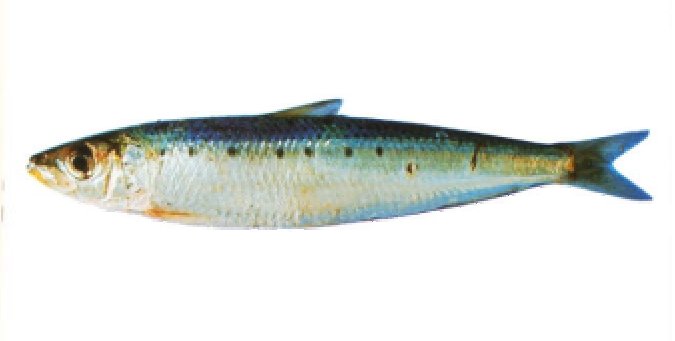
PILCHARD (*Sardinops sagax*) MORTALITY EVENTS IN AUSTRALIA AND RELATED WORLD EVENTS



FRDC Project 99/227

Alexandra C. Gaut

FISHERIES RESEARCH & DEVELOPMENT CORPORATION

PIRSA
Primary Industries and Resources South Australia

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PREFACE AND DISCLAIMER

As part of the investigations into the 1995 and 1998-99 pilchard mortality events in southern Australian waters, Australia's Consultative Committee on Emergency Animal Diseases (CCEAD) endorsed a national strategic research program developed by its Joint Pilchard Scientific Working Group (JPSWG) in October 1998. In addition to the research projects, JPSWG identified the need for a comprehensive report on the mortality events. In February 1999, PIRSA Fisheries submitted a project proposal to the Fisheries Research and Development Corporation to obtain funds to produce such a report. Funding was provided in October 1999. A consultant was appointed in February 2000, and a preliminary draft of the report was submitted to JPSWG in August 2000.

The report was compiled by the author on behalf of PIRSA Fisheries but represents the collective views of members of the JPSWG. All members of the committee made substantial contributions to the report. Throughout their deliberations, members of the JPSWG had utmost respect for each other's views, but there was not always unanimous agreement on all matters. Individual members or organisations represented do not necessarily agree with each of the statements and interpretations made in the report.

JPSWG submitted the draft final report to CCEAD in October 2000. CCEAD members endorsed the report, however, substantial comments on the contents were received from both South Australia Animal Health and from Fisheries Western Australia. The author considered all submissions when preparing the final version, but incorporation of all comments from all reviewers was not possible, especially where views expressed by different reviewers were diametrically opposed. In particular, some of the changes suggested by SA Animal Health and Fisheries WA were not incorporated. Advice regarding the nature of these suggested changes can be obtained from the organisations.

PIRSA Fisheries and the JPSWG have taken all reasonable steps to ensure that the information contained in the report is accurate at the time of production. Readers should ensure that they make appropriate inquiries to determine whether new information is available.

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1 ACRONYMS AND ABBREVIATIONS

AAHL	Australian Animal Health Laboratory
AAHU	Aquatic Animal Health Unit
ABARE	Australian Bureau of Agricultural and Resource Economics
ABWMAC	Australian Ballast Water Management Advisory Council
AFFA	Agriculture, Fisheries and Forestry, Australia
AFHRL	Australian Fish Health Reference Laboratory
AHC	Animal Health Committee
AHWMC	Animal Health and Welfare Management Committee
AMSA	Australian Maritime Safety Authority
ANZECC	Australia and New Zealand Environment and Conservation Committee
ARMCANZ	Agriculture and Resource Management Council of Australia and New Zealand
AQIS	Australian Quarantine and Inspection Service
ASP	Amnesiac Shellfish Poisoning
BRS	Bureau of Resource Sciences
CALM	Conservation and Land Management (WA)
CBD	Convention on Biological Diversity
CCEAD	Consultative Committee on Emergency Animal Diseases
CITES	Convention on International Trade in Endangered Species of Wild Fauna and Flora
CSIRO	Commonwealth Scientific and Industrial Research Organisation
CCVO	Commonwealth Chief Veterinary Officer
CVO	Chief Veterinary Officer (State)
DPIE	Dept Primary Industries and Energy
DPIF	Dept Primary Industries and Fisheries (now DPIWE, Tas)
DPIWE	Dept Primary Industries, Water and the Environment (Tas)
DSP	Diarrheic Shellfish Poisoning
EMAI	Elizabeth Macarthur Agricultural Institute (NSW)
FAO	Food and Agriculture Organisation
FDC	Fish Diseases Commission
FEHC	Fisheries Environment and Health Committee
FHCG	Fish Health Coordinating Group
FL	Fork length
FRDC	Fisheries Research and Development Corporation
GATT	General Agreement on Tariffs and Trade
ICES	International Council for the Exploration of the Seas
IMO	International Maritime Organisation
IRA	Import Risk Analysis
JPSWG	Joint Pilchard Scientific Working Group (of CCEAD)
LPD	Livestock and Pastoral Division
MAFRI	Marine and Freshwater Research Institute (Victoria)
MEPC	Maritime Environment Protection Committee
NACA	Network of Aquaculture Centres in Asia-Pacific
NFA	National Food Authority
NSP	Neurolytic Shellfish Poisoning
NTFIFFP	National Task Force on Imported Fish and Fish Products
OCVO	Office of the Chief Veterinary Officer

OIE	Office International des Epizooties (World Animal Health Organisation)
PSP	Paralytic Shellfish Poisoning
SARDI	South Australian Research and Development Institute
SARFAC	South Australian Recreational Fishing Advisory Council
SAFIC	South Australian Fishing Industry Council
SARLAC	South Australian Rock Lobster Advisory Council
SBT	Southern Bluefin Tuna
SCARM	Standing Committee on Agricultural and Resource Management
SCFA	Standing Committee on Fisheries and Aquaculture
SCFH	Sub-Committee on Fish Health
SPS	Agreement on the Application of Sanitary and Phytosanitary Measures
TBOAA	Tuna Boat Owners Association, Australia
TBT	Agreement on Technical Barriers to Trade
VFRI	Victorian Fisheries Research Institute
VIAR	Victorian Institute of Agricultural Research
VIAS	Victorian Institute of Animal Science
VPS	Veterinary Pathology Services (now IDEXX/VPS)
WHO	World Health Organisation
WTO	World Trade Organisation

2 NON-TECHNICAL SUMMARY

99/227 Pilchard (*Sardinops sagax*) mortality events in Australia and related world events.

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OBJECTIVES:

1. To provide a comprehensive summary and appraisal of pilchard mortality events in Australian waters prior to 1995 and mass mortality events in the world's oceans to the present time, including an analysis of current knowledge of pilchard diseases and marine herpesviruses.
2. To provide a comprehensive summary of technical information on the 1995 pilchard mortality event.
3. To provide a comprehensive summary of technical information on the 1998 pilchard mortality event and compare the pathology and epizootiology of the 1995 and 1998 events.
4. To identify and assess the direct ecological and economic effects of the pilchard mortality events.
5. To describe and evaluate procedures for coordinating research and management of the 1995 and 1998 pilchard mortality events, and identify and assess options for improving responses to a possible future pilchard mortality event.
6. To identify and evaluate hypotheses regarding the cause, trigger and origin of the pilchard mortality events.
7. To assess the implications of the hypotheses regarding the cause, trigger and origin of the pilchard mortality events for the agencies and industries responsible for the management and utilisation of Australia's marine ecosystems and resources.

This report documents pilchard mortality events that occurred in March-June 1995 and October 1998-May 1999 across the entire distribution of the Australian pilchard population. These two events are unique in recorded history because of the extensive geographic range they covered and the large quantities of pilchards that died.

Examination of affected pilchards in 1995 identified gill lesions that caused the fish to die of asphyxiation. A herpesvirus was consistently associated with these lesions. There was an upwelling event off the western Eyre Peninsula 2-3 weeks before the beginning of the mortality event in 1995. This is thought by some to be involved in the event, although evidence suggests that environmental anomalies were irrelevant.

In 1998, research was more focussed because it was suspected that a herpesvirus might again be involved. Examination of affected pilchards identified gill lesions similar in appearance to those in 1995, as well as the presence of a herpesvirus in association with the lesions.

Several hypotheses were proposed as to the cause of the pilchard mortality events, including environmental factors, phytoplankton and an infectious agent. The herpesvirus found consistently in the gill lesions of affected pilchards from both epizootics is considered to be the most likely cause of the mortalities.

The origin of the herpesvirus(es) associated with the mortalities is a contentious issue. Several theories have been proposed including introduction by seabirds, ballast water or imported baitfish. Other hypotheses suggest that the virus has emerged from an enzootic origin.

Minimal research effort has been directed into monitoring the ecological effects of the pilchard mortality events. Since 1995 numbers of little penguins (*Eudyptula minor*) returning to Phillip Island, Victoria, have decreased, and their diet, which prior to 1995 included a significant quantity of pilchard, has changed to include only a minimal quantity. The pilchard content of the diet of gannets (*Morus serrator*) in Port Phillip Bay, Victoria, decreased from ~50% to 5% immediately after the 1998 mortality event. A positive ecological impact was increased food availability for opportunistic feeders such as rock lobster (*Jasus edwardsii*) and New Zealand fur seals (*Arctocephalus forsteri*). Impacts on large, long-lived predators such as marine mammals and seabirds may not be seen for some time due to 'lag effects' in biological systems. There has been an increase in the abundance of some pilchard competitors such as anchovies (*Engraulis australis*).

The economic impacts of the mortalities have been most strongly felt in the various pilchard fisheries. The estimated loss to these fisheries after the 1995 mortality event was approximately $12 million. Two of three south coast pilchard fisheries in WA had zero quotas set for 2000, after the 1998/99 mortality event. The pilchard fishery in Port Phillip Bay, Victoria has had continuously low catches since 1995. Bait retailers around Australia also suffered loss of income due to a decline in recreational fishing and a shortage of pilchard supply.

The 1995 mortality event alerted Australian authorities to the deficiencies in aquatic animal health policy and the need for a national aquatic animal disease emergency response plan. The event precipitated the formation of the National Task Force on Imported Fish and Fish Products, to report on aquatic animal imports. In its report, the task force recommended the development of a strategic plan for Australian fish health issues. In 1999, AQUAPLAN was launched. It is a comprehensive series of programs each comprising several projects to implement management plans, financial resources, awareness campaigns, international linkages and other areas relating to aquatic animal health. A series of manuals will make up AQUAVETPLAN, which will detail protocols for responding to aquatic animal disease emergencies.

AQIS conducted risk analyses on imported aquatic animals (live and dead) and aquatic animal products and in December 1999 instituted new import policies.

3 INTRODUCTION

3.1 BACKGROUND

In 1995 and 1998 two unprecedented and large-scale pilchard mortality events occurred around the southern temperate coastline of Australia. The mortality events are the largest known fish mortality events in recorded human history. In both cases, pilchards started dying in South Australian waters and mortality 'waves' spread both east and west against all prevailing currents, throughout the entire Australian pilchard population.

The pilchard mortality event of 1995 resulted in pilchard deaths extending from Western Australia to the east coast, as far north as Brisbane, Qld. The mortalities were first recorded on the western Eyre Peninsula, SA. State and Commonwealth authorities were, arguably, ill prepared for the event and although numerous investigations were conducted, it remains uncertain as to how well they were coordinated.

It was concluded that the most likely cause of the mortalities was a herpesvirus which infected the gills of the pilchards (Hyatt *et al*., 1997; Whittington *et al*., 1997). However, the source of the virus remains unknown. It is considered that although the herpesvirus is the most probable cause of the pilchard deaths, other factors cannot yet be discounted.

Despite the numerous attempts to investigate the 1995 mortality, no consolidated report has been produced. The WA Department of Fisheries produced a report focussing on the mortality events in Western Australia (Fletcher *et al*., 1997). A National Pilchard Mortality Task Force produced an interim and a final report on the pilchard mortalities but these reports have never been published (Anon. 1995; Anon. 1999). A number of specific scientific papers were subsequently published (Hine, 1995; Smith, 1995; Smith *et al*., 1996; Whittington, 1996; Griffin *et al*., 1997; Dann *et al*., 2000).

In October 1998, pilchard mortalities were again recorded in Spencer Gulf, SA. As in 1995, the mortalities spread both westward and eastward. A Joint Pilchard Scientific Working Group was established under the auspices of the Consultative Committee on Emergency Animal Diseases to monitor and investigate the 1998 mortality event. There have been few papers published in relation to this event (Gaughan *et al*., 2000; Murray *et al*., 2000; Bunce and Norman, 2000).

It soon became apparent that the lack of a consolidated report, suitably refereed and supported by comprehensive records of field data, was a major impediment to the efforts of the Working Group. It was fortuitous that many individual scientists, who had participated in the 1995 response were available to provide the necessary background and interpretation.

3.2 NEED

1. After and during both the 1995 and 1998 mortality events there was considerable debate about whether or not similar mass mortalities of pilchards had been recorded previously. There was therefore a need to collate all previous records of pilchard mortalities in Australia and overseas. It was also noted by participating scientists that

current knowledge of pilchard diseases and marine herpesviruses had not been synthesised, and that this was an impediment to their investigations.

2. Investigation of the 1998 mortality event was hampered by the lack of a comprehensive national report on the 1995 pilchard mortality event. Although this problem was partially ameliorated by involving scientists who were also involved in the 1995 event, it was still necessary to compile information from 1995, as these scientists may not be available in the future.

3. In order to ensure that investigations of possible future marine mass mortality events are not impeded by the lack of a comprehensive national report of the 1998 event, it was necessary to collate all the available information about the 1998 event from all affected States.

4. Pilchards are a key component of temperate pelagic ecosystems (Bakun, 1996) and concern was expressed that the loss of a significant quantity of pilchard biomass may significantly affect ecosystem structure and function (Gaughan *et al.*, 2000). However minimal research effort has been directed into this issue (Bunce and Norman, 2000; Dann *et al.*, 2000; Ward *et al.*, in press). No comprehensive analysis of the potential ecological effects of the mortality events has been produced.

The two mortality events significantly affected the economic viability of pilchard fisheries of SA and WA, and recreational bait and tackle retailers in southern States. For example two of three pilchard fisheries on the south coast of WA had their quota cut, from 1500 tonnes (Albany) and 1900 tonnes (Bremer Bay), to zero for the year 2000, effectively closing the fisheries after the 1998/99 mortality event. Although the economic impacts of the mortality events were significant, these effects have not been formally assessed.

5. After the 1995 event, concern was expressed that coordination of the investigation was poor. However, there has been no detailed review examining the overall response to the event. The coordination of the 1998 event is considered to have been much improved, however, this response has not been reviewed. A thorough review of the responses to both events is required in order to suggest improvements for responses to future marine mass mortality events.

6. Many hypotheses have been proposed as to the cause and trigger of the events, and to the origin of the virus (Anon., 1995; Fletcher *et al.*, 1997), however no report has provided a detailed examination of all the hypotheses proposed for both events. Such an assessment is needed in order to provide directions for future research.

3.3 OBJECTIVES

The original proposal objectives were:

1. Prepare a comprehensive report of pilchard mortality events in Australian waters prior to 1995 and in waters world wide to the present time.
2. Provide a summary of the 1995 pilchard mortality event.
3. Prepare a comprehensive and competent technical report on the pilchard mortality event in 1998.
4. Describe and evaluate the coordinating and managing approaches taken in the 1995 and 1998 pilchard mortality events.

5. Assess the implications of the pilchard mortality events
6. Evaluate the conclusions, which can be drawn from various hypotheses as to the cause, origin trigger and epizootiology of the events.
7. Assess options for managing future pilchard mortality events.

The revised objectives are:

1. To provide a comprehensive summary and appraisal of pilchard mortality events in Australian waters prior to 1995 and mass mortality events in the world's oceans to the present time, including an analysis of current knowledge of pilchard diseases and marine herpesviruses.
2. To provide a comprehensive summary of technical information on the 1995 pilchard mortality event.
3. To provide a comprehensive summary of technical information on the 1998 pilchard mortality event and compare the pathology and epizootiology of the 1995 and 1998 events.
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6. To identify and evaluate hypotheses regarding the cause, trigger and origin of the pilchard mortality events.
7. To assess the implications of the hypotheses regarding the cause, trigger and origin of the pilchard mortality events for the agencies and industries responsible for the management and utilisation of Australia's marine ecosystems and resources.

4 MASS MORTALITY EVENTS IN MARINE FISHES

Objective: To provide a comprehensive summary and appraisal of pilchard mortality events in Australian waters prior to 1995 and mass mortality events in the world's oceans to the present time, including an analysis of current knowledge of pilchard diseases and marine herpesviruses.

This objective was achieved by reviewing scientific literature on marine mass mortality events, pilchard diseases and herpesviruses. Pilchard mortality events have been observed in Australia and New Zealand since the mid-19th century, however no previous event moved as a "bushfire-like" wave, killed a large quantity of fish or affected the entire population like the 1995 and 1998/99 mortality events. Similarly, no mortality events affecting pilchard populations overseas have displayed these characteristics. A mortality event of marine catfish (4 species) that began near a port in Babitonga Bay, Brazil is the only other mass marine mortality reported that has moved in a wave-like manner with a front travelling against prevailing currents. However, in comparison with the Australian pilchard mortality events, the Brazilian event was relatively small.

Marine mortality events have been reported overseas for the last four centuries. There is evidence to suggest they are occurring with increasing frequency. Harvell *et al*. suggested in a 1999 issue of the journal 'Science' that climate change and anthropogenic factors are significant. The article also suggests that mass mortality events are usually due to disease, and that new diseases are usually the result of existing pathogens that are introduced to new hosts because of translocation.

Virtually nothing is known about pilchard diseases. Viral haemorrhagic septicaemia virus was responsible for a pilchard mortality event in Canada (1998; Glavin, 1999), but this was the first time this virus has been associated with pilchards. Herpesviruses affect a broad range of marine organisms. There are 23 herpesviruses that affect 26 species of fish, but there are no prior records of a herpesvirus affecting any species of clupeoid fish.

4.1 AUSTRALIA AND NEW ZEALAND PRIOR TO 1995

4.1.1 Australia

Pilchard mortalities have been recorded in Australia since the mid-19th century, but prior to 1995, these were all exclusively localised, small-scale events. There is also no evidence to suggest that mortality events which occurred prior to 1995 moved in a "bushfire-like" wave around the entire distribution of the Australian pilchard population, or that affected the same large numbers as the 1995 and 1998/99 mortalities.

Pilchard mortality events occurred in Tasmania in 1844 and 1867 (Waite, 1923). The 1867 event took place on "Bruni" (Bruny) Island in southern Tasmania. It was said, "an immense shoal was driven … by larger fishes in such numbers that they absolutely suffocated each other". It was estimated that "at least 100 tons there and fully 200 more at the bottom of the water, all dead."

There are no other accounts of pilchard mortality events until the middle of the twentieth century. Mr Dinko Lukin recalls "steaming through continuous dead pilchards from Ward Island to St. Francis Island" (SA) in the late 1970s. Another story from South Australia is from Mr G. Gibble and Mr R. Hall, who both remember

a time in the late 1950s when thousands of tonnes of pilchards washed up along the south east coast and the Coorong. No other details of these events are available.

In January 1982 thousands of pilchards washed up on Hawley Beach in northwestern Tasmania (Copas, 1982). The fish were adults (15-25 cm) and had washed up with their stomach burst, their intestines lost before they washed ashore, and still bleeding from the gills and anus. According to the Tasmanian Fisheries Development Authority these were symptoms associated with crushing. A spokesman speculated that it was possible that the fish were caught in a net, dragged and crushed, then dropped out, however, no cause has ever been assigned to this case.

In 1984, in Victoria, there was a multispecies mortality event in Port Phillip Bay, which appears to have gone on for several months and included a small amount of pilchards. During this time several pilchards were submitted to the then Australian Fish Health Reference Laboratory, Benalla, Victoria (now AAHL Fish Diseases Laboratory) for pathological examination. The pilchards all had skin lesions but none of the pathology of the other species was consistent.

In March 1990 pilchards were found in the stomachs of King George whiting caught in Investigator Strait, South Australia (Griffin *et al.*, 1997). King George whiting is usually a benthic feeder, so it was highly unusual to find pilchards, a pelagic fish, in their stomachs. This phenomenon was also noted in the 1995 mortality event when pilchards were found in the stomachs of deep sea flathead which are also benthic feeders.

Just under a year later in January 1991, juvenile pilchards were killed in the Port River Estuary, Adelaide. These deaths were tentatively associated with a mixed dinoflagellate bloom of the toxic phytoplankton, *Alexandrium minutum*, and *Scrippsiella* spp., a non-toxic genus, which lowered the dissolved oxygen content of the water (Griffin *et al.*, 1997).

4.1.2 New Zealand

New Zealand has a history of pilchard deaths dating back to 1891, but none of these are similar to the event that occurred there in 1995 or in Australia in 1995 and 1998/99. Until 1995, all pilchard mortality events in New Zealand were localised occurrences that were associated with either phytoplankton blooms or predators.

Graham (1956) cites pilchards being washed ashore "in large quantities" in 1900-1902, 1921 and 1932. He cites other instances of pilchards "being washed ashore during the summer and autumn in large quantities". During May and June the pilchards were apparently chased ashore by Barracouta (*Thyrsites atun*) and forced on to the rocky coastline where "they were dead and dying in their hundreds". Nearby residents collected and sold the fish at market, either fresh, salted or smoked. No pathology is given for any of the deaths. The author also observed that "pilchards die more quickly than any other fish". He attributed this to either the loss of easily disturbed scales, or that "once out of the water they are apparently unable to close the gill-covers as tightly as most other fish and thereby die of suffocation". There are other reports of pilchards dying in the 1920s and in 1937 (Smith *et al.*, 1996).

In December 1993 approximately half a tonne of pilchards died in a small lagoon in Wellington Harbour (Jones & Rhodes, 1994). The pilchards died of anoxia caused by a combination of reduced oxygen levels and the phytoplankton *Tetraselmis*, sticking to and clogging the gill lamellae. No other fish species were affected. The fish were 12-14 cm (fork length) and showed slight haemorrhages and moderate mucous production from the gills. There was also some enlargement and oedema of the gill epithelium.

4.2 OVERSEAS

Marine finfish mass mortalities have been recorded for at least four centuries (Brongersma-Sanders, 1957). Harvell *et al.* (1999) state that reports of the frequency of marine epizootics and the number of new diseases have increased recently, and may be linked to global climate changes and anthropogenic factors. The authors noted that marine mass mortalities are often due to disease. They suggest that the emergence of apparently new diseases is not due to new pathogens but rather the translocation of existing pathogens to new hosts, or by extension of their geographic range because of changing climate regimes.

Griffin et al (1997) documented two mortality events involving several species of marine catfish in 1992/93 off Sierra Leone, and in 1994-95 in coastal waters off Brazil/Uruguay. In Brazil, mortalities of four species of adult catfish began near a port in Babitonga Bay, and spread both north with prevailing currents, and south against prevailing currents. The front of mortalities spread at an average rate of 2.5–3 km/day and eventually covered 1700km of coastline. Phytoplankton or toxins were not observed. The bacterium, *Aeromonas hydrophila*, was present but is very common in stressed fish. There was some suggestion that a virus, isolated from the posterior kidney, was the cause, as there were no contiguous phytoplankton blooms. The definitive cause is unknown (Griffin *et al*., 1997). This is the only other mass marine mortality reported that has moved in a wave-like manner with a front travelling against prevailing currents, similar to the Australian pilchard mortality. It must be noted that in comparison with the scale of the Australian pilchard mortalities, this Brazilian event was relatively small.

Recent communication with Dr. Jan Landsberg (Florida Marine Institute, pers com, September, 2000, with Dr. Keith Jones, SARDI) has resulted in the documentation of similar wave-type mortality events of predominantly hardhead catfish (*Arius felis*) in the marine coastal waters of northern Gulf of Mexico (Florida - Texas) in 1995-96 and more recently in 2000. Similar to the previous catfish mortalities, virus-type particles were isolated from the posterior kidneys of moribund and dead catfish in these latest events. The possibility that all these catfish mortality events are related is appealing; however, insufficient research has been undertaken to verify such a connection.

Mass mortalities of pilchards were first reported in 1941 (Sindermann, 1990). However no evidence suggests that pilchard mortality events of the scale observed in Australia have ever been recorded in any other country (Jones *et al.*, 1997; Ward *et al.*, 1999).

In British Columbia (BC), Canada, during January – March, 1941, “dead fish [pilchards] were observed floating at the surface, washed up on the shore, and on the

bottom, and schools of sluggish, abnormal fish were seen at the surface near shore." (Sindermann, 1990). External signs and post mortem examination indicated pathology inconsistent with the Australasian pilchard mortalities. There was no evidence of other fish species being involved. Although no cause has ever been determined, some authors believe the most likely cause was viral haemorrhagic septicemia (VHS) (Fletcher *et al.*, 1997; Whittington *et al.*, 1997; Glavin, 1999).

Another pilchard mortality occurred in January and February of 1942 off Vancouver Island, BC, in smaller numbers than 1941, but Pacific herring were also found dying in the same area. The signs that were described are similar to those observed in Atlantic herring in which the bacterium *Vibrio* sp has been found responsible for generalised bacteraemia. In winter 1998, another pilchard mortality event occurred around the Johnstone Strait area, between Vancouver Island and mainland British Columbia. This time the cause was definitely VHSV (Glavin, 1999). All of these events were much smaller and more localised than the Australian pilchard mortalities.

Large mortalities of Atlantic herring (*Clupea harengus*) have been observed and reported since 1898. Two causes have been identified: (1) a combination of predator pressure and lethal physical factors, and (2) outbreaks of epizootic disease, caused by *Ichthyophonus hoferi*.

Mortalities of Pacific herring (*Clupea h. pallasi*) have been observed in British Columbia and Alaska in 1949, 1985 and 1994 (Meyers *et al*., 1986; Whittington *et al.*, 1997). Brongersma-Sanders (1957) records a clupeoid mortality on the west coast of Prince of Wales Island, Alaska, in January 1913. A mass stranding occurred where Pacific herring "were left in a solid mass over the beach to a depth of several feet."

In January 1991, in a bay in Greece, there was a mass mortality of multiple species, including marine invertebrates. Of the fish that died, nearly all were the sardine, *Sardinella aurita.* This event has been linked to a massive and very quick cold water event and it is concluded that the fish died from thermal shock (Economidis and Vogiatzis, 1992). The event was highly localised and small scale.

Mortalities of clupeoid fishes are common around the world (eg. Glavin, 1999; Sindermann, 1990), although many are not officially documented. Those that are reported have been small-scale, localised events. The most significant difference between these mortality events and the Australian pilchard mortality events is the way in which, in both Australian events, the mortality front moved in a wave-like manner. This has only ever been reported once before in the catfish mortality in Brazil (Griffin *et al*., 1997).

4.3 DISEASES AND PARASITES OF PILCHARDS

4.3.1 Diseases

Virtually nothing is known of pilchard diseases (Whittington *et al*., 1997; Jones, 2000). In 1998 viral haemorrhagic septicemia virus (VHSV) was found to be responsible for a pilchard mortality event in British Columbia (Glavin, 1999). This event took place under exceptional circumstances and it appears that sudden exposure to extremely cold water made the pilchards susceptible to disease and allowed the virus to become pathogenic (Glavin, 1999). To date VHSV has not been observed in Australian clupeoids, but has been reported in a wide range of marine fishes overseas.

4.3.2 Parasites

There are many known parasites of clupeoid fish. Table 4.1 lists those that have been observed in Australian clupeoids. Some of these parasites (eg. *K. thyrsites, Anisakis* spp.) are commonly found in a wide range of marine fish families around the world.

Table 4.1 Parasites observed in Australian fish of the family Clupeidae.

Parasite species	**Location within host**	**Reference**
Goussia clupearum	Liver, spleen, kidney.	Whittington *et al*., 1997
Kudoa thyrsites	Muscle tissue.	Langdon *et al*., 1992
Parahemiurus merus		Beumer *et al*., 1982
Aphanurus stossichi		Korotaeva, 1969
Pseudobacciger harengulae		Korotaeva, 1969
Anisakis simplex	Liver	AAHL, unpublished data (1996).
Nerocila orbignyi		Hale, 1926
Lernaeenicus spp.	Eye, lateral muscle, heart, or muscle at the base of fins.	Rousset and Raibaut, 1989
Peroderma cylindricum	Kidneys	Ben-Hassine *et al*., 1990
Phocanema decipens	No records of observation in *Sardinops*, but human infections occurred after eating undercooked *Sardinops*.	Grabda, 1991
Hysterothylacium spp.	Visceral cavity, liver.	Korotaeva, 1969
Neomazoraes rohdei	Gills?	Williams, 1988
Mazocraeoides australis	Gills?	Williams, 1988
Unnamed pyriform microsporidian spore	Muscle myofibres.	Langdon *et al*., 1992

4.4 HERPESVIRUSES OF OTHER AQUATIC ORGANISMS

Herpesviruses are known to occur in a wide range of organisms from humans to phytoplankton and are found in a diverse range of fish species. They consist of a double strand of DNA, surrounded by a protein capsid, surrounded by a lipoprotein envelope with projections. The capsid is icosadeltahedral, approximately 100-120 nm in diameter, with 162 capsomeres, most of which are hexameric, but those at the vertices are pentameric (Roizman, 1996). Herpesviruses are known for their ability to establish latent infections (Roizman, 1996). They survive freezing better at lower temperatures (-80°C) than higher temperatures (-20°) (L. Stannard, pers.com.). Some viral particles die during freeze-thaw cycles, but those remaining “alive” will still be infective. After many freeze-thaw cycles, the viral titre may be sufficiently depleted that although still infective, it is insufficient to induce disease (B. Jones, pers.com.).

Herpesviruses are also known to be highly species-specific i.e. once a herpesvirus is established in its host species, it is very rare for it to transfer to a different species. However, some herpesviruses can cross species eg. cercopithecine herpesvirus 1 (B

virus) of Old World monkeys can be pathogenic for humans (Hyatt *et al.*, 1997). There is no evidence to suggest that herpesviruses mutate and evolve faster than other DNA viruses and although there are some data suggesting that interspecies transmission of herpesviruses has occurred in evolutionary time, such events are believed to be exceedingly rare (Davison and McGeoch, 1998; McGeoch and Davison, 1999). A list of known herpesviruses of marine organisms is given in Table 4.4.

4.4.1 Fish

There are 23 known fish herpesviruses that affect 26 species of fish (Tables 4.2 and 4.3). They are found in a wide range of fish from all over the world. In the Australian mortality events, epithelial hyperplasia was consistently seen in dead pilchards from Australia and New Zealand during the two epizootics and is reported as a consistent histopathologic change associated with a large number of fish herpesviruses (Hedrick and Sano, 1989; Wolf, 1988; Iida *et al.*, 1989; McAllister & Herman, 1989; Yamamoto *et al.*, 1983).

Watson *et al*. (1995) report that herpesvirus-infected white sturgeon (*Acipenser transmontanus*) displayed not only epithelial hyperplasia but also hyperplasia of the branchial epithelium and signs of osmoregulatory failure, also seen in Australian pilchards affected in the epizootics. A newly recognised herpesvirus affecting koi carp *(Cyprinus carpio*) produced gill lesions (Hedrick *et al*., 2000) that appear strikingly similar to the gill lesions seen in affected Australian pilchards.

The salmonid herpesviruses (Table 4.3) Yamame Tumour Virus (YTV) and Nerka Virus Tawada Lake, Akita prefecture (NeVTA) are thought by Wolf (1988) to be sufficiently similar serologically and histopathologically, to be included in the same classification as Oncorhynchus masou virus. Several species of salmonids are vulnerable to both Type I and Type II herpesviruses.

Table 4.2 All reported non-salmonid fish herpesviruses.

Hosts species	**Herpesvirus name**	**References**
Channel catfish, *Ictalurus punctatus*	Channel Catfish Virus (CCV)	Wolf and Darlington (1971)
Walleye, *Stizostedion vitreum*	*Herpesvirus vitreum*	Kelly *et al.* (1983)
White sturgeon, *Acipenser transmontanus*	White Sturgeon Herpes Virus (WSHV-1 & -2)	Watson *et al*. (1995)
Turbot, *Scophthalmus maximus* (*Psetta maxima*)	*Herpesvirus scophthalmi*	Bloch and Larsen (1994)
Japanese eel, *Anguilla japonica* Eel *Anguilla anguilla*	*Herpesvirus anguillae* (HVA)	Ueno *et al*. (1996)
Carp, *Cyprinus carpio* Golden ide, *Leuciscus idus* Roach, *Rutilus rutilus* Tench, *Tinca tinca*	*Herpesvirus cyprini* (CHV)	Hetrick and Hedrick (1993) Humphrey (1995) Humphrey (1995)
Koi carp, *Cyprinus carpio*	Koi herpesvirus, KHV-I (Israel), KHV-U (USA)	Hedrick *et al*. (2000)
Goldfish, *Carassius auratus*	Goldfish Haematopoietic Necrosis Virus (GFHNV)	Jung and Miyazaki (1995)
Smooth dogfish, *Mustelis canis*	Shark HV	Leibovitz and Lebouitz (1985)
European smelt, *Osmerus eperlanus*	Smelt papilloma HV	Anders and Möller (1985)
Sheatfish, *Silurus glanis*	Sheatfish HV	Wolf (1988)
Pacific cod, *Gadus macrocephalus*	Pacific Cod HV	Wolf (1988)
Japanese flounder, *Paralichthys olivaceus*	Japanese flounder HV	Iida *et al*. (1989, 1991)
Angelfish, *Pterophyllum altum*	Angelfish HV	Mellergaard and Bloch (1998)
Pike, *Esox lucius*	Pike epidermal proliferative HV	Yamamoto *et al*. (1983)

4.4.2 Reptiles

Amongst reptiles, herpesviruses appear to be particularly prevalent in the Chelonia, especially amongst pond turtles (Emydidae). The only marine reptile to be affected however, is the green sea turtle (*Chelonia mydas*) which is host to a herpesvirus associated with skin lesions (Shortridge, 1989).

4.4.3 Marine mammals

Marine mammal herpesviruses have been isolated from harbour seals (*Phoca vitulina*), beluga whales (*Delphinaterus leucas*), sea otters (*Enhydra lutis*) and from a single Californian sea lion (*Zalophus californianus*). Each of these herpesviruses except the harbor seal herpesvirus (SeHV) was associated with epithelial lesions.

Internal signs associated with the SeHV were emphysema and pneumonia (Borst *et al*., 1986).

In sea otters, the lesions were oral. Although, in some animals, the lesions covered extensive areas of the buccal, labial, gingival and glossal mucosa and appeared under the tongue, the animals rarely showed a reluctance to eat (Moeller, 1996). The beluga whale examined showed a mild dermatitis with skin lesions that regressed and scarred over. There were no other clinical signs of infection and the disease was not fatal (Barr *et al.*, 1989). Over a three-month period prior to its death, the Californian sea lion displayed recurring skin lesions, lethargy, anorexia and alopecia. A retrovirus was however, also present in this animal and it is impossible to attribute the disease signs and death to either virus (Kennedy-Stoskopf *et al*, 1986).

4.4.4 Bivalves

Given the economic significance of oysters, monitoring of stocks has led to these being the first bivalves reported to have a herpesvirus (Farley *et al*., 1972). Sindermann (1990) reports that recent evidence suggests that the viral agents are latent in natural populations but may grow to epizootic proportions under conditions of environmental stress.

4.4.5 Crustaceans

Three herpesviruses have been identified in crustaceans, one occurs in king blue crabs, *Paralithodes platypus,* as well as *P. camtschatica* and *Lithodes aequispina*. A second is found in *Rhithropanopeus harrisii* (Sindermann, 1990) and a third was identified by Johnson (1976) in blue crabs, *Callinectes sapidus.*

4.4.6 Phytoplankton

There has been one record of herpesvirus-like particles in *Platymonas,* a species of phytoplankton. The particles were hexagonal in cross-section, measured between 51-57.5 µm in diameter, and had a single electron-dense core surrounded by a lighter matrix all enclosed by a shell. The particles were only seen in the nucleus (Pearson and Norris, 1974). This was the first evidence for intranuclear virus-like particles in marine algae (Lauckner, 1980).

The particles observed were similar to a herpes-type virus that infects American oysters, *Crassostrea virginica*. In addition, viral pathogens of fish have been found in rotifers (Comps *et al*., 1991). As stated by Pearson and Norris (1974), the occurrence of virus-like particles in a phytoplankton species and of similar agents in oysters provide a basis for speculation that such marine algae may act as vectors for diseases of marine animals. Alternatively, the tendency of filter feeders such as oysters to accumulate various materials may provide a reservoir for a range of viruses.

Table 4.3 Salmonid herpesviruses.

