evaluation should therefore explicitly be considered within the *Risk assessment* steps for aquatic commodities.

The second *Risk assessment* step, *Exposure assessment*, allows consideration of, for example, human and animal demographics, the quantities of a proposed

commodity to be imported and the intended use of a commodity. The *Consequence assessment* step addresses potential biological, environmental and economic impacts of disease incursion. Production losses are an example of direct consequences of disease incursion, while adverse impacts to the environment are an example of indirect consequences. The final step in the OIE risk assessment process is *Risk estimation*. This step integrates the results from the *Release*, *Exposure* and *Consequence assessments* to produce an overall assessment of the risks associated with the identified hazard. The IRA then proceeds to the next steps of reporting and reviewing the risk assessment.

Because the *Release assessment* and *Exposure assessment* steps are closely linked, sharing the same sub-headings of inputs that may need to be considered (i.e., *Biological factors*, *Country factors* and *Commodity factors*) this immediately allows some basic principles to be examined. In the case of species-specific pathogens, the level of risk is higher when the product being transported has populations on both sides of a geographic boundary. The risk becomes even higher if isolation has persisted long enough for the separate populations to have developed different levels of immunity to various pathogens; the risk of spreading disease may not necessarily increase in this case, but the risk of serious damage to a naïve population will increase. This was the case with the devastating *G. salaris* epidemic that spread through much of the wild salmon of Norway following an introduction that resulted from transfer across the Scandinavian Mountains during the practice of transporting juvenile salmon for aquaculture purposes (Johnsen and Jensen, 1991). The Scandinavian Mountains form a geographic barrier between the salmon populations in Norway and Sweden, on either side of which different levels of immunity to *G. salaris* have developed (Malmberg and Malmberg, 1993). In Sweden, *G. salaris* was in balance with the salmon population but in Norway the salmon had little resistance and thus underwent a significant population decline.

While WTO members are encouraged to abide by the international standards (OIE, 2001a) when considering sanitary and phytosanitary measures within a framework of international trade, the OIE also recommends that WTO members be allowed to adopt a higher level of protection if supported by scientific evidence or if the level of protection being applied is considered inappropriate (OIE, 2001a, Article

1.4.1.2). Aquatic-resource managers involved in international disease-risk management must therefore actively pursue consideration of case histories, recognize that disease translocations are already happening, and realize that such translocations will continue to occur even when processes designed specifically to minimize this risk are in place.

Because consequences of disease translocation cannot be predicted if the identity of potential pathogens is not known, there appears to be ample justification to adopt a high level of protection in those cases where disease status of a commodity is poorly known; the precautionary approach in such instances would be to assume that devastating epidemics will result. However, as this position will be difficult, if not impossible, to defend if applied in general, I propose that smaller steps first be taken to alleviate the more obvious threats posed by unidentified diseases and translocation across geographic boundaries. In this article I also contend that identification of a hazard should not be restricted to dealing only with identifiable diseases, but should also embrace case histories of epizootic events linked to (a) previously unidentified pathogens and (b) translocation of particular species between geographically separate populations (Arthur, 1995; Jones, 2000).

I provide an overview of the Australian *Sardinops* mass mortalities of 1995 and 1998/99 as an example of a recent and dramatic marine epizootic and apply the above arguments for utilizing qualitative information for risk assessments.

Mass mortality of Australian *Sardinops*

The two epizootics

Between October 1998 and May 1999 a mass mortality of *Sardinops sagax* (Clupeidae) spread through most of the species' distribution (Figure 1) around the temperate and subtropical coastline of Australia from a starting location along the central coast of South Australia (Gaughan et al., 2000; Jones, 2000). Pathological assessments indicated that a herpesvirus capable of severely damaging gills was associated with moribund and dead *Sardinops* collected during the mortality event. The mortality rate off the coasts of Western Australia and South Australia, the regions with Australia's largest *Sardinops* fisheries, were independently estimated to be around 60–70% of the spawning biomass (Gaughan

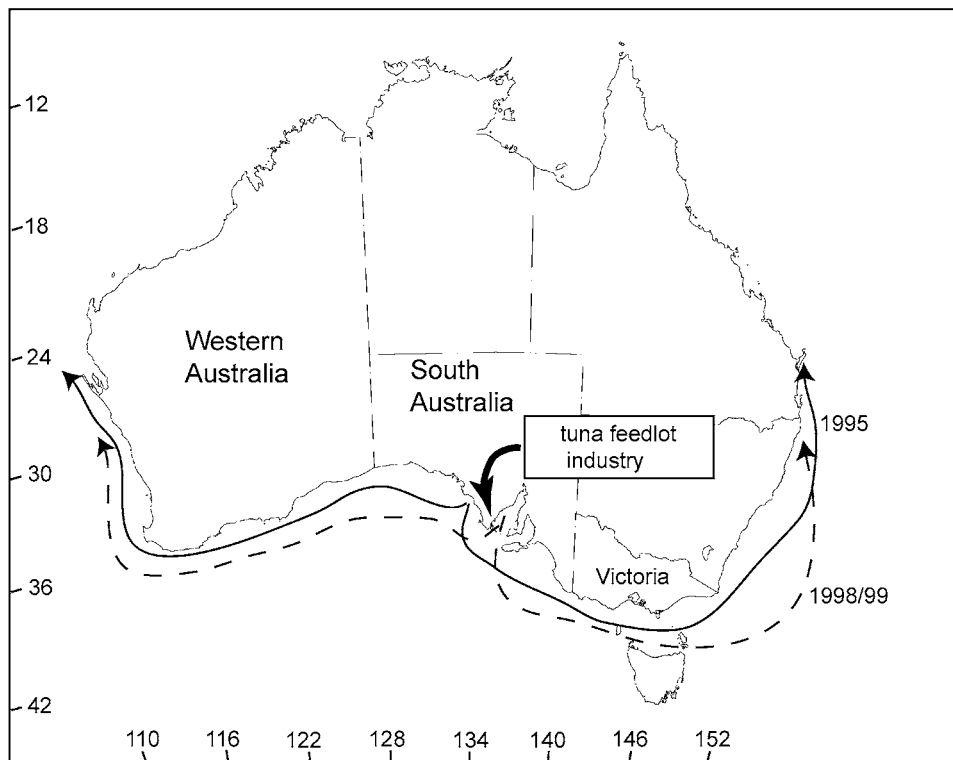


Figure 1. Extent of the 1995 and 1998/99 mass mortalities of *Sardinops sagax* around Southern Australia from starting points along central South Australia. Latitudes and longitudes are degrees south and east respectively.

et al., 2000; Ward et al., 2001), with the variance recognized in both cases as being large. In March to June 1995, a mass mortality event also spread throughout the range of *S. sagax* in Australia (Figure 1) from a starting point along the central coast of SA (Fletcher et al., 1997; Griffin et al., 1997; Jones et al., 1997) and likewise was associated with a herpesvirus (Hyatt et al., 1997; Whittington et al., 1997). These studies postulated that the behavior of the epizootic (i.e., no previous similar events, focal origin, dramatic spread through entire range) was indicative of an exotic pathogen to which Australian *Sardinops* had not previously been exposed.

The herpesvirus(es) associated with the epizootics has been identified, and named pilchard herpesvirus (PHV, Hyatt et al., 1997) after the local common name for *Sardinops*. Following the studies arising from the 1995 and 1998/99 mortalities, doubts were raised as to whether PHV had been shown conclusively to be the fatal agent. This is not particularly surprising: fulfilling cause-and-effect requirements developed for veterinary and human medicine (Koch's postulates) can be impossible for aquatic species (Bakke and

Harris, 1998). However, an important aspect of the epizootic was that, regardless of the identity of the cause, it still behaved as if an exotic pathogen was responsible. Although it was clear that scientists thought there was a link between the 1995 epizootics and imported *Sardinops* (Harvell et al., 1999), the lack of a formally identified causative agent stalled, and eventually stopped, any effectual risk-management action. Imported *Sardinops* were again implicated in the 1998/99 epizootic (Harvell et al., 1999; Gaughan et al., 2000; Ward et al., 2001). PHV was again present in dying *Sardinops* in 1998/99: there is no doubt that this virus was the pathogen responsible for both epizootics, causing hyperplasia of gill epithelium which lead to death by asphyxia (Jones et al., 1997).