Host species	**Herpesvirus**	**References**
Type I		
Chum salmon, *Oncorhynchus keta* Chinook salmon, *O. tshawytscha* Rainbow/steelhead trout, *Salmo gairdneri*	*Herpesvirus salmonis*	Wolf (1988) Hedrick and Sano (1989) Sindermann (1990)
Type II		
Kokanee salmon, *O. nerka*	Nerka Virus Tawada Lake, Akita prefecture (NeVTA)	Wolf (1988)
Masu/Yamame salmon, *O. masou* Chinook salmon, *O. tshawytscha* Chum salmon, *O. keta* Coho salmon, *O. kisutch* Rainbow/steelhead trout, *Salmo gairdneri*	*Oncorhynchus masou* Virus (OMV)	Wolf (1988) Hedrick and Sano (1989) Sindermann (1990)
Masu/Yamame salmon, *O. masou*	Yamame Tumor Virus (YTV)	Hedrick and Sano (1989)
Coho salmon, *O. kisutch*	Coho Salmon Tumor Virus (CSTV)	Hedrick and Sano (1989)
Type III		
Lake trout, *Salvelinus namaycush*	Epizootic epitheliotropic disease virus (EEDV)	McAllister and Herman (1989)
Not typed		
Atlantic salmon, *Salmo salar*	Atlantic salmon papillomatosis	Hetrick and Hedrick (1993)

Table 4.4 Herpesviruses of marine organisms other than fish. (N = not named)

HOST SPECIES	HERPESVIRUS NAME	LOCATION IN HOST	REFERENCE
Phytoplankton:			
Platymonas spp.	N	Intranuclear	Lauckner, 1980
Bivalves:			
American oyster, *Crassostrea virginica*	N	Cellular aggregates surrounding haemolymph sinuses; mantle epithelium in juveniles.	Farley *et al*., 1972; Buchanan and Richards, 1982
Pacific oyster, *C. gigas*	N	In larvae and spat	Hine *et al*., 1992; Nicolas *et al*., 1992; Renault *et al*., 1994.
Hard-shell clam, *Mercenaria mercenaria*	N	Gonad	Sindermann, 1990
Clam, *Katelysia scalarina*	N		Munday and Owens, 1998
Flat oyster, *Ostrea angasi*	N		Wilson *et al*., 1993
Crustaceans:			
Blue king crab, *Paralithodes platypus* *P. camtschatica* *Lithodes aequispina*	N (presumptive)	Epithelium of bladder, antennal gland and hindgut	Sindermann, 1990
Rhithropanopeus harrisii	N	Mesodermal cells of male gonad	Sindermann, 1990
Blue crab, *Callinectes sapidus*	N	Gill epithelia and haematopoietic tissues	Johnson, 1976
Reptiles:			
Green sea turtle, *Chelonia mydas*	Grey-patch virus (GPV)	Epidermis	Shortridge, 1989

Table 4.4 Continued

Marine mammals:			
Sea otter, *Enhydra lutis*	N	Oral tissues	Moeller, 1996
Harbor seal, *Phoca vitulina*	Harbor seal herpesvirus (SeHV)	Lungs, brain, liver, kidneys.	Borst *et al*., 1986
Beluga whale, *Delphinaterus leucas*	N	Epithelial cells	Barr *et al*., 1989
Californian sea lion, *Zalophus californianus*	N	Lungs	Kennedy-Stoskopf *et al*, 1986

5 1995 TECHNICAL OVERVIEW

Objective: To provide a comprehensive summary of technical information on the 1995 pilchard mortality event.

This objective was achieved by reviewing scientific papers and unpublished reports relating to the mortalities, by examining files from agencies that undertook research into the mortality event and by interviewing scientists and other personnel involved in the research.

The only pathology consistently observed in affected pilchards were gill lesions. A herpesvirus was associated with affected cells in the gill lesions. This herpesvirus could not be cultured, but molecular studies have determined the sequence of 342 base pairs of virus DNA. Only adult pilchards were affected. Transmission trials were conducted in NSW using salmonid fish. The fish remained healthy and showed no signs of disease.

There was no phytoplankton involvement, no biotoxins and although some bacteria were present in affected pilchards, bacteria were also present in pilchards with little or no pathology.

An upwelling event off the western Eyre Peninsula approximately 2-3 weeks prior to the beginning of the mortality event was thought to be linked to the onset of the event, but it is now considered largely irrelevant.

A survey of imported pilchards undertaken by the AAHL found no major fish viruses. There were some bacterial isolates and a few samples of the parasite *Anisakis simplex*.

5.1 INTRODUCTION

In March 1995, an extraordinary and unprecedented epizootic of adult pilchards (*Sardinops sagax*) began in southern Australian waters. There are conflicting reports concerning the first sightings of dead pilchards, but initial reports cluster around the eastern Great Australian Bight, with one of the first coming from Anxious Bay on the western Eyre Peninsula, SA (see Appendix 3 for all dates of reports).

On March 10th, five days before the pilchard mortality event began (March 15th), there was a report of a multi-species mortality event in the outer Coffin Bay area of South Australia. Mortalities included abalone, cockles, stingrays and multiple fish species, but not pilchards. This event overlapped with the pilchard mortality for about a week, but occurred in a different area. The initial multi-species incident in Coffin Bay is thought to have been caused by an algal bloom (P. Christy, pers.com.). The coincidental timing of this incident with the beginning of the pilchard mortalities led many to believe that the pilchard deaths were linked to the bloom (eg. Jones and Rhodes, 1994). The dinoflagellate that was responsible for the multi-species mortality in Coffin Bay was *Gymnodinium mikimotoi*. *G. mikimotoi* is a known fish killing species and it occurs normally in low numbers around the lower Eyre peninsula coasts (and elsewhere). The massive increase in population density in March 1995 was associated with an upwelling of nutrient rich cold water from the deeper layer of the ocean. The G. mikimotoi bloom was extensive and noticeably discoloured the water throughout the bay. However, the inner bays (Mt Dutton Bay and Kellidie) were not significantly impacted by the dinoflagellate because only relatively low numbers were brought in by tidal flows. Although there was initial suspicion that the pilchard mortality may have been connected to the *G. mikimotoi* bloom, subsequent analysis showed that environmental conditions surrounding mortalities elsewhere were normal and that the algae bloom was not the likely cause (Griffin *et al*., 1997).

From SA the pilchard mortalities spread east and west over several months reaching Carnarvon (WA) and Noosa Heads (Queensland) in the middle of June (Anon., 1995). Mortalities began in North Island, New Zealand, in June and continued sporadically until September (Appendix 4; Hine, 1995; Smith, 1995; Smith *et al*., 1996). The Australian epizootic moved progressively, in a "bushfire-like" manner, along more than 5000km of Australia's coastline, which represents the total range of pilchards in Australia. The mortality events traveled against the prevailing Leeuwin and East Australian currents and were not stopped by numerous storm events (Griffin *et al*., 1997). The fronts were observed to move at a rate of approximately 21 km/day to the west and 40 km/day to the east (Murray *et al*., 2000). No other species, including predators, appeared to be involved and only adult pilchards (>10 cm total length) were affected. Pathological examination showed affected pilchards to have gill lesions that caused asphyxiation (Whittington *et al*., 1997). Examination of the lesions by electron microscopy revealed the presence of herpesvirus particles (Hyatt *et al*., 1997).

5.2 TECHNICAL INFORMATION

5.2.1 Histopathology

5.2.1.1 Gills

Affected pilchards showed moderate to severe inflammation in the gills (Whittington *et al*., 1997). There was enlargement of the gills due to an increase in the size of the cells (hypertrophy), as well as abnormal multiplication of the cells (hyperplasia). Secondary lamellae were folded, distorted and apparently shortened (Fig. 5.1, 5.2). There was also sloughing of epithelial cells and general oedema. The ultimate cause of death was asphyxiation, a conclusion supported by blood gas analysis which showed elevated carbon dioxide levels (hypercapnia), decreased oxygen levels (hypoxaemia) and a significantly reduced blood pH (Whittington *et al*., 1997).

The primary lesion was inflammatory; the epithelial cells were hypertrophic and detached from the basement membrane. The subepithelial space was empty, or contained fibrillar and granular material and/or a mixed inflammatory cell exudate with small numbers of a variety of cells (eg. macrophages, neutrophils, lymphocytes and chloride cells) and cell debris. There was also leakage of protein from pillar capillaries into the subepithelial space. Chloride cells detached from their basal location, moved into subepithelial spaces along the secondary lamellae and probably proliferated. In fish from all locations in Australia the gill was consolidated by epithelium. The lesions were initially focal but progressed to become generalised over about 4 days. These lesions were unlike those that are associated with toxic or siliceous algae, physiochemical factors, fungi, bacteria, dinoflagellates, amoebae, protozoa or metazoa. (Whittington *et al*., 1997.)

The morphogenesis of the lesions in pilchard gills and the development of lamellar fusion through contact of attenuated cell extensions followed by hyperplasia were remarkable and have not been described before (Whittington *et al*., 1997). The lack of both mucous cell activity and epithelial degeneration and necrosis were also notable.

A report from New Zealand (Hine, 1996) states the most noticeable difference between the pilchards that died in New Zealand and those in Australia, was that the Australian pilchards showed epithelial hyperplasia in the gills, whereas the New Zealand pilchards only displayed "simple disruption" and loss of epithelium. The gills showed some loss of the epithelial cells, and disorganisation and loss of chloride cells

in the troughs between secondary lamellae. There was less consolidation of the gills of affected pilchards in New Zealand.

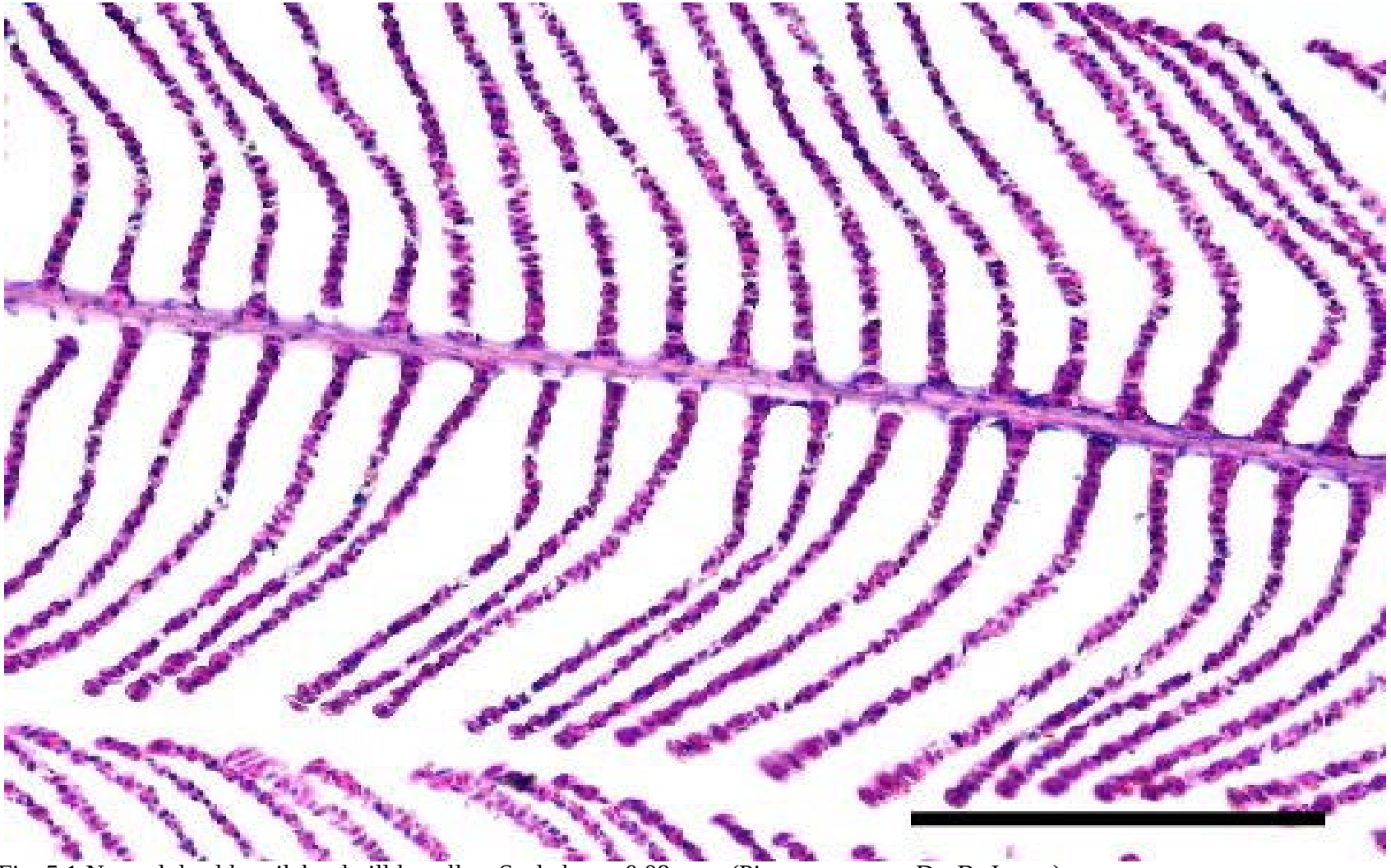

Fig. 5.1 Normal, healthy pilchard gill lamellae. Scale bar = 0.08 mm. (Picture courtesy Dr. B. Jones.)

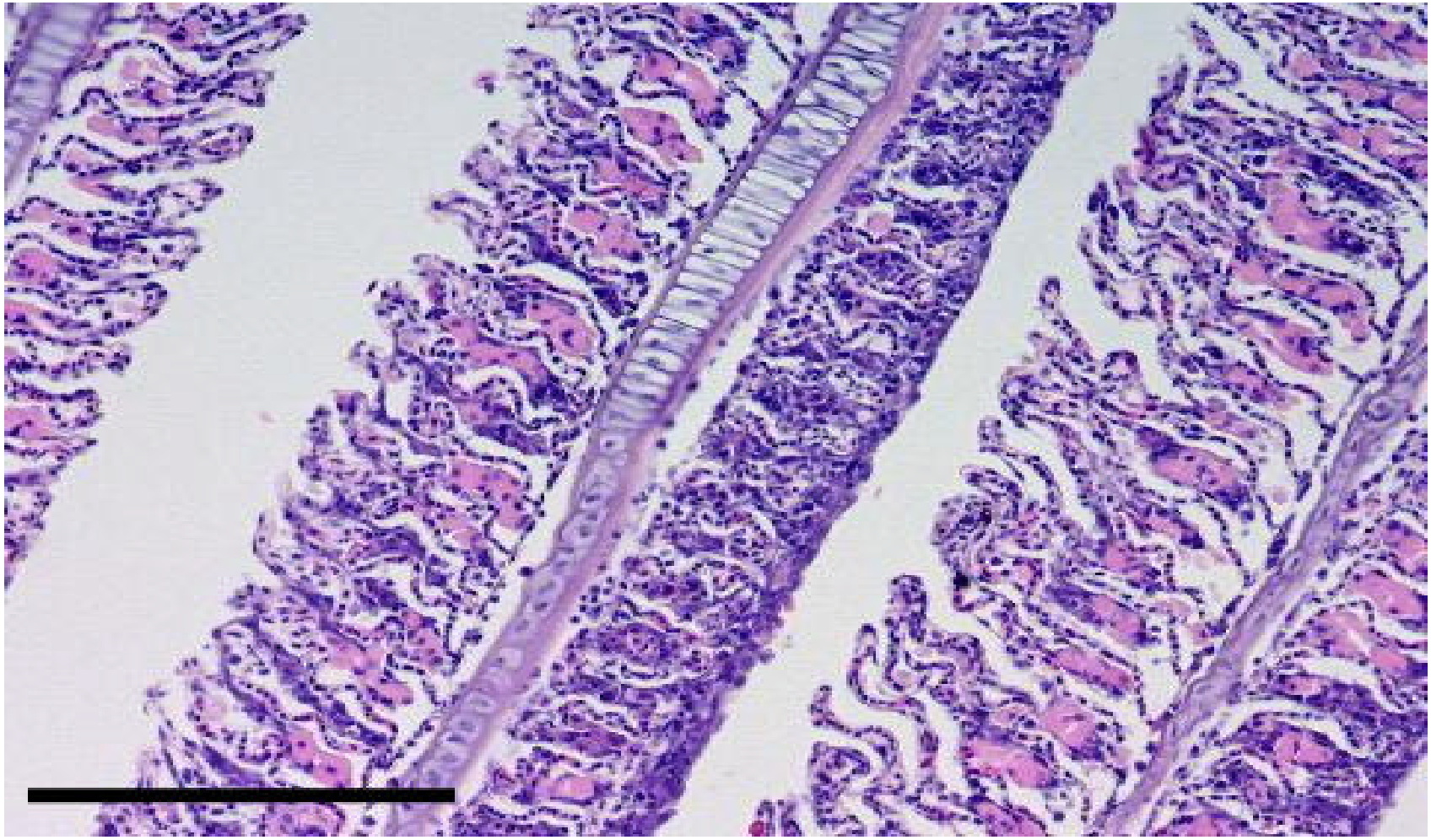

Fig. 5.2 Consolidation of the secondary gill lamellae seen in the gill lesions of affected pilchards from both epizootics (picture from 1995). Scale bar = 0.14 mm. (Picture courtesy Dr B. Jones.)

5.2.1.2 Other tissues

Liver, kidney, spleen, brain, heart, pancreas, gastrointestinal tract, gonad, skin and skeletal muscle were examined from pilchards from NSW (Whittington, 1996). There were occasional insignificant abnormalities in a range of tissues including microsporidian cysts, metazoan parasites, intestinal coccidia and scattered inflammatory foci in various organs. The exocrine pancreas was autolytic or scant in many fish. The large spleens were engorged with erythrocytes, while small spleens contained few. There was mild to marked vacuolar change of the liver in most fish and mild to severe autolysis of the gastrointestinal tract (Whittington, 1996). There were no significant findings in most affected pilchards from WA, although the parasites *Kudoa thyrsites and Goussia clupearum* were found in the musculature and liver of some (Fletcher *et al*., 1997).

There were no lesions in the viscera or brain of affected pilchards in New Zealand (Hine, 1996).

5.2.2 Cell culture

No cytopathic effect typical for a herpesvirus was observed in any of the inoculated cell cultures at any of the laboratories (Hyatt *et al.*, 1997). Some homogenates were confirmed by negative contrast electron microscopy to contain herpesviruses prior to inoculation onto BF-2 monolayers at the Elizabeth Macarthur Agricultural Institute. These cultures did not produce a cytopathic effect during either the first or second passage. Examination of the supernatants and cells by electron microscopy did not find a replicating virus.

A cytopathic-like effect was observed in cultures inoculated with samples from apparently healthy fish obtained from locations ahead of and behind the front and in imported pilchards from California. However, no virus was visualised by electron microscopy in any of the apparently healthy or imported fish (Hyatt *et al*., 1997).

5.2.3 Polymerase Chain Reaction Analysis

Initial specific salmonid (Type I and II) and ictalurid (Channel Catfish Virus) herpesvirus polymerase chain reaction (PCR) primers yielded negative results with tissue samples from affected pilchards as well as partially purified pilchard herpesvirus DNA.

New Zealand oligonucleotides were claimed to yield positive PCR products (Tham and Moon, 1996) but these PCR primers yielded negative results in PCR tests, conducted by the AAHL, on partially purified DNA as well as known infected gill materials.

PCR primers based on the Channel Catfish Virus thymidine kinase gene sequence were generated, tested and shown to yield a product of approximately 250 base pairs. From the sequence of this DNA fragment, specific oligonucleotides for a hemi-nested PCR specific for this sequence were devised to give a 180 base pair product, which was subsequently cloned. Tissue samples obtained from affected pilchards yielded positive results by PCR using these primers. Moreover, both partially purified virus and DNA from infected gills containing herpesvirus were strongly positive by PCR (AAHL, 1999).

To date, AAHL has sequenced 342 base pairs of the 1995 herpesvirus.

5.2.4 Electron Microscopy

Electron microscopy of affected pilchard tissue demonstrated the absence of adhering algae or algal spines and showed no evidence of mechanical damage in the gills (Hyatt *et al*., 1997). Herpesvirus particles were visualised within the affected epithelial cells of fish from WA, NSW, Victoria and New Zealand. The virus was present as naked icosahedral capsids and as enveloped virions. Viruses were not observed from homogenates prepared from unaffected fish. A temporal study undertaken at Iluka, NSW showed a statistically significant correlation between the presence of herpesvirus, histological lesions and mortalities (Hyatt *et al*., 1997).

A few viral capsids were observed in the tubular system of detached and lysed chloride cells and around the debris of chloride cells. Detached mucous cells containing a single virus particle were also observed. The nuclei of infected epithelial cells were of variable shape with dilation of the nuclear membranes. A large ovoid-to-round dark granular inclusion was frequently observed near the centre of the nucleus and electron-dense bodies, 50 nm in diameter, were present in or near patches of fine granular material. Viral capsids were seen in close proximity to these 50 nm dense bodies that appeared to enter the capsids to form nucleocapsids. The nuclear membrane of some epithelial cells appeared fragmented or could not be resolved. Capsids and nucleocapsids were not observed passing through the nuclear membrane but were frequently observed in the cytoplasm. Viral egress occurred by degeneration of the plasma membrane and by the budding of capsids and nucleocapsids through the plasma membrane. (Hyatt *et al*., 1997.)

In New Zealand infection of epithelial and chloride cells by herpesvirus particles was observed (Hine, 1996), but the degree of infection was very variable and in some cases very light. Capsids and nucleocapsids were found in low numbers in the nucleus. In the cytoplasm, nucleocapsids were observed around fine granular masses and close to vesicles that may have been dilated endoplasmic reticulum or dilated Golgi cisternae. Similar vesicles also contained enveloped virions with a dense tegument. Virions were not observed budding from the plasma membrane, which appeared to fragment with the loss of cytosol. Detached epithelial cells often contained only low numbers of nucleocapsids or virions (Hine, 1996).

As with the Australian pilchards, there were many moribund chloride cells in herpesvirus-infected gills. Very few viral replication stages were observed in these cells. Infected cells were invariably detached and had one to three nucleocapsids closely associated with the cell tubular system (Hine, 1996).

5.2.5 Protozoology

Immunofluorescence Antibody Tests (IFAT) demonstrated the absence of significant bacteria, but detected the presence of amoebae in affected pilchard gills from Tasmania, Victoria, WA and NSW. The tests included antisera to 4 amoeba species. The amoebae were not *Paramoeba*, which includes species pathogenic to salmon. The most likely candidate was thought to be a *Vanella* sp., but the identity was never confirmed. There were very large numbers of cells deep in the gill tissue and sometimes many on the surface (Handlinger, 1995, unpublished).

It is noteworthy that unaffected pilchards from Queensland had amoebae present in the gills with little pathology (AAHL, 1995, unpublished).

5.2.6 Phytoplankton and biotoxins

There is no consistent correlation between the presence and abundance of any phytoplankton species and the pilchard mortality events. There is also no evidence to suggest that biotoxins were involved.

A strong correlation was identified between mortality sites in NSW and Victoria and the occurrence of an unidentified species of *Thalassiosira*. In WA, *Thalassiosira* was present in variable but low to moderate numbers on the south coast and was largely absent from the west coast (Fletcher *et al.* 1997). In South Australia, there was also appeared to be a strong initial correlation of pilchard mortalities with water of greater than normal turbidity.

Phytoplankton sampling in WA and gut content analysis by Fletcher *et al.* (1997) showed no consistent pattern in densities within and outside areas where dead pilchards were found. Average phytoplankton densities were generally low with no substantial blooms of any species and no consistent composition amongst locations.

In New Zealand the findings were similar. A routine monitoring program, conducted by the Cawthron Institute, showed no evidence of unusual phytoplankton species or events in the days, weeks or months preceding the pilchard mortalities. Only a few specimens of noxious or potentially ichthyotoxic microalgae were observed (Mackenzie and Todd, 1995).

Pilchards and bivalves tested by bioassay for biotoxins, eg. Amnesiac Shellfish Poisoning, Paralytic Shellfish Poisoning and Neurolytic Shellfish Poisoning, showed negative results in WA, SA and Victoria. At the CCEAD meeting on 16th May 1995, Victoria reported that they had examined the liver, heart and spleen of gulls that had been eating the dead pilchards, for toxicity, but this also proved negative.

5.2.7 Environmental Data

There is no evidence to suggest that environmental influences had any role in the pilchard mortality event (Griffin *et al*., 1997; Fletcher *et al*., 1997). Physical processes which could have potentially played a role in the pilchard mortality include unusually strong upwelling, anomalously low or high water temperatures, or some nutrient variability leading to potentially harmful phytoplankton blooms. The two hypotheses that are associated with environmental influence are that either an environmental anomaly was directly responsible for the mortality event or that an environmental anomaly was a trigger for a latent infection causing the mortality event. Environmental data were also examined for a mechanism that could transport the virus against all prevailing currents.

An upwelling event predated the outbreak of mortalities by 2-3 weeks (Griffin *et al.*, 1997). Upwelling brings water to the surface that is often cold, has low oxygen levels and is high in nutrients. Each of these differences can stress fish and can cause direct mortalities due to thermal shock (Economidis and Vogiatzis, 1992), suffocation, or can make the fish more susceptible to infection by pathogens.

Satellite data showed that the temperature of the upwelled water near Cape Carnot was 4°C cooler than the surrounding surface water (Appendix 6). It was suggested that the initial mortalities observed from Anxious Bay to Fowlers Bay, SA were

linked with upwelling off Cape Carnot, southern Eyre Peninsula, however this hypothesis includes the assumption of a 3 week incubation period for an infectious agent (Griffin *et al*., 1997). A latent period of this length does not correlate with data from a chronological histopathological study that was undertaken at Iluka, NSW (Whittington *et al*., 1997). This study showed that prior to 4 days before death the pilchards showed no lesions in the gills. Modeling of the 1998 epizootic demonstrated that models that best fit the data have a latent period of only 4-5 days, which correlates with the Iluka study (Murray *et al*., 2000).

Temperature changes recorded in NSW were within normal seasonal variability for the Sydney region (Griffin *et al.,* 1997). A cold water intrusion was also noted, extending north of Newcastle, NSW (Anon., 1995). This was unusual but not unique and is thought to be a feature of the Eastern Australian Current.

Sea surface temperatures (SSTs) for the south and west coasts of WA, for March, April and May 1995, were well within normal seasonal ranges (Fletcher *et al*., 1997). Current measurements have shown that the net flow along the Western Australian continental shelf tends to be southwards at about 0.2 m/s in winter (April – August) and northwards at about half that speed in summer (November – March). Numerous other data concerning a mechanism for transporting an infectious agent against the prevailing Leeuwin and other coastal currents are all inconclusive (Fletcher *et al.*, 1997).

The main factor in concluding that cold upwelling or other environmental anomalies were not directly responsible for the mortalities is the fact that only pilchards died. Mass mortalities as a result of thermal shock (Economidis and Vogiatzis, 1992), or other environmental disturbances, usually affect a range of fish species and sometimes also invertebrates. There are several occasions prior to March 1995 when upwelling events off the western Eyre Peninsula have been similar, stronger and/or colder, (Griffin *et al.,* 1997; Appendix 6) and have not resulted in mass mortality events.

It is more difficult to prove the hypothesis that upwelling or some other environmental anomaly acted as a trigger for a latent infection that subsequently caused the mortality event. An example of this sort of trigger occurred in Canada, in 1998, where a localised pilchard mortality event appears to have been triggered by the sudden exposure of the fish to extremely cold water. It appears that the exposure to the cold water increased the pilchards susceptibility to viral haemorrhagic septicaemia virus (Glavin, 1999), which generally causes disease in water temperatures less than 15°C (Kahn *et al*., 1999).

There are several problems with the hypothesis that the mortality event was triggered by the reduction in surface water temperature associated with the upwelling event off the western Eyre Peninsula, SA, in March 1995. For example, the hypothesis assumes that the cool water increased the susceptibility of pilchards to disease. During summer (November – March) cool water (~14°C) underlies warm shelf waters (~20°C) in South Australia creating a thermocline at 20 to 60 m (Ward and McLeay, 1998, 1999a). This stratification is not seen in satellite pictures that only display sea surface temperatures. Localised upwelling is commonly observed during late summer and autumn in areas such as the western Eyre Peninsula. Pilchards are thought to occur both above and below the thermocline (T. Ward, pers.com.), exposing them to this range of temperatures around South Australian shelf waters. Exposure of schools of

pilchards to the cool water brought to the surface by the upwelling event in March 1995 would not have been an unusual event for the fish and probably did not increase the susceptibility of pilchards to disease.

The latent virus trigger hypothesis requires that the virus must already be present in the population awaiting a 'trigger'. There is no evidence that the pilchard population had been exposed to the virus before 1995 and the behaviour of the epizootic was that of a novel agent infecting a naive population.

In New Zealand, the sea surface temperatures were above average in April and May, then fell rapidly in late May/early June to average levels (Smith *et al*., 1996). May 1995 was the warmest on record and the April average was very high. The June temperature was close to the long term average, but the drop in temperature between May and June was 2-3 times faster than normal and was unusual, but many fish species face daily temperature fluctuations of 1-2°C in the environment. Water temperatures recorded in the Tasman Bay (South Island) in late August to early September 1995, were 11.2-11.8°C and are typical spring (September/October) temperatures.

5.3 TRANSMISSION TRIALS

The Elizabeth Macarthur Agricultural Institute attempted to transmit the virus to both rainbow trout (*Oncorhynchus mykiss*) and Australian salmon (*Arripis trutta*). Fingerlings were inoculated by oral, trans-gill or intraperitoneal routes with material shown by electron microscopy to contain pilchard herpesvirus. All fish remained healthy for the duration of the trial. Serum and tissue samples have been retained for future testing when tests become available. (R. Whittington, pers. com.)

5.4 IMPORTED PILCHARD SURVEY 1995-1996

Bacteriological, virological and parasitological examinations were conducted at the AAHL, on a total of 63 batches of imported, frozen pilchards (total of 160 individual fish) submitted by the Australian Quarantine and Inspection Service offices in Adelaide and Melbourne. No fish viruses such as Oncorhynchus masou virus, viral haemorrhagic septicaemia virus or infectious pancreatic necrosis virus were detected, although a cytopathic effect was observed in some of the cultures. Bacterial isolates were identified as various *Vibrio* spp. and *Aeromonas* spp, but not *A. salmonicida*.

In a few samples, forms of the parasite *Anisakis simplex* were observed in or around the liver. The observation of a motile form in one fish raised the issue of conditions of freezing during shipment because this parasite is usually susceptible to freezing. Batches of imported frozen fish must remain below a temperature of -18°C. In the event of a refrigeration failure during the voyage to Australia, the container of spoiled bait is processed into blood and bone meal at 136°C and the cartons and plastic liners are buried in the municipal landfill (Jones and Gibson, 1997).

The AAHL report noted that there was a variable extent of tissue autolysis in the fish samples submitted, probably associated with different methods of freezing, length of time frozen and method of storage.

Although the diagnostic survey undertaken did not reveal the presence of any major fish pathogens, "absence of these or other pathogens from the samples and the whole batches cannot be concluded" due to a number of factors (AAHL, 1996, unpublished):

- Small samples size related to the number and size of whole batches
- Frozen state of samples, which impairs growth of many possible pathogens
- Necessarily limited number of tests applied

In addition to the laboratory diagnostic work undertaken at AAHL, in August 1995 AQIS conducted an extensive tariff-code based review of available customs data on importation of frozen baitfish. One of the conclusions of the review was that tariff codes were not adequately specific to allow identification of product imported for bait and/or tuna feed purposes and therefore different sources of product could not be identified. Limited data subsequently obtained by AQIS in 1995/6 during a two month enhanced monitoring period and for the same year from one importer, identified that the bulk of pilchards imported for bait and/or tuna feed was sourced from the United States and Mexico, while North Sea herring was sourced from the Netherlands and Sweden.

5.5 DISCUSSION

The data collected during the first mortality event did not support a role for a toxic chemical spill, or phytoplankton involvement in the mortalities. However, insufficient data is available to determine the role, if any, of environmental factors. Although it is the conclusion of this report and Griffin *et al.* (1997), Fletcher *et al*. (1997) and Jones *et al*. (1997) that an environmental factor was not involved, there are still some personnel involved with the 1995 mortality event who consider the upwelling event relevant.

As noted in Item 5.4, the number of imported frozen pilchards fish surveyed for the presence of various fish pathogens was extremely small given the large tonnages imported for use as bait and aquaculture feed. Testing was also restricted to exotic fish pathogens for which diagnostic tests were available primarily for the detection of pathogens of salmonid finfish. From the laboratory perspective conducting detailed diagnostic analyses (including tissue culture) on 160 individual finfish is very resource intensive. Frozen-thawed fish specimens do not provide the most suitable material for diagnostic analysis; this is particularly true for detection of gross physical abnormalities as well as microscopic lesions. On balance, it appears unlikely that further sampling and diagnostic work on frozen imported baitfish would have identified a pathogen that would not otherwise have been detected in fresh, chilled, clinically affected specimens submitted during the 1995 mortality event.

6 1998 TECHNICAL OVERVIEW

Objective: To provide a comprehensive summary of technical information on the 1998 pilchard mortality event.

This objective was achieved mainly through personal contact with scientists involved in research of the 1998 mortality event.
There was a single report published outlining some of the research undertaken by the AAHL. Affected pilchards had gill lesions similar in appearance to those seen in affected pilchards from 1995. Pancreatic lesions were particularly significant and were not observed in affected pilchards from 1995. The spleens were, on occasion, severely depleted of lymphocytes and were grossly haemorrhagic. There was also inflammation in the dermis. A herpesvirus was associated with affected cells in the gill lesions but in smaller quantities than in 1995. This herpesvirus could not be cultured. Molecular studies have led to 422 base pairs of the virus DNA being sequenced.
Mainly adult pilchards were affected, with some juveniles also affected in SA and Victoria. SA reported no unusual phytoplankton activity, but no other State undertook sampling. There were no environmental anomalies associated with the onset of this mortality event.
Transmission trials were undertaken in WA with inconclusive results. WA also took blood sera and faecal samples from seabirds seen consuming dead pilchards.

6.1 INTRODUCTION

The mortality event of 1998/99 began in October 1998 in the Spencer Gulf region of South Australia and lasted almost eight months (Appendix 5). The last report of dead pilchards in Western Australia was in May 1999, near Geraldton, at almost the same location that the last mortalities were recorded in 1995. In the east, after only five to seven days of mortalities in NSW, the eastern 'wave' of the event ended in January 1999; Queensland experienced no mortalities. There is evidence to suggest that pilchard abundance in southern Queensland may be low during the summer (November – March; T. Ward, pers.com.). The eastern and western 'waves' traveled at 10.7 km/day (Murray *et al*., 2000). In South Australia and Victoria some juveniles were found and assumed to have been killed as part of the main mortality event. Examination of the SA juveniles to determine the cause of death proved inconclusive, however samples from Victoria were observed to have herpesvirus in the gills (P. Hooper, pers.com.). No other juveniles were observed or collected.

6.2 TECHNICAL INFORMATION

6.2.1 Histopathology

6.2.1.1 Gills

Tissues from Victoria, Tasmania, SA and NSW were examined by AAHL in 1998. The gills showed severe necrotising inflammation which was very extensive and accompanied by mild to moderate multiplication of cells (hyperplasia) (AAHL, 1999). Gills also exhibited primary lamellae with hyperplasia of the basal epithelium. There were few cells active under the epithelium, eg. chloride cells and amoebae. Secondary lamellae had similar epithelial lesions and there were interconnections (anastomoses) of adjacent secondary lamellae. The primary and secondary lamellae were significantly deformed. There was mild atrophy of primary and secondary lamellae and complete lamellar atrophy in some areas (P. Hooper, pers.com.). The presence of

atrophy is only a matter of degree in the disease process and may occur because of a more prolonged and possibly more localised diseased process. The disease process was consistent with viral infection (AAHL, 1999).

Pilchards collected by AAHL staff from Lakes Entrance, Victoria, approximately one month ahead of the disease front, were found to show focal lesions in the gills consistent with the main outbreak (AAHL, 1999). Although the cause of these changes could not be determined, this observation raises the possibility that the infectious cause of the pilchard disease travels much further ahead of the associated mortality front than is generally hypothesized (AAHL, 1999).

6.2.1.2 Other tissues

Pancreatic lesions, similar to other viral pancreatopathies were observed. Although gill tissues from WA showed typical lesions associated with the herpesvirus, the pancreatic lesions were not observed in affected pilchards from WA. The most common lesion was the loss of pre-enzyme (zymogen) granules and apparent replacement by a form of 'cytoplasmic microvesiculation' (P. Hooper, pers. com.). There was mild inflammation, contraction of nuclear contents (pyknosis) and many cells contained nuclei which were enlarged with marginated chromatin. In some cases, there were numerous nuclei surrounded by zones of vacuolation resembling haloes (AAHL, 1999). The remaining lesions consisted of enlarged nuclei somewhat resembling large Adenoviral inclusion bodies, and mild infiltration of macrophages and lymphocytes. The appearance of the latter was significant in terms of confidence that the pancreatic lesions were pathological rather than artefactual (P. Hooper, pers.com.) because macrophages and lymphocytes are part of the immunological defence system.

The spleens were on occasion severely depleted of lymphocytes (AAHL, 1999). The spleens were grossly haemorrhagic and appeared to be necrotised as part of the viral pathogenesis. One spleen was severely depleted, consistent with severe (adrenocortical-mediated) stress (P. Hooper, pers. com.).

There was also inflammation in the dermis with mild hyperplasia. One fish displayed haemorrhage and a lesion in the pharynx; this lesion was thought to be continuous with those of the gills (P. Hooper, pers.com.).

6.2.2 Electron Microscopy

Eight sets of tissues from Victoria and South Australia were submitted for examination by electron microscopy (AAHL, 1999). Examination of ultrathin sections revealed the presence of variable ultrastructural pathology. Epithelial cells were observed fusing with similar cells on adjacent lamellae. Associated with epithelial cells were herpesvirus particles. The presence of virus particles in affected pilchards in WA, was confirmed by negative staining transmission electron microscopy (B. Jones, pers.com.).

The viral particles were associated with cells in variable stages of necrosis and in the nuclei and cytoplasm of infected cells. Proliferation of the nuclear membranes was occasionally observed, as were the electron-dense cytoplasmic inclusions that are associated with the acquisition of viral integuments. In the cytoplasm, the viruses possessed either single or double membranes and the cores were electron dense. The

number of virions and infected cells were not extensive and negative contrast electron microscopy was not as efficient in screening individual fish gills. The numbers observed were, however, significant and associated with the described lesions (H. Westbury, pers. com.).