The apparent introduction to Australia of the pathogen responsible for the 1995 epizootic was hypothesized to have occurred via either ballast water, sea birds or imported baitfish, with only a remote possibility of the epizootic being due to activation of a latent herpesvirus (Whittington et al., 1997). These same hypotheses were resurrected for the 1998/99 event. Mutation of the previously observed PHV was also

postulated as the cause of the second event. The probability of such a mass mortality event starting randomly within any particular 250 km unit of coastline within this species' 7,700 km range in Australia is 0.032 (i.e., $250/7,700$). Although not inherently significant, this probability is low and indicates that such events should be rare for any one region. The probability of a second mass mortality starting randomly within the same 250 km stretch of coast is about 0.001 (i.e., 0.032^2). This very low probability indicates that the focal origin, in central South Australia, of the two mass mortality events was not random, as has already been suggested by Jones (2000). The non-random origin suggests against the ballast water, sea bird and mutation hypotheses. Although Hyatt et al. (1997) invoked the need for a vector to explain the pattern of spread of the *Sardinops* mortality in 1995, subsequent modeling of the wave-like progression of the disease from a focal origin indicates that no vectors were involved in its spread around southern Australia (Murray et al., 2000). This finding also provides evidence against sea birds having brought the disease into Australia.

Although the origin of the implicated PHV has not been formally determined, it appears more than coincidental that both outbreaks began in the same region (Figure 1) where mature *Sardinops* are abundant and where thousands of tonnes of imported, untreated (frozen whole) *Sardinops* are placed annually into the marine environment to feed caged southern bluefin tuna (*Thunnus maccoyii*), a practice which began in the early 1990s (Whittington et al., 1997; Jones, 2000; Gaughan et al., 2000; Ward et al., 2001). In 1995, the quantity of *Sardinops* imported was around 10,000–16,000 tonnes; this has gradually increased and more recently the amount imported for tuna feed was 40,000–50,000 tonnes, and possibly up to 70,000 tonnes.

The pattern of usage of *Sardinops* at the tuna feedlots in central South Australia, which involves introducing large amounts of feed into relatively small areas, is intuitively more risky to Australian *Sardinops* than the introduction of smaller quantities over wider areas (Jones, 2000), with the latter type of usage being the case for the majority of other consumers of *Sardinops* in Australia, who use this species as bait for line fishing.

In contrast to the bulk usage of baitfish to feed caged tuna in South Australia, the Western Australian rock lobster industry also imports about 10,000 tonnes of frozen bait annually, albeit with none of this

consisting of *Sardinops*, but uses smaller quantities over wider areas (Jones and Gibson, 1997). Since about 1980, when large quantities of bait were first imported for the lobster industry, there have been no indications of exotic disease events associated with this type of usage (Jones and Gibson, 1997). This contrasts markedly with the two epizootics of *Sardinops* in Australian waters that have occurred within the six year period over which *Sardinops* has been imported in large quantities and used in relatively small areas (Jones, 2000).

Socio-economic implications

The loss of *Sardinops* in southern Australia has seriously impacted the commercial fishery, including fish-processing establishments. Socio-economic effects of the mass mortality in southern WA have thus been significant (Gaughan et al., 2000). Despite this, there has been a general lack of concern by the recreational fishing sector, the largest consumer of *Sardinops* in Australia, which highly values *Sardinops* as bait. This has partly arisen because the gap left in the market by the decline in local fisheries has, ironically, been filled by imported *Sardinops*. Although the southern Australian pelagic ecosystem can be considered large (van der Lingen et al., in press), it has relatively small *Sardinops* populations due to much lower productivity than the globally significant upwelling systems of South Africa and the Americas that support huge *Sardinops* fisheries (Schwartzlose et al., 1999; Gaughan et al., 2001). As such, the general community appears to have a very poor understanding of, or interest in, this pelagic ecosystem. Surprisingly, there has also been little comment from the marine conservation movement, perhaps because increasing numbers of seals and whales in Western Australian waters has led to a certain level of satisfaction.

Ecosystem implications

Because *Sardinops*, as with other abundant clupeoids, typically form an important component in pelagic food webs and account for much of the energy transfer between lower and higher trophic levels (e.g., Bakun, 1996; Cole and McGlade, 1998), the sudden loss of *S. sagax* from the pelagic ecosystem of southern Australian shelf waters would be expected to result in changes to that ecosystem, including negative impacts on higher trophic levels (e.g., Mackinson et al., 1997; Cury et al., 2000). Direct knock-on effects that negatively impact predatory species

following pathogen pollution (Daszak, 2000) of prey can be expected. Furthermore, more cryptic knock-on effects that impact biodiversity, if not the important exploited or forage species, are also likely (Daszak, 2000). Although such impacts may never be formally described, it is not ethical to pretend they do not exist. Prediction of specific effects is difficult. For example, due to the complexity of interactions within marine ecosystems, some predator species may recover quickly while others may take many years to recover following a major decline in abundance of their dominant prey (Cury et al., 2000). Although populations of predators can respond to periodic changes in the abundance of prey resources (Bakun, 1996), predator populations may not be “buffered” against long-term changes (Cury et al., 2000). Likewise, patterns in the population dynamics of predators would be unlikely to include strategies to deal with very sudden decreases in prey populations that can result from mass mortality events associated with exotic pathogens. Including a plan to minimize risk of disease translocation, through a risk analysis and research framework, as part of the management of exploited species would be prudent.

Given the paucity of community concern over the mass mortality of *Sardinops* in Australia, there has been little impetus to examine either any of the potential effects on the ecosystem or of the risk of the continuing, and expanding, practice of introducing imported *Sardinops* into the marine environment occupied by Australian stocks of this species. Peer reviewed studies, which address impact of either of the mass mortality events, the two largest yet recorded for aquatic organisms, have thus far been scarce, with the first taking five years to appear (Bunce and Norman, 2000; Dann et al., 2000; Gaughan et al., 2000; Ward et al., 2001). Dann et al. (2000) recently concluded that the 1995 *Sardinops* epizootic was responsible for the deaths of a considerable number of little penguins (*Eudyptula minor*) and a significant decrease in their breeding success off the central Victorian coast in south-eastern Australia; this appears to be the only study published in the primary scientific literature that describes an ecological impact of the 1995 epizootic. Following the 1998/99 epizootic, Bunce and Norman (2000) described a dramatic decrease in the proportion of *Sardinops* in the diet of the Australasian gannet (*Morus serrator*) and suggested this would cause a decrease in breeding success and survival. In contrast to the 1995 mass mortality, two studies that estimated the impact on the *Sardinops* stocks following the

1998/99 epizootic (Gaughan et al., 2000; Ward et al., 2001) were relatively rapidly completed.

The need for a review of international standards

Infection by a pathogen that was translocated across geographic boundaries has apparently been the case for *Sardinops* in Australian waters, regardless of the origin and identity of the fatal agent, although we now know it was PHV. Because the pathogen responsible for 1995 and 1998/99 epizootics appears to have been species-specific (Fletcher et al., 1997; Gaughan et al., 2000) it is relevant that the imported *Sardinops* are the same species as occur in southern Australian waters: the two populations have been geographically separate for 200,000–500,000 years (Grant et al., 1998). Due to the duration of separation, the populations of *S. sagax* which contribute to each of three subgroups (Japan-China-Korea-Russia, North and South America, Australia-South Africa) would be expected to have divergent immunity profiles (Grant et al., 1998).