Associated with the affected gill epithelium were fat droplets and areas containing small discrete particles. These particles (which do not have the structural characteristics of viruses) were not electron dense and were also found in granulocytes. The significance of these structures is not known. There was the possibility that these structures may be glycogen granules (AAHL, 1999); subsequent histological staining indicated that this was not the case (A Hyatt, pers.com.).

The apparent microvesiculation associated with the gills and pancreatic tissues when examined under electron microscopy appeared to be extraordinary structures known as *Rhabdospora* or "rodlets". They were observed in pilchards from South Australia and Victoria. These structures were not seen in samples examined in 1995. The following details have been supplied by the AAHL (A. Hyatt, pers.com.):

(a) The parasite-like organism was present on the surface (and intracellular) of gills and within the diffuse pancreatic tissues.
(b) The organism was present within the lumen and walls of the caeca.
(c) The wall of the "cell" appeared elastic in structure composed of filaments (contractile?).
(d) The capsule was interior to a plasma membrane and appeared to degenerate when the contents of the "cell" were being released.
(e) The "cell" appeared to have the capability of penetrating the walls of the caeca and no cellular junctions were apparent.
(f) Within the "cell" a membrane bound nucleus was present at one end; Golgi, rough endoplasmic reticulum, ribosomes were also present as were vacuoles; mitochondria were not observed.
(g) The main feature of the "cells" were "rodlets" that consisted of dense cores surrounded by a dense fine granular to amorphous region and then an electron translucent area that was membrane bound. There were variable numbers of these membrane bound structures present (eg. up to and in excess of 10). These rodlets appeared to mature over time whereby the outer electron translucent region becomes electron dense.
(h) All of the rodlets appeared to run in a single direction (i.e. along the length of the "cell"). The cells were approximately 5µm in length and 3 to 5 µm in diameter.

There has been, and still is, some controversy as to the identity of these structures; i.e. endogenous cells or parasites. The AAHL reports that recent literature and discussion with colleagues indicate that the "cells" may be part of a cell-mediated immune response; i.e. the number and location of these cells may be a response to infection (A. Hyatt, pers.com.).

6.2.3 Pilchard cell culture

Pilchard tissues collected for primary culture were gill, kidney, liver, spleen and heart. Gill, kidney and spleen cultures did not survive. By March 2000, the liver cell cultures had undergone 29 passages and the heart cell cultures 23 passages. Both cultures have been incubated at 22°C. Aliquots of liver and heart cells were stored in liquid nitrogen

and cell viability following recovery of stored aliquots was good. It is noteworthy that as the cultures are maintained for longer periods it appears that the cells are becoming more robust as they adapt to the *in vitro* conditions (M. Crane, pers.com.). The establishment of new cell lines was a significant achievement.

These cultures were tested with tissue culture supernatants from primary pilchard cell cultures displaying cytotoxicity and gill homogenates from affected pilchards from WA and SA. Although cytopathic effect-like areas were visualised, no virus particles were detected by electron microscopy (M. Crane, pers.com.).

The cultures have also been inoculated with six different fish viruses:

- Viral Haemorrhagic Septicaemia Virus (VHSV)
- Infectious Haematopoietic Necrosis Virus (IHNV)
- Infectious Pancreatic Necrosis Virus (IPNV)
- Oncorhynchus masou Virus (OMV)
- Epizootic Haematopoietic Necrosis Virus (EHNV)
- Atlantic salmon reovirus

These experiments produced similar results with cytopathic effect-like areas, but no virus was detected. It is possible that either the cells are refractory to infection or that any virus that does grow produces cytopathic effect but the virus titre is too low to be detected by electron microscopy (M. Crane, pers.com.). [1]

6.2.4 Polymerase Chain Reaction (PCR) Analysis

Using sequence data from other aquatic animal herpesviruses (eg. Channel Catfish Virus, Salmon Herpesvirus-1), universal primers were designed for the conserved region 'open reading frame-62' of herpesvirus DNA. When these primers were used with DNA derived from herpesvirus-infected pilchards, products were obtained which were homologous with these other aquatic animal herpesvirus sequences. By sequencing the pilchard herpesvirus products, a 422 base pair sequence of pilchard herpesvirus has been obtained and pilchard herpesvirus-specific primers have been designed. The specificity of these primers is undergoing evaluation. The new pilchard herpesvirus primers and universal primers were tested on DNA derived from gill scrapings from affected pilchards in WA and the viruses shown in Table 6.1.

Table 6.1 Viruses that were tested using pilchard herpesvirus and universal primers.

Channel Catfish Virus (CCV)
Salmon HerpesVirus-1 (SHV-1)
White Sturgeon HerpesVirus-1 (WHSV-1)
WSHV-2
Oncorhynchus masou Virus

The universal primers produced PCR products from reactions with Channel Catfish Virus, Salmon Herpesvirus-1 and Oncorhynchus masou Virus whilst the pilchard

[1] Note added in proof: Subsequent to endorsement of this report by CCEAD, recent results from AAHL have shown that the pilchard liver cell strain developed as part of FRDC Project Number 99/226 is, under specific conditions, susceptible to certain finfish viruses including EHNV, IPNV and pilchard orthomyxo-like virus. Further studies to determine the range of viruses to which these cells are susceptible are on-going.

herpesvirus primers did not yield any product from reactions with any of the DNA samples. The lack of any product from the pilchard herpesvirus-primer driven reactions indicates that the pilchard herpesvirus primers are specific. However, PCR with DNA from the pilchard gill scrapings yielded no products. It is possible that the gill scrapings did not yield sufficient virus to be detected. A positive control is needed for the pilchard herpesvirus PCR. Two samples were identified as potential positive controls: semi-purified virus supplied by WA, stored from the 1995 mortality event, and DNA from semi-purified virus stored at the AAHL (M. Crane, pers.com.).

The primers developed from the 1995 mortality yielded positive results on tissues derived from affected 1998 pilchards. It is of significance that the genetic sequence of the product was identical to that obtained from material derived during 1995, this indicates that the two herpesviruses may be identical (M. Crane, pers.com.).

The full details of this process are explained in the AAHL Research Status Report (1999).

6.2.5 Phytoplankton and Biotoxins

Harmful species of phytoplankton were not detected in any samples collected from South Australian waters during the 1998 event and total phytoplankton abundance was low at all locations (Ward *et al.*, 1999).
Because the 1995 mass mortality was not deemed to be associated with phytoplankton, neither WA or NSW conducted phytoplankton sampling in 1998(R Fletcher, pers. com.; D. Gaughan & B. Jones, pers. com.).

6.2.6 Environmental Data

There are no evident environmental anomalies associated with the outbreak of the 1998 mortality event. Upwelling does not usually occur in South Australia during spring (September/October; Appendix 7). The satellite image of the southern Australian coastline around the time dead pilchards were first sighted in 1998 indicates no sign of upwelling. Scientists at CSIRO Marine Research Laboratories (Hobart) concluded that oceanographic factors were unlikely to have had an influence in the second disease outbreak (D. Griffin, pers.com.).

6.3 WA TRANSMISSION STUDIES

6.3.1 Background

Following the second pilchard mortality event, Fisheries WA was requested by the Joint Pilchard Scientific Working Group (JPSWG) to undertake specific investigatory work into this disease. It was considered important to fulfill Koch's postulates to conclusively demonstrate the virus was the aetiological agent. In order to fulfill Koch's postulates in this case it is necessary to:

- isolate the virus in pure form from infected pilchards
- show that purified virus can be experimentally transmitted to pilchards;
- demonstrate that the experimentally infected pilchards develop lesions characteristic of the disease (gill lesions);
- demonstrate that the virus can be recovered from the experimentally infected pilchards

Demonstration and isolation of the virus in infected pilchards was originally achieved in 1995 by the Fish Health Laboratory of Fisheries WA, working in conjunction with the AAHL and NSW Agriculture. The virus was again isolated in 1998 by Fisheries WA and AAHL.

In order to test these postulates, a transmission trial was conducted at Bremer Bay, WA. Uninfected pilchards, held under controlled conditions, were exposed to gill homogenate from dying pilchards and maintained for a period of 23 days.

If gill lesions characteristic of the disease found in wild stocks occurred, samples were to be forwarded to the AAHL for further virus identification work using *in situ* hybridisation and polymerase chain reaction testing.

6.3.2 Objectives

The objectives for these trials were:

1. To demonstrate that an infective agent can cause pilchard mortalities.
2. To demonstrate that the pilchard herpesvirus is responsible for the gill lesions and subsequently the death of the pilchards.
3. To provide a stock of pilchard gill samples from which the virus could be recovered.

6.3.2.1 Transmission trials

Objective 2: To demonstrate that the pilchard herpesvirus is responsible for the gill lesions and subsequently the death of the pilchards.

Methods

Six tanks were established in a factory in Bremer Bay to house the pilchards, 3 tanks for the treatment and 3 for the controls (for detailed methods see Appendix 10). The pilchards were acclimatised to a commercial aquaculture feed before commencing the experiment and the tanks were monitored daily.

During the trial, the treatment pilchards were fed commercial feed pellets coated with viral particles in a suspension medium. The control pilchards were fed the commercial feed coated in the same medium without viral particles.

Approximately 400 pilchards were also kept in a sea cage in Bremer Bay marina and fed on the same pellet feed.

Results

A total of seven pilchards within the treatment tanks and eight within the control tanks died during the 23 days of the trial.

Following completion of the trial (day 23), gills from all pilchards within the treatment tanks were examined. Lesions tentatively associated with early stages of the disease were identified in 6 out of 60 virus-exposed pilchards (similar lesions were not detected in control pilchards). Wax blocks containing tissue from these fish were forwarded to the AAHL for further testing using polymerase chain reaction primers. These tests were negative (AAHL, 1999).

Although pilchards collected on days 13 and 23 showed gill lesions thought to be consistent with early stages of the disease; definitive pilchard herpesvirus disease was not reproduced. Reasons for this may include:

- The virus was not viable;
- The virus was viable but not infective;
- The viral titre was not sufficient to establish an infective dose;
- The virus did not cause the mortalities seen;
- The pilchards were not susceptible to the virus infection at the time of the trial;
- The oral route might not have been an infective pathway;
- Other co-factors may be required to produce severe disease.

Subsequent modeling of the pilchard mortality epizootic of 1995 and 1998 demonstrated that the most successful models used an infectious period of only 1 day before death (Murray *et al.*, 2000). If this is the case, then it is quite likely that the viral titre in the purified virus preparation used in these studies was too low to reproduce disease.

Only 10 pilchards remained in the sea-cage when it was examined. Deaths were reported to have begun 10 days after carcasses of wild pilchards began washing into the marina. Lesions were noted on the gills of the surviving pilchards and these were consistent with lesions observed in other affected pilchards that died as a result of the epizootic.

The only lesions found to be confidently consistent with pilchard herpesvirus were those on the gills of the sea-cage pilchards. These fish were almost certainly exposed to the virus in the open ocean, but this does not satisfy Koch's postulates. It was concluded that until the viability of the virus could be determined, there was no point in continuing to expose pilchards to virus of unknown viability. The current level of technology did not enable the viability of the virus to be confirmed by cell culture. The only pilchard cells associated with the herpesvirus are the gills, but the only pilchard cell lines to have been successfully cultured are heart and liver cultures. At the time of these trials, a PCR was not available to determine whether virus was present in the gills of potentially infected animals; this work is on going at AAHL.

6.3.2.2 Seabird sampling

Objective 3: Sample blood and faeces from seabirds that have been feeding on dead pilchards in WA.

During the 1995 mortality event, deaths of pilchards moved at an average rate of 30 km/day. The disease fronts moved along the coast without the expected interruption from storms, the Leeuwin Current, or the East Australian current. One theory suggested explaining this rapid and continuous movement of the disease front was the mechanical transfer of the virus via the faeces of seabirds feeding on pilchards.

Pathogenic fish viruses can be transmitted by herons (Peters and Neukirch, 1986). To investigate, the mechanisms of spread of the herpesvirus between pilchard populations, the Fish Health Laboratory (WA) collected blood sera and faecal samples

from seabirds known to consume pilchards. The presence of antibody in the blood would show that the birds had been exposed to live virus passing through the gut.

Samples from a number of seabird colonies along the WA coast between Bremer Bay and Albany were collected at the time when pilchard deaths were occurring and when the birds involved had been seen to be feeding on dead pilchards (Table 6.2).

Appendix 11 lists the serum and faecal samples collected from seabirds, all of which were forwarded to the AAHL on 5 May 1999 for storage. In November 1999, the Laboratory reported that the faecal samples were tested with a herpesvirus-specific polymerase chain reaction primer and all samples were negative. The negative results do not mean the virus was not present. It may have been degraded during passage through the gastro-intestinal tract or the quantity of virus may have been below the detection limit of the polymerase chain reaction technique. However, these negative results are consistent with modeling of the epizootic, which indicated that a transmission vector, such as a large, fast-travelling predator, was not necessary for the virus to travel at high speed (Murray *et al.*, 2000).

Table 6.2 Seabirds from which faecal and blood samples were taken.

Common name	**Scientific name**
Crested tern	*Sterna bergii*
Pacific gulls	*Larus pacificus*
Silver gulls	*Larus novaehollandiae*
Australian pelican	*Pelecanus conspicillatus*
Black-faced cormorant	*Leucocarbo fuscescens*
Great-winged petrel	*Pterodroma macroptera*
Flesh-footed shearwaters	*Puffinus carneipes*

No suitable test has been developed to test for antibodies in the seabird sera. The technology to do this exists at several Australian laboratories, but the Joint Pilchard Scientific Working Group determined that the priority for using the limited amounts of virus available should be the DNA sequencing work.

6.3.2.3 Collection of infective material

Objective 4: To provide a stock of pilchard gill samples from which the virus could be recovered.

The lack of sufficient quantities of viable pilchard herpesvirus was a major constraining factor for ongoing research following the 1995 pilchard mortality event. Consequently, the Fish Health Laboratory (WA) took every opportunity, during the 1998/99 event, to collect appropriate tissues from pilchards thought to be infected with the herpesvirus. Staff regularly accompanied commercial fishermen at Esperance, Bremer Bay and Fremantle to collect as much fresh and fixed material as possible.

- **Esperance**: Fresh gills from 600 clinically affected pilchards were collected off Esperance during January 1999. All gills were placed immediately into viral transport media. Half of this material was used during the transmission trial; the rest is stored at -80°C at the Fish Health Laboratory, WA.

- **Bremer Bay**: Approximately 120kg of apparently healthy pilchards caught one day prior to reports of dead pilchards was collected and stored in freezers at Bremer Bay. However, due to a power failure the majority of this sample defrosted and only 10kg previously brought to Perth was salvaged. Gills from this 10kg were stored at -80°C at the AAHL.

- **Fremantle**: Staff from the Fish Health Laboratory accompanied commercial vessels fishing for pilchards around Perth waters on six occasions during April 1999. On each occasion, fresh, frozen and fixed material was collected. 54kg of frozen pilchards caught one day prior to the closure of the fishery was forwarded to the AAHL. Sampling trips continued for a further two days following the closure of the fishery. Histology from these catches indicated that gill pathology was consistent to that observed with other affected pilchards.

6.4 COMPARISON OF 1995 AND 1998 MORTALITY EVENTS

6.4.1 Histopathology

The appearance of the gills and lesions of pilchards affected in 1995 and 1998/99 were similar. There were however, some differences and these are described in Table 6.3. There were more gills with interconnections (anastomoses) of the secondary lamellae, and the primary and secondary lamellae were more deformed in 1998, than in 1995.

It is noteworthy that the pancreatic lesions were obviously present in 1998 but not in 1995 (P. Hooper, pers. com.). This is consistent with the negative findings by Whittington *et al.* (1997).

Table 6.3 Comparison of histopathology of the gills of *Sardinops sagax* killed in the pilchard mortalities of 1995 and 1998 and examined at AAHL (P. Hooper pers. com.).

Specific sign	**Comparison with 1995**
Cellular exudate	less in 1998
Chloride cell numbers	much less in 1998
Consolidated epithelium/anastomoses	more in 1998
Leakage of protein	less in 1998
Hyperplasia	much increased in 1998
Epithelial cell detachment	probably less in 1998
Secondary lamellae distortion	much increase in 1998

It was considerably more difficult to find herpesvirus particles by electron microscopy in 1998 than in 1995 (AAHL, 1999). However, insufficient pilchards from 1998 were examined to indicate whether this was representative of all infected pilchards in 1998.

There were discrete particles associated with the affected gill epithelium of affected pilchards from 1998. These structures were not a major ultrastructural characteristic of gill epithelium derived from Australian pilchards from the 1995 epizootic (AAHL, 1999).

6.4.2 Epizootiology

The two pilchard mortality events differed in several respects:
1) the speed of the spread of mortalities;
2) the apparent virulence of the epizootics;
3) the time of year of the mortality events and;
4) the effects on juvenile pilchards.

The spread of the mortalities in 1998 was approximately half the speed (~15 km/day) of the 1995 epizootic. The path of the eastern 'wave' of the 1998/99 epizootic finished in New South Wales early in January 1999, but in WA the western 'wave' continued up the west coast to the northern limit of the population, finishing in May 1999. There is evidence to suggest that the pilchards migrate from northern NSW into southern Queensland to spawn during autumn and winter (April – August; Fletcher, 1990), then migrate back to northern NSW in summer (November – March) when the water temperature rises above optimal levels (T. Ward, pers.com.). This would account for the eastern 'wave' of the mortality event stopping in northern NSW waters, because there may have been few, if any, pilchards further north.

Although the spread of the deaths was much slower in 1998/99, higher mortality rates were observed, particularly in WA, where approximately 70% of the pilchard biomass on the south coast died (Gaughan et al., 2000) compared with an estimated 10-15% in 1995. The 1995 mortality estimate may be an underestimate, and the actual figures may be as high as 20-30% (D. Gaughan, pers.com.), but this is still substantially below the estimate for 1998/99.

In contrast, in Victoria the number of affected pilchards appears to have decreased during the 1998/99 mortality event. In 1995, affected pilchards were readily available over large stretches of coastline, but in 1998 it was very difficult to find affected pilchards for laboratory studies (M. Crane, pers.com.).

In 1998/99, juvenile pilchards were affected in SA and Victoria, but none were found during the 1995 mortality event. Another difference was the time of year of the mortality events. In 1995, the mortality occurred during March – June, and in 1998, October - May.

Disease outbreaks tend to be influenced by the interaction of three major components: (1) the pathogen, (2) the host and (3) the environment. Differences observed between the 1995 event and the 1998/99 event may be due to any number of factors related to each of these three major components. The pathogen, the herpesvirus, may have been different, i.e. different herpesviruses may have been involved in the two events or the virus in the 1998/99 event may have been a mutant of the 1995 virus. Viral evolution is continuous and variation in the genome of a virus can alter its phenotypic character, which can increase the disease potential of the virus (Murphy and Nathanson, 1994).

The age profile or health status of the host populations may have been different in the two epizootics, and this may have influenced the pattern of mortality. Finally, environmental factors (eg. water temperature, water quality) may have been different prior to or during the two epizootics. The variable aetiology of spring viraemia of carp caused by the rhabdovirus, *Rhabdovirus carpio* demonstrates how environmental

influences can affect the severity of a disease outbreak (Ahne, 1986; Crane and Eaton, 1997).

6.5 DISCUSSION

There was much less diversity of research during this epizootic because it was suspected from the outset that a herpesvirus may have been involved. The gill lesions were similar in appearance to those seen in affected pilchards in 1995. The results of the polymerase chain reaction primer work suggest that the two herpesviruses may be similar, however further sequence data is necessary to confirm this. Definitive conclusions cannot be drawn from the small amount of sequence currently available. Sequencing of the DNA from both isolates of pilchard herpesviruses is continuing.

Collaboration between the States, Commonwealth and industry, facilitated by the JPSWG, was effective in rapidly establishing a comprehensive research program. The JPSWG should be viewed as an effective model for future management of disease events.

7 DIRECT EFFECTS

Objective: To identify and assess the direct ecological and economic effects of the pilchard mortality events.

Most of the information relating to the potential ecological effects of the mortality events has been gained from discussion with researchers from the southern Australian States. Scientific papers related to the diets of pilchard predators were also read to understand the ecology of the various predators. There has been little research effort directed into the potential ecological effects of the mortality events. Papers discussing the population relationships of pilchards and anchovies were reviewed to understand the interaction of these two competitors in temperate pelagic ecosystems around the world. There is a plethora of information about some predators (eg. penguins), but paucity about others (eg New Zealand fur seals). A large number of fairy penguins died in Victoria shortly after the 1995 mortality event had passed through the State. In a paper written about this event, the researchers could not relate the penguin deaths to the pilchard mortality events. Fairy penguin numbers remain low at Phillip Island (Victoria) and their diets changed, and continue to remain changed, so that instead of the previously high quantity of pilchard (pre-1995) in the diet, pilchard now only comprises a very small component of the diet and has been replaced by other fish species. Gannets in Port Phillip Bay also changed their diet composition immediately after the pilchard mortality event of 1998, almost entirely replacing pilchard with barracouta. Australian anchovies, which are the main competitors of pilchards in South Australian waters, appear to have become more abundant since 1998. Similar fluctuations of populations of competing clupeoid fish species have been observed in other temperate pelagic ecosystems.

Research into the economic effects of the mortality events involved examining fisheries agencies files, talking to fisheries managers and fisheries scientists. A small survey of Adelaide bait retailers ascertained that there had been a negative impact on their businesses. Discussion with scientists in other States about this issue suggests that the survey reflects loss of income to bait and tackle retailers around southern Australia. The most strongly affected area is the south coast of WA, where two of the three pilchard fisheries have had their quota cut to zero for the year 2000. The industry in WA expects recovery to be slow and Fisheries WA has instigated a 'Re-Establishment Grant' to help fishers relocate or change business.

7.1 ECOLOGICAL RAMIFICATIONS

7.1.1 Introduction

Pilchards are a key component of temperate pelagic ecosystems (Bakun, 1996). They have few (in both species and numbers) comparably sized, planktivorous, pelagic counterparts and usually only one species is dominant at any particular time. In contrast, there are a very large number of species at the lower trophic levels and a substantial number of species that feed at the highest trophic levels. This creates what is known as a 'wasp-waist' ecosystem (Fig. 7.1) where the major control is neither "bottom up" nor "top down" but rather both up and down from the centre (Bakun, 1996). Thus the highly variable populations of small pelagic planktivorous fish can have major effects on the upper trophic levels, which depend upon these fish as a food source, and also on lower trophic levels, which are fed upon by the wasp-waist populations. The clupeoids that comprise this mid-trophic level in temperate pelagic

ecosystems are vital indicator species of the health of those ecosystems (Bakun, 1996).

Reductions in the abundance of dominant clupeoid species can significantly affect pelagic ecosystem structure and function, especially their competitors and predators. For example, the Black Sea once supported 26 commercial fishes in the 1960s. Most of the large predatory fish disappeared leading to an increase in small predatory fishes, so that by the 1980s only 5 commercial fish species were left. In 1982 the comb jelly *Mnemiopsis leidyi*, was accidentally introduced to the Black Sea and entered the Sea of Azov a few years later. This organism has now succeeded in nearly completely replacing the anchovies (*Engraulis encrasicolus*) in the Sea of Azov as the terminal zooplankton consumer and is also very important in the ecosystem of the entire Black Sea (Mills, 1995). Two mechanisms exist by which the ctenophore could be responsible for the decrease in anchovy stocks. The ctenophore feeds on the anchovy eggs and larvae, as well as being able to out compete them for plankton, the main food of the anchovy (Berdnikov *et al.*, 1999). However, the modeling by Berdnikov *et al.* (1999) showed that even if *M. leidyi* is absent the fish stocks still decrease, so the authors suggest that the observed collapse and the comb jelly dominance could be "a combination of highly intensive catches in 1987-1988 and unfavourable exogenous factors."

Populations of small pelagic fish species fluctuate in abundance due to several influences including competition for resources, depletion by fisheries (Batchelor and Ross, 1984), or in response to environmental changes (Southward *et al*., 1988), eg. El Niño Southern Oscillation (ENSO) (Lluch-Cota *et al*., 1997). When one kind of weather regime dominates the Pacific, one species of small clupeoid will dominate the other. When the opposite weather regime ('La Niña') dominates, the species with the previously low population will become the more abundant.

A model designed to assist the development of management principles for exploited pelagic ecosystems, predicted that a large decrease in stock size of small pelagic fish is likely to result in increased biomass of their food and competitors, and a decrease in the population sizes of their predators (Mackinson *et al*., 1997). The higher trophic levels took the longest time to recover in the model, presumably because these levels are comprised of longer-lived animals, with longer breeding cycles.

Ward *et al*. (1998) summarised the findings of many overseas studies of the effect of clupeoid fisheries on predatory animals. During development of the Peruvian anchovy fishery, seabird numbers decreased by more than an order of magnitude and following collapse of the stocks in 1972, guano bird populations fell to ~20% of their previous numbers. These and other impacts can be translated into a general context of clupeoid depletion and would occur whether loss of clupeoid biomass was man-made or due to natural impacts.

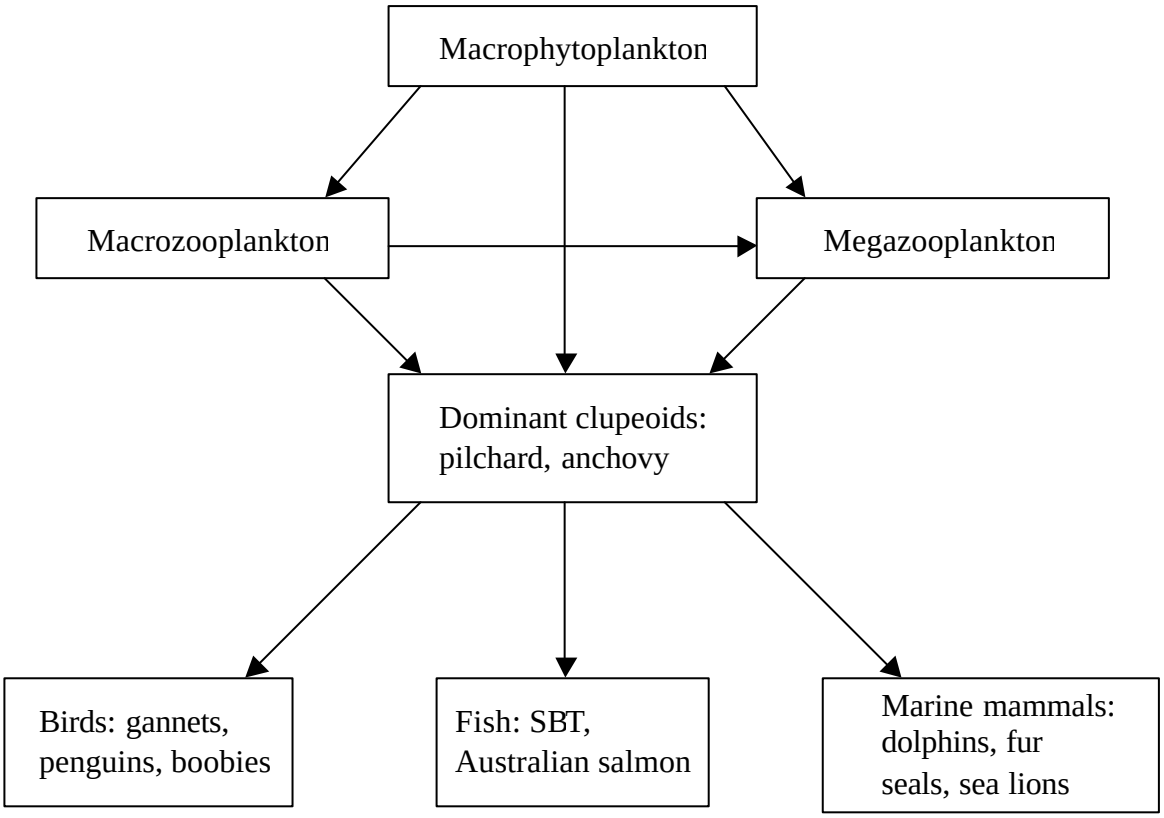

Fig. 7.1 Major pelagic feeding relationships in southern Australian temperate waters, demonstrating the 'wasp waist' ecosystem.

7.1.2 Impact on pilchard competitors

Due to a lack of competition for resources from pilchards, there may be an increase in biomass of other pelagic, planktivorous clupeoid fish (eg. Australian anchovy, *Engraulis australis*) and invertebrates. In South Africa, when the pilchard fishery collapsed, the diet of Cape Gannets became predominantly composed of anchovy instead of pilchards (Batchelor and Ross, 1984). This fluctuation of dominant clupeoids has also been observed in the pilchard and herring fisheries of south-western England over the last four hundred years and has been linked to climate change (Southward *et al*., 1988).

In Australia, a fluctuation in pilchard and anchovy abundance appears to have been induced by the massive reduction in the pilchard stock from the mortality events. Effects are likely to be different between regions. In South Australia, where there are relatively few species of planktivorous fishes, the anchovy population appears to have expanded (T. Ward *et al*., in press). There also appears to have been an increase in the anchovy stocks and catch in Port Phillip Bay, Victoria (S. McCormack, pers. com.).

In WA and States other than South Australia, there is a greater diversity of pelagic planktivorous fish that compete with pilchards. Anecdotal evidence from fishers suggests that the numbers of herring (*Arripis georgianus*), maray and anchovies may well have increased in WA (D. Gaughan, pers.com.). However, recent research on herring in WA indicates that the recently observed high abundance (1996 year class) was linked to an environmentally determined pre-recruit index as far away as eastern SA (Ayvazian *et al*., 2000).

7.1.3 Impact on pilchard predators

Pilchards are eaten by many species (see Table 7.1) and they occupy a pivotal role of energy transfer by turning photosynthetic energy from phytoplankton into abundant, high-calorie food.

Table 7.1 Some of the common large vertebrate predators of pilchards in Australia.

Western Australian salmon	*Arripis truttacea*
Southern Bluefin Tuna (SBT)	*Thunnus maccoyi*
Sharks	Elasmobranchii
Australasian gannets	*Morus serrator*
Shearwaters	*Puffinus* spp.
Cormorants	*Phalacrocorax* spp.
Little penguins	*Eudyptula minor*
Australian fur seals	*Arctocephalus pusillus doriferus*
New Zealand fur seals	*Arctocephalus forsteri*
Australian sea lions	*Neophoca cinerea*
Bottlenose dolphins	*Tursiops truncatus*
Common dolphins	*Delphinus delphis*

Little is known about the diets of many of the large predators, however most appear to have varied diets, and flexibility in their prey selection. These predators (eg. gannets) can substitute pilchards with other species (Bunce and Norman, 2000). Pilchards however, are a particularly high-energy food source that may be difficult to replace unless the predators take a higher quantity of lesser quality prey, for which they may have to expend greater foraging effort (Charnov, 1976). This greater effort may negatively impact on growth rates, breeding success, mortality rates and population numbers of the predators (Cairns, 1987).

Concern was expressed that the herpesvirus or other pathogens carried by the pilchards may harm predators that ate the moribund and dead fish. Pilchards weakened by an initial pathogen (probably the herpesvirus) may have been more susceptible to infection by other pathogens and parasites. Consuming any pilchard pathogens may affect the health of animals eating the dead pilchards as well as their breeding success and mortality rates.

Pathogenic fish viruses can be transmitted by herons (Peters and Neukirch, 1986), so it was not unreasonable to suppose that seabirds consuming the pilchards may well act as mechanical vectors of the virus. However, modeling work by Murray *et al*. (2000) indicates that the involvement of a vector is unlikely.

Large quantities of moribund and dead pilchards provided readily available food. Anecdotal evidence reports extremely well fed seabirds, and lower catch rates of rock lobster (J. Prescott, pers.com.) and sand crab (Westlake and Jones, 1999) presumably because so much food was available outside the pots. These effects can be significant. After the 1954-1956 fungal disease outbreak in herring in the Gulf of Saint Lawrence, cod fed on moribund herring to such an extent that their growth rate during the epizootic period exceeded anything previously recorded. Cod landings in the southern Gulf of Saint Lawrence almost doubled during the immediate post-epizootic period,

due almost entirely to increased weight of individual fish caught, rather than to increased numbers of fish taken (Sindermann, 1990).

7.1.3.1 Fish

No published data are available and no studies were undertaken to examine the effect of the pilchard mortalities on commercially important species such as SBT (*Thunnus maccoyi*) and western Australian salmon (*Arripis truttacea*). Food webs in southern Australian waters are often simple and include only a few species of small planktivorous fish, so although piscivorous fish may be able to utilise a wide range of prey species, the opportunities to switch prey may be limited (Ward *et al.*, 1998).

Pilchard stocks are highly significant for juvenile (2-4 years old, J. Gunn, pers.com.) SBT feeding in waters of Western Australia and South Australia, where they consume large quantities of clupeoid fish (Serventy, 1956; Young *et al.*, 1997). Research on northern bluefin tuna (*Thunnus thynnus*) suggests that its local abundance may be positively correlated with local abundance of *S. sagax* (Ward *et al.*, 1998).

Massive fluctuations in clupeoid abundance have been recorded for hundreds of years (Southward *et al.*, 1988). The predators that rely on these abundant clupeoid fish have had millions of years to adapt to their prey's yearly fluctuations and seek alternative food sources, so perhaps it is not unexpected that there has been no noticeable negative impact on SBT stocks .

7.1.3.2 Penguins

Western Australia

There is no monitoring of little penguin (*Eudyptula minor*) numbers in WA. A population on Penguin Island is studied but no number counts are taken. In 1995 during the pilchard mortality event, the penguins were so gorged with pilchards that researchers had difficulty emptying the stomach contents, which are normally easily taken by turning the birds upside down (R. Wooler, pers.com). There are no data available for breeding success or adult weights.

South Australia

Data collected in SA suggests that there was no measurable impact on the little penguin population of SA. There are several colonies of little penguins in SA, but monitoring in 1995 and 1998 was only conducted on four colonies, two on Kangaroo Island and two near Victor Harbor, on Granite and West Islands.

The population of Kangaroo Island penguins has been monitored continuously for 6-7 years. The Kingscote population showed a 14% decrease in average total numbers in both 1996 and 1999. The Penneshaw population showed slight increases in numbers in the years after the mortalities and this would indicate that the decrease in the Kingscote colony is probably not due to a lack of pilchard biomass (Rowley, 2000, unpublished data).

The data from the Victor Harbor area are more comprehensive but interpretation is hampered because some data were not recorded until the middle of the breeding season. Adult penguin weights vary between 0.9 kg to 1.3 kg during the breeding season. The average weights of the Granite Island colony, August to January 1996/97, were 0.905 kg to 1.75 kg, which showed no significant decrease in average weight.

Breeding data (no. clutches, no. eggs, no. chicks, etc.) were also recorded in 1990 and 1991. The latter year was a poor year for the penguins and both 1995 and 1996 were more successful.

West Island does not have the human disturbance levels found on Granite Island which is a popular tourist destination. The breeding success of the silver gulls and the subsequent increase in the predation of penguin eggs and chicks appear to have had some impact on the viability of egg hatching and fledgling success. The silver gulls have a more aggressive nesting behaviour that also impacts on the availability of nesting habitat for the penguins. The average adult weight range for this colony was 0.85 kg to 1.52 kg, slightly less than the Granite Island population, but not significantly different from the average. It was not possible to compare breeding data for this colony because reliable baseline data was not available (Geschmay *et al*., 1998, unpublished.)

After the 1995 pilchard mortality, there were greater than normal deaths of adult penguins. This may have been due to an unusually high mortality of yearling penguins, which are generally the most susceptible to environmental change (C. Halstead, pers. com.).

Victoria

Of all the penguins in Australia, those of Phillip Island and Bass Strait have been most intensively studied. Breeding success of little penguins in southeastern Australian has been related to annual variations in clupeoid abundance (Cullen *et al.*, 1992). A paper by Dann *et al.* (2000) covers in detail the close monitoring of the penguins coming ashore at Phillip Island, during and after the pilchard mortality event. This report is extremely comprehensive and covers all aspects of the consequences. Not only was there a significant decrease in penguins coming ashore during winter 1995, but also a large number (855) of predominantly adult birds washed ashore at Discovery Bay, between June and September 1995. Of 29 that were autopsied, 26 died of starvation associated with mild to severe gastro-intestinal parasitism. Other measurable effects were that the breeding season was delayed by approximately 2 weeks and that breeding success was exceedingly poor at 0.3 chicks fledged per pair, substantially less than the long-term mean of 1.0 ± 0.4 chicks fledged per pair. The authors noted that only pilchards >10cm died, but fish of this size are not usually taken by little penguins. Most penguin prey items are <13cm long, so there was only a small portion of the cohort of pilchards that some of the adult birds may have fed on.

Stomach samples taken between September and December, 1995, showed that returning adults were taking a large range of prey that excluded pilchards, and again in the 1996-97 breeding season, the presence of pilchards was markedly reduced compared with previous studies (Montague and Cullen, 1988; Cullen *et al.*, 1992).

Dann *et al*. (2000) conclude that "the coincidence of the decline in numbers of penguins at Phillip Island with the onset of the pilchard mortality "front" and the subsequent unseasonal and substantial mortality of adult penguins followed by a late and significantly unproductive breeding [season] and the marked increase in first-year mortality suggests that several population parameters were closely linked to the pilchard mortality". They are unable to make a more defined link between the pilchard deaths and these penguin population parameters.

Analysis at Phillip Island after the 1998/99 pilchard mortality event has shown no obvious mortalities or breeding failures during or immediately post-mortality. Number counts have shown that, on average, the number of penguins coming ashore at night have continued to be low since the 1995 event. Penguin diets also continue to contain low amounts of pilchard since 1995. Pre-1995 reports show that pilchards comprised 22-33% of the dietary intake (Montague and Cullen, 1988; Cullen *et al*., 1992) but recent (1998) stomach content analysis at Phillip Island has shown that pilchard intake is substantially reduced, now only comprising 2-9%. Wahou (*Seriolella brama*) has now largely replaced pilchard, comprising 37% of the diet, with red cod (*Pseudophycis bachus*) and barracouta (*Thyrsites atun*) also making up significant portions of the diet (P. Dann, pers. com.).

Given the lack of evidence suggesting impacts on other penguin colonies, it is possible that the Phillip Island penguins were affected a number of factors in addition to the lack of pilchard biomass.

Tasmania
Although there is a substantial penguin population on Tasmania's hundred or so islands, there is no regular monitoring. Shortly after the pilchard mortalities, in June-July 1995, the Baron oil spill devastated a local colony. However, there is no available data for any of the colonies (Marine Unit, Parks & Wildlife, pers.com.).

New Zealand
Large numbers of little penguins were washed up on Ninety-Mile Beach, North Island, in mid-August 1995, about a month after the pilchard mortality events. It was suggested at that time that these birds died of starvation, possibly linked to the loss of pilchards, however, no formal study was undertaken and none of those birds was autopsied (Smith, 1995).

7.1.3.3 Other seabirds
The only seabirds, other than penguins, that have been studied with direct relation to the pilchard mortalities are Australian gannets (*Morus serrator*, Bunce and Norman, 2000). Prior to the mortalities of 1995 and 1998/99, pilchards comprised approximately 50% of the diet of gannets breeding in Port Phillip Bay, Victoria. However, in 1999, the proportion of pilchards declined to only 5% of the diet, with other species forming a major component. The authors state that the effect of the mortalities on gannets was "minimised by foraging flexibility in gannets as evidenced both in size of prey consumed and range of species taken … pilchards were replaced with a variety of species, particularly barracouta".

The authors also discuss the consequences of a lower quality diet, stating that greater foraging effort and food consumption is required which may "ultimately affect the reproductive success and survival of gannets". The authors highlight a significant mortality of Australasian gannets, the largest on record, occurring between August and November 1995 in New Zealand, immediately after the mortality had ceased there.

Anecdotal evidence from WA suggests that flesh-footed shearwaters (*Puffinus carneipes*) had a "difficult" time in 1999. These birds are normally present behind

pilchard fishing boats, feeding off the catch, but in 1999 fishermen reported that there were no shearwaters at all (R. Wooler, pers. com.). Several reasons could be postulated for this observation, however, the coincidence in timing with the pilchard mortality indicates there may have been a relationship.

Numerous species of seabird including gulls, terns, boobies, gannets and shearwaters have been observed taking pilchards from catches in Australia. Lack of pilchards may impact on breeding success and juvenile learning patterns. Field observations suggest that feeding preferences of most seabirds are more related to prey size than prey type (Ward *et al*., 1998). Small birds such as terns and petrels feed on a range of juvenile clupeoids, while larger birds such as gannets and albatross feed on adult pilchard and herring. Thus it is possible that these larger species of seabird may have been significantly impacted, probably necessitating a change in diet, as with the gannets in Port Phillip Bay (Bunce and Norman, 2000).

Studies have identified a relationship between fluctuations in marine food supplies and seabird populations. Poor to moderate availability of food can reduce adult body weight, clutch size, breeding success, growth rates of chicks, colony attendance and guano production (Cairns, 1987). For example, many populations of Peruvian guano birds did not breed after the collapse of anchovy stocks and seabird numbers decreased by more than an order of magnitude during development of the Peruvian anchovy fishery (Ward *et al.*, 1998). Reductions in the abundance of anchovies off the west coast of North America were correlated with reduced breeding success of brown pelicans (Anderson *et al*., 1980).

7.1.3.4 Pinnipeds

Numbers of the Australian sea lion (*Neophoca cinerea*) have declined drastically since European colonisation and they are now listed as Rare under the IUCN's Red List of Threatened Animals (Shaughnessy, 1999). In South Australia, where 70% of the population now resides, National Parks & Wildlife was commissioned to undertake pup counts of Australian sea lions at Dangerous Reef and the Pages, SA, between January – August 1999. Any pups that died were autopsied but there was no evidence of a lack of food (R. Allen, per.com.). No connection could be made between these deaths and the pilchard mortalities.

The pupping events of 1996 and 1999 at Dangerous Reef and the Pages showed relatively high pup mortalities but they could not be linked to the pilchard deaths (P. Shaughnessy, pers.com.). The pupping rate of this species has been in decline for a decade, in both SA and WA (N. Gales, per.com.), and recent declinescould not be confidently attributed to decreased pilchard abundance because the decline was noticed well before the pilchard mortalities.

New Zealand fur seals (*Arctocephalus forsteri*) inhabit waters in both Western Australia and South Australia. Although no formal study was conducted, large amounts of pilchard were seen in NZ fur seal vomit (N. Gales, per.com.). Vomiting is a natural reaction of this species to overfeeding. This was highly unusual; the only other prey item to be seen in vomit previously was squid. However, little is known about other populations of NZ fur seals in relation to the pilchard deaths.

The Australian fur seal (*Arctocephalus pusillus doriferus*) has been little studied. The major breeding colonies are all in Victoria. It is primarily a benthic feeder and as such, pilchards constitute only a small part of its diet. Even when there were dead pilchards on the seafloor, it is considered highly unlikely that this species would prey on dead and moribund fish. Although diet composition is well known, little is known of this animal's feeding habits despite several studies. (J. Arnould, pers.com.)

Pilchards are part of the diet of two of the three pinniped species permanently living around the coast of Australia. However, although pilchards are often in the top 10 fish species taken, pinnipeds do not appear to be dependent on pilchards as a major diet component and it is suggested that they are flexible in their dietary choice (Gales and Cheal, 1992; Gales and Pemberton, 1994).

7.1.3.5 Dolphins

Nothing has been reported about the impact on the several dolphin species that inhabit southern Australian waters. The SA Museum has regularly autopsied dead, washed up dolphins for many years, and although pilchards have been found in their stomachs, there is no evidence to suggest that any of the deaths could be linked to the pilchard deaths. No other States have examined dolphins in relation to the pilchard mortality.

7.2 ECONOMIC EFFECTS

7.2.1 Introduction

The economic impacts of the two mortality events not only affect the pilchard fisheries, the fishers and their livelihoods, but also the processors, retailers and other flow-on businesses upon which they rely to provide goods and services. These impacts varied between regions where there are differences in pilchard production. In the period 1995-98 WA and SA produced the largest quantities of pilchards in Australia. Victoria and NSW both fished pilchards but only in small-scale, unmanaged fisheries. The 1995 mortality event was estimated to have done around $12 million damage to the entire Australian pilchard fishery over 3 years (Daszak *et al*., 2000).

Apart from these immediate impacts it must be considered that there may now be a latent infection established in the Australian pilchard population. This may have future consequences and introduces an element of uncertainty into pilchard fisheries.

7.2.2 Fisheries

There are three main economic effects of a mass epizootic of commercially important finfish (Sindermann, 1990), which are relevant to fisheries and the markets they supply. Each of these will be examined in relation to the pilchard mortalities:

A. Reduction in numbers of food fish available to the fishery;
B. Effects on other species, positive (eg. increased growth rate and weight) as well as negative;
C. Rejection by consumers, often combined with subsequent loss of interest in fish as food.

A. Reduction in numbers of food fish available to the fishery

7.2.2.1 Western Australia

The reduction in pilchard biomass has had significant negative economic effects on the Western Australian fisheries. The 1999-2000 quota was originally set as in Table 7.2. When the full impact of the mortality was estimated (~60% biomass) the quota was immediately reduced. In 1999, the annual catch amounted to only 730 t the expected catch was 4000-5000 t. The 2000-2001 quota was drastically reduced.

Due to a number of factors, including the pilchard mortalities, a number of fishers from the south coast area have either relocated or gone out of business. A "Re-Establishment Grant" has been approved this year (2000) specifically for those fishers who have been hardest hit in the south coast purse seine fishery. It is estimated that the economic impact in the southern region is a loss of $12 million to $15 million, including loss of income for fishers and loss of business income for flow-on goods and service providers.

Table 7.2 Quota for south coast pilchard fishery, WA, for 2000. (Figures supplied by Fisheries WA.)

Zone	1999-2000 quota (tonnes)	2000-2001 quota (tonnes)
Albany	1,500	0
Bremer Bay	1,900	0
Esperance	1,800	1060

The small west coast fishery became a managed fishery in 1989. At that time, an annual catch of approximately 400 tonnes of pilchards was recorded. Since then, catches increased to the extent that the annual catch of pilchards peaked at almost 4000 tonnes in 1996. Beyond that point however, catches decreased sharply and during the 2000-2001 licensing period, fishermen on the west coast are permitted to take no more than 260 tonnes of pilchards.

It is estimated that approximately 5100 tonnes of pilchards died on the West Coast as a result of the 1999 epizootic (D. Gaughan, pers. com.). This is significant when considered in conjunction with the estimated spawning biomass of 5,275 tonnes.

7.2.2.2 South Australia

In South Australia the pilchard quota was reduced from 1999 because there was substantially less stock as evidenced by the spawning biomass estimate(see Table 7.3). The spawning biomass figures for 2000 and 2001 were 91,000 tonnes and 142,000 tonnes respectively. Pilchard stocks appear to be capable of almost tripling in one year (1996-1997). Clupeoid populations are somewhat resilient to population reductions (Ward *et al.*, 1998), but preliminary investigations indicate that the rate of recovery of the South Australian pilchard population after the 1998/99 mortality is somewhat slower than after the 1995 mortality.

Table 7.3 Spawning biomass, Total Allowable Catch and estimated catch for the South Australian pilchard fishery. * Experimental years of the fishery. (Figures supplied by SARDI.)

Year of estimation	Spawning biomass estimate (tonnes)	TAC (tonnes)	Estimated catch (tonnes)
1995*	59,000	3,500	2,597
1996*	18,000	3,500	3,531
1997*	59,000	3,500	3,500
1998	95,000	11,500	7,312
1999	38,000	6,000	4,080
2000	91,000	3,800	3,290
2001	142,000	9,100	6,600

To maximise the returns from the resource, pilchard fishers in SA have begun value adding their product to sell to more lucrative markets such as the human consumption market and the recreational fishing bait industry (B. Loiterton, pers.com.). Anchovies are also caught by SA pilchard fishers as part of their pilchard quota (B. Loiterton, pers.com.) and anchovy numbers appear to be increasing following the mortality events.

7.2.2.3 Victoria

The majority of pilchards in Victoria are caught in Port Phillip Bay. There are 52 licensed fishers (decreased from 112 last year) who are permitted to use a purse seine net not exceeding 460 metres in length. The fishery is too small to justify sophisticated management, so there is no quota or stock assessment process. Prior to 1996/97, average annual landings from all Victorian waters were in the order of 2,500 tonnes with the bulk of the catch coming from Port Phillip Bay. Under reporting of the catch from the bay was significant and may well have been by more than 50%. Catches are currently (year 2000) about 500 to 700 tonnes a year and, although this year (2000) is apparently the best for several years, the stocks show little sign of improving.

Fishers in the bay are dependent on pilchards moving in from the ocean. This now happens less frequently and may be due to a reduction in pilchard stock as a result of the mortality events. Gaughan *et al*. (2000) suggest that unusually high water temperatures coincident with La Niña conditions may have "caused shifts in distribution of pilchards away from warm water" and into cooler offshore waters, possibly contributing to the lack of pilchards in the Albany and Bremer Bay areas of WA. Whether this is the case for Victoria is not known.

Most of the Victorian pilchard catch was sold for pet food, or to the tuna farming industry, but most of the small catch is now going to the fresh fish market in Melbourne, where they are sought after and attract an improving price (~$3 per kg). Fishers are pessimistic about the fishery and only one or two operators actively target pilchards. Others have diversified or handed in their licences in a buy-out last year. If the catches improve then Fisheries Victoria will have to examine controls on the fishery, but this is unlikely to be an issue in the next 2 years. (S. McCormack, pers. com.)

7.2.2.4 New South Wales

The NSW pilchard catch has always been only a few hundred tonnes per year and hence is not a managed fishery. In 1995 catches were at about 50 % of their normal level four months after the event had passed through the state and in 1998 the fishery was closed for approximately one month around the time the mortalities arrived in NSW (January 1999). Catches remain at a few hundred tonnes (R. Fletcher, pers.com.), so it would appear that there has been no long-term effect.

B. Effects on other species, positive as well as negative

This issue of potential impacts on pilchard predators and competitors was largely examined in the previous section. Some commercially valuable species such as rock lobster may have benefited from large amounts of dead pilchards. Lobster and sand crab fishers reported that catch rates decreased, probably because the lobsters and crabs were feeding on dead pilchards outside the pots.

C. Rejection by consumers, often combined with subsequent loss of interest in fish as food

In WA, there is a cannery for pilchards for human consumption. This cannery would have stopped canning due to the voluntary closure of the fishery in the south except that frozen stock was available and this allowed production to continue (R. Stevens, pers.com.). The majority of pilchards in Australia are used for tuna feed, pet food and fishing bait for both recreational and commercial fisheries, with a small amount going to the fresh fish market. The pet food industry in affected States was aware of the situation in both cases, and relied on imported pilchards, for the duration of the mortality events.

7.2.3 Retailers

Through research for this report it became evident that several bait retailers in the Adelaide area experienced loss of income and business due to a lack of pilchard supply. There was also a general decrease in recreational fishing due to concern amongst fishers that other fish species may be infected. All the replies stated that pilchards were unavailable during or after the pilchard mortalities. One reply stated that customers were concerned that other species were contaminated and not edible, so both boat and beach recreational fishing decreased. Another reply stated that their business suffered a decrease in their annual sales because pilchards comprise a large portion of the sales.

Interviews with the Adelaide public during the 1995 mortality event indicated that pilchards washed up onto beaches were collected and frozen for personal bait usage (G.K. Jones, pers.com.). This reduced the need for purchase of bait from tackle dealers. However, the extent of this occurrence is not known.

Bait suppliers in northern New South Wales and southern Queensland were similarly affected by loss of supply and income (J. Staunton-Smith, pers. com.).

7.2.4 Economic impact of disease introduction

Because of the pilchard mortality events, the stock was reduced by between 10-70% and this severely impacted on the economic viability of the fisheries. In wild fish stocks, the economic impact of a disease will only be felt if there is a significant

change in population abundance because of an epizootic (Jones and Gibson, 1997). Exotic pathogens in the marine environment do not impact only on the affected species. Flow-on effects in the ecosystem impact both positively and negatively on the affected species' predators, competitors and other opportunistic species, any of which could be commercially valuable, eg. rock lobster, SBT.

7.2.5 Discussion

Due to the lack of specific studies, it is difficult to assess the overall impact of the mortalities on the marine ecosystem. Several papers have documented clupeoid population fluctuations (Kawasaki and Kumagai, 1984; Southward *et al*., 1988; Lluch-Cota *et al*., 1997; Batchelor and Ross, 1984), either due to climate change or overfishing, or a combination of both.

The lack of penguins returning to shore at night and the unsuccessful breeding season at Phillip Island, reported by Dann *et al*. (2000) may be associated with the pilchard mortality event of 1995, however only the coincidental timing links the two events for this period.

It seems that opportunistic predators that feed on moribund and dead fish like New Zealand fur seals and rock lobsters may have benefited from the extra food, but others, eg little penguins, may have had a temporarily depleted food source. Some predators, eg gannets, temporarily switched prey (Bunce and Norman, 2000) and the diets of little penguins still have not returned to contain the high portion of pilchard seen before 1995 (P. Dann, pers.com.).

Some effects may not be seen for several years, due to "lag effects" in biological systems (Smith, 1995), as the impacts slowly filter through the marine food webs and through future generations. Some of these animals are particularly long-lived and any effects may not be seen in these populations until some time in the future. Pinnipeds, for example, breed at intervals of up to 18 months, so any change in diet composition possibly affecting the quality or quantity of the breeding adults may not be seen until the next breeding seasons. Any reductions in food availability are also likely to impact on the pre-breeding mortality of juvenile seabirds. Most seabirds do not breed until they are several years of age, so a delayed response in the decline of seabird numbers at breeding colonies is possible (A. Bunce, pers.com.).

Ward *et al.* (1998) state that the need for ecological research is most pressing in South Australia, where there are fewer species of clupeoid fish than any other State (T. Ward, pers.com.). South Australia also supports Australia's largest pilchard fishery as well as globally significant populations of Australian sea lions, little penguins and SBT. Increased monitoring of populations of pilchard predators that reproduce on land and are long-lived, eg pinnipeds and seabirds, could provide valuable insights into ecosystem function and the effects of natural or man-made fluctuations in clupeoid abundance.

The negative economic impacts of the mortality events are most readily seen in the pilchard fisheries through loss of quota and short-term closure. The closed pilchard fisheries on the south coast of WA have been severely affected with some fishers having to relocate or re-establish a different business. Bait retailers also suffered lost income through a decrease in recreational fishing.

Control or eradication of exotic infectious agents in the marine environment is not possible (Jones and Gibson, 1997). To avoid further stock depletion and economic hardship to the fishers who depend on the fisheries due to possible future outbreaks, the Australian pilchard stocks must be managed conservatively.

8 LESSONS LEARNED

Objective: To describe and evaluate procedures for co-ordinating research and management of the 1995 and 1998 pilchard mortality events, and identify and assess options for improving responses to a possible future pilchard mortality event.

This objective was achieved by examining the files of agencies involved in the responses to the mortality events and interviewing personnel involved in the investigations.

In retrospect, the response to the 1995 mortality event appears poor. However, as the event was unprecedented, its seriousness could not be determined until it was over. The *ad hoc* response that was eventually conducted utilised the Consultative Committee on Emergency Animal Diseases (CCEAD), which consists of State Chief Veterinary Officers. It also brought together fisheries managers and scientists from numerous agencies with a potential interest in the implications of the event. Communication within and outside the management and research agencies was poor, and speculation was rife in the media. Coordination of the response to the 1998 mortality event was much improved, partially because of the involvement of many personnel that participated in 1995, and partly because of the experience that the fisheries agencies had gained in participating in the CCEAD mechanism. There was less involvement from some States and agencies and less research on some issues. For example, environmental factors were given only scant attention as it was suspected from the outset that a herpesvirus was involved again. Communication was also improved in 1998/99 due to familiarity with the CCEAD mechanism and an early agreement about media relations. In the future, marine mass mortalities will be managed and coordinated using the CCEAD mechanism and will incorporate Fisheries Directors from relevant States.

In 1998 AQUAPLAN was launched. The Fish Health Management Committee oversees implementation of this joint industry/government strategy. Parts of AQUAPLAN include initiating emergency management plans, aquatic animal health awareness, international linkages and other initiatives to support Australia's increasingly valuable fisheries and aquaculture industries.

8.1 RESPONSE TO THE 1995 PILCHARD MORTALITY EVENT

The mass pilchard mortality of 1995 was totally unprecedented, not only in Australia, but also in the world. The Standing Committee on Agriculture and Resource Management Incursion Management report (1996) commented that overall the lack of a national aquatic animal disease management plan led to "significant problems as contact points were not easily identifiable and communications with media organisations could not be controlled". The report also commented that on an operational level, resources both nationally and within States were stretched. The lack of trained aquatic animal health personnel is reiterated in various reports dealing with aquatic animal health management and is seen as a severe constraint in all States' ability to react to an aquatic animal health emergency (Humphrey, 1995; Crane and Rawlin, 1996; Jones, 1996; Nairn *et al.*, 1996; SCARM, 1996). At the time, the overall response appeared to be poor, however - in retrospect - it was only when the event had progressed significantly that it was seen as a national emergency and a response was formulated. Response personnel were always in the position of 'playing catch-up' once the significance of the 1995 epizootic was realised.

The lack of an event of such proportions anywhere in the world makes an analysis of the management extremely difficult, as there are no guidelines or regulations for comparison. Hence, comparison with terrestrial animal disease emergency management provides a guide for analysis of the management of the mortality event.

In a terrestrial animal disease emergency, there are four main areas of management:

- **Coordination**: research, testing, disease experts, decision-making, management, eg how many samples, where from. These people decide what 'Operations' personnel carry out.
- **Operations** (field officers):
  - Investigations: eg. sample collection, surveys.
  - Infected premises: eg. slaughter, disinfection, and disposal.
- **Resources and logistics**: administration, finance, legislation, liaison with other agencies eg. SES, police.
- **Media/PR**: defined contact points, control of press releases.

Analysis of the management of the 1995 mortality event will be divided into two sections. Firstly, management will cover coordination, operations, and resources and logistics. Secondly, communication will cover both internal, using the Consultative Committee for Emergency Animal Diseases (CCEAD) network and interactions between organisations, and external communications such as media releases, public relations and reports. There is also a brief overview of the outbreak in New Zealand.

8.1.1 Management

8.1.1.1 Coordination of the response

National approach

At the time of the 1995 outbreak (March), the only mechanism with a structure equipped to deal with a national animal health emergency was the CCEAD, which was established in 1941. Until 1995 CCEAD had only dealt with terrestrial livestock disease emergencies. The CCEAD was not activated until 8 weeks after the first outbreaks in South Australia. At this time, the outbreak had spread to Victoria, WA, NSW and Tasmania and had developed into a national emergency.

One of the basic tenets of terrestrial animal disease emergency management is to use the precautionary principle: activate every emergency mechanism and alert all agencies at the start, then downgrade the response if the emergency does not warrant so much attention. It is more desirable to 'over-report' than 'under-report'. Minor intrastate emergencies can have national implications (eg. trade issues), especially if the cause is exotic, so an immediate and urgent response is necessary. In retrospect it would seem that this principle was not applied in the first epizootic. However, as an emergency of this nature had never been experienced before, it was impossible to predict that the pilchard deaths would spread so far so rapidly.

The CCEAD, convened under the then Animal Health Secretariat (AHS), is the communication network of State and Territory Chief Veterinary Officers. This being the first aquatic emergency that the CCEAD had ever managed, it was decided that specialists were needed, so numerous agency representatives were invited to participate (Table 8.1)

Table 8.1 Participants of the initial 1995 CCEAD.

State Chief Veterinary Officers
State Fisheries Directors
Fish health experts
Fisheries scientists
Oceanographers
Public health officers
CSIRO/AAHL
Fisheries Policy Branch (then DPIE)
Bureau of Resource Sciences (then DPIE)
Livestock and Pastoral Division (then DPIE)
Australian Quarantine and Inspection Service

Typically, many of these personnel had no prior experience with animal health emergencies, therefore there were difficulties in using an unfamiliar mechanism and in identifying and involving all relevant agencies. On the other hand, personnel experienced with animal health emergencies did not have experience with aquatic animal diseases. Convening such a multidisciplinary meeting was an *ad hoc* arrangement and had not been practiced in relation to an aquatic incident. This led to inevitable delays and initial communication was difficult. Once established, however, communication was facilitated by the use of the CCEAD mechanism.

Continuity of meetings

There was no sense of ownership or accountability in the meetings held during the response to the 1995 mortality event. The difficulty in identifying the relevant agencies is reflected by the large number of agency representatives at some of the early meetings; this made the meetings difficult and lengthy.

During the series of management meetings, several issues were raised that were not necessarily progressed at subsequent meetings. At the first meeting (see Table 8.2) the issue of the disease being introduced through imported baitfish was raised, but not further discussed. The possibility of the disease being associated with environmental stress was also mentioned.

Table 8.2 Dates of meetings and teleconferences.

	Date	**Group**
1.	May 10	CCEAD
2.	May 16	CCEAD
3.	May 19	Fisheries experts, VIAS
4.	May 26	CCEAD
5.	June 1	Coordinator meeting in Melbourne
6.	June 8	CCEAD
7.	June 15	Coordinators
8.	July 3	Task Force
9.	July 14	CCEAD

It was stated that public health interests needed to be a “matter of focus” in the investigations, although at that stage, two months after the first reports, no State had yet conducted toxicity testing. The risk to public health should have been identified

earlier and testing for Amnesiac Shellfish Poisoning, Paralytic Shellfish Poisoning and Neurolytic Shellfish Poisoning undertaken before May. Victoria undertook these investigations.

The Fisheries Policy Branch would liaise with industry and prepare a position paper. Importantly, the need was expressed to get industry involved at an early stage. However, it was stated that the tuna industry needed to develop its own media response to the mortality events.

The need to assess the impact of the outbreak on the viability of the pilchard fisheries was expressed but not progressed. It was suggested that an assessment of the effect of the mortalities on fish stocks and industry should be conducted expeditiously, but this was not done. States that had valuable pilchard fisheries (SA and WA) undertook their annual biomass surveys as usual to determine quotas for the next year, but no detailed analysis of the economic implications was undertaken.

Diagnostic approach

On 16th May, nearly nine weeks after the mortalities were first reported, it was decided that four coordinating groups should be established to assist with laboratory coordination of the investigation. These were designated as indicated in Table 8.3.

Table 8.3 Agency responsibilities for coordinating research in 1995

Agency	**Research responsibility**
Victorian Institute of Animal Science	pathology/toxicology
CSIRO Marine Research, Hobart	oceanography/planktonology
Australian Animal Health Laboratory	virology and electron microscopy
Australian Quarantine and Inspection Service	public health and food safety

The role of each of these coordinators was to both coordinate research and to provide advice to all interested parties on their areas of responsibility. Thus, research activities in response to the mortality event were coordinated and the various laboratories carried out important activities in a cooperative manner. In addition, individual laboratories undertook other investigations, using local expertise available at the laboratories, in attempts to ensure that all options were covered. Unfortunately, while every effort was made to submit appropriate samples to the laboratories, many of the fish samples were unsuitable for laboratory investigation. Due to the remoteness of some locations from where affected pilchards were collected and a lack of resources to support these efforts, some samples had deteriorated and could not be examined.

Duplication of research was important for independent verification of results. There were at least three experienced laboratories working on cell culture, each using different systems. The fact that none of these laboratories could culture the herpesvirus is an indication that methodological or human errors are not an issue. There was much communication between all the laboratories undertaking diagnostic research in a mass effort to find a cause for the mortality event.

There seemed to be confusion in some agencies over the correct procedure for submission of samples to the AAHL so that some samples were “lost in the system” and went to the wrong department or staff member. Appropriate labeling procedures should have been supplied by AAHL and disseminated by CCEAD.

In mid-May it was suggested that a consolidated report of the progress of the various research groups was required.

Further surveillance
At a third CCEAD meeting on 8th June 1985, when the 1995 mortality event had nearly finished, SA and Tasmania both reported some "unexplained" pilchard deaths. On 4th June 1985, new pilchard mortalities were reported from Coffin Bay, SA. Samples were collected and sent to WA and the South Australian Research and Development Institute (SARDI). Scientists at SARDI confirmed that the affected fish were juvenile pilchards and gross examination revealed no significant gill damage. Tasmania had a report of a small amount of pilchards washed up on Maria Island on 29th May 1995. The report, from a member of the public, stated that there were about 70 fish. The person making the report collected 4 fish for laboratory submission. No further dead fish were seen during an aerial survey of the area that day or during an extensive investigation of the shore the next day. Large flocks of gannets were seen in the area, so it is thought that they probably consumed the fish. The samples collected were very autolysed and findings suggested focal lesions in the gills with focal amoeba aggregates. It is considered unlikely that either of these events is linked to the main epizootic.

8.1.1.2 Operational aspects of the response
On March 10th 1995 the first reports of discoloured water in Coffin Bay, SA and along the west coast along with reports of dead cockles in Coffin Bay were received. SARDI staff from Port Lincoln, SA were contacted to initiate investigations into this incident. Water sampling confirmed the presence of a bloom of the algae *Gymnodinium mikimotoi*. Other animals that died included black-lipped abalone, stingrays and multiple fish species, excluding pilchards. There was a period of approximately one week when this bloom was still occurring near Coffin Bay and the time when pilchards started dying along the western Eyre Peninsula, SA (around the 15th March). This event in Coffin Bay appears to be unrelated to the start of the pilchard mortality (Griffin *et al*., 1997).

The pilchard mortalities began over five years ago and inadequate records have lead to uncertainty concerning the location and timing of the first specimens collected. To obtain fresh specimens, collections needed to be made at sea where the fish were dying, not from beaches, where specimens were autolysed. The *RV Ngerin*, a South Australian government research vessel, had completed a pilchard egg survey in the area of, and only 5 days prior to, the first report of pilchards dying in the west coast waters off the Eyre Peninsula. During the next research cruise by this vessel in the same area, during April 4th –9th, the scientists were directed to make observations and collect any samples of dead pilchards. Dead pilchards were observed on the bottom at depths of 10-15 metres during diving surveys. Some of these were collected, however, when the samples arrived at the then VETLAB (now Veterinary Pathology Services) at the beginning of April, the pilchards were too decomposed for autopsy. Water samples were also taken and passed onto PIRSA to arrange examination.

On April 20th 1995 more samples were analysed by VETLAB, who were unable to determine the cause of death, although it was noted that the gills were severely deteriorated. A SARDI inspection also failed to provide a diagnosis. Two days later

further samples were delivered to VETLAB, an examination of which also showed severe gill damage but other pathology was inconclusive. SARDI also collected water samples and conducted dissolved oxygen and temperature profiles. On April 24th, numerous samples were examined by VETLAB but all samples showed badly decomposed internal organs and examinations remained inconclusive.

No South Australian samples were sent to AAHL or Tasmania for virology or bacteriology. It is unfortunate that that no fresh specimens were made available to these laboratories for further examination, especially as the epizootic began in SA and pilchards were dying in SA for nearly 6 weeks.

The epizootic arrived in WA on April 18th, with the first reports coming from the Cape Arid and Middle Island area. The first samples were collected on May 3rd and sent to AAHL. WA also sent other fish species to AAHL (Table 8.4). These were sent frozen or as treated in one of four ways (see Table 8.5).

Table 8.4 Fish species sent to AAHL by WA, other than pilchard.

Common name	**Scientific name**
Maray	*Etrumeus leres*
Yellowtail scad	*Trachurus novaezelandiae*
Southern mackerel scad	*Decapturus muroadsi*
Russell's mackerel scad	*Decapturus russelli*
Fusilier sweep	*Caesioscorpius theagenes*
Blue mackerel	*Scomber australasicus*
Blue sprat	*Spratelloides robustus*

Table 8.5 Four treatments recommended for fixing pilchard gills.

2.5% glutaraldehyde in phosphate buffer
10% formalin + skin sample
2.5% glutaraldehyde in 0.1M cacodylate buffer
0.45µ seawater in 1.0% glutaraldehyde

Other samples were sent to the Fish Health Laboratory (WA) where they were examined by a fish pathologist. The autopsy included gut content analysis as well as pathological examination. Extensive phytoplankton testing and temperature profiling was undertaken by Fisheries WA as the mortality front moved through the State. Researchers also examined sea surface temperatures using satellite data. The WA response to the mortality event was a large-scale effort and is to be commended.

In Tasmania, pilchard samples were examined at the Mount Pleasant Laboratories where extensive bacterial and protozoal testing (Indirect Fluorescent Antibody Tests) was undertaken on pilchard samples from several States. Although no significant bacteria were found, the tests identified amoebae in the gills of pilchards from four States, but the results were inconsistent so this line of work was not continued after the discovery of the herpesvirus. CSIRO Marine Research (Hobart) were responsible for examining oceanographic data at the beginning of the epizootic in March and subsequent data taken by other States.

In Victoria, pilchard samples went to AAHL and the Victorian Institute of Animal Science for examination.

NSW also sent samples to AAHL and the Elizabeth Macarthur Agricultural Institute (NSW) undertook histopathology examinations and transmission tests to salmonid fish. Virology was also conducted at the Elizabeth Macarthur Agricultural Institute in conjunction with the electron microscopy undertaken at AAHL. Fisheries in NSW undertook extensive phytoplankton surveys and temperature profiling of the Eastern Australian Current.

Samples were collected from the small amount of pilchards affected in Queensland, some went to the Queensland Museum for identification, and unaffected pilchards were sent to AAHL. Staff from the Southern Fisheries Centre obtained water and plankton samples and undertook temperature profiles.

Closure of fisheries
Catches in Victoria, SA and NSW were withheld and WA had a voluntary moratorium on fishing in the south coast purse seine fishery. Bremer Bay also voluntarily stopped all pilchard exports. Queensland was advised that it should make its own decision regarding a temporary halt in importing interstate pilchards. These decisions were made by each State, not by the Consultative Committee on Emergency Animal Diseases.

8.1.1.3 Resources and Logistics
All States affected by the mortality event were under resourced and responded accordingly. The lack of a financial assistance scheme or cost-sharing structure, similar to that which has been used for decades in terrestrial livestock disease emergencies complicated the decision-making process. There were no defined guidelines on how to share or divide the financial burden of research costs, salaries, resources, travel and other expenses. Uncertainties about funding issues can delay an effective response.

Administration was largely provided by the then Animal Health Secretariat as well as fisheries departments in each State.

8.1.2 Communication
8.1.2.1 Internal communication
It is vital in any national emergency that clear lines of communication between agencies at all levels are established and formally delineated in an approved response plan. State Fisheries agencies had not previously been involved in an animal health emergency and so were unaware of the procedures established under AUSVETPLAN. This resulted in confusion and difficulty in the initial stages of the epizootic. Fortunately the SA and WA State Veterinary Laboratories deal with both aquatic and terrestrial animals and are aware of their obligation to inform the State Chief Veterinary Officer of potential animal health emergencies. In WA, there has been a long-standing agreement that the fish pathologist would report fish disease issues to both the State Chief Veterinary Officer and the Director of Fisheries.

The main factor contributing to the late activation of the CCEAD was the uncertainty of the seriousness of the epizootic. Pilchards had died in relatively small numbers in South Australia previously, so it was not initially believed to be a major emergency. By the time CCEAD was activated, the emergency had spread to other States and was

well established. There is also a hesitancy to activate the CCEAD to deal with aquatic animal health emergencies, until a disease is shown to be responsible rather than an environmental factor.

There was a significant flow of information between the various laboratories undertaking research and between scientists in Australia and New Zealand. There was excellent cooperation between scientists from five States and New Zealand to produce several papers and reports (Fletcher *et al*., 1997; Griffin *et al*., 1997; Hyatt *et al*., 1997; Whittington *et al*., 1997). New Zealand also published their own reports in 1995 and 1996 (Hine, 1995; Smith *et al*., 1996). There was also some healthy dispute amongst the scientific community about the possible cause of the event; this kind of discussion leads to the formation of new hypotheses and is to be encouraged.

The first documentation of the discovery of the herpesvirus is from AAHL in an update provided to the then Animal Health Secretariat (AHS) on 23rd May. At that stage, AAHL had detected virus in pilchards from WA, NSW and Victoria. At the meeting on 26th May, AAHL provided CCEAD with a report covering virology and microscopy. AAHL agreed to provide the herpesvirus to laboratories that required it. Tasmania advised that amoebae had been detected in gill tissue from four States.

During the months of May and June several agencies circulated documents via the AHS, outlining numerous theories, lists of facts and papers that might be relevant, and tabling arguments in support of several theories.

All other communication and dissemination of information occurred through the then AHS. Usually, documents that needed circulating amongst the participants of the CCEAD and the Task Force were faxed to the then AHS by the author and the AHS subsequently faxed the documents to the participants. This avenue of communication, once established, worked well considering the unfamiliarity of the agencies involved with this mechanism.

8.1.2.2 External communication

In May-June, a web page was established by CSIRO Division of Fisheries. This provided the public with access to information on the latest reports of dead pilchards and a map that showed the positions of reported sightings with the date sighted. During the sightings, some formal reports concerning the mortalities and progress updates from the various agencies involved (eg. AAHL) were also prepared. In addition, there were some media links (eg. Promed), but some of these were not available in the correct format. Summary pages from each State involved in the emergency and updates on research were also included on the web site. There was also a summary of pilchard ecology and an overview of historical fishing patterns for pilchards in the Great Australian Bight.

On May 26th, CCEAD decided that liaison with industry concerning pilchard mortality issues should be managed directly between State and industry representatives rather than through industry participation in the proceedings of the Management Committee.

In addition, on 26th May, AQIS confirmed that testing and field reports provided no evidence that the mortalities posed a risk to public health and supported a media release attesting to this.

At the end of June, the *Interim Report on Pilchard Kills in Australian Waters* was released.

Although at the time of the pilchard mortality, the draft Office International des Epizooties' (OIE) International Aquatic Animal Health Code – which would stipulate reporting obligation on aquatic animal disease emergencies – was still being finalised, the Australian OIE delegate (the Commonwealth Chief Veterinary Officer) provided OIE with a note on the event. The Commonwealth Chief Veterinary Officer also provided contingency briefings to overseas posts.

8.1.2.3 Media

There was national coverage of the event. The first reports in Adelaide's 'The Advertiser' were in early April and portrayed a convincing picture of "killer algae". It was reported that the bloom started in Coffin Bay, SA and killed a variety of aquatic animals, however, this is thought to be a separate incident and not related to the pilchard deaths. 'The Advertiser' accurately reported that pilchards were the only fish affected.

There were few media releases, which led to some misreporting. Misleading and accusatory statements were also made by a Senator, and scathing articles published in professional fishing journals. One article also suggested the involvement of the dangerous dinoflagellate *Pfisteria piscimorte.* It was responsible for killing millions of fish in a North Carolina estuary in 1991-92 and also affected human health. This theory was also postulated by a Tasmanian scientist but subsequently dismissed.

On May 10th, CCEAD decided that the contact points for the media would be the Fisheries Directors in each State. During the previous two months, communication with media had been uncontrolled, so interviews had been conducted, articles published and news broadcasts aired with no formally approved releases. The absence of an agreed media response resulted in uncontrolled and conflicting media coverage. This lack of a media strategy gave the emergency response the appearance of being highly uncoordinated.

The ABC program "Four Corners" produced an episode ("Death in the Water") dedicated almost exclusively to the pilchard mortality. The report highlighted the allegedly diminishing resources of AQIS and the changes to quarantine policy that could result in the introduction of exotic pests, threatening Australia's clean image. The reporter spoke to a number of senior officers involved in pilchard research in both Australia and New Zealand.

In general, media management was uncoordinated and substandard. Because CCEAD became involved late into the event, the media had already interviewed several fisheries personnel and scientists. The absence of regular press releases resulted in substantial speculation in reporting.

8.1.2.4 Reports and Publications

An interim report from the Task Force was produced on June 22, 1995. The lack of a report prior to this time received severe criticism from governmental and non-governmental bodies. The delay between the beginning of the mortality in March and the production of a report in June seemed excessive. However, at the time, the extent of the emergency was not foreseen and this resulted in a delay prior to the commencement of the nationwide research effort. In retrospect, given the uncertainty surrounding the cause of the emergency and the coordination required to produce the report, the report was produced by the Office of the Chief Veterinary Officer within an acceptable time frame.

A few brief reports were published in 1995 and 1996. During the two years following the event, many papers and reports were published (see below). The reports by Fisheries in NSW and WA were produced independently from the Task Force because they did not agree with the conclusions of the interim report and wished to discuss further the issue of imported baitfish.

1. Anon. (1995) Interim report on pilchard kills in Australian waters. Pilchard Mortality Task Force.

2. Anon. (1999) Final Report of the Pilchard Mortality Task Force: Summary of Findings in Relation to the 1995 Pilchard Mortality Events.

3. Crane, M.S. and Hyatt, A.D. (1996) Herpesvirus linked to fish fatalities. *Microbiol. Australia*. p. 13.

4. Dann, P., Norman, F.I., Cullen, J.M., Neira, F.J. & Chiaradia, A. (2000) Mortality and breeding failure of little penguins, *Eudyptula minor* in Victoria, 1995-6 following a widespread mortality of pilchard, *Sardinops sagax*. *Mar. Freshwater Res.* **51**: 355-362.

5. Fletcher, R., Jones, B., Hosje, V. & Pearce, A. (1996) Patterns of pilchard deaths. *Microbiol. Australia*. pp. 12-13.

6. Fletcher, W.J., Jones, B., Pearce, A.F. & Hosja, W. (1997) Environmental and biological aspects of the mass mortality of pilchards (Autumn 1995) in Western Australia. Fisheries Research Report No. 106, Fisheries Department of Western Australia.

7. Griffin, D.A. (1997) The 1995 mass mortality of pilchard: no role found for physical or biological oceanographic factors in Australia. *Mar. Freshwater Res.* **48**: 27-42.

8. Hine, P.M. (1995) Herpesviruses associated with mortalities among pilchards (*Sardinops neopilchardus*) around New Zealand. *Surveillance.* **22**(3): 45-48.

9. Hyatt, A.D., Hine, P.M., Jones, J.B., Whittington, A.J., Kearns, C., Wise, T.G., Crane, M.S. & Williams, L.M. (1997) Epizootic mortality in the pilchard *Sardinops sagax neopilchardus* in Australia and New Zealand in 1995. II. Identification of a herpesvirus within the gill epithelium. *Dis. of Aq. Orgs*. **28**:17-29.

10. Jones, J.B., Hyatt, A.D., Hine, P.M., Whittington, R.J., Griffin, D.A. & Bax, N.J. (1997) Special topic review: Australasian pilchard mortalities. *World J. of Microbiol. & Biotechnology*. **13**: 383-392.

11. Mackenzie, L. and Todd, K. (1995) Phytoplankton and water quality associated with mass mortalities of pilchards (*Sardinops neopilchardus*) in Tasman Bay, August-September 1995. Publication No. 309. (Cawthron Institute: Nelson, New Zealand.)

12. O'Neill, G. Ocean anomaly triggers record fish kill. *Science.* **268**: 1431.

13. Smith, P. (1995) Pilchard deaths in New Zealand. *Seafood New Zealand.*

14. Smith, P.J., Holdsworth, J., Anderson, C., Hine, P.M., Allen, D., Gibbs, W., McKenzie, L., Taylor, P., Blackwell, R.H. & Williamson, S.H. (1996) Pilchard deaths in New Zealand, 1995. New Zealand Fisheries Data Report No. 70, Wellington.

15. Trajstman, A.C. (1995) Pilchard Herpesvirus Status and Virus-Wave Front at Various Locations Along the Australian Coast. CSIRO, IAPP, Clayton, Victoria.

16. Whittington, R.J. (1996) Epizootic mortality in pilchards *Sardinops sagax neopilchardus* in Australasia in 1995. NSW Fisheries, 20 Feb.

17. Whittington, R.J., Jones, J.B., Hine, P.M., & Hyatt, A.D. (1997) Epizootic mortality in the pilchard *Sardinops sagax neopilchardus* in Australia and New Zealand in 1995. I. Pathology and epizootiology. *Dis. of Aq. Orgs.* **28**:1-16.

8.2 NEW ZEALAND

The pilchard deaths in New Zealand started four weeks after a shipment of frozen pilchards was received from Bremer Bay, WA (Fletcher *et al*., 1997). The container of potentially infected pilchards was primarily used for long-lining on the eastern side of North Island. This is the site where the New Zealand outbreaks started. These fish had been caught about 10 days before the mortality front reached the area where they were caught. Although both the disease aetiology and modeling suggest that fish developed lesions in only the last 4 days before death (Whittington *et al*., 1997) and were only infectious on the last day before death (Murray *et al.*, 2000), it is possible that a latent period could have been much longer (4-12 days, Murray *et al.*, 2000; up to 3 weeks, Griffin *et al*., 1997).

8.3 RESPONSE TO THE 1998 PILCHARD MORTALITY EVENT

The experience gained during the 1995 mortality ensured that the response to the 1998/99 mortality event was rapid, smooth and well coordinated, and handled with

much greater urgency and efficiency than in 1995. Once again, the Consultative Committee on Emergency Animal Diseases (CCEAD) mechanism was activated but this time in an immediate response. Many of the same personnel were involved, so lines of communication were clearer, smoother and benefited from the prior experience of handling an aquatic animal health emergency. Reactions were much quicker in many areas. Funds were immediately available, enabling boat surveys and other monitoring strategies to be implemented quickly. The relative lack of interest shown by some States and agencies was disappointing, but in general the response was more than satisfactory.

8.3.1 Management

8.3.1.1 Coordination of the response

National approach

The first reported sighting of dead pilchards was on 7th October, although a retrospective report came a little later notifying authorities of a sighting of some dead pilchards on 2nd October. The CCEAD was activated, convened on 15th October, only two weeks after the second epizootic began, and this time the membership was much smaller than in 1995.

This first CCEAD meeting was agreed to establish a small multi-disciplinary scientific Working Group to "undertake a scoping exercise and report on the diagnostic requirements, surveillance and research needs to determine the nature of the event". The Joint Pilchard Scientific Working Group met on 16th October and numerous meetings followed (Table 8.6).

Table 8.6 Dates of major management meetings.

	Date	**Year**	**Group**
	15th Oct.	1998	CCEAD teleconference
1	16th Oct.	1998	JPSWG
2	30th Oct.	1998	JPSWG
3	17th Nov.	1998	JPSWG
	30th Nov.	1998	CCEAD teleconference
4	15th Dec.	1998	JPSWG
5	19th Jan.	1999	JPSWG
6	23rd Feb.	1999	JPSWG
7	30th March	1999	JPSWG
8	12th May	1999	JPSWG
9	2nd Aug.	1999	JPSWG
10	6th March	2000	JPSWG
11	21st July	2000	JPSWG

The actions of the Working Group meetings are listed in Appendix 8. Participants of the meetings are given in Table 8.7. The inaugural meeting of the Working Group decided to invite the Australian Quarantine and Inspection Service to be a member of the group and the agency has been represented since 30th October 1998.

Table 8.7 Participants of CCEAD and JPSWG meetings.

Date	Group	Participants
15/10/98	CCEAD	Office of the Chief Veterinary Officer (OCVO) Commonwealth Chief Veterinary Officer (CCVO) State Chief Veterinary Officers (CVOs) Aquatic Animal Health Unit (AAHU) Australian Quarantine and Inspection Service (AQIS) Department of Primary Industries and Energy CSIRO Australian Animal Health Laboratory (AAHL) CSIRO Marine Research Directors of Fisheries for WA, SA, NSW, Queensland, Victoria and Tasmania.
16/10/98	JPSWG	Director of Fisheries SA, Fisheries SA representative, Aquatic Animal Health Unit (AAHU), Fisheries WA, AAHL, SARDI Aquatic Sciences, SA Health Commission, Primary Industries and Resources South Australia (PIRSA), Veterinary Pathology Services (VPS) SA.
30/10/98	JPSWG	Director of Fisheries SA, AAHU, Fisheries WA, AAHL, SARDI Aquatic Sciences, SA Health Commission, AQIS, VPS, Dept. Agriculture Fisheries and Forestry.
17/11/98	JPSWG	Director of Fisheries SA, AAHU, Fisheries WA, AAHL, SARDI Aquatic Sciences, SA Health Commission, VPS, CSIRO Marine Research
30/11/99	CCEAD	OCVO, CCVO, State CVOs, AAHL, SA Fisheries, WA Fisheries, NSW Fisheries, Victoria Fisheries, Queensland Dept Primary Industries, Fisheries & Aquaculture Branch (AFFA), Bureau of Resource Science, Livestock and Pastoral Division
15/12/98	JPSWG	Director of Fisheries SA, AAHU, Fisheries WA, AAHL, SARDI Aquatic Sciences, VPS, CSIRO Marine Research, PIRSA, Marine And Freshwater Research Institute (Victoria)
19/1/99	JPSWG	AAHU, A/Director Fisheries SA, Fisheries WA, SARDI Aquatic Sciences, AQIS, VPS, CSIRO Marine Research
23/2/99	JPSWG	Director Fisheries SA, AAHU, Fisheries WA, AAHL, SARDI Aquatic Sciences, AQIS, VPS, PIRSA
30/3/99	JPSWG	Director Fisheries SA, AAHU, Fisheries WA, AAHL, SARDI Aquatic Sciences, AQIS, VPS, NSW Fisheries, CSIRO Marine Research
12/5/99	JPSWG	Director Fisheries SA, AAHU, Fisheries WA, AAHL, SARDI Aquatic Sciences, VPS, PIRSA, CSIRO Marine Research
2/8/99	JPSWG	Director Fisheries SA, AAHU, Fisheries WA, AAHL, SARDI Aquatic Sciences, VPS, AQIS, PIRSA, CSIRO Marine Research
6/3/00	JPSWG	Director Fisheries SA, AAHU, Fisheries WA, AAHL, SARDI Aquatic Sciences, VPS, AQIS, PIRSA, CSIRO Marine Research
21/7/00	JPSWG	A/Director Fisheries SA, AAHU, Fisheries WA, AAHL, AQIS, PIRSA

It was agreed at the first meeting that more and better samples of dead and dying fish were essential for diagnostic work. Other priorities identified included the sequencing of the viral DNA, development of a polymerase chain reaction test, testing of

imported fish using the PCR test, transmission trials, development of pilchard gill cell lines, and import risk analyses of all imported fish.

There was a considerable amount of forward planning by Fisheries WA, who held a number of meetings to prepare research and management plans. In 1995, the path of the mortalities was very difficult to predict. In 1998, the experience of the 1995 mortality event gave Fisheries WA confidence in predicting that not only would a westbound mortality front reach WA, but also would probably go past Cape Leeuwin and through any storm fronts it encountered. WA had over a month to prepare and predicted accurately that the mortalities would reach WA on 6-7 November. The first report of mortalities in WA was on 10 Nov. Estimates and guidelines were given for the costs of sampling, transmission trials, quantification of the mortality event, size and age studies, and reporting, even before the event reached the State.

Continuity of meetings
Compared to 1995, the series of Working Group meetings had a smooth continuity and follow-up. Actions items were listed in tables (Appendix 8) and progress on each item was reported at subsequent meetings.

There was a significant commitment from participating agencies to appoint a representative to the Working Group and support their attendance at as many meetings as possible. This created a much greater sense of continuity and ownership amongst the group, and ensured that actions were completed. In 1995 some of the meetings contained so many agency representatives, they were difficult to participate in and the same representatives were not present at continuous meetings.

Diagnostic approach
In contrast to the haphazard approach used in 1995, the formation of the Joint Pilchard Scientific Working Group was accepted by all parties in 1998. This made coordination of research issues easier because decisions were made and implemented by one group. The research was well organised and generally proceeded smoothly.

Because of the virus found in 1995, the AAHL (AAHL) concentrated on various virological studies, using histopathology, electron microscopy and cell cultures. Although the working group recognised that other issues had to be explored, a recurrence of the herpesvirus could not be ruled out.

The South Australian Research and Development Institute (SARDI) was responsible for coordinating sample collection in SA and for sending samples to other laboratories who requested samples. SARDI also assessed methods for the non-destructive sampling of gannets, but this line of work was not pursued due to a lack of funds.

WA Fisheries were responsible for conducting transmission trials and purifying virus from affected pilchards, as well as monitoring the mortality front in WA.

Further surveillance
The longer time frame of the second emergency meant that it was in the public eye for longer, so more fishers, beachgoers and other ocean users were aware of the event and of the need to report sightings of dead pilchards. There was extensive surveillance in WA, mostly by WA Fisheries staff, but members of the public and family members

were also utilised in monitoring the spread of the mortality and in studies to estimate the number of pilchards affected (Gaughan *et al*., 2000).

Fisheries WA was conditionally approved a grant from the Fisheries Research and Development Corporation for a project titled "Regrowth of pilchard (*Sardinops sagax*) stocks off southern WA following the mass mortality event of 1998/1999." This will allow close monitoring of the recovery of the south coast pilchard stocks which was severely depleted after the 1998/99 mortality.

8.3.1.2 Operational aspects of the response

Dead pilchards were collected on 7th October, and delivered to VPS on the 9th. SARDI immediately notified WA Fisheries. An initial aerial survey was conducted on 13th October, with further aerial surveys conducted throughout the period that pilchards died in South Australia. These were done using helicopter and small aeroplanes. By 14th October, AAHL had received pilchard samples from SA.

Because of the first meeting, an expanded sampling program was set into place. PIRSA immediately diverted the SARDI vessel to west coast areas to assist in sampling. Surveillance and sample collection were facilitated by pilchard fishers who allowed SARDI and Fisheries staff on their boats and transported them to where the pilchards were dying in the sea. SARDI was to co-ordinate sampling for all national requirements and AAHL sent staff to South Australia to assist in sampling. A significant effort was made by South Australian agencies to monitor the deaths and collect samples.

Toxicity testing commenced shortly after the first meeting. This quickly revealed that the presence of toxins such as Neurolytic Shellfish Poisoning or Amnesiac Shellfish Poisoning was highly unlikely. This was a much quicker response than in 1995.

Samples of dead pilchards from all affected States were sent to AAHL promptly.

WA Fisheries collected a huge amount of dead and moribund pilchards. Some of these are still in storage at -80°C in Perth and some of have been sent to AAHL for use in the ongoing polymerase chain reaction and sequencing work. Considerable effort was made by WA agencies to monitor the deaths and estimate the biomass killed through beach transects, aerial surveys and sea floor surveys using an underwater video camera towed under a boat (Gaughan *et al*., 2000).

The transmission trials undertaken at Bremer Bay, WA, were a good example of collaboration between State government and industry. The government conducted trials were housed in a commercial manufacturing warehouse with the support of the owner. Pilchards were also held in a sea cage in Bremer Bay marina.

Closure of fisheries

In 1998, closure of the SA and WA pilchard fisheries was by official order. In SA, the fisheries were closed from 14th October to 21st November by the Director of Fisheries. From 16th–31st October, The Australian Fisheries Management Authority also prohibited taking of local pilchards by tuna operators, who are subject to Commonwealth management.

The south coast purse seine fisheries in WA were closed down sequentially. A series of notices prohibiting fishing in clearly defined zones was produced by the Director of Fisheries in WA. WA released notices of fishery closure between 30th Dec.1998 and 8th March 1999 (Table 8.8).

Table 8.8 The dates and details of zone closures in the south coast pilchard fisheries of WA.

30/12/98	Zone 4 until 29/1/99
31/12/98	Zone 4 E of line 11 nm E of western boundary until 29/1/99 Yesterday's notice repealed.
27/1/99	Zones 1 & 3; Zone 2 E of 117° E longitude until 26/2/99
11/2/99	Zone 4 W of 120°6' E longitude until 26/2/99
16/2/99	Zone 2 W of 117° E longitude; Zone 5 until 17/3/99.
8/3/99	Zone 2 W of 117°58' E longitude; Zone 5 until 17/3/99 Notice of 16/2/99 repealed.

On 16th April, dead pilchards were reported around the Rottnest Island area, off Perth, so the West Coast Pilchard Fishery was closed until further notice. Other issues associated with the management of this fishery influenced the decision to close it for an unspecified period.

In Victoria, fishers agreed to a voluntary closure of the fishery (S. McCormack, pers.com.) whilst in NSW, there was an official closure for approximately a month during the time of the pilchard deaths (January 1999, R. Fletcher, pers.com.).

8.3.1.3 Resources and logistics

Funds for monitoring operations were rapidly made available, so that boat and aerial surveys and sample collection could commence immediately and be conducted on a regular basis. A summary of funding sources for priority research is presented in Table 8.9. These funds are for specific projects and do not include the large cost of personnel salaries, travel expenses, overheads and other costs absorbed by the agencies involved.

Table 8.9 Funding sources for priority projects in 1998/99.

Project Title	**Funding source and project host**	**Status**
Pathology of pilchards and associated marine fauna	Funded from existing sources with PIRSA, WA, AAHL	Completed
Pilchard virus transmission trials	South Australian Biosecurity fund and WA	One trial completed but unsuccessful.
Monitoring of pilchard mortalities	Funded from sources within States	Completed
Generation of diagnostic reagents for pilchard herpesvirus	FRDC Grant $98,800 CSIRO AAHL	3 year project (commenced July 1999)
Development of a model of the spread of the pilchard mortalities	FRDC Grant $46,250 CSIRO Marine Research, Hobart	Completed
Report: Pilchard mortality events in Australia and related world events	FRDC Grant $24,730 PIRSA	Completed

8.3.2 Communication aspects

8.3.2.1 Internal communication

Involvement of personnel from agencies that had experienced the CCEAD mechanism in 1995, enhanced communication. There was greater co-operation and information sharing between agencies and quicker decision-making due to an enhanced understanding of CCEAD mechanisms.

8.3.2.2 External communication

SA Fisheries, and later WA Fisheries, published daily pilchard bulletins on the departmental websites, for access by the public. This was an excellent means of providing information to the public. The Office of the Chief Veterinary Officer posted notifications on Promed, an Internet medical news service, at regular intervals.

Within the first two weeks of the mortality, the SA Health authority issued a general public warning.

This second pilchard mortality was reported to the Office International des Epizooties in January 1999, as part of the normal annual report. A separate report was made to the Fish Diseases Commission.

8.3.2.3 Media

On 15th October, CCEAD agreed that a disciplined approach to media contact was important. The SA Director of Fisheries was appointed as the initial media contact point and other State Fisheries Directors and Chief Veterinary Officers referred media requests to SA. When the mortalities spread to other States, CCEAD agreed that each State's Fisheries Director would act as the media contact for queries within the State. This approach meant that there was adherence to a common message.

8.3.2.4 Reports and publications

Fewer reports have been published about the second mortality event because they would largely be repetitive. As expected, there has been a two year time lag between the start of the mortality event (1998) and publication of papers. Further papers are expected in 2000 - 2001. The reports published in 2000 have a substantially different focus to that of the papers relating to the 1995 event.

1. AAHL (1999) Status report on research program. Report to the Joint Pilchard Scientific Working Group. CSIRO Animal Health. June 1999. 36 pp.

2. Bunce, A. and Norman, F.I. (2000) Changes in the diet of the Australasian gannet (*Morus serrator*) in response to the 1998 mortality of pilchards (*Sardinops sagax*). *Mar. Freshwater Res.* **51**:349-353.

3. Gaughan, D., Mitchell, R.W. and Blight, S.J. (2000) Impact of mortality, possibly due to herpesvirus, on pilchard *Sardinops sagax* stocks along the south coast of Western Australia in 1998-1999. *Mar. and Freshwater Res.* **51**(6): 601-612.

4. Murray, A. G., O'Callaghan, M. and Jones, B. (2000) Modeling the pilchard mass mortality events of 1995 and 1998/9. Report to the Fisheries Research and Development Corporation. Project no. 99/225.

5. Murray, A.G., O'Callaghan, M. and Jones, B. (in press) Simple models of massive epidemics of herpesvirus in Australian (and New Zealand) pilchards. *Environ. Int*.

6. Murray, A.G., O'Callaghan, M. and Jones, B. (in review) A model of transmission of a viral epidemic among schools within a shoal of pilchards. *Ecol. Modell.*

7. Murray, A.G., O'Callaghan, M. and Jones, B. (to be submitted) A model of spatially-evolving herpesvirus epidemics causing mass mortality in Australian pilchards (*Sardinops sagax*).

8. Murray, G. (1999) Update on southern Australia's latest outbreak of pilchard mortalities. *Australian Veterinary Journal*. **77**: 59.

9. Ward, T.M., Westlake, M., McLeay, L.J. and Jones, G.K. (1999) Pilchard Mortality Events in South Australia. Final Report to the Joint Pilchard Scientific Working Group.

8.4 MANAGING AQUATIC ANIMAL DISEASE EMERGENCIES

8.4.1 A historical perspective on aquatic animal health management

In 1992, the Australian Quarantine and Inspection Service identified the lack of aquatic animal quarantine policies in "Aquatic Animal Quarantine in Australia". In 1994, the Standing Committee on Fish and Aquaculture (SCFA) identified the need for the development of national plans and mechanisms for the management of aquatic disease emergencies. The Fish Health Coordinating Group was formed with representatives from the Animal Health Committee, SCFA's Environment and Health Committee, the Aquaculture Committee, the fishing industry and the Commonwealth Chief Veterinary Officer. The Fish Health Coordinating Group commissioned a report, "Managing the National Response to Fisheries and Aquaculture Emergencies"

(Jones, 1996), which was released in 1996 after the pilchard mortality event of 1995. The pilchard deaths were a timely and significant alarm, alerting many Commonwealth and State agencies of the need to expedite the formulation of emergency management plans.

The 1995 pilchard mortality event precipitated the formation of the National Task Force on Imported Fish and Fish Products, which was established at the end of June 1995, and had its first meeting in July. The Task Force was to investigate and report on the implications arising from aquatic animal imports. The Task Force had a number of Terms of Reference including examining the effectiveness of current import-related legislative controls and practices at the Commonwealth and State levels, identifying areas of concern such as the impact of exotic disease, and developing a proposal for a transparent, documented import risk assessment policy and process. A detailed report, produced in December 1996 (DPIE, 1996) recommended the development of a strategic plan for Australian fish health issues.

In December 1995, the Commonwealth Government announced that an independent review of Australia's animal and plant quarantine policies and programs was to be undertaken by the Nairn Committee. The Committee's Terms of Reference were wide and in 1996, this committee also produced a report detailing a number of recommendations on quarantine policies (Nairn *et al*., 1996).

The Standing Committee on Agriculture and Resource Management (SCARM) established a Task Force to further assess the issues of emergency response strategies for incursions of exotic pests, terrestrial and marine weeds and diseases. The Fish Sub-Committee Task Force published a report in 1996 entitled "Task Force into Managing Incursions".

In 1995, an FRDC grant was approved to undertake an assessment on aquatic animal disease preparedness. This resulted in another report in 1996 dealing with an issue relevant to the pilchard mortality event (Crane and Rawlin, 1996).

Each of these reports highlighted the lack of attention surrounding the related issues of aquatic animal quarantine policy and aquatic animal disease emergency response plans. In 1997, SCARM recommended the establishment of the Australian Fish Health Management Committee, with a 2-year lifespan. At the inaugural meeting, the Fish Health Management Committee agreed to develop a 5-year strategic plan ('AQUAPLAN') to establish a national aquatic animal health system. As part of AQUAPLAN, there were projects to identify the roles and responsibilities of key agencies and address issues such as awareness of aquatic animal health and the integration of the private sector. An initial document was produced to develop contingency plans for aquatic animal disease emergencies (Crane and Rawlin, 1997), which set in place the foundation for a series of manuals and AQUAVETPLAN was initiated.

8.4.2 AQUAPLAN (1998 – 2003)

In the future, all aquatic animal health emergencies of national significance (including any further pilchard mortalities) will be managed using the procedures detailed in AQUAPLAN and AQUAVETPLAN. The full text of AQUAPLAN and an update of the projects can be found on the AFFA website under 'Aquatic Animal Health'. The Executive Summary of AQUAPLAN states:

"AQUAPLAN is a broad, comprehensive strategy that outlines objectives and projects to develop a national approach to emergency preparedness and response and to the overall management of aquatic animal health in Australia.

AQUAPLAN was developed in response to the *Report of the National Task Force on Imported Fish and Fish Products* (1996) and *Australian Quarantine: A Shared Responsibility* (1996). Funding was allocated to the Commonwealth Department of Agriculture, Fisheries and Forestry — Australia, AFFA (previously the Commonwealth Department of Primary Industries and Energy) to co-ordinate the development of a strategic plan for aquatic animal health management.

AQUAPLAN has been jointly developed by Government and private industry sectors and seeks to build and enhance capacity for the management of aquatic animal health. It has been developed in a manner consistent with existing arrangements in the terrestrial animal sector and, wherever possible, links into existing State/Territory Government and industry health management arrangements.

The Ministerially appointed Fish Health Management Committee (FHMC) is the body, which oversees the development of AQUAPLAN. The FHMC is chaired by Gardner Murray, the Managing Director of the National Offices of Animal and Plant Health and Food Safety and comprises representatives from the Standing Committee on Agriculture and Resource Management (SCARM), the Standing Committee on Fisheries and Aquaculture (SCFA), the Commonwealth Scientific and Industrial Research Organisation (CSIRO) Livestock Industries, the Australian Seafood Industry Council (ASIC), recreational fisheries (RecFish Australia) and representatives from the peak aquaculture industry bodies of Australia.

AQUAPLAN comprises eight key programs under which government and private sectors have identified priority projects to achieve the program objectives. Together these objectives will assist in maximising Australia's ability to control aquatic animal disease outbreaks, maintain market access, support quality assurance and improve the productivity and sustainability of Australia's aquatic animal production industries. Wherever possible, AQUAPLAN projects link into existing terrestrial animal health arrangements in order to avoid duplication and to maximise sensible use of resources."

The eight programs that comprise AQUAPLAN are:

1. International Linkages
2. Quarantine
3. Surveillance, Monitoring and Reporting
4. Preparedness and Response

5. Awareness
6. Research and Development
7. Legislation, Policies and Jurisdiction
8. Resources and Funding

8.4.2.1 AQUAVETPLAN

AQUAPLAN Project number four, "Preparedness and Response", includes the formulation of a series of manuals detailing the procedures for an emergency response to an aquatic animal disease outbreak. Modelled on AUSVETPLAN, these manuals are collectively called AQUAVETPLAN. They comprise an "Enterprise Manual", several "Operational Procedures Manuals", "Disease Strategy Manuals" and a "Control Centre Manual". They deal with four different aquatic systems:

- Open: neither animal movement or water flow is controlled eg. wild fisheries
- Semi-open: animal movement is restricted but not water flow eg. net pen culture
- Semi-closed: control of animal movement and some control of water flow eg. pond culture, race culture
- Closed: both animal movement and water flow are controlled: eg. aquaria

The Enterprise Manual is the first completed and has recently been endorsed by SCFA and SCARM and approved for final publication.

The Control Centre Manual is nearing completion. It clearly defines the lines of communication and management during an incident. Aquatic disease management has been incorporated into the Consultative Committee on Emergency Animal Diseases (CCEAD) mechanism, so if the disease is of national importance, the State Chief Veterinary Officer and/or Director of Fisheries will activate the CCEAD via the Commonwealth Chief Veterinary Officer. In the case of aquatic animal disease management, the CCEAD will comprise State Chief Veterinary Officers as well as the Fisheries Directors of each State/Territory and the Commonwealth. The CCEAD will advise on communication to international agencies if the emergency is of international significance. Such communication is the responsibility of the Commonwealth Chief Veterinary Officer on behalf of Australia. AQIS is responsible for informing overseas trading ports and partners.

The State Disease Control Head Quarters (DCHQ) is the central combat authority and coordinates the Local Disease Control Centre, State Emergency Services, etc.

A task force or disease management team including personnel with relevant expertise and industry representatives may be formed. Liaison with other services eg. State Emergency Services, Police, the Environment Protection Agency, would be through the task force. The task force may form a local control centre, as is the procedure for terrestrial animal emergencies. The management of the incident is led by the State Chief Veterinary Officer through the State Veterinary Laboratory and/or the Director of Fisheries.

The Enterprise Manual states "Timely release of accurate information is the key to good information management. The aim of communications should be to keep relevant parties informed, reduce the spread of inaccurate information by rumour, and protect any appropriate trade (domestic or international) position."

An outline of the coordination response is shown in Fig. 8.1.

Animal Health Committee (AHC) protocols for submission of diagnostic samples to AAHL have been available for some time but were written before the AAHL Fish Diseases Laboratory (AFDL) was established at AAHL and, until recently, only covered submissions from terrestrial disease outbreaks. Submission of samples for fish disease diagnosis was effected by the fish pathologist from the submitting State communicating directly with AFDL staff. Until the pilchard mortality in 1995, there had been no aquatic animal epizootic of national importance and only relatively few aquatic animal samples had been submitted to AAHL. Thus, when pilchard samples were submitted to AAHL by agencies unfamiliar with AFDL a few of the samples were misdirected. This caused some confusion and delay in sample processing. Currently, the protocols for submission of samples to AAHL are under review, and revised versions will cover submission of samples from both terrestrial as well as aquatic animals.

The AQUAVETPLAN Disease Strategy Manuals stipulate that samples sent to them to AAHL must be labeled in the correct way, directing them to the AFDL. At this point, a member of staff from AAHL should be identified to whom the packages should be sent and their name made clearly and widely known to the relevant agencies. However, for new and emerging diseases, such details should also be given in a generic operational manual.

8.4.2.2 Funding aquatic animal health emergencies

There is a good discussion of funding issues in Bernoth *et al.* (1999), from which this discussion is mostly taken.

The funding of management of emergencies is complex. Uncertainties may arise during emergencies responses can be delayed until funding issues are resolved. Industry may have little incentive to report disease, unless there is some certainty about funding support, including compensation. Funding principles include:

- Agreement on which diseases will qualify for compensation;
- Agreement on which activities the money will be available to fund (eg. the cost of restocking);
- Agreement on a fund-raising procedure (eg. levies, cost-sharing between the government and private sector)
- Encouragement of early reporting of disease;
- The principle that those affected by a disease for which compensation is provided should be neither better nor worse off as a result of receiving compensation.

In the terrestrial animal sector, there is a cost-sharing arrangement between Commonwealth and State/Territory Governments applied when reimbursing costs are incurred in the control of a small number of exotic diseases. This approach is being re-examined and a different arrangement, based on the nature of the disease, is being developed.

The situation in the aquatic animal sector is very complex for several reasons:

- Both aquaculture and fisheries have been relatively free from disease to date;
- Aquaculture is a new and emerging industry with many small farms;
- Large aquaculture ventures such as pearling, Atlantic salmon and SBT farming can afford the costs of insurance, but smaller individual farmers cannot; although wild catch fisheries could probably afford insurance, they do not see the need;
- Effective aquatic animal disease emergency plans must involve both the wild catch fishing sector and the recreational sector;
- There is reluctance within the fisheries sectors to contribute to a compensation fund involving aquaculture;
- Aquatic species do not occur homogeneously in Australia, eg. SBT is farmed in South Australia and Atlantic salmon is farmed predominantly in Tasmania;
- Diagnosis of aquatic animal diseases is difficult and time consuming. This complicates funding and compensation issues.

The issue of funding emergency responses to aquatic animal diseases has been given the highest priority in AQUAPLAN. The Federal Budget has offered funding to target disease prevention, raise awareness, and improve Australia's ability to respond to emergencies. The entire sum available from financial year 2000/2001, for the next four years is approximately $3.4 million. These funds are to be managed by external organisations with a view to coordinating the interests of industry and Government; they will not be made available to Agriculture, Fisheries and Forestry – Australia (AFFA) (Murray, 2000).

8.4.2.3 Increasing public awareness of aquatic animal diseases and emergency management

The extensive media coverage and ready accessibility of dead pilchards on metropolitan beaches highlighted aquatic animal diseases to the public for the first time.

The Aquatic Animal Health Unit, part of the then National Office of Animal and Plant Health (within AFFA), was created in 1998 and is part of an increasing commitment to the previously neglected area of aquatic animal health. The Aquatic Animal Health Unit recently released the *Australian Aquatic Animal Disease Identification Field Guide* as one of the projects under program 5 of AQUAPLAN - Awareness. It has been produced "in the interest of public awareness in aquatic animal disease management in Australia and in support of AQUAPLAN".

The AFFA Website http://www.affa.gov.au/outputs/animalplanthealth.html under 'Aquatic Animal Health' describes the Guide:
"The Field Guide provides an informative, sometimes graphic, account of the diseases and organisms that threaten Australia's aquatic animal industries. It also has information on diseases in other parts of the world and how they could affect us if they were to occur here.

The book is waterproof, tear resistant and ultra-violet light tolerant. It provides easy-to-read information on diseases of aquatic animals for those whose livelihoods or interest mean they are well situated to spot changes in aquatic animal populations.

The addition of a spiral binding makes for easy one-handed operation, whether on a boat or in the laboratory."

Other projects under the 'Awareness' program are shown in Table 8.10. All awareness projects are monitored and primarily run by the Aquatic Animal Health Unit, who act to expedite the highest priority projects. States also run their own awareness campaigns.

Table 8.10 Projects included in the Awareness program of AQUAPLAN.

1.	Posters, pamphlets and videos for industry, veterinarians and the public
2.	Publication of regular columns in industry or trade journals
3.	A disease Hotline for use by industry and the general public
4.	Inclusion of aquatic animal health in veterinary curricula and other tertiary education
5.	Post-graduate training of veterinarians, aquatic animal health specialists and industry groups in recognition of disease syndromes
6.	Training for aquaculturists in aquatic animal health management and improving farm practices to promote disease prevention
7.	Registration and minor use permit approval for the use of drugs and chemicals in aquaculture
8.	Encouraging the development of programs to promote the safe, effective and minimal use of drugs and chemicals

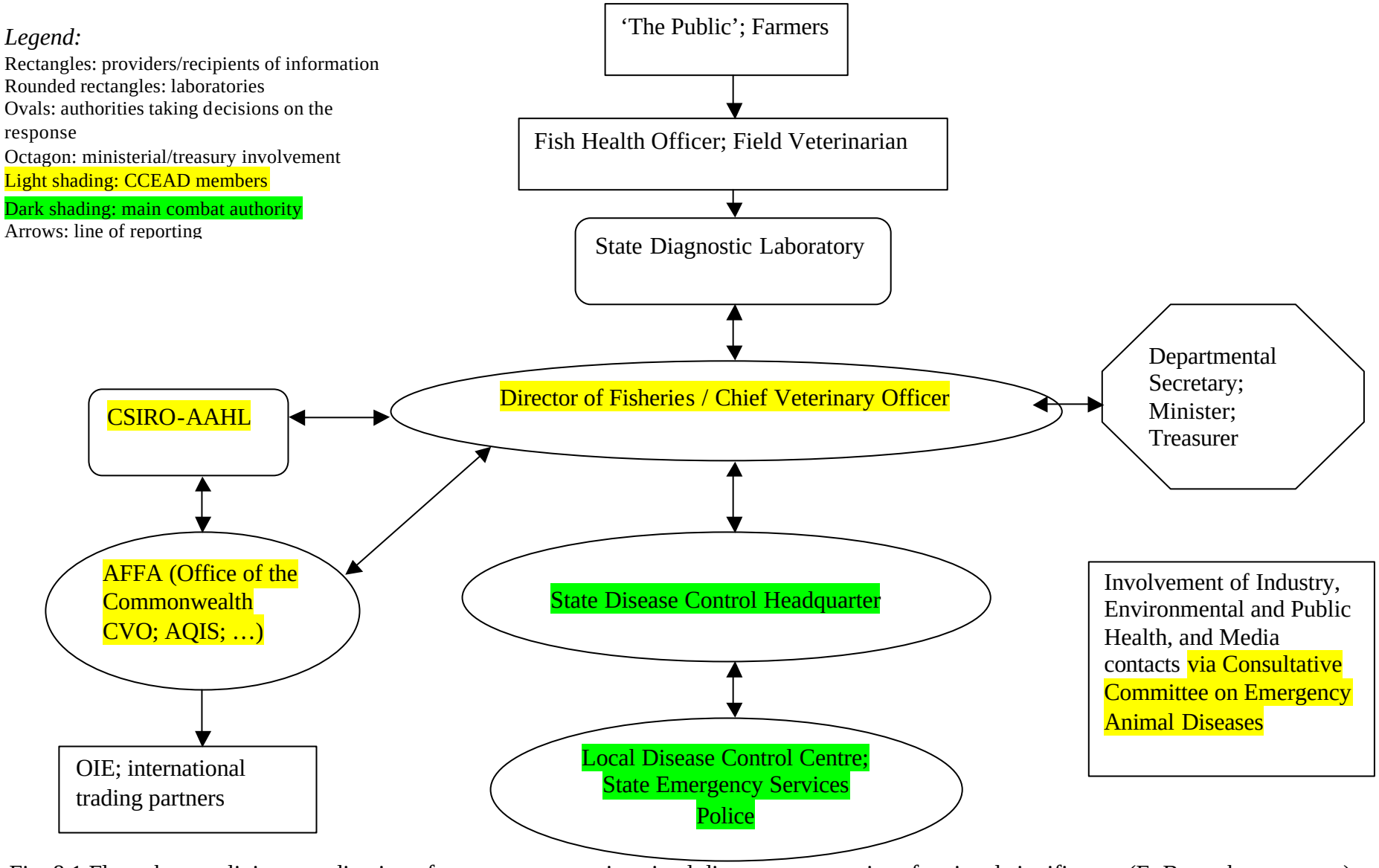

Fig. 8.1 Flow chart outlining coordination of response to aquatic animal disease emergencies of national significance (E. Bernoth, pers.com.)

8.5 RESPONSE OPTIONS FOR A POSSIBLE FUTURE PILCHARD MORTALITY EVENT

8.5.1 Coordination

In a manner similar to the 1998/99 event, response to a possible future pilchard mortality event would be coordinated by the 'aquatic' CCEAD, which incorporates Fisheries Directors, and other aquatic animal health personnel.

Currently, AQUAVETPLAN is still under development and if an epizootic were to occur, not all response procedures are yet developed or documented. However, the procedure of activating CCEAD is now well understood by the aquatic industry and associated government agencies, so a response to an aquatic disease emergency would be better coordinated than previously. Although many components of AQUAVETPLAN are still in draft form or at various stages of endorsement, those procedures thus far developed may well be brought into use to facilitate the response to an emergency. In the past five years, many States have updated existing legislation or introduced new legislation to address disease emergencies in the aquaculture and fisheries sectors.

8.5.2 Monitoring

Ward *et al.* (1999) suggested that improved aerial surveys would result in more effective monitoring during future mortality events. Experience showed that small aeroplanes flew too fast and too high to permit accurate identification of small fish floating on the sea surface. Helicopters proved to be superior aircraft for surveillance. However, the inherently high costs and fuel limitations of helicopters are major constraints to conducting these surveys. The authors suggest that it is more useful to conduct a few helicopter surveys at critical times than to conduct numerous, relatively unproductive surveys from small aeroplanes.

Ward *et al.* (1999) also observed that the use of the same, trained observers would substantially increase the success of surveys. They suggested that designated officers be identified at locations throughout each State and be provided with equipment and training to ensure that high quality samples and data are collected quickly and efficiently. It is also suggested that boats suitable for collecting fish for diagnostic studies be identified. The authors stated that the "establishment of a protocol for sampling and data collection is particularly important as it is possible that more pilchard mortality events will occur".

The authors also discuss sampling for diagnostic studies:

> "many of the pilchards initially provided for veterinary studies had been collected from beaches and were too badly decomposed to provide useful information. In the future, priority must be given to the collection of large samples of freshly dead and dying pilchards as soon as it is clear that a mortality event has begun. Well-fixed gills are critical for histopathology associated with toxic events and algal blooms as well as viruses. Gill must be fixed within 1-2 minutes of death for transmission electron microscopy and 3-5 minutes of death for light microscopy. Samples should be collected by designated officers using the vessels and equipment described above, or by the trained staff of animal health agencies. Samples should be stored on (but not in contact with) ice, or fixed before being transported to the South Australian Veterinary Pathology Services and/or

CSIRO's Australian Animal Health Laboratories. Additional samples and alternative preservation methods that may be required should be collected under the direction of officers from these agencies."

The establishment of a written protocol for emergency response sampling in a fish mortality event should be a priority for States. These procedures should be provided to all fisheries agencies field officers and relevant researchers (eg. South Australian Research and Development Institute, Marine and Freshwater Research Institute), who comprise the on-site operational personnel of an emergency response.

Gaughan *et al*. (2000) stated that sea floor counts, using underwater video, enabled the determination that "pilchard densities averaged 11.45 times higher (…) on the sea-floor than on the sea-surface". This large number enabled more accurate estimation of the numbers and proportion of pilchards killed on the south WA coast. It would be useful if other affected States could also utilise this method for surveillance. It would be impractical however, in States like SA, where the pilchard population is widely distributed (T. Ward, pers.com.) and underwater surveys would take a prohibitively long period.

8.5.3 Diagnostic capability

Australia has diagnostic capability for all diseases listed on Australia's *National List of Reportable Diseases of Aquatic Animals* which contains all diseases listed on the Network of Aquaculture Centres in Asia-Pacific (NACA) and Office Internationale des Epizooties (OIE) lists. Although there is diagnostic capability for all diseases on three different diagnostic levels (see Table 8.11), this does not mean that every laboratory in Australia has this capability. For example, diagnostic tests that require the use of viable exotic pathogens are usually restricted to the AAHL where they can be carried out under microbiologically secure conditions.

Table 8.11 The three levels of diagnosis of disease.

Level 1	Observation of animal and environment; gross clinical examination
Level 2	Parasitology, bacteriology, mycology and histopathology
Level 3	Virology, electron microscopy, molecular biology and immunology

Diagnostic capability does not necessarily imply that the diagnostic methods are fully standardised and validated, or are in total accordance with the internationally recommended methods as outlined in the *OIE Diagnostic Manual for Aquatic Animal Diseases*. Australia does, however, invest considerably into the standardisation of its diagnostic methods for aquatic animal diseases. As part of the new federal budget initiative (section 8.4.2.2), over $1.6 million is available over the years 2000/2001 to 2003/2004 to strengthen diagnostic capability in the aquatic animal sector (E. Bernoth, pers.com.).

To maximise the integrity of collected data, the development of Standard Diagnostic Techniques and Standard Operating Procedures is regarded as a high priority for AQUAPLAN. To date, the Standard Operating Procedure for virus isolation is near completion. A range of further Standard Diagnostic Techniques and Standard Operating Procedures for finfish sampling and for survey techniques are under development. Each of these techniques and procedures follows the OIE recommended methods as outlined

in the *OIE Diagnostic Manual for Aquatic Animal Diseases* where possible (E. Bernoth, pers.com.).

8.5.4 Management

A discussion on management options is given by Murray *et al*. (2000) in a report documenting the modeling of the epizootics. This discussion will be largely summarised from that report.

Schooling clupeoid fish are vulnerable to disease because, even at a low population density, a virulent disease can be transmitted effectively due to the high contact rates of schooling fish. The modeling showed that the disease was only weakly dependent on population density. Even if very low densities of the adult population could be brought about by human intervention and manipulation (eg. increased fishing effort), the disease would still be transmitted effectively. In addition, the actual position of the epidemic front would be 140 – 400 km ahead of the mortality front, therefore a 'firebreak' would most probably be unsuccessful.

The culling of birds, mammals or large predatory fish is also unlikely to be an effective means of limiting the spread of the virus. The modeling showed that such vectors lack a critical role in transmission.

The spread of the virus is very sensitive to patterns of movement. If some form of containment or barrier could be formed to inhibit pilchard movement, this may be able to contain the disease, although, as stated above, the disease front might be several hundred kilometres ahead of the mortality front. Vectors may also transport the virus over the barrier, even if they are not normally an important method of transmission.

Juvenile pilchards, although susceptible to the virus in 1998/99, do not shoal with adult pilchards and live in coastal embayments. Mortality of juveniles was very limited in the 1998/99 event. It is suggested that preservation and separation of juvenile stocks would appear to be the most effective means of ensuring rapid pilchard recovery. This may be achieved by limiting fisheries based on juveniles and by ensuring as many pilchard nursery areas as possible are preserved as healthy environments. Murray *et al*., (2000) state "It is better, for this purpose, to preserve many ecosystems in reasonable condition than a few in pristine condition".

The most sustainable, long-term option for mitigating future pilchard mortalities is to preserve coastal embayments of particular significance to juvenile pilchards and limit or prohibit juvenile fisheries. Schooling clupeoid fish have highly variable population numbers and have been shown to increase threefold in just one year (Table 7.3) and are known to fluctuate by orders of magnitude (Southward *et al*., 1988; Kawasaki and Kumagai, 1984).

Table 8.12 Spawning seasons of Australian pilchards around the Australian coast. (Data from Fletcher *et al*., 1997.)

Location	**Spawning season**
Western WA	August and February
Albany	July and December to January
Bremer Bay	June to July
Eastern WA and Great Australian Bight	April to July
South Australia	February to March
Victoria	November
New South Wales	Summer (Nov-Feb), progressing north.

8.6 DISCUSSION

The improved national response to aquatic animal health emergencies is demonstrated by:

- The creation of the Aquatic Animal Health Unit and its participation in numerous committees,
- the publication of the Field Guide for aquatic animal diseases,
- federal budget funding for aquatic animal health issues,
- the creation of AQUAPLAN and
- new Australian Quarantine and Inspection Service import policies

These steps reflect and support the rapidly expanding and increasing value of Australia's aquatic animal industries. The development of AQUAPLAN will significantly improve preparedness and response capacity in the event of future aquatic animal disease emergencies.

In 1997, the Government published the booklet "Australian Quarantine - A shared responsibility: The Government Response". This publication details the Government's response to the Nairn Report (Nairn *et al*., 1996) and the National Task Force on Imported Fish and Fish Products (DPIE, 1996). The response includes detail of funding, acceptance or rejection of various recommendations of both reports and is a major commitment to maintaining and enhancing the quarantine system.

9 HYPOTHESES ON THE CAUSE, TRIGGER AND ORIGIN OF THE MORTALITIES

Objective: To identify and evaluate the hypotheses regarding the cause, trigger and origin of the pilchard mortality events.

This objective was achieved by reviewing papers and reports associated with the mortality events and analysing all the data and evidence presented in Sections 5 and 6. There was also much discussion with scientists and researchers involved in the investigations of the events.
The herpesvirus associated with gill lesions of affected pilchards in both mortality events is considered to be the most likely cause of the events. Some personnel involved with the 1995 mortality event still consider an upwelling event in March 1995 to be linked to the beginning of the mortality event, however this report considers the upwelling in 1995 irrelevant, although trigger factors may be involved. There are several pathways for introduction of an exotic pathogen to Australia's marine environment: ballast water, migratory seabirds, and imported baitfish. Alternatively the herpesvirus could have emerged from an enzootic source.

There are two main issues relating to the pilchard mortalities: 1) What was the aetiological agent; and 2) How did it happen? The origin of the infectious agent has sparked controversy over quarantine measures and is an important issue arising as a result of the pilchard mortalities.

Many hypotheses for the cause of the pilchard deaths were proposed during the early stages of the 1995 epizootic (see Table 9.1). A toxic chemical spill was quickly ruled out because this would have caused a multi-species mortality event and the pathology was not consistent with a chemical cause. The remainder can be grouped into two general hypotheses: an infectious agent or non-infectious (i.e. environmental) mechanisms.

Table 9.1 Some of the hypotheses proposed as a cause of the pilchard mortalities.

Viral agent.
Amoeboid agent.
Toxic chemical spill.
Water temperature change due to upwelling.
Physical damage by plankton eg. *Chaetoceros, Thalassiosira.*
Chemical damage by plankton eg. *Gymnodinium.*
Overpopulation (lemming effect). Evidence for regular occurrence of smaller fish kills when high populations observed.
Combined events. Possible combined effects of any of the following: upwelling (or other environmental anomaly), amoeba, dinoflagellate/diatom, viral agent.

9.1 CAUSE

9.1.1 Environmental mechanisms

There are two environmental factors implicated by various hypotheses: oceanographic factors and phytoplankton. The major oceanographic factor proposed as a cause was upwelling of cold, low-oxygen, deep oceanic water causing thermal shock. The issue of phytoplankton is discussed later.

Thermal shock has killed clupeid fish in the Mediterranean (Economidis and Vogiatzis, 1992) and in waters near British Columbia, Canada (Glavin, 1999). Sudden changes in water temperature, whether an increase or decrease, are known to be detrimental to fish health. Fish physiology is, in general, designed to cope with small and gradual temperature changes within specific ranges optimal to their survival. Most fish tolerate 1-2°C temperature changes on a daily basis (Smith *et al.*, 1996).

A detailed examination of environmental factors is given in section 5.2.7. The influence of upwelling, which has been much discussed in relation to the 1995 mortality event, is still considered by some to be an important factor in the initiation of the pilchard deaths. However, this hypothesis is not supported by the species-specificity of the mortality events. If the deaths were indeed due to some kind of environmental anomaly, it would have initiated a multi-species mortality and there is no evidence of this. Another fact that argues against the influence of environmental factors is that the same area of the western Eyre Peninsula has had more extreme upwelling events in previous years without associated pilchard mortalities. Appendix 6 contains satellite images of the Eyre Peninsula showing the upwelling event in March 1995 and compares it with upwelling events from previous years. There is also no consistent environmental anomaly that was detected around the entire coastline of southern Australia in March 1995.

In addition, the upwelling event was localised to the inshore area of the western coast of the Eyre Peninsula. Modeling by Murray *et al*. (2000) demonstrated that the virus had a minimum incubation period of 4 days. In 4 days, a school of pilchards may travel more than 700 km (given a pilchard school swimming speed of 7.5 km/hr, Fletcher *et al*., 1997). Therefore the initial mortalities at Anxious Bay, SA may have contracted the aetiological agent anywhere in a 700 km radius from the western Eyre Peninsula and may not have been exposed to the upwelling event.

Further, the cold water seen upwelling in the satellite images in Appendix 6 underlies South Australian shelf waters during summer (November – March), but is not seen by satellites as these only display sea surface temperatures. The presence of this cold water (~14°C) under the warmer surface water (~20°C) creates a thermocline at approximately 20 m to 60 m depth (Ward and McLeay, 1998). Pilchards are thought to occur above and below the thermocline (T. Ward, pers.com.) therefore schools of pilchards are exposed to this range of water temperatures in South Australian shelf waters. Exposure of schools of pilchards to the cool water brought to the surface by the upwelling event in March 1995 would not have been unusual for the fish and probably did not act as a trigger for a latent infection.

Examination of satellite images (Appendix 7) and other environmental data from the time of the 1998 outbreak, shows marine environmental variables to be within normal parameters (A. Cheshire, pers.com.). Because of this, environmental data has largely been ignored with reference to the 1998/99 mortality event.

There are five mechanisms by which phytoplankton can kill fish: mechanical damage, asphyxiation due to oxygen depletion, chemical toxicity due to ichthyotoxins, increased seawater viscosity due to secretion of mucilages, and gas bubble trauma due to supersaturation (Jones and Rhodes, 1994).

Physical damage to the gills of planktivorous fish by spiny diatoms, or by production of excess mucilage that clogs gills and suffocates fish, is not uncommon. Asphyxiation due to oxygen depletion is also not unknown, for example in New Zealand in December, 1993, pilchards died from asphyxiation due to an algal bloom in a partially enclosed marine environment (Jones and Rhodes, 1994). There are several known species of plankton which produce ichthyotoxic chemicals (eg. *Gymnodinium* spp.), but usually are found in extremely small numbers in the water and do not often induce fish mortalities on a large scale. When numbers become high enough to induce a fish mortality event, it is usually a multi-species mortality, sometimes also killing invertebrates.

The theory of "killer algae" was particularly popular in the media, in early April 1995, supported by the bloom of an ichthyotoxic phytoplankton in Coffin Bay, SA shortly before the pilchard deaths began, but this was an isolated multi-species incident and unrelated to the pilchard deaths. The theory of phytoplankton involvement was quickly dismissed when pilchards were examined and consistently found to have no phytoplankton in the gills. The histopathology was also inconsistent with phytoplankton-induced death in both mortalities (P. Hooper, pers.com.). Any physically damaging phytoplankton would probably also have affected anchovies, which are a similarly sized planktivorous fish.

The hypothesis of phytoplankton damage was not supported by the results of phytoplankton tows that were conducted in several States (and in New Zealand in 1995; Mackenzie and Todd, 1995) that showed no species that could be either physically dangerous to fish gills, or that were ichthyotoxic. Numbers were generally low and gut content analysis by Fletcher *et al*. (1997) showed no consistency in phytoplankton species within and outside areas where dead pilchards were observed.

9.1.2 Infectious agent

An infectious agent is the most likely cause of the mortalities because of the specificity of the deaths. Amoebae were found in some of the dead pilchards but were quickly dismissed as a possible cause because findings were not consistent across samples from the various affected States. In a parliamentary report on the pilchard fishery, Dr Harvey Westbury, from AAHL, said "the disease in 1995 acted like it was a virus in what we call an immunologically naive population". Other reports have expressed similar opinions that the disease swept through the pilchard population as if it were naive to the disease (Anon., 1999; Fletcher *et al*., 1997).

A herpesvirus was consistently found in affected pilchards in Australia in the 1995 and 1998 epizootics and in New Zealand in 1995. Other fish herpesviruses are consistently associated with epithelial hyperplasia, which was seen in the gill lesions of affected pilchards.

The pilchard herpesvirus is the most likely cause of both epizootics. The herpesviruses found in affected pilchards from both events are very similar and the lesions in the gills of affected pilchards from both events were also similar in appearance (P. Hooper, pers.com.). Herpesvirus particles were seen in the gill lesions, but not in other tissues. Although Koch's postulates have not been - and may never be fulfilled - the overwhelming coincidence of the presence of herpesvirus in the gills of all affected pilchards (and absence from unaffected pilchards) from all States and

New Zealand is hard to ignore. The presence of the herpesvirus is the most plausible hypothesis for causing the mortality events.

AAHL is continuing work to clarify whether the herpesviruses found in 1995 and 1998 are the same. Polymerase chain reaction analyses and DNA sequencing of the viruses may allow development of a pilchard herpesvirus-specific DNA probe. This probe may allow direct testing of stored pilchard gill material from the mortality events, instead of having to purify the virus first. If a successful DNA probe is developed, it would be used in the future to test imported baitfish, and in diagnosis and research if there is a future pilchard mortality event.

9.2 TRIGGER

The issue of a trigger is a moot point. One of the definitions of 'trigger' is given as: "An event that precipitates other events; to set off; initiate" (Source: *The American Heritage® Dictionary of the English Language, Third Edition*). This suggests that one factor could change in such a way as to solely contribute to the onset of disease, or to the initiation of a series of events leading to an epizootic. One theory is that the Coffin Bay, SA, multi-species incident, brought about by an algal bloom associated with upwelling, was in some way linked to the beginning of the pilchard deaths. The hypothesis is that the stress of upwelling and the algal bloom could have sufficiently lowered the resistance of the pilchards so that they became susceptible to the virus. This hypothesis relies on the proposal that once initiated in the pilchard population, the virus does not need further environmental events to sustain the epizootic. Data collected by all States demonstrated that there was no continuous or contiguous series of environmental anomalies that spread around the entire southern coastline of Australia. The data examined by Griffin *et al*. (1997) suggest that no environmental anomalies were significant enough to have been responsible for triggering the outbreak of the mortalities.

One difficulty with this hypothesis, with relation to the 1995 mortality event, is that the herpesvirus was new to the Australian pilchard population and the fish would have no immunity to the virus. It was likely therefore, that the epizootic would have occurred regardless of whether the fish were stressed by environmental factors. To date, triggers for the 1995 and 1998/99 events have not been identified.

There were no detectable environmental changes of any significance recorded in South Australian waters at the beginning of the 1998 mortality event which could have stressed the pilchards sufficiently to trigger another outbreak of the herpesvirus (see Appendix 7 for satellite image). However, if the virus had mutated or evolved like human influenza viruses, it could have caused disease although the population was healthy.

Environmental mechanisms capable of transporting an infectious agent against all prevailing currents and through storm events were not identified. The modeling by Murray *et al*. (2000) demonstrated that the deaths spread slower than the maximum swimming speed of pilchards and therefore the virus might have been spread by school-to-school contact rather than by an oceanographic factor. Insufficient data about the migration habits of Australian pilchard stocks is available to support this theory and research into migratory behaviour should be a high priority, especially as it

is known that South American and South African pilchard stocks undertake very large migrations each year (eg. Torres *et al*., 1985).

9.3 ORIGIN OF THE VIRUS

This is one of the most important issues arising as a consequence of the pilchard mortalities. The identification of herpesvirus particles raised many questions regarding their origin. Hypotheses regarding the origin of the herpesvirus (es) fall into two general categories: either the virus was introduced from overseas, or it was enzootic.

It is unusual that this particular virus has never been seen before. However, new diseases of marine organisms are observed each year as disease results from the global transport of host and disease organisms (Harvell *et al*., 1999). Emerging infectious diseases are driven by various factors including global transport, urbanisation, ecological manipulation, agricultural intensification, human encroachment and biomedical manipulation (Daszak *et al*., 2000).

Two major questions regarding the origin of the virus are: 1) If the virus was latent, then why have similar epizootics not previously been seen in Australia? 2) If the virus was introduced from overseas, why has the virus never been seen in any of the countries from which Australia imports pilchards? These issues will be discussed.

9.3.1 Enzootic hypotheses

There are three hypotheses that were proposed:

- The herpesvirus was latent in the population (Jones *et al*., 1997)
- The herpesvirus has transferred from another species (Jones *et al*., 1997)
- The herpesvirus mutated from a non-pathogenic virus (Jones *et al*., 1997)

9.3.1.1 Latent infection

There is circumstantial evidence to suggest that the infection was not latent. The fact that the population acted as if it were immunologically naive to the disease suggests that the virus had been introduced to the population recently. Although herpesviruses can cause latent infections (Roizman, 1996), a previous infection is a prerequisite for latency (Jones *et al*., 1997). Pilchards have a short life cycle. If there is no infection to shed viral particles and ensure the continuity of the virus then, within approximately 6 years, the virus would die out due to the absence of an infected generation of pilchards (B. Jones, pers.com.). Nevertheless, there is no direct evidence to support either the presence or absence of a latent infection. However, there is no example of a herpesvirus that lacks a latent cycle (Davidson and McGeoch, 1998).

During the last two hundred years of European settlement in Australia, pilchard deaths on such a massive scale have never been reported before. This argues against the hypothesis of a latent infection. If the infection were latent, it is likely that there would have been much more frequent events, like the ones seen in 1995 and 1998/99. The increase in human population during this time could have resulted in an undetectable anthropogenic trigger for a latent infection, although there is no evidence for this.

9.3.1.2 Species transference

The specificity of herpesviruses to their hosts (Hyatt *et al*., 1997)), particularly in non-salmonid fish (see Tables 4.2 and 4.3), makes it highly unlikely that the virus has transferred from another species. Before now, only two other herpesviruses have been identified in fish in Australia:

- A herpes-like virus associated with severe necrosis of the haematopoietic tissues of black moor (*Carassius auratus*) imported into quarantine in Australia (Humphrey, 1995).
- A herpes-like virus associated with mortalities of catfish (*Tandanus* sp.) (Humphrey, 1995).

These are both freshwater fish and are not closely related to pilchards, so it is highly unlikely that the herpes-like viruses infecting these fish could evolve sufficiently to become pathogenic for an entirely different species of marine fish.

9.3.1.3 Mutation from a non-pathogenic form

The hypothesis that the herpesvirus may have mutated from a non-pathogenic form does not exclude the possibility of recent introduction into Australia. A non-pathogenic form may have existed in Australian pilchards prior to the mortality events, however it is also possible that a non-pathogenic form was introduced through seabirds, ballast water or imported baitfish, and subsequently become pathogenic for Australian pilchards.

The emergence of a pathogenic virus from a non-pathogenic form has been described twice in terrestrial animals in the last sixteen years. A pathogenic mutant of Australian Newcastle Disease virus has recently emerged from a non-pathogenic form. (H. Westbury, pers. com.). This mutated virus spread quickly through a naive chicken population despite its non-pathogenic progenitor being prevalent in the Australian chicken population for many years without causing major problems.

Rabbit Haemorrhagic Disease Virus (RHDV) is the official name for the Australian rabbit calicivirus released to control rabbits in Australia in 1995. RHDV was first discovered in China in 1984 in Angora rabbits. In 1986, the disease was recognised in Italy and soon spread to most countries in Europe (Fenner and Fanitini, 1999). There were three possible sources of the virus but two were ruled out when Capucci *et al.* (1996) discovered a non-pathogenic calicivirus (called RCV) that produced seroconversion and protected rabbits against RHDV. This suggests that the calicivirus emerged from the non-pathogenic RCV. They state: "RHDV may have evolved from a virus already present in rabbits before 1984." Other evidence that strongly supports this suggestion includes a retrospective study of rabbit sera collected between 1975 and 1985, well before RHD became widespread in Europe, which "demonstrated the presence of anti-RHDV antibodies" (Rodak *et al.*, 1990).

The common factor linking the chicken, rabbit and pilchard viruses is the way they emerged suddenly and spread through apparently naive populations. Fantini and Fenner (1999) state that "viruses do not appear by spontaneous generation". The theory that RHDV and Australian Newcastle Disease evolved from non-pathogenic viruses gives credence to the theory that the same may have happened with the pilchard herpesvirus.

Analyses of herpesvirus gene sequences confirm a distant relationship between avian and mammalian isolates and indicate that mammalian and avian herpesviruses have evolved in parallel with their host species. Piscine herpesviruses on the other hand, display little or no genetic relationship to other herpesviruses. The typical herpesvirus morphology suggests that mammalian/avian and fish herpesviruses probably evolved from a common ancestor but the two groups have diverged so far that a genetic relationship between them is no longer detectable. There is no evidence to suggest that herpesviruses mutate and evolve any faster than other DNA viruses and although there are some data suggesting that interspecies transmission of herpesviruses has occurred in evolutionary time, such events are believed to be exceedingly rare (Davison and McGeoch, 1998; McGeoch and Davison, 1999).

These recent data do not prove or disprove the theory that the herpesvirus was introduced into Australian waters through one of the pathways mentioned previously. It is quite possible that a non-pathogenic herpesvirus specific to pilchards was introduced from overseas and conditions were such that they facilitated the virus evolving into a virulent strain. Alternatively the virus may have caused disease in Australian pilchards without mutating. It is also possible that the herpesvirus exists in overseas pilchard populations in an endemic form. The virus may have been present for a sufficiently long time that the pilchards are naturally immune and thus it is non-pathogenic for these populations and epizootics have not been observed. Further research would be required to elucidate the mechanisms involved.

9.3.2 Introductory hypotheses

Three pathways have been identified through which an exotic pathogen may be introduced into Australian waters:

- Migratory seabirds (Hyatt *et al*., 1997; Jones *et al*., 1997)
- Ballast water (Hyatt *et al*., 1997; Jones *et al*., 1997)
- Imported baitfish (Whittington *et al*., 1997)

9.3.2.1 Migratory seabirds

Introduction of the virus via seabirds is possible given that pathogenic fish viruses can be transmitted by birds and remain viable after travelling through the birds intestinal tract (Peters and Neukirch, 1986).

Unfortunately the diagnostic tools to test seabird blood serum, collected in WA during the 1998/99 mortality event, for herpesvirus antibodies, have not yet been developed. AAHL have the seabird sera in storage awaiting the time when these diagnostic tools can be developed. It may take several years to develop such tools and even when technology is at a stage when it is ready to undertake such tests, there are no positive controls to test against, so negative results may be misleading.

9.3.2.2 Ballast water

The issue of ballast water is discussed in greater detail in section 10.2.2.4. Ballast water is a potential entry point for numerous exotic marine pests and diseases. There are ~150 million tonnes of ballast water dumped into Australian waters every year from 8,000 vessels over 25m long (F. Michealis, pers.com.).

Ballast water is potentially responsible for introducing marine pests such as the Pacific sea star (*Asterias amurensis*) and has the potential to introduce more exotic marine organisms to Australian waters. One of the most highlighted in recent times is the dinoflagellate *Pfiesteria piscimorte*, a highly dangerous organism to both fish and humans, found on the northern Atlantic coast of North America. Another organism that is causing devastation in California at the present time is the algae *Caulerpa toxifolia*, which can establish very quickly in marine ecosystems and dominate large areas of the benthos in a short time. It is toxic to most sea life and can destroy coastal habitats.

A virus cannot be introduced in ballast water on its own, but must be transmitted by its host, or inside a neutral vector such as plankton. To date, the only known host for pilchard herpesvirus is *Sardinops sagax*. Pilchards feed largely on zooplankton which, due to lack of sunlight, could not survive for extended periods in ballast water tanks, so it is highly unlikely that pilchards could survive the long journey between international ports, despite the ballast water being changed mid-journey.

There is one record of a clupeoid fish (*Hyperlophus vittatus*) surviving in ballast water in Australia (Middleton, 1982). The captain of a bulk carrier that travelled between WA and NSW reported the occurrence of "literally millions" of small fishes swimming in the ballast water tanks. Although the captain considered this to be a rare event, it provides evidence that small fish can survive in ballast water for "long periods" (Middleton, 1982).

The discovery of a herpes-like virus in phytoplankton (*Platymonas* spp.; Pearson and Norris, 1974) and a similar agent in American oysters (*Crassostrea virginica*; Farley *et al*., 1972) led to the speculation that marine algae, phytoplankton in particular, may act as vectors for diseases of marine animals. If so, although no zooplankton species has ever been discovered with a herpesvirus, it raises the possibility that planktonic organisms could be neutral refuges for viruses in ballast water. This would allow the transport of a pathogenic agent between international ports independent of the host.

The large size of the Sydney and Melbourne ports may make them more likely places for introduction of exotic diseases through ballast water, than South Australian ports. Also, if ballast water were the pathway for introduction then outbreaks of pilchard mortalities would have jumped between ports. However, if a ship emptied all its ballast at one port, this may contain enough viral particles to start the mortality and not see outbreaks at other ports.

Research into testing ballast water is significantly under-funded, relying on funds raised from a shipping levy introduced in 1998. If a project were to find just one live pilchard in ballast water from foreign waters, the project would be justified and would highlight this issue and indicate that further research should be undertaken. It is a highly plausible mechanism for exotic disease agents, carried in hosts, to enter Australian waters, and warrants further investigation.

9.3.2.3 Imported fish

This issue has received a lot of attention. The pilchard mortalities suddenly alerted many agencies and authorities to the possibility of exotic marine disease incursion

through unregulated imports of whole fish for human consumption, bait or for feed in aquaculture.

It is impossible to know whether the virus was introduced through imported baitfish. One fact that suggests that imported baitfish were involved is that both epizootics began in SA, where large quantities of imported pilchards are used to feed tuna. Tuna are kept in sea cages between January, at the very earliest, to August-September of the same year (B. Jeffriess, pers.com.). At the time of the 1998 outbreak, there may not have been any tuna in the pens at Port Lincoln, hence it was unlikely that pilchards were going into the water as feed at that time.

WA have imported baitfish of a dozen different species, for the rock lobster industry, from all over the world for 17–22 years without strict quarantine measures and without incident (Jones and Gibson, 1997). SA had only imported pilchards in large quantities as tuna feed for 2-3 years before the epizootic in 1995 occurred. The major difference between WA and SA is the way the imported fish are used. In WA, the fish enter the water one at a time in lobster pots distributed over a wide area, whereas in SA the fish are put into the water in large amounts in a small area. This difference in risk between the two industries has been noted by Jones (2000). The OIE's International Aquatic Animal Health Code states that, in general, the larger the volume of any given commodity traded in a specified time, the higher the potential risk of a disease incursion. According to this rationale, SA became potentially more vulnerable to disease incursion as the volume of imported pilchards placed into the marine environment increased, particularly given the relatively restricted locality of tuna aquaculture operations.

The use of imported fish as aquaculture feed is a potential route of entry for a virus to enter Australian waters, especially since herpesviruses can, depending on the conditions, survive freezing. If future technology allows detection of pilchard herpesvirus in imported baitfish, it will support the contention that the original introduction took place via such imports.

9.4 SCENARIOS

These scenarios are designed around the four pathways for a virus to enter Australian waters and incorporate the possibility that the upwelling event of 1995 may have influenced the disease process. The scenarios are all based upon events in 1995.

9.4.1 Seabirds

Migrating seabirds returning from overseas bring with them an infectious fish herpesvirus from feeding on foreign pilchards. The birds defecate in the ocean whilst feeding on a school of pilchards in the Great Australian Bight. The still viable virus comes into direct contact, via the bird faeces, with an entire school of Australian pilchards. Then either the Australian pilchards are naive to the virus and thus are susceptible to disease, or due to introduction to a new ecosystem (Australian pilchards), environmental influences select a sub-population of viruses from the quasispecies (Domingo, 1999), which is subsequently pathogenic for Australian pilchards. The concept of quasispecies is used by virologists “to describe dynamic distributions of non-identical but closely related mutant and recombinant viral genomes subjected to a continuous process of genetic variation, competition and selection and which act as a unit of selection” (Domingo, 1999).

Before the pilchards become moribund, they travel to the western Eyre Peninsula to feed on large amounts of zooplankton which have proliferated due to the nutrient-rich upwelling. Here two things could have happened: either they simply start dying and are blown onshore, or the thermal shock of the upwelling event speeds up, or in some other way aids, the disease process so that the pilchards still die but perhaps quicker or in larger numbers than if there had been no upwelling event. The upwelling may also have made another school of uninfected pilchards more susceptible to the virus, by lowering their immunity and allowing propagation of the virus in this naive pilchard population.

9.4.2 Ballast water

An international shipping vessel dumps its ballast water at a port in the Great Australian Bight or in SA. The ballast water has transported live pilchards from another part of the world and they are released into the Australian marine environment. These foreign pilchards come into contact with Australian pilchards anywhere in the Great Australian Bight and subsequently transmit to Australian pilchards a viable herpesvirus, which for the foreign pilchards is non-pathogenic. The same two possibilities as above also exist in this scenario that (1) the virus is immediately pathogenic to the pilchards or (2) the virus evolves and the scenario proceeds similar to the seabird scenario.

9.4.3 Imported baitfish

In mid-March, tuna farmers are daily feeding their tuna tonnes of frozen pilchards. A batch of imported pilchards from overseas contains pilchards that have a herpesvirus, either pathogenic or, more likely, non-pathogenic for the foreign pilchards. It is not known how close schools of pilchards will approach cages of tuna, but the pathway for infection requires close contact. When the tuna eat the frozen pilchards, particulate matter is dissipated into the surrounding water and not all the feed will be consumed. Dead pilchards break down very quickly; water currents will pick up so particulate matter from the fish that fall to the bottom and from tuna faeces. The wild pilchards may feed on this matter or inadvertently swim through it and thus come into contact with infective tissue. It is important to note that contact with the virus is not the only requirement for infection, but the pilchards must receive a high enough dose of viral particles for the virus to become infective.

Again the possibility exists that the pilchards subsequently travel to the western Eyre Peninsula where they are or are not affected by the upwelling event.

9.4.4 Mutation from a non-pathogenic virus

Although this hypothesis has been incorporated into the previous scenarios, the option of a non-pathogenic virus previously existing in Australian pilchards cannot be ruled out. There are two possible options: (1) Australian pilchards carried a non-pathogenic herpesvirus for millions of years, which by some evolutionary change or mutation (possibly influenced by environmental factors) became pathogenic, or (2) an exotic herpesvirus was introduced by any of the methods described above and then became infectious for Australian pilchards.

Further research is required to determine whether any of these scenarios was applicable to the pilchard mortality events.

10 IMPLICATIONS FOR INDUSTRY AND REGULATORY AGENCIES

Objective: To assess the implications of hypotheses regarding the cause, trigger and origin of the pilchard mortality events for the agencies and industries responsible for the management of Australia's marine ecosystems and resources.

This objective was achieved by examining the changes made by government agencies and industry as a result of the mortality events. There was much discussion with personnel involved with investigation of the events and with implementing the changes that resulted from the mortality events.

There is a fear that imported pilchards could be banned in the future, which would have serious implications for the tuna farming industry in South Australia. However under the World Trade Organization's Sanitary and Phytosanitary Agreement, it is unlikely that Australia could justify a prohibition of pilchard imports based solely on the pilchard herpesvirus. In cases where the available scientific information is insufficient, Article 5.7 of the SPS Agreement allows a WTO Member to base provisional sanitary measures on available information, for example in cases of emergency. In such cases, Members must seek to obtain the additional information necessary for a more objective risk assessment, and must review the measure, within a reasonable period of time, in accordance with that new information. Such interim measures would generally only be applied where a specific hazard is identified in an exporting country that is not present in an importing country and about which little technical information is available.

The tuna farming industry is aware of its dependence on one major food source (pilchards) and has for the last two years been working with the Aquaculture Co-operative Research Centre to develop artificial pellet feeds to replace the use of imported fish.

In 1999, the Australian Quarantine and Inspection Service undertook an import risk analysis for marine finfish products, as recommended by the National Task Force on Imported Fish and Fish Products (DPIE, 1996) and subsequently implemented new quarantine requirements for finfish products. The use of imported pilchards for tuna feed continues to be allowed under the new quarantine requirements.

The pilchard mortalities have not resulted in changes to procedures for the management of ballast water but have raised the profile of the potential for entry and establishment of exotic pathogens due to incursion by exotic marine organisms.

The Department of Agriculture, Fisheries and Forestry – Australia created the Aquatic Animal Health Unit, which instigated AQUAPLAN (1998-2003) in 1999. It is a five-year plan with eight programs relating to aquatic animal health in Australia. Part of the plan is to design a series of manuals that detail the appropriate management, including exact lines of communication and response options in the event of an aquatic animal disease emergency.

10.1 ECONOMICS

10.1.1 Introduction

In addition to the direct negative impacts on fisheries, fishers and retailers, there are other potential economic impacts that must be considered. There has been much attention focussed on the imported pilchards used for feeding farmed tuna in Port Lincoln. Tuna farming has brought substantial economic stability to the Eyre

Peninsula region of South Australia so impacts on income, flow-on effects and employment will be examined.

The reliance of the tuna farming industry on pilchards for tuna feed is also the focus of much attention and is of particular concern to the aquaculture industry, government departments and research scientists. Research into the use of alternative fish species and the development of alternative feeds is discussed.

10.1.2 Potential economic impacts of a quarantine ban on imported pilchards

It has been suggested that the herpesvirus identified in the pilchard gills may have been introduced via the feeding of imported pilchards to farmed tuna (Fletcher *et al.*, 1997). The National Task Force on Imported Fish and Fish Products (NTFIFFP) identified the use of imported pilchards for tuna feed as a high risk activity for which risk reduction measures should be undertaken (DPIE, 1996). Imported pilchards are also used as recreational fishing and lobster fishing bait, but not in such large quantities or in as concentrated a manner as for tuna. For 2000/2001 the tuna farming industry is predicting to export 7000 tonnes of tuna worth around $255 million (B. Jeffriess, pers. com.). The direct value added generated in the Eyre Region by the industry in 1998/99 was $98 million (EconSearch, 1999). If no substitute feeds were found, a ban on imported pilchards would significantly reduce the tuna farming industry, and the flow-on benefits to other businesses.

The Australian Bureau of Agricultural and Resource Economics (ABARE) wrote a minute and a full report (Thorpe *et al.*, 1997) modeling the impacts on the tuna farming industry of a ban on pilchard imports. The model predicted that if the minimum price for a tuna feed substitute (herring, mackerel, pellet feed) is greater than the pilchard import price of $870/tonne (1997 price, currently $A732/ tonne (from the USA), Tasmanian mackerel minimum $A900/tonne), there would be a negative impact on tuna farmers and other pilchard users. The modeling (Thorpe *et al.*, 1997) predicted that, over the next twenty years, pilchard fishers would be better off, with an increase in the price of domestic pilchards. However, the total reduced economic welfare of tuna farmers, related industries and flow-on businesses would substantially outweigh the marginal increase in domestic pilchard profits.

If Australia wished to implement a quarantine ban or alternative sanitary measures on imported pilchards, there are three criteria that would need to be fulfilled under the SPS Agreement of the World Trade Organisation:

1. it must be demonstrated that specific disease(s) of quarantine concern to Australia is/are likely to be present in the imported commodity;
2. it must be demonstrated that the animal/plant population at risk in the importing country (Australia) are free of the specific disease(s);
3. it must be shown that a pathway for transmission of the disease(s), from the imported commodity to the population at risk, exists in the importing country.

Alternative sanitary measures could also be applied (eg evisceration) that may effectively provide an equivalent level of biosecurity to prohibition but again would need to be based on a demonstrated risk.

In cases where the available scientific information is insufficient, Article 5.7 of the SPS Agreement allows a WTO Member to base provisional sanitary measures on

available information, for example in cases of emergency. In such cases, Members must seek to obtain the additional information necessary for a more objective risk assessment, and must review the measure, within a reasonable period of time, in accordance with that new information.
At the present time, the Australian pilchard population is the only pilchard population in the world known to host a herpesvirus. This fact would make it extremely difficult for Australia to sustain either a prohibition or alternative sanitary measures on imported baitfish on the basis of pilchard herpesvirus.

10.1.2.1 Employment.

Port Lincoln and the Eyre Peninsula region are heavily dependent on the tuna industry for jobs and it is the basis of a growing local economy. The industry directly employs over 580 people and has created approximately 800 other jobs in flow-on business activity. The direct output generated by tuna farms in the Eyre Peninsula region was over $166 million in 1998/99, with flow-on effects to other sectors adding over $90 million in business income (EconSearch, 1999). If tuna farming suddenly ceased, industry employees would lose their jobs, regional employment would fall significantly and the regional economy would be severely affected.

10.1.3 Alternative tuna feeds

The reliance of the tuna farming industry on imported pilchards means that it is vulnerable to market prices fluctuations, supply and changes in Australia's quarantine policy on baitfish. For example, in 1998 thousands of tonnes of imported pilchards were delayed due to industrial action, and SBT farmers were forced to ration their feed supplies (Glencross *et al.*, 1999). There is also an awareness that artificial feeds can increase the health of the fish and the environment, and the colour of the flesh, which makes the tuna more attractive to the Japanese market. Therefore using a manufactured feed would give the industry greater control over the quality of the product (Glencross *et al.*, 1999).

Important elements for a tuna feed are a high fat content, such as that of pilchards, as well as moisture content and freshness (S. Clarke, pers.com.). Fish that can be swallowed whole are preferred because fish that must be cut into portions increase waste in the water and leach oil.

Other species of imported baitfish such as North Sea (Baltic) herring (*Clupea harengus*) can provide a suitable alternative to pilchards (S. Clarke, pers.com.), but their use is largely dependent on price. In conducting its import risk analysis, the Australian Quarantine and Inspection Service (AQIS) considered the risk of entry and establishment of viral haemorrhagic septicaemia virus (VHSV) in Australia via the use of North Sea herring as bait or fish feed, as unacceptably high. Herring are a clupeoid fish and therefore related to pilchards. VHSV was the cause of a pilchard mortality event on the Pacific coast of Canada in 1998 (Glavin, 1999). However, this event happened under exceptional circumstances due to abnormal northward migration of pilchards followed by sudden exposure and entrapment of the pilchards in a body of cold water (<10ºC). VHSV causes disease at water temperatures of 1-12°C but not above 15°C (Kahn *et al.*, 1999).

Coastal water temperatures around Australia are generally considerably warmer than those required for transmission of VHSV. Accordingly, herring (and other species capable of carrying VHSV, eg. *Clupea* spp.) may be used as aquaculture feed and bait in Australia subject to complying with quarantine requirements. In Western Australia, herring can be used as rock lobster bait all year round (the fishery is closed between July-October) because the mean daily water temperature (>18ºC) is too warm to allow transmission of VHSV. In South Australia, herring may only be used as tuna feed between December and April. At this time, the mean daily water temperature in Boston Bay SA is consistently 17ºC or greater thus precluding the transmission of VHSV. Herring are utilised by the tuna farming industry as a supplement to pilchards during these months. The industry also uses a small amount of imported squid to supplement the tuna diet (B. Jeffriess, pers.com.).

The Aquaculture Co-operative Research Centre is one of the largest of the Co-operative Research Centres around Australia. Under their Nutrition and Feed Development Program, one specific project aims at developing and evaluating cost-effective feeds for the farming of SBT. The aims of the project can be viewed at the Aquaculture Co-operative Research Centre website:
http://www.aquacrc.uts.edu.au/index.html

The Fisheries Research and Development Corporation (http://www.frdc.com.au/) and the tuna industry have funded research and development trials of pellet feeds for tuna at Port Lincoln in 1998. The results were encouraging but leave room for significant improvement. An article by Glencross *et al*. (1999) details the trials and the following information is summarised from the paper. Tuna were fed one of three diets: pilchards, dry pellets or moist pellets. After 16 weeks, growth of fish fed the moist pellet diet was slightly, but not significantly, better those on the pilchard diet. In addition, survival was significantly better than tuna fed either the dry pellet or pilchard diets. The feed conversion ratio (FCR) measures the kilograms of feed required to grow one kilogram of fish. The FCR for the moist pellet was 8:1, whereas the pilchard FCR was 13:1. At these rates, feed costs are approximately the same because the pellets are more expensive than pilchards. The researchers state “If a moist-pellet feed that had a FCR of 7:1 or lower was developed, then feed costs would favor a change to moist pellets as the preferred feed for the industry." The researchers suspect that the differences in FCR are due to the different protein to lipid ratios in the different feed sources (Table 10.1). Differences in moisture content of the two diets may also affect acceptability and ultimately the intake of total dry matter.

Table 10.1 Protein, fat and moisture contents of three feeds used in tuna trials. Values are means ± SEM of proximate analysis based on 3 to 7 samples (from Glencross *et al*., 1999).

	Pilchards	Dry pellets	Moist pellets
Protein (%)	17.7 ±1.8	46.4 ± 1.1	35.0 ±1.1
Fat (%)	7.4 ±2.6	13.1 ±0.6	12.2 ±1.0
Moisture (%)	68.9 ±2.1	6.0 ± 0.3	38.4 ±0.8

The researchers state that although pilchards support good growth, the possibility of disease transmission remains. This issue is relevant to all finfish grown in aquaculture and fed on other fish. The superior health benefits, seen in increased survival, may already favour moist pellet technology. The researchers are confident that moist

pellets offer considerable potential as a replacement for pilchards in the near future. Further research will focus on improving the pellet formulation by increasing the lipid content of the moist pellet and examining the effects of minor feed restriction. An increase in dietary lipid, as an alternative to protein, as an energy source and adoption of restricted feeding regimes will help reduce any negative environmental impacts of the industry.

The issue of transmissible spongiform encephalopathies and the growing ban on the feeding of animal-derived meals to related species may also impact eventually on aquaculture.

There is the potential for substantial replacement of imported baitfish with locally-resourced under-exploited small pelagic fish, for example anchovy (*Engraulis australis*), jack mackerel (*Trachurus declivis*), saury (*Muranosox saurus*) and blue mackerel (*Scomber australasicus*). The costs of local fish are slightly higher, but tuna farming is highly lucrative, and local fish would carry none of the disease risks perceived to be associated with imported fish. Before the use of species other than pilchards are accepted by the aquaculture industry, it would be necessary to undertake detailed biochemical analyses of their nutritional quality and assess their general suitability for use as tuna feed. The diet of wild SBT consists of a wide variety of prey including fish, squid, crustaceans and macrozooplankton (Young *et al*., 1997), so a more varied diet for farmed tuna may prove beneficial.

10.2 TRADE AND QUARANTINE ISSUES

10.2.1 International obligations

As a member of the World Trade Organization, Australia is required to ensure that quarantine measures applied to protect animal, plant and human health are consistent with guidelines provided in the Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement). The SPS Agreement provides member countries the right to determine the sanitary and phytosanitary measures they wish to apply to protect animal, plant and human health within their borders. Measures must be based either on international standards or scientifically based import risk analysis. The SPS Agreement also requires that sanitary and phytosanitary measures are applied in a consistent manner that minimises trade restrictions and does not arbitrarily discriminate against imported products.

Assuming a herpesvirus caused each of the pilchard mortality events, even if the virus was introduced through imported pilchards, it is unlikely that AQIS could justify a quarantine ban on imports primarily because at this time there is no evidence that the pilchard herpesvirus is present in overseas fish stocks. Furthermore, it is likely Australia would be deemed to have the pilchard herpesvirus and Australia does not apply any official controls on the movement of domestically caught pilchards with respect to this disease agent. Application of a quarantine prohibition or alternative sanitary measures on imported pilchards due to pilchard herpesvirus would therefore not be supported by the available scientific information and would be deemed by the WTO to arbitrarily discriminate against imported products.

Australia's obligations as a WTO member appear to somewhat conflict with a guiding principle of the National Strategy for Ecologically Sustainable Development, known as the Precautionary Principle. The Precautionary Principle indicates that "where

there are threats of serious or irreversible environmental damage, lack of full scientific certainty should not be used as a reason for postponing measures to prevent environmental degradation." Under SPS guidelines, a precautionary approach may be adopted in the absence of available scientific evidence, through the application of interim sanitary measures. The implementation of such interim measures does however appear to be restricted to circumstances where a specific hazard of quarantine concern to an importing country is identified in an exporting country about which little technical information is available with respect to disease prevalence, epidemiology and the susceptibility of the disease agent to inactivation.

10.2.2 Methods for entry and establishment of disease

This section will explore the two main potential pathways for exotic disease introduction through international trade – (1) imported aquatic animals and aquatic animal products, and (2) ballast water. It will also review the new AQIS import conditions for whole, frozen marine fish for aquaculture feed, implemented last year as a result of an import risk analysis.

Dumping of ballast water is an important issue for Australia, which has a diverse and unique marine fauna, and is a mechanism by which incursions of marine pests have occurred. It is possible that these introduced marine species could carry exotic pathogens, so prevention of incursions of marine pests is one way of also preventing exotic disease incursion. Management of ballast water is the responsibility of AQIS and various mechanisms are slowly being introduced to minimise the risk posed by ballast water. These mechanisms will be discussed in this section.

For a disease to be noticeable at the population level, the causative agent must be present in sufficient numbers and be passed on to other members of the community at a rate that would allow the disease to become established. If a mechanism to transmit the infection between hosts does not exist in the new environment, then the host will become diseased and die before being able to pass on the infection. This means that not only does the population have to become infected but the means to sustain and transfer the infectious agent must also be present (Jones and Gibson, 1997).

10.2.2.1 Imported aquatic animals and aquatic animal products

Australia is free of many of the major diseases of fish and aquatic invertebrates and this status is a "national asset worthy of protection" (Humphrey, 1995). Australia's disease-free status is continually threatened by foreign imports. Importation of live aquatic animals presents the greatest risk of disease introduction to aquatic animals. Humphrey (1995) states "the use of live fish for bait or for food for carnivorous fish constitutes a major risk of disease transmission, and of introduction of exotic disease if the live species is imported". He also states "A high risk of exotic disease incursion exists in circumstances whereby live [imported] fish, aquatic invertebrates, or products or wastes derived from such animals are directly exposed to a natural, open aquatic environment containing susceptible host species".

Whole, frozen fish used as bait, or to feed high-value species, present a high risk as vehicles of contamination because they are unprocessed (Hine and MacDiarmid, 1997). The risk of introducing gastrointestinal parasites and pathogens can be reduced by gutting and heading fish, and by extracting the kidney, liver and spleen, because these organs are major sites of viral and bacterial infection. There are further body

parts that can be removed to reduce infections of the nervous system and haemorrhagic septicaemias, however, viral and bacterial pathogens may remain in fillets. Any extra work to process fish will, of course, incur increased production costs and increase the overall price of the commodity.

The NTFIFFP identified a number of imported aquatic commodities, including pilchards for tuna feed, for which risk reduction action was deemed desirable (DPIE, 1996). The 1995 pilchard mortality event occurred just before the NTFIFFP was established, adding weight to concerns of a potential deficiency in quarantine policy with regard to risks presented by imported fish and fish products.

Baitfish species have been imported into Australia as rock lobster bait for at least 22 years, apparently without incident. Quantitative data on the importation of pilchards are lacking but it is recognised there has been a substantial increase in the use of imported pilchards in association with the expansion of tuna farming off Port Lincoln. In 1995, the quantity of pilchards imported for tuna feed was estimated at approximately 16,000 tonnes and approximately 18,000 tonnes in 1998. The quantity of pilchards imported for use as tuna feed in 1999/2000 is estimated at approximately 45,000 tonnes (I. Peebles, pers.com.). The apparent significant increase in imports in 1999/2000 suggests that earlier estimates were conservative but may also be due to a combination of expansion in the tuna farming industry and lack of availability of Australian pilchard stocks.

Pilchards have predominantly been imported from the United States but imports have also been sourced from Mexico, Japan, Spain and northern Africa.

10.2.2.2 Import Risk Analysis (IRA)

Quarantine policies for aquatic animals and aquatic animal products are intended to minimise the risk of exotic disease introduction and establishment while complying with Australia's international obligations. Under the auspices of the SPS Agreement, member countries of the WTO are required to have quarantine policies that are either based on recognised international standards or supported by scientifically based import risk analyses. WTO members are obliged to be consistent in their quarantine decision-making with respect to imported commodities. The use of risk analysis is a means of ensuring such consistency.

AQIS is the Commonwealth agency responsible for the development and implementation of quarantine policy in accordance with Australia's international obligations. The Quarantine Act 1908 (and subordinate legislation) provides the legislative powers under which AQIS administers its quarantine functions.

In conducting a review of Australia's quarantine policies and administration the Nairn Report (1996) outlined seven fundamental principles that should apply to an IRA process:

- It should be a clear and understandable process (transparent)
- There must be consideration of all relevant scientific information
- There should be a review of scientific assessment by peers
- Consultation with all affected parties is essential

- There should be clear and effective liaison between Quarantine and Environment Agencies
- The decision maker should be visibly independent
- There should be a consistency in decisions.

Hine and MacDiarmid (1997) reiterate that "Importation decisions should be based on the outcome of risk analyses". They comment that by "breaking down the overall risk into various components (…), a risk assessment is designed to focus attention on the specific criteria which must be satisfied before a disease introduction occurs." Risk analysis, regarding the likelihood of disease incursion, takes into account "the prevalence of pathogens in the population from which the finfish products are derived, the probability of their surviving in the product during the process of importation, the probability of the pathogen coming into contact with local fish stocks after importation and the repercussions of such contact."

Jones (2000) stated that the most neglected part of the risk analysis process is an evaluation of the effect of the Sanitary and Phytosanitary measures that might be applied to reduce risk. The author states "It is necessary to explicitly evaluate the relative effectiveness of the measures applied in reducing the overall disease risk. For diseases about which little is known this is a very difficult exercise, and especially so when heading and evisceration is not an option". No single measure is a guarantee of risk minimisation, so it is suggested that each measure needs to be assessed individually to analyse its effectiveness in reducing risk. The IRA guidelines given by the Office Internationale des Epizooties are now under review to address these flaws. The evidence-based guidelines for import risk analyses provided by the SPS Agreement mean that biosecurity risks presented by as yet unidentified hazards may only be managed in a passive manner, (ie if risk management measures for an identified hazard are also effective in minimising risks associated with an unidentified hazard).

AQIS IRA for non-salmonid marine finfish

In July 1999, AQIS completed an IRA on non-viable salmonids and non-salmonid marine finfish (Kahn *et al.*, 1999). The IRA covered a wide range of fish and fish products and included a comprehensive review of relevant scientific literature. Following completion of the IRA, new quarantine requirements for non–salmonid finfish were implemented in November 1999. Under the new quarantine requirements the importation of pilchards for use as bait or tuna feed continues to be allowed subject to each consignment being accompanied by appropriate health certification. The IRA and resulting import policies can be found on the AQIS website under http://aqis.gov.au/import/index.htm.

AQIS ensured the scientific validity of the risk analyses by considering the reports of consultancies on identified gaps in information relating to these risk analyses. AQIS also contracted 14 independent scientists in Australia and overseas, to review the draft reports.

To ensure that the process fulfilled the Government's commitment to a transparent, open and consultative approach to import risk analysis, AQIS held public meetings in five capital cities and two meetings of key stakeholders. AQIS also made each chapter of the draft reports available to the public for comment.

Western Australian IRA

In 1997, the Western Australian Fishing Industry Council (WAFIC) produced the report *Risk Analysis for the Practice of Importing Frozen Fish as Bait* (Jones and Gibson, 1997). WAFIC undertook this IRA to "specifically address the risk posed to the marine environment by frozen bait imported into Australia from New Zealand and Europe." The methodology used was modified from the Guidelines for Risk Assessment issued by the Office International des Epizooties in the International Aquatic Animal Health Code. It examined in detail numerous factors; the main issues are given in Table 10.2. They examined data relating to 12 different imported fish species. This IRA did not specifically address risks associated with the importation and use of pilchards (*Sardinops sagax*) for rock lobster bait, as pilchards are not an important baitfish species for the rock lobster industry.

Table 10.2 The main issues covered by the WA import risk analysis (Jones and Gibson, 1997).

1.	Assessment of the disease risks associated with the importation from New Zealand and Europe of wild-caught, frozen fish for bait usage by the rock lobster industry.
2.	A discussion of risk reduction measures available.
3.	A socio-economic assessment of the potential impact on Australia of a reduction in rock lobster bait imports, caused either by disease or by a policy change.
4.	The nature of the commodity to be considered for import.
5.	The risk of disease entry and establishment as a consequence of importation.
6.	The consequences of disease entry and establishment.
7.	The measures that may be taken to reduce the level of risk and/or consequences.

The WAFIC IRA has no regulatory standing in terms of Australia's quarantine policy. It is a science based risk analysis that supports the position of the WA rock lobster industry that the risk of entry and establishment of an exotic disease via the importation of baitfish for rock lobster bait is sufficiently low that the practice should not be banned.

Jones and Gibson (1997) identified a number of criteria for a micro organism to become established in a new environment from a bait source.

The micro organism must:

- be present in the waters of origin
- be present in the fish caught to be processed as bait
- be present in the imported bait
- be present in the bait tissue when it passes import inspection
- survive storage
- be taken in by a susceptible host in sufficient quantity to create an infection

The authors observe:

> "since 1979 we have inadvertently conducted a large scale experiment off Western Australia where around 5 million pots have been set with imported

bait and 5 million with locally-sourced bait. The absence of any fish mortality event during this period suggests that the results from these two groups are indistinguishable. This experiment has been repeated 17 times with the same results each year leading us to draw the conclusion that the risks posed by imported bait, if they exist, are no different from those posed by locally sourced bait."

The authors comment "either the risk [from imported baitfish] is relatively high, and the fact we have observed no incident in 17 to 22 years is a lucky quirk of nature; or that the risk is very low and the absence of a fish kill is to be expected". Their final conclusion says they "cannot conclude that there is no risk of introducing an exotic disease, only that the risk of introducing an exotic disease which is capable of producing a large scale fish kill is either very low or does not exist at all".

It must be emphasised that the above statement referred to baitfish imported for use as rock lobster bait and that the WAFIC IRA did not pertain to pilchard volumes or feeding practices associated with the tuna aquaculture industry.

10.2.2.3 Import conditions

The pilchard mortality of 1995 pointed to a gap in quarantine policy with regard to imported fish and fish products that the Australian Quarantine and Inspection Service (AQIS) had previously identified in 1992 (DPIE, 1996).

Following completion of the IRA on non-salmonid finfish, AQIS implemented new quarantine requirements for the importation of baitfish in November 1999. The overall purpose of the measures is to strengthen Australia's fish quarantine laws in a way which limits the quarantine risk to a level that is acceptably low and complies with Australia's international obligations.

Pilchards (*Sardinops* spp., *Sardinella* spp., and *Sardina* spp.) for use as bait or as aquaculture feed are classified by AQIS under 'Finfish – whole (specified and non-specified species)'. The fish may be partially processed (eg. head-on, gutted product), or unprocessed (eg. whole, round fish). The condition, number C9156, can be found on the AQIS website.

Each consignment of frozen fish must be accompanied by a valid import permit and Quarantine Entry. AQIS has classified fish into specified and non-specified species, based on the quarantine risk each group represents. Permits will generally only be granted for non-specified species, which includes all pilchard and sardine species. An import permit is usually granted for non-specified species, based on the provision of specific information. Additional conditions apply to fish on the specified species list, which are considered high risk. The following conditions apply to imports of non-salmonid finfish from all countries except New Zealand.

> "The importer must obtain a permit to import non-salmonid product into Australia from the Director of Animal and Plant Quarantine (herein called the Director) prior to the product first being imported.

Unless otherwise specified, each consignment must be accompanied by an official certificate in English and, if required, the language of the exporting country providing the following information/attestation:

- source of the fish and confirmation that the fish or fish from which the product is derived were wild caught;
- identification of fish species (scientific name and common name) in the consignment;
- that the consignment does not contain other fish species;
- that the fish were processed in premises approved by and under the control of the Competent Authority;
- that the fish were inspected under the supervision of the Competent Authority; and
- that the product is free from visible lesions associated with infectious disease.

The certificate must bear the name(s), address(es) and approval number(s) of establishment(s) at which the finfish were processed and the name and address of the consignor and the consignee. The certificate must be signed by a person authorised by the Competent Authority and bear an impression of the official stamp on each page."

For pilchards (and other baitfish) imported from New Zealand the following conditions apply:

"An import permit is not required for the importation of non-salmonid finfish product originating in and exported from New Zealand.

Consignments exported to Australia must be accompanied by MAF certification that the fish was caught in New Zealand's EEZ or adjacent international waters.

The certificate must bear the name(s), address(es) and approval number(s) of establishment(s) at which the finfish were processed and the name and address of the consignor and the consignee. The certificate must be signed by a MAF-authorised person and bear an impression of the official stamp on each page."

10.2.2.4 Ballast Water

Infected 'exotic' pilchards could have inadvertently been introduced into Australia's coastal waters via discharge of ballast water from ocean-going vessels (Jones *et al*., 1997; Ward *et al*., 1999). It is known that fish can survive the journey between international ports in ballast water and subsequently populate a new environment.

The history of research into ballast water goes back to the early 1900s, when organisms such as diatoms, hydroids, isopods and crabs were thought to be carried to other countries on the hulls of ships or in their ballast (Carlton, 1985). From the late 1960s during construction of the Panama Canal, there was much debate on the potential biological and ecological changes that might result from the exchange of ballast between the Atlantic and Pacific sides of the Canal. In 1973, the MARPOL Convention was drawn up, part of which required research into ballast water as a transport mechanism for marine organisms.

Management of biosecurity risks presented by discharge of ballast water is primarily focussed on preventing the entry and establishment of microscopic organisms (*eg* toxicogenic bacteria, dinoflagellates, diatoms) and aquatic invertebrates (eg black-striped mussel, *Mytellopsis sallei*) that have potential to cause adverse ecological/economic consequences.

The likelihood of pilchards being taken up in ballast water appears low (Ward *et al.*, 1999) given that pilchards are typically an evasive pelagic species (i.e. are unlikely to school in the vicinity of a vessel berthed at port). In addition, the ability of pilchards to survive in ballast water for an extended period of time is uncertain given that they feed largely on phytoplankton which require sunlight for photosynthesis. Ballast tanks are sealed and thus no light would be available for photosynthesis.

There is no formal sampling program for the detection and identification of finfish in ballast water. Research identifying the range of organisms that gain access to Australian waters through ballast water is essential to maintaining the biodiversity of the unique Australian marine fauna. There have already been incursions of exotic marine pests that it is not yet possible to control (eg. Pacific sea star, *Asterias amurensis*). Unfortunately, ballast water research is under-funded and there is high demand for research into many groups of marine organisms. Research (aimed at collection and identification of finfish in ballast water) is required to provide data identifying finfish species that can survive and be transferred into Australian coastal waters via ballast water.

Detection and identification of a single live pilchard in ballast water would support the hypothesis that 'exotic' pilchards may have been introduced into Australian coastal waters via discharge of ballast water. The pilchard mortalities have not resulted in changes to procedures for the management of ballast water but have raised the profile of the potential for entry and establishment of exotic pathogens due to incursion by aquatic vertebrates.

It was not possible to gain detailed information on when or if large, international commercial shipping vessels dumped ballast water in South Australian waters around the time of the pilchard mortality outbreaks. However a list of ships' movements in and around South Australian water was obtained. The records only show that a ship has 'arrived from sea', it does not detail whether this is the ships' first port of call in Australia or if it has visited other States first. The combination of uncertainty surrounding the epizootiology of the pilchard herpesvirus and the lack of available information in relation to discharge of ballast water mean it is not possible to draw any meaningful conclusions about the movement of ocean-going vessels in South Australia around the time of each mortality event.

AQIS's role

AQIS is the lead Commonwealth agency responsible for the management of biosecurity risks presented by the discharge of ballast water from vessels entering Australia's coastal waters.

AQIS has explicit ballast water requirements for ships' Masters on their website at: www.aqis.gov.au/docs/ballast/mastersreq.htm

The information in this section is summarised from the AQIS website: www.aqis.gov.au/docs/ballast/bprogram1.htm

In 1995, AQIS finalised and released a Management Strategy for ballast water. It is part of the Australian Ballast Water Program. The Strategy points to the need for an integrated research and development program to address the ballast water problem and the introduction of ballast water management controls for coastal shipping. AQIS has administered Ballast Water Management Guidelines since 1991. These include mandatory requirements for reporting, access to sampling points and disposal of sediments. In September 1999, the Minister for Agriculture, Fisheries and Forestry announced that Australia would unilaterally implement strict new rules for ballast water management to reduce the risk of introduction of invasive marine species. This new Management Strategy was signed in mid-June 2000 and will come into force on July 1st, 2001 (F. Michaelis, pers.com.).

A Strategic Ballast Water Research and Development Program was implemented in 1996 and has been funded a number of ways: in 1996/97 AMSA funded research, in 1997/98 the Prime Minister dedicated $1 million to fund the Program, and in addition the Australian Ballast Water Management Advisory Council agreed to introduce a levy on shipping, implemented on 1 July 1998, to collect a further $2 million for 1998/99 and 1999/2000.

Australia's Oceans Policy, adopted in December 1998, considers ballast water to be a major source of harmful marine pests. AQIS is the lead Commonwealth agency responsible for the development of a single national regime for ballast water management applicable to both Commonwealth and State waters. The Policy also supports AQIS as the lead Commonwealth agency to develop such a regime. The AQIS website provides full details of the specific sectoral measures in relation to ballast water as reflected in the Oceans Policy.

Decision Support System

A key element of the new ballast water management arrangements is the implementation of the Australian Ballast Water Decision Support System (DSS). The objective of the DSS is to assess each vessel that intends to enter an Australian port in terms of potential biosecurity risks that the discharge of ballast water may present. This will provide a risk assessment tool for application to each vessel voyage, and will allow Australian authorities to manage ballast water discharges more effectively. The System is generic in nature and could have international applicability.

In the event that the ballast water is considered to present an unacceptable biosecurity risk, vessels will be required to treat ballast water prior to port entry, in a manner approved by AQIS. The criteria used for assessment are outlined in Table 10.3.

Table 10.3 Factors given consideration to assess the risk of a vessel visiting an Australian port.

1.	Where the vessel has taken up its ballast water.
2.	Where it is intending to discharge its ballast water.
3.	The port environment.
4.	Similarity of uptake and discharge ports.

5.	Time of year.
6.	Duration of voyage.

10.2.3 International agreements

There are over 100 international agreements to which Australia is a signatory, many of which include provisions that influence Australia's quarantine policy. In addition to these agreements (outlined in Appendix 9), Australia is also a signatory to the United Nations Convention on the Law of the Sea and the Convention for the Protection of the Natural Resources and Environment of the South Pacific. These multilateral and regional agreements include an obligation to protect natural environments, including their flora and fauna. Thus, there is a responsibility to ensure that import controls including quarantine policies and procedures are adequate to manage the threat of exotic pests and diseases that could damage these environments.

In its report (DPIE, 1996) the Task Force on Imported Fish and Fish Products points out that in addition to these international obligations, "the Australian community is now demanding greater protection of its unique natural environment by responsible agencies", pressuring Commonwealth agencies to take tighter precautionary measures.

10.2.4 Discussion

With regard to biosecurity, the nature of the mortality events is highly suggestive of exposure of a naive population to a new and highly pathogenic disease agent. The importation of large volumes of baitfish provides a clearly identifiable pathway by which the Australian pilchard population may have been exposed to an exotic pathogen. There is a lack of available information on the sourcing of imported pilchards prior to the 1995 mortality to enable any informed comment on whether pilchards had recently been imported from a "new" source.

AQIS's development of quarantine policy must be conducted in accordance with Australia's international obligations as a member of the WTO. Under the principles outlined in the SPS Agreement, Australia would have difficulties justifying a quarantine ban on the importation of frozen pilchards on the basis of pilchard herpesvirus because that pathogen has only ever been recorded in Australia and New Zealand. Should the herpesvirus subsequently be shown to be the cause of the mortalities and to have been introduced via imported pilchards there may be significant implications for international trade in terms of how to address biosecurity risks associated with as yet unrecognised pathogens. This is an area of particular concern for trade in aquatic animal products in which there is often little or no scientific information available in relation to the occurrence and prevalence of disease agents.

The mortality events have highlighted a lack of information about the possible role of ballast water as a pathway for introduction of exotic pathogens. While the introduction of an exotic pathogen of pilchards via ballast water appears unlikely, appropriate research is needed to obtain baseline data on finfish species present in ballast water of international ocean-going vessels that enter Australian ports.

Research into pelleted feeds for use in tuna farming is currently ongoing, and preliminary indications are that a moist pellet could be developed to replace the use of imported pilchards (Glencross *et al*., 1999).

11 SUMMARY AND DISCUSSION

11.1 SUMMARY

The pilchard mortality events are unique and unprecedented in recorded history. Such a large scale fish mortality event, especially with the wave-like patterns of spread seen in both 1995 and 1998, has not been reported previously anywhere in the world. These events highlight a significant lack of knowledge of the diseases of wild aquatic animals, even in species that are exploited worldwide.

The first mortality event lasted from mid-March to mid-June, 1995. It started in South Australia, then spread east and west to the northern most limits of the Australian pilchard population in Western Australia (~25°S) and southern Queensland (~26°S). Only adult pilchards (>10cm) appeared to have been affected. The deaths spread at approximately 21 km/day to the west and 40 km/day to the east (Murray *et al*., 2000). In South Australia, approximately 60% of the pilchard spawning biomass was killed, whereas in Western Australia the mortality was estimated at only 10-15% (Fletcher *et al*., 1997). The Western Australian figure is now thought to be a significant underestimation of the actual loss of biomass. Some upwelling was identified just prior to the first reported deaths near the Eyre Peninsula, but is considered largely irrelevant to the pilchard deaths (Griffin *et al*., 1997). Samples of phytoplankton were collected and analysed in all affected States for the presence of toxigenic species. However, there was no consistent association of phytoplankton with pilchard mortalities, and the gill pathology was not consistent with damage by phytoplankton (Whittington *et al*., 1997). A herpesvirus was consistently identified in the gill lesions of affected pilchards (Hyatt *et al*., 1997).

In 1998 the mortality started at the beginning of October and lasted eight months. It also started in South Australia, spreading in both easterly and westerly directions from Spencer Gulf. In Western Australia, mortalities continued up to the northern limits of the Australian pilchard population, but on the eastern seaboard mortalities were not recorded beyond northern New South Wales (~33°S). The mortality waves travelled at about 10 km/day (Murray *et al*., 2000), and affected adults and small numbers of juveniles in SA and Victoria (P. Hooper, pers.com.). In SA, approximately 70% of spawning biomass was killed (Ward and McLeay, 1999a) while in WA mortalities were considered to be much more severe than in 1995, killing 60-70% of the pilchard biomass (Gaughan *et al*., 2000). In Victoria, a very low level of pilchard mortalities was reported, possibly due to the fact that the pilchard population had not recovered from the 1995 mortality event. There were no environmental anomalies and no phytoplankton associated with this mortality event (Ward *et al*., 1999). A herpesvirus was consistently identified in the gill lesions of affected pilchards (A. Hyatt, pers.com.).

The ecological impacts of such large-scale mortality events affect both pilchard competitors and predators. In SA, the biomass of Australian anchovy has increased since 1998 (T. Ward, pers.com.). In WA, there is no direct monitoring of other pilchard competitors, but anecdotal evidence from fishers suggests that there are more herring and maray around this year (2000) (D. Gaughan, pers.com.). In Victoria fishers in Port Phillip Bay have also commented that there are increased numbers of anchovies (S. McCormack, pers.com.).

There has been no noticeable impact on the numbers of pilchard predators. However, a significant number of adult penguins died in Victoria just after the 1995 pilchard mortality swept through the State (Dann *et al*., 2000). The penguin deaths could not be linked to the pilchard mortality event, however the dietary intake of penguins monitored in Victoria appears to have changed significantly since 1995. Pilchard was a significant dietary component prior to 1995 (22-33%; Montague and Cullen, 1988; Cullen *et al*., 1992), but since then, pilchards continue to be replaced by other fish species such as wahou, red cod and barracouta (P. Dann, pers.com.). Gannets in Port Phillip Bay, Victoria, also significantly changed their diets, with the large amount of pilchard usually seen in gannet diets (~50%), replaced by barracouta immediately after the mortality in 1998. However, there was no decline in gannet numbers following the pilchard mortality in 1998 (Bunce and Norman, 2000).

Some pilchard fisheries were closed during the 1995 event, but all were closed during the 1998 event. The economic viability of the WA south coast fisheries was particularly affected. Two of the three fisheries were effectively closed when quotas of zero tonnes were set for 2000 (D. Gaughan, pers.com.). The industry does not expect any significant exploitation for the next few years and anticipates slow stock recovery (Gaughan, 2000). The small pilchard fishery in Victoria has become even smaller, taking only a few hundred tonnes each year (S. McCormack, pers.com.). In SA, the quota is now set at 12.5% of the estimated spawning biomass (B. Loiterton, pers.com.). Due to the loss of biomass associated with the mortality events, the quota available in SA declined significantly in 1999 and again in 2000. Stock assessments of the South Australian pilchard population indicate that the population may be capable of a quick recovery (Table 7.3). However, ongoing uncertainty regarding the potential for further mortality events in the future has created uncertainty over the viability of Australia's pilchard fishery.

Based on a questionnaire conducted in the Adelaide area and some evidence from NSW and southern Queensland bait suppliers and retailers were negatively affected by a shortfall in pilchard supply and loss of income through loss of business because recreational fishing declined.

National coordination of the response to the 1995 event was perceived to be poor. The delayed response can be partly explained by the unprecedented scale of the pilchard mortality. However, despite the hesitant start, considerable data were collected resulting in numerous articles being published in the following two years. At the time of the 1995 mortality event, Australia had no national plan for managing an aquatic animal disease emergency. Fisheries staff had no prior experience in the management and coordination of a national emergency of this kind and were incorporated into the Consultative Committee on Emergency Animal Diseases. Responsibility for responding to animal disease emergencies continues to lie with States.

When the 1998 mortality event began, personnel involved in the 1995 event were swiftly contacted and samples collected as soon as possible. A working group was established to coordinate the investigation into the event. Communication between States and other agencies was generally better than in 1995 and coordination between laboratories was smoother. The better response in 1998 was facilitated by the experience of personnel involved in the 1995 event. From a diagnostic perspective, the possible involvement of a herpesvirus was suspected and quickly confirmed. This

limited the range of laboratory analysis required. Communication was vastly improved with both SA and WA Fisheries publishing almost daily updates on their websites.

After the experience of the 1995 event, which was a timely alarm to Australian authorities, several committees were established to investigate aspects of aquatic animal quarantine and importation policies, aquatic animal disease emergency preparedness and other issues. In response to the recommendations from these various committees, the Federal Government funded the development of a comprehensive strategic plan for aquatic animal health. In 1999, AQUAPLAN (1998-2003) was launched: a five year strategy to develop and implement aquatic animal health policy in Australia including management and financial issues for handling local and national aquatic animal disease emergencies, and to raise awareness of aquatic animal health in Australia. Developed as one of the eight programs that comprise AQUAPLAN, the AQUAVETPLAN manuals deal specifically with issues related to aquatic animal disease emergencies. The manuals are under preparation at the present time.

The main hypotheses proposed to explain the cause of the pilchard mortality events were: 1) an environmental anomaly such as upwelling or a sudden temperature change was directly responsible for the events; 2) an environmental anomaly acted as a trigger for a latent infection; 3) an exotic infectious pathogenic agent.

The fact that only pilchards died, the consistent detection of a herpesvirus in association with the gill lesions of affected pilchards, and the histology of the lesions in both events, strongly suggest that the herpesvirus is the most likely cause of the mortality events. There is no evidence to suggest that any detectable environmental anomaly was either directly responsible for the mortality events, or indirectly responsible by acting as a trigger for a latent infection (Griffin *et al*., 1997; Fletcher *et al*., 1997). However, it is possible that environmental factors, as yet unidentified, or other trigger factors may be involved.

The hypotheses on the origin of a herpesvirus are divided into two groups, those that propose an enzootic origin and those that propose an exotic origin. The enzootic hypotheses include the transfer of the virus from another species, a latent infection, or mutation from a non-pathogenic form. The hypotheses that propose an introduced pathogen recognise three potential pathways for introduction of a pathogen into Australian waters: seabirds, ballast water and imported baitfish.

There are only two other reports of herpesvirus-like particles in fish in Australia, both in freshwater species, one of which was detained in quarantine. The host-specificity of herpesviruses (Roizman, 1996) indicates that it is highly unlikely that the herpesvirus observed in Australian pilchards would be related to either of the reports in freshwater fish. There is no evidence for a latent infection in Australian pilchards, the epizootiology of the mortality events behaved like a new pathogen in an immunologically naive population.

Some pathogenic fish viruses can pass through the intestinal tract of fish-eating birds and remain pathogenic (Peters and Neukirch, 1986). However, the technology to test for the presence of viable and pathogenic pilchard herpesvirus in tissues, or for

antibodies to the virus, for example, in blood samples collected from seabirds is not yet available (M. Crane, pers.com.).

Ballast water is capable of introducing exotic organisms to new environments around the world and is difficult to control or monitor. Australia has already seen incursions of exotic marine pests, most likely introduced via ballast water (eg. Pacific sea star, *Asterias amurensis*). Ballast water research is under-funded, however the potential for the introduction of exotic pests and diseases via ballast water is a major concern. Some funding was created by a shipping levy, introduced in 1998 to assist in the development of a ballast water decision support system (DSS) as a risk management tool for vessels that enter Australia from international waters. This levy was discontinued in 2000 once the necessary funds for the DSS had been raised.

The hypothesis that the herpesvirus has evolved or mutated from a non-pathogenic virus would explain why it has never been seen anywhere in the world before. This kind of change, from a harmless to pathogenic virus, has been reported with rabbit calicivirus (RHDV, Fanitini and Fenner, 1999) and Newcastle disease virus (H. Westbury, pers.com.).

The potential negative economic impacts of a ban on imported pilchards would be most strongly felt in the Eyre Peninsula region of South Australia where the tuna farming industry relies heavily on imported frozen pilchards. The industry has generated very significant economic benefits to the region and employs an increasing number of people (EconSearch, 1999). With regard to international trade obligations, Australia would have difficulty justifying a ban on imported pilchards on the basis of pilchard herpesvirus as this virus has only been detected in pilchards in Australia and New Zealand.

There is a pressing need to develop alternative tuna feeds. The industry is aware that its dependence on imported pilchards make it vulnerable to market price fluctuations and any kind of supply stoppages, for example, industrial action. The Aquaculture Cooperative Research Centre is undertaking development of artificial pellet feeds. There is a specific program for the development of pellet feeds that can improve the growth, survival and health of SBT, and the health of the environment. Trials have been running for at least two years and research is on-going (Glencross *et al*., 1999).

In December 1999 AQIS put in place new policies for the importation of aquatic animals and aquatic animal products, including frozen pilchards. These policies are among the strictest protocols in the world. The import risk analysis process that AQIS undertook in developing these quarantine requirements took account of available scientific knowledge, in accordance with Australia's international obligations as a member of the WTO. There is however, a lack of scientific knowledge on diseases of many aquatic animals and this may present a serious problem to quarantine services in their evaluation of the biosecurity risks associated with trade in aquatic animal commodities (Jones, 2000).

11.2 DISCUSSION

These unique mortality events have alerted Australian agencies to the potential of exotic marine pathogens to cause significant and serious damage to Australia's increasingly valuable fisheries and aquaculture industries. These impacts can come

from a number of sources. The responses of various government agencies to the mortalities have only come after significantly devastating events which demonstrated the vulnerability of the marine environment and the economically valuable industries which rely on that environment, to incursions of exotic marine pests and diseases.

The major conclusions of this report are:

- The pilchard mortality events are unique because of their geographic coverage and the large quantity of fish that died.
- The response to the 1995 mortality event was haphazard and slow. However, in retrospect, the event was unprecedented, and the seriousness, spreading pattern and aetiology of the event were unknown.
- The response to the 1998 event was by comparison much improved; the response was immediate, lines of communication were better delineated and research was well coordinated, mainly due to the experience gained from the 1995 event.
- Herpesvirus is the most likely cause of the mortalities.
- The origin of the herpesvirus(es) is either enzootic or exotic. Hypotheses on an enzootic origin are: (1) the virus transferred from another species, (2) the virus was latent in the population, or (3) the virus mutated/evolved from a non-pathogenic form. The pathways identified for introduction of an exotic virus are: (1) migratory seabirds, (2) ballast water and (3) imported baitfish.
- Ballast water research needs extra funding to be a major focus of research over the next few years if Australia is to protect its marine biodiversity.
- There is a lack of scientific knowledge of diseases of many aquatic animals, which presents a serious potential deficiency for science-based risk analyses on imported aquatic animals and their products.
- The impacts on southern Australian marine ecosystems are unknown.
- The economic impacts of the two mortality events primarily affected purse seine fishers with those based on the south coast of WA most seriously affected.
- AQUAPLAN and the AQUAVETPLAN manuals will provide guidelines for managing future aquatic animal disease emergencies.

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14 APPENDICES

APPENDIX 1: INTELLECTUAL PROPERTY

There is no intellectual property arising from this project.

APPENDIX 2: STAFF

Ms Alexandra Gaut
Mr Will Zacharin
Mr Ben Loiterton
Dr Tim Ward

APPENDIX 3: DATES OF ALL REPORTS OF MORTALITIES FROM 1995

Date	Place name	Co-ordinates
	Black = SA; Red = Vic; Blue = WA; Green = NSW; Plum = Tas Pink = Qld	
15/03/1995	Anxious Bay	
16/03/1995	Offshore, SW of Ceduna	
17/03/1995	Great Australian Bight (GAB), ref. SARDI time series; Fowlers Bay	
22+23/3/95	GAB, KI, Coffin Bay	33.5S, 133E; 127.65E
24/03/1995	GAB, ref. SARDI maps	
26/03/1995	" " " "	
28/03/1995	" " " " ; Point Sinclair	
29/03/1995	Port Lincoln, Taylor's Island; GAB	
30/03/1995	Point Bell, nr. Ceduna; St. Francis Island; Cape Adieu	
1/04/1995	Western side of entrance to Spencer Gulf; Fowlers Bay; Sth KI	
2/04/1995	Point Whidbey	
3/04/1995	W of Flinders Is.	
4/04/1995	ESE Pearson Island; north Innes NP; Point Sir Isaac	
5/04/1995	Topgallant Island, Pt. Neill Beach; NW of Flinders Is.; Point Sir Isaac; Anxious Bay	
6/04/1995	Cape Radstock; entrance to Venus Bay; Whidbey Is	
7/04/1995	Sheringa Beach; Althorpe Island; Streaky Bay; W of Innes NP	
8/04/1995	Cape Carnot; Browns Beach	
10/04/1995	Corny Point; Kingston; SW off Innes NP	
11/04/1995	Daly Head	
12/04/1995	Althorpe Island; SW off Innes NP	
13/04/1995	Marion Bay; SW of Flinders Is.	
14/04/1995	Cape Spencer, Stenhouse Bay; Cape Torrens (KI); S of Flinders Is	
15/04/1995	Althorpe Island; Point Sir Isaac; Anxious Bay; W of Cape Jaffa	
16/04/1995	Franklin Is.	
17/04/1995	Althorpe Island;	
18/04/1995	Middle Island and Cape Arid; south of KI	
19/04/1995	Backstairs passage; Kingston; Sellicks Beach; Young Rocks; Victor Harbor; O'Sullivans Beach; W of Cape Jaffa	
20/04/1995	North coast KI; Adelaide metropolitan beaches; W of Cape Jaffa; Topgallant Is.; several small areas south of KI; Esperance	
21/04/1995	Middle Island; Gulf St. Vincent	
22/04/1995	Adelaide northern metro beaches; Kingston; D'Estrees Bay (KI); Vennachar Point (KI); Point Sir Isaac	
24/04/1995	Adelaide central metro beaches; W of Cape Jaffa Immediately east of Esperance	
25/04/1995	Gambier Is.; W of Cape Jaffa	
26/04/1995	Backstairs Passage; S of KI; Wof Cape Jaffa; 50 km W of Esperance	
27/04/1995	W of Cape Jaffa	
28/04/1995	W of Cape Jaffa	

29/04/1995	Bremer Bay	
30/04/1995	D'Estrees Bay (KI); Bremer Bay	
1/05/1995	Cape Knob	
2/05/1995	Mid Bass Strait (Bream platform)	38.5S, 147.77E
3/05/1995	Albany. Wesley Vale, Port Sorrell. 35 nm offshore Lakes Entrance	
4/05/1995	Albany. NW Tas.	
5/05/1995	Cape Vancouver. Lakes Entrance.	
6/05/1995	Torbay. Lakes Entrance	
7/05/1995	Snapper platform	38.12S, 148.02E
8/05/1995	Goat Island, Nuyts Archipelago. (Marlin?boat?)	38.25S, 148.25E
9/05/1995	Wynyard, Port Latta.	40.51S, 145.23E
10/05/1995	Albany, Torbay. Mallacoota. Eden.	37.44S, 149.3E
11/05/1995	Merimbula; GAB.	
15/05/1995	Port Hacking, Botany Bay, Sydney. Point D'Entrecasteaux, Nornalup Estuary.	
16/05/1995	Augusta	
17/05/1995	Newcastle	
25/05/1995	Bunbury. Coffs Harbour	
29/05/1995	25m NNE Rottnest. Coffs Harbour.	
30/05/1995	Bunbury, 10m NE Rottnest.	
31/05/1995	Rottnest. Coffs Harbour, Iluka.	
1/06/1995	Rottnest.	
2/06/1995	Cockburn Sound, Rockingham Sound.	
3/06/1995	Coolum.	
5/06/1995	Kingscliff/Tweed Heads, Teewah, offshore Coolum.	
6/06/1995	Moore River. Noosa.	
12/06/1995	Dongara, Lancelin.	
13/06/1995	Dongara.	
14/06/1995	Dongara, Horrocks.	
16/06/1995	Horrocks.	
End June	Carnarvon.	

APPENDIX 4: DATES OF REPORTS OF MORTALITIES IN NEW ZEALAND IN 1995

NEW ZEALAND

North Island

16/06/1995	Cape Colville
19/06/1995	Firth of Thames; Coromandel area
16-23/6/95	W side of Great Barrier Island; Leigh, Omaha: Mangawhai Heads
18-20/6/95	Marsden Point
22/06/1995	Tapu, Te Puru.
20-29/6/95	Bay of Islands
28/06/1995	Tapu, Te Puru.
3-7/7/95	Doubtless Bay
"mid July"	North Cape
15-late/7/95	Ninety Mile Beach

South Island

29/08/1995	Farewell Spit, Separation Point
1-5/9/95	Tasman Bay, whole shoreline
4-10/9/95	French Pass
14-20/9/95	S + W Arapawa Island
17-28/9/95	**Wellington Harbour, North Island**

APPENDIX 5: DATES OF ALL REPORTS OF MORTALITIES FROM 1998/99

Date	**Place name** (*Italics = fish with dead pilchards in stomach*) Black = SA; Red = Vic; Blue = WA; Green = NSW; Plum = Tas	Ref.	Coordinates
2/10/1998	Offshore from Arno Bay	PB3	
4/10/1998	NE of Corny Point, Spencer Gulf	PB1	
7/10/1998	Moonta Bay, Point Riley; 6 miles W Althorpe Island	E-mail; PB4	
8/10/1998	Port Victoria, Tickera, Port Broughton	SARDI report	
12/10/1998	Coffin Bay	SARDI report	
13/10/1998	Cape Jervois to Marino Rocks; N coastline KI; Investigator Strait; Port Augusta to Thistle Island; Maslins Beach, Stokes Bay	PB1; SARDI report	
14/10/1998	Between Blanche Harbour and Point Lowly; Adelaide to Cape Jervis; Sellicks Beach; Smiths Bay (Nth coast KI)	SARDI report	
15/10/1998	Silver Sands, Adelaide to Cape Jervis, Farm Beach, Convention Beach, Picnic Beach nr. Pt. Drummond; NW KI; few kms E of Cape Jervis.	PB2; SARDI report	
16/10/1998	Sultana Point (Edithburgh); offshore nr. Cape Finnis & Waldegrove Is.; Investigator Strait btwn. Marion Bay and Stokes Bay (N KI)	PB4; SARDI report	
18/10/1998	Streaky Bay, Elliston; Glenelg, Brighton and Hove.	PB5; SARDI report	
19/10/1998	Waitpinga Beach; Henley Beach	SARDI survey	35.42'80"S 138.27'01"E
20/10/1998	Waitpinga Beach; still washing up in metro areas as far N as Henley Beach.	PB6	
	Venus Bay to Elliston.	PB7	
21/10/1998	Port Brown	SARDI report	
22/10/1998	Point Bell; Coffin Bay; St. Vincent Gulf; Goolwa	SARDI report	
23/10/1998	Venus Bay; fisheries officers Located areas in SVG with fresh mortalities.	PB8; SARDI report	
24/10/1998	Murray Mouth	SARDI report	
25/10/1998	Mount Dutton Bay	SARDI report	
26/10/1998	Goolwa, 30m SE Murray mouth; Streaky Bay	PB9; SARDI report	
27/10/1998	Surf beaches of Coorong; Victor Harbor; NW Kingston; Bales Bay (S KI); Seaford, Southport.	PB10; SARDI report	
28/10/1998	Dead juvs. @ Streaky Bay and Seaford.	PB10	
29/10/1998	28 Mile Crossing btwn. Salt Creek & Kingston; Cape Jervis; Carrickalinga; Smoky Bay to Franklin Is.; dead juveniles at Hog Beach & Antechamber Bay (N KI); Goolwa; Waitpinga Beach.	SARDI report	
30/10/1998	*70m W Cape Jaffa;*	PB12	
31/10/1998	D'Estrees Bay (KI)	SARDI report	
1/11/1998	Franklin Is.; Point Bell; *Head of the Bight;* Tea Tree Crossing (Coorong); D'Estrees Bay, Antechamber Bay, Bales Beach, Pennington Bay & Penneshaw (KI); Brighton; Fowlers Bay; Sheoak Flats	PB13; PB14; SARDI report	
2/11/1998	Fowlers Bay; Sheoak Flats; Black Point Bay; Myponga	PB13; SARDI	

		report	
3/11/1998	Wandilla Beach; Black Point Bay; Sheoak Flats; Scott Bay, Fowlers Bay; Perforated Is.; Purdie Is.; *Purdie Is.*; S Pine Pt.	PB14; SARDI report	
4/11/1998	Coorong; Combyra Beach (E Head of Bight); Discovery Bay.	PB14; F. Neira	
5/11/1998	Ceduna; Goat Is.; Sinclair Beach; Point Bell, Bielamah Beach; Surfers Beach btwn. Middleton & Goolwa; Cape Jaffa; Victor Harbor; Scotts Bay; Clare Bay; Combyra Beach; Fowlers Bay	PB15; SARDI report	
6/11/1998	Rapid Bay, Carrickalinga Beach; Discovery Bay.	PB16; PB19; SARDI	
7/11/1998	Off KI; South Neptune Is.; Station Beach nr. Cape Otway.	PB16; SARDI; F. Neira	
8/11/1998	Yalata Beach & surrounding area; Greenly Is.; offshore W of KI, offshore S of KI; Goolwa; Moana.	PB16; SARDI report	
10/11/1998	Yalata Beach; Eucla; Investigator Strait - Troubridge Pt to Kingscote; Goolwa & Murray mouth; Morgans Beach.	PB17	
11/11/1998	Port Campbell/Twelve Apostles; Apollo Bay; Eucla; D'Estrees Bay (S KI); Apollo Bay.	PB18; SARDI report	
13/11/1998	Swan Lake, Discovery Bay; 30 km W Portland; Lorne.	PB19	
14-15/11/98	Point Addis, Anglesea, Big Hill/Cinema Pnt; Eucla; Pennington Bay (S KI).	PB19	
17/11/1998	30 kms W Eucla; btwn. Lorne & Anglesea & Pt. Lonsdale, btwn. Pt. Nepean & Cape Shanck; 1.5nm off entrance to Pt Phillip Bay, & inside Pt Phillip Bay;	PB20	
	Parsons & Waitpinga beaches (unconfirmed).	PB21	
19/11/1998	45m SW Eucla (27 fathoms).	PB21	
20/11/1998	Port Phillip Bay.	F. Neira	
22/11/1998	60 nm S Eucla	SARDI report	
23/11/1998	55 km W Eucla; 129-130 longitude, 60nm S Eucla.	PB22	
25/11/1998	No further reports from SA.	PB23	
27/11/1998	Wanteen 55 km W Eucla.	PB25	
1/12/1998	15-20nm off Lakes Entrance; btwn. Seaspray & Lake Tyers.	PB26	
2/12/1998	Ninety Mile Beach, beaches W Lakes Entrance.	PB26	
	150 km offshore from Eyre Bird Observatory.	PB27	33.11'S 126.45'E
5/12/1998	Lakes Entrance.	PB29	
8/12/1998	Floating btwn. Lakes Entrance & Marlo, 2.5nm offshore.	PB29	
10/12/1998	120 km E Middle Island.	PB30	33.39'89"S 134.29'14"E
13/12/1998	Straight line from sighting on 10/12 to coordinates;all 5-10 km offshore.	WAPB37	33.39'47"S 124.12'44"E
14/12/1998	Eyre Bird Observatory and 8 km offshore from there.	PB31	
17/12/1998	Western most point at coordinates, 5-10nm offshore.	PB33	33.44'S 123.58'E

	South of Israelite Beach in water in rows running N-S.		33.07'S 124.08'E
21/12/1998	Flight observed scattered pilchards at coordinates	WAPB43	33.46'S 123.49'E
29/12/1998	5-8nm offshore; Cape Pasley to Salisbury Is., 25nm offshore.	WAPB47	34S 122.38'E
31/12/1998	Cape Arid	WAPB49	
1/01/1999	Thomas River	WAPB50/ PB36	
3/01/1999	Alexander Bay	WAPB50/ PB36	
4/01/1999	Wharton; Bellerive Beach	WAPB50/ PB36/SM	
5/01/1999	Lucky Bay; Cremorne (offshore), Tranmere Beach	WAPB51/ PB36/SM	
6/01/1999	Thistle Cove; Taroona, 7 Mile Beach, Frederick Henry Bay	WAPB52/ PB37/SM	
7/01/1999	All beaches btwn Thistle Cove & Esperance. Kingston beach, 7 Mile Beach, Park beach, Hinsby beach, Norfolk Bay/Connellys Marsh, Carlton beach, Blackmans Bay Howrah beach, Primrose Sands.	WAPB53 Sandy Murray	
8/01/1999	Western most observation at coordinates, 12nm S of	WAPB54/PB37	34.19'S
	Observatory Island. Frederick Henry Bay, Droghty Pnt, Summers Bay, Murdunna, Primrose Sands.	Sandy Murray	121.59'E
11/01/1999	11 Mile Beach, Twilight Cove; 10nm W of position on 8/01.	WAPB55	
12/01/1999	4 Mile Beach, 11 Mile Beach, 14 Mile Beach, Butty Head, Roses		
	Beach.	WAPB56	
13/01/1999	Westernmost observation at coordinates, 13nm offshore.	WAPB57/ PB38	34S, 121.32'E
	Modrain Island area. Beaches N Newcastle & reef 5 km offshore.	PB38	
14/01/1999	Stockton & Merewether beaches, Blackhead.	PB39	
15/01/1999	Western front at coordinates, 23nm offshore.	WAPB59	121.30'E
	Seven Mile Beach.	WAPB59	
17/01/1999	Skippy Rock & Margaret Cove. Old Bar; offshore Hallidays Pnt.	WAPB60/ PB40/41	121.01'E
18/01/1999	Front at coordinates, 10 km offshore.	WAPB61	120.57'E
20/01/1999	Starvation beach (40kms E Hopetoun); Recherche Archipelago.	WAPB63	
24/01/1999	Point Ann	WAPB65/ PB42	
25/01/1999	Western front at coordinates, Bremer Bay.	WAPB65/ PB42	34.29'S 119.27'E
27/01/1999	Bremer Bay	WAPB66	
31/01/1999	Westernmost sighting 30nm SW Bremer Bay & 35nm NE Bald Is Dillon Bay.	WAPB69/ PB44	

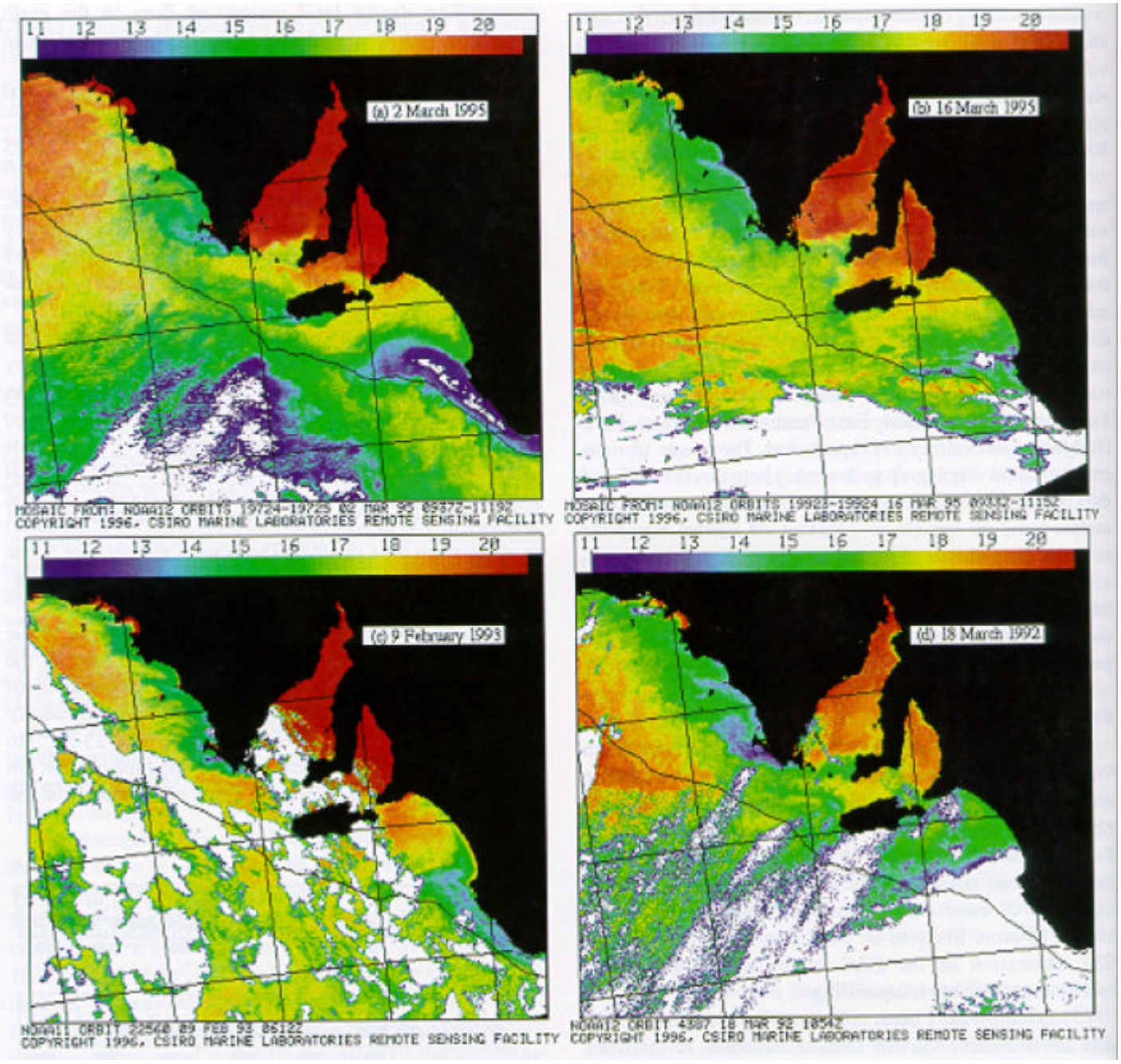
5/02/1999	Cape Riche, Dillon beach.	WAPB73/74	
8/02/1999	Cheynes beach.	WAPB74/ PB46	
10/02/1999	King George Sound, Seal Is. to old whaling station.	WAPB76	
11/02/1999	Whaleworld, Nannerup beach to E Oyster Harbour.	WAPB77	
12/02/1999	Nannerup beach, King George Sound.	WAPB78	
15/02/1999	All beaches in King George Sound, Torbay Head.	WAPB79	
27/02/1999	Nornalup beach	WAPB89	
3/04/1999	Smiths Beach at Yallingup, and in water nrby.	WAPB108	
7/04/1999	WNW Bunbury 10-45 nm offshore.	WAPB109/ PB56	
8/04/1999	Northernmost extent dead fish at coordinates, 12 nm offshore.	WAPB110	33.10'S
9/04/1999	W of Bunbury and as yesterday.	WAPB111	
16/04/1999	Garden Island; btwn Carnac & Rottnest Is; SW Parker Pnt. 5nm offshore Observation City towards Rottnest;	WAPB116	
	25nm W Mandurah; Coventry Reef/Warnbro Sound; N & S of Rottnest; 6nm offshore btwn Hillarys & Ocean Reef.	WAPB117	
19/04/1999	8nm offshore from Mindarie. Btwn Hillarys & Two Rocks.	WAPB117/118	
24/04/1999	Over continental shelf.	WAPB122	31.27'S to 31.18'S
	Large wind row extending close to beach at		31.09'S
24-25/4/99	Just N of Lancelin	WAPB126	
6/05/1999	20-30 fathom, W Seaward Ledge off Jurien Bay	WAPB128	
21/05/1999	26 fathoms, Dongara to Geraldton	WAPB131	

References

PB: Pilchard Bulletins produced by SA Fisheries
WAPB: Pilchard Bulletins produced by WA Fisheries
SARDI report: Preliminary report to the Joint Pilchard Scientific Working Group on the 1998 Pilchard Mortality Event. 1998. T.M. Ward, M. Westlake, L.Y. McLeay and G.K. Jones
Sandy Murray: Dr Alexander Murray, formerly with CSIRO Hobart
F. Neira: Dr Fransisco Neira, MAFRI

APPENDIX 6: SATELLITE IMAGES OF UPWELLING AROUND THE EYRE PENINSULA AND IN THE GREAT AUSTRALIAN BIGHT ON VARIOUS DATES (1992 TO 1995).

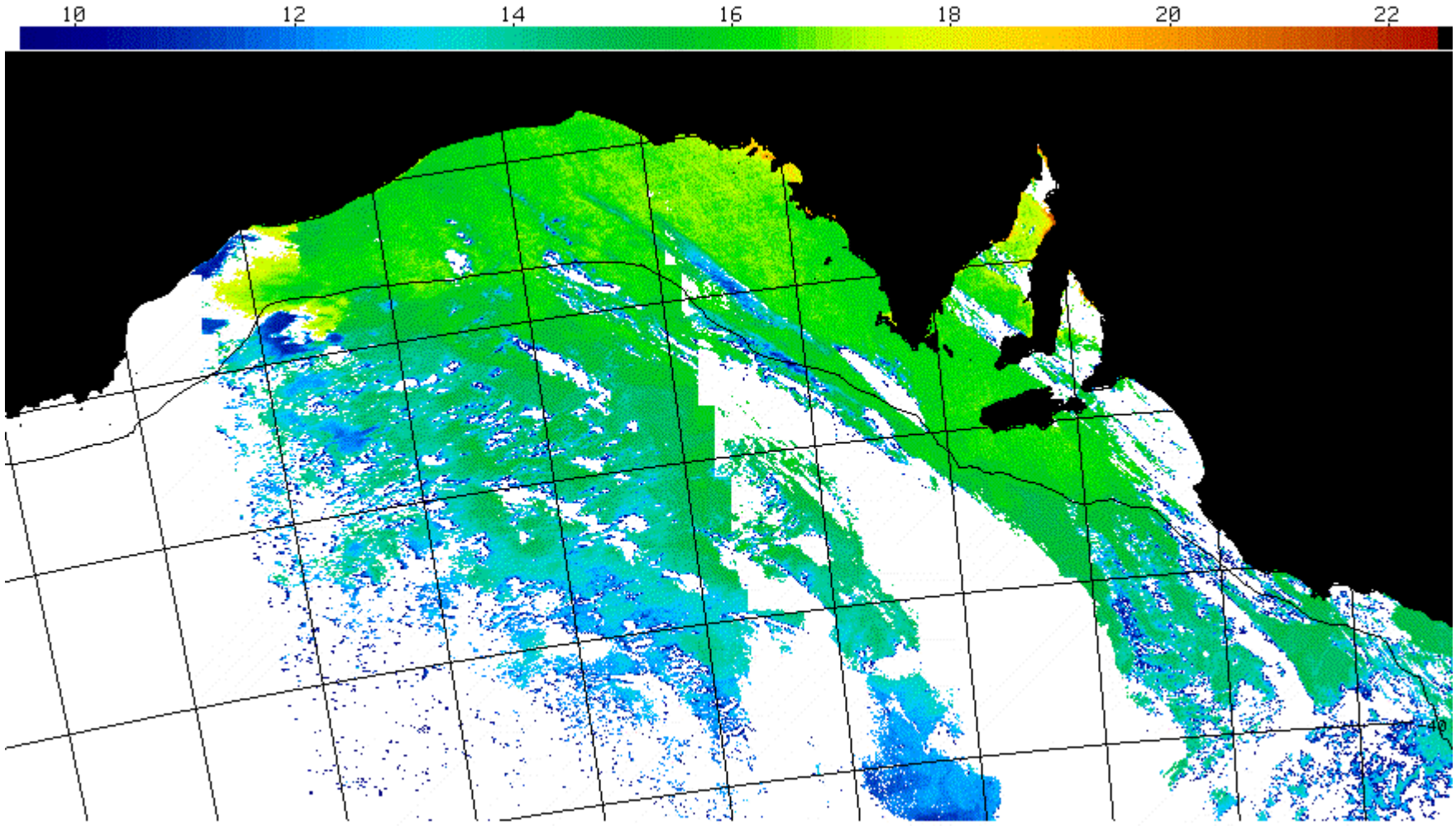
(Reproduced with permission from Griffin et al., 1997)



APPENDIX 7: SATELLITE IMAGE OF GREAT AUSTRALIAN BIGHT ON 2 OCTOBER 1998.

This is the first date that anyone reported seeing dead pilchards in 1998.

It can clearly be seen that the sea surface temperature is uniform (around 16°C) close to the South Australian coast and over much of the Great Australian Bight. This temperature is within 'normal' parameters for spring (September/October) in southern Australian temperate waters. (Image on following page).



NOAA12 SST mosaic 02 Oct 1998 0706Z-0846Z

APPENDIX 8: ACTION TABLES FROM ALL JOINT PILCHARD SCIENTIFIC WORKING GROUP (JPSWG) MEETINGS

JOINT PILCHARD SCIENTIFIC WORKING GROUP
ACTION ITEMS FROM MEETING No. 1 – 16 October 1998

ITEM	ACTION	ACTION BY	STATUS
1.	The group **agreed** that AQIS would be notified of the work of the JPSWG and be invited to participate.	PIRSA Fisheries will contact AQIS.	
2.	It was **agreed** that the age structure of the dead pilchards should be determined as soon as possible.	SARDI to undertake this, with assistance from MAFRI in Victoria, if necessary.	
3.	The group also **agreed** to alert the agencies (Environment Australia and SA Department of Environment, Heritage and Aboriginal Affairs) with an interest in the Great Australian Bight Marine Park of the event.	PIRSA Fisheries	
4.	The group **agreed** that more and better samples of dead and dying fish were essential for diagnostic and biological work. As a result, an expanded sampling program was agreed upon with the SA fisheries patrol vessel being diverted to the West Coast areas immediately to assist in this work. Dr Ward would co-ordinate sampling for all national requirements and AAHL would send staff to participate in the sampling exercise.	PIRSA Fisheries SARDI AAHL	
5.	The group **agreed** to an expansion of the range of diagnostic tests that would include transmission tests. SARDI would make available isolated tanks at its West Beach facility and co-ordinate sampling of fish in order to undertake the transmission tests.	SARDI	
6.	A precautionary pathology sampling of imported pilchards was **recommended**.	PIRSA Fisheries to facilitate this.	

JOINT PILCHARD SCIENTIFIC WORKING GROUP
ACTION ITEMS FROM MEETING No. 2 – 30 October 1998

ITEM	ACTION	ACTION BY	STATUS
1.	Additional sampling of dying pilchards	Dr Ward to coordinate with other groups, particularly AAHL	
2.	Sampling requirements for ballast water	AQIS to report back to the group.	
3.	Non-destructive sampling of Australian gannets	Dr Ward to coordinate sampling and ensure through PIRSA that environmental agencies were fully informed.	
4.	Proposal to be prepared for Fisheries Research & Development Corporation for funding the preparation of a comprehensive report into the incident.	Dr Bernoth to prepare report based on the outline developed by the group	
5.	Objective 1 Pathological examination of small fish, survivors and fish not yet impacted by the mortality event.	Dr Reuter to coordinate this work.	
6.	**Objective 1** Disease transmission tests	SARDI and WA Fisheries to jointly prepare a proposal for undertaking this work.	
7.	**Objective 2** *Pending result of Objective 1 (previous page)* Herpes Virus DNA from New Zealand and from 1995 & 1998 pilchard mortality events would be sent to AAHL for genetic fingerprinting.	AAHL to incorporate this work into its research proposal.	
8.	**Objective 2** *Pending result of Objective 1 (previous page)* Pilchards from overseas countries would be tested for the presence of Herpes Virus. In the meantime, overseas pilchards scientists would be contacted and requested to exam/collect gill samples during and pilchard mortality events.	DAFF agreed to coordinate this work.	
9.	**Objective 4** Development of diagnostic tools	AAHL agreed to prepare a proposal for undertaking this work.	

ITEM	ACTION	ACTION BY	STATUS
10.	Development of mathematical models related to the epidemiology of the mortality event.	WA Fisheries to prepare a proposal for this work.	

JOINT PILCHARD SCIENTIFIC WORKING GROUP
ACTION ITEMS FROM MEETING No. 3 – 17 November 1998

ITEM	ACTION	ACTION BY	STATUS
1.	Additional sampling of dying pilchards	Dr Ward to coordinate with other groups, particularly AAHL	
2.	Sampling requirements for ballast water	AQIS to assist if sampling required	
3.	Non-destructive sampling of Australian gannets	Dr Ward to follow up with WA Fishery Authorities. Dr Keesing to coordinate required samples in SA waters	
4.	Proposal to be prepared for Fisheries Research & Development Corporation for funding the preparation of a comprehensive report into the incident.	Dr Morgan to contact Mr Glen Hurry for a copy of the submission	
5.	Pathological examination of small fish, survivors and fish not yet impacted by the mortality event.	Dr Reuter to coordinate this work from WA and Vic. Dr Keesing to coordinate collection from SA.	
6.	Herpes Virus DNA from New Zealand and from 1995 & 1998 pilchard mortality events would be sent to AAHL for genetic fingerprinting.	AAHL to incorporate this work into its research proposal.	
7.	Pilchards from overseas countries would be tested for the presence of Herpes Virus. In the meantime, overseas pilchards scientists would be contacted and requested to exam/collect gill samples during and pilchard mortality events.	Dr Morgan to seek an update from Fisheries and Aquaculture Branch of DAFF and circulate.	
8.	Development of diagnostic tools	AAHL agreed to prepare a proposal for undertaking this work.	
9.	Deaths of Salmon smolt	Dr Reuter agreed to examine for indications of pancreatic damage.	
10.	Cause of mortalities	PIRSA to fund continuation of work.	
11.	Development of a clear understanding of the epidemiology of the infection	Dr Morgan seek CCEAD endorsement of the research program and liase with funding agencies for project funding.	

JOINT PILCHARD SCIENTIFIC WORKING GROUP