The likelihood for divergent immunity profiles between geographically separate populations, the fact that two mass mortalities of *Sardinops* occurred in Australian waters within the six year period that this same species has been imported in large quantities (Jones, 2000) and the non-random origin of both epizootics close to where the largest quantities of imported *Sardinops* enter the water, all provide qualitative evidence that imported *Sardinops* were the source of PHV. Of the number of hypotheses regarding the origin of PHV in southern Australia, the imported-*Sardinops* hypothesis is thus the most difficult to dismiss (Gaughan et al., 2000).

PHV has not, however, been traced back to imported *Sardinops* (Hyatt et al., 1997; Jones et al., 1997; Gaughan et al., 2000). This fact is reiterated here because it appears to have underpinned the lack of a specific IRA on *Sardinops* in Australia, despite the frequency and magnitude of the two epizootics. Therefore, inconclusive knowledge of the fatal agent represents a crucial factor in the management of *Sardinops* in Australia, a process which must, however, also recognize obligations pertaining to international biodiversity legislation.

Given the flaw in the OIE approach to risk assessment (Jones, 2000), whereby risk of disease incursion can exist in an imported commodity without the offensive pathogens having been identified, the

lack of quantitative evidence of a link between the two epizootics of Australian *Sardinops* and imported *Sardinops* should not necessarily negate the need for a formal IRA nor the consideration of qualitative evidence if an IRA was instigated. Because a qualitative approach to IRA is also consistent with OIE policy, *Sardinops* should perhaps be given special quarantine status rather than be included under a general umbrella for the plethora of species for which there is poor information on disease status. Under international law, there are few restrictions on trade of these species specifically because of the poor information on disease status. As OIE policy promotes the need for flexibility in risk assessment (OIE, 2001a, Article 1.3.2.3.1), it provides a means for international law to recognize special cases of high-risk commodities even if particular diseases have not been formally identified in the exporting country. This allows continuation, or instigation, of an IRA in the absence of disease identification.

The problem of apathy regarding global-scale viral traffic described by Morse (1993) in regards to human health issues, an area which for humanitarian reasons has received considerable attention, warrants that the potential for viral transport within the world trade in aquatic organisms be approached proactively. While the Australian *Sardinops* and the Norwegian and Scottish salmon epizootics may be seen as extreme examples, it is such cases that cause the most damage and which must therefore be considered within international risk-management plans (see Jones, 2000). These rare events have been the most problematic to describe in a scientific manner because of the inherent difficulty in establishing statistical robustness in non-experimental situations.

While estimating quantities of animals killed in an aquatic mass mortality event is difficult, the methods are theoretically straightforward. In contrast, our understanding of the population dynamics of viruses is still expanding. In the case of the 1998/99 Australian *Sardinops* epizootic, it was essentially previous experience (observation of the wave – like spread of the disease in the 1995 event coupled with the same staff being present) that allowed design of a sampling program to quantify the impact of the disease in WA by Gaughan et al. (2000), whereas Ward et al. (2001) used “before and after” estimates of spawning biomass to assess the impact in SA. In direct contrast to the results obtained in these studies, attempts in WA to experimentally infect *Sardinops* with viral extracts from samples collected at the wave-front of the mass

mortalities failed. The experience gained in 1995 assisted in setting up the experiment, with *Sardinops* acclimatized within experimental aquaria in southern WA well before the epizootic arrived. But what was not known at the time of the trial infection was that maximum PHV concentrations in *Sardinops* preceded the wave-front (i.e., preceded death) by four days and that individual fish were a source of infection for only a short period (Murray et al., 2000). This meant that the best chances of obtaining high levels of PHV for extraction was to collect *Sardinops* several days before the mass mortality appeared in any one region, which was not the protocol undertaken. Thus, the infection-trial failed due to a flaw in its design that resulted from poor understanding of the nature of PHV, not because the wrong disease agent had been implicated. Because it could not be shown in an experimental situation that PHV was the pathological agent, various interested parties incorrectly interpreted this to mean that PHV was therefore still only one of several possible factors causing the mass mortalities, a view that continues to persist against scientific opinion. Indeed, some aspects of our understanding of disease are still so poor that salmon infected with Infectious Salmon Anemia were moved, under veterinary supervision, between farms in Scotland, thereby spreading the disease (Stagg et al., 2001). Consequently, poor understanding of many diseases and associated uncertainties in their epizootiology, particularly regarding the origin of disease, have been a key feature of the conclusions of investigations on the recent epizootics of Australian *Sardinops* and Scottish salmon.

While the broader issue of disease translocation needs greater exposure to combat the negative effects of apathy (Morse, 1993), inconclusive evidence over the origin of a particular disease tends to dominate the communication of research results. An inconclusive result is thus seen as more correct than unsubstantiated, but expert scientific opinion. Having uncertainty as a key part of the formal conclusion provides a negative focus for the understanding of mass mortality events and hampers the ability to draw comparisons between meso-scale epizootic events. The potential insights that could be gained from such comparisons could likely be applied to managing the risk of disease and as such, should also be considered during IRAs.

Given the economic, nutritional and social importance of fishing to some regions, a concerted effort to undertake research into minimizing the risk of disease transfer, i.e., gaining greater control over viral traffic (Morse, 1993) of aquatic resources, should be

a priority for the R&D (research and development) strategies of the fishing industry. That is, high priority should be given to adequately funding research into disease transfer in aquatic commodities and the major commercial producers and users of the commodity should provide the funds, particularly in those cases where profit levels are sufficient to permit the R&D investment. Pathogen invasion was recently highlighted as one of the four broad threats posed by aquaculture activities to the world's ocean and coastal resources (Naylor et al., 2000). A negative result for an IRA for aquaculture, either for the cultured commodity or for an imported food material, should not be viewed as the end point for any particular aquaculture industry. Rather, it should be the impetus to invest in the longer term success of an industry by funding research that alleviates the risk of disease translocation.

Conclusions

Infection by a pathogen that was translocated across geographic boundaries has apparently been the case for *Sardinops* in Australian waters, regardless of the origin and identity of the fatal agent, although we now know it was PHV. Hazard identification, which OIE views as a crucial first step in its guidelines for risk assessment, should not be restricted to dealing only with identifiable diseases, but should also embrace case histories of epizootic events linked to unidentified pathogens. Thus, failure to identify a specific pathogen should not always be sufficient reason to conclude an IRA, as is currently the case. Similarly, the example provided by the two *Sardinops* epizootics in Australia indicate that existence of the same species at both country of export and country of import warrants consideration as a special case in deciding whether or not a formal IRA should be instigated, without the need for knowledge of the disease status of the species involved. In particular, recognition of high risk associated with creating artificial links between geographically separate populations within a particular species and between similar species should not await quantitative evidence. As anthropogenic factors have been strongly linked to emerging infectious diseases of wildlife, including marine organisms (Harvell et al., 1999; Daszak et al., 2000), it is time to recognize that trade in untreated commodities is in fact the hazard; attaching identities and names to the hazardous components is clearly a secondary concern

in managing disease translocation. We cannot wait for conclusive results from controlled experiments to show us that potential for disaster exists. The potential already exists: the risk of disease should not be ignored just because a particular disease cannot be detected (Hoffman, 1970; Bakke and Harris, 1998).

Once the problem of transporting diseases to naïve populations is addressed at an international level, then perhaps the local-scale managers, those who interface directly with industry and undertake deliberations on resource sustainability, will have the support required to make the appropriate decisions. In the case of the Southern Australian states, such decisions have the broader capability of reducing the risk currently facing the warm-temperate, pelagic ecosystem of an entire continent, and which constitutes one of world's large marine ecosystems that support *Sardinops* populations (van der Lingen et al., in press).

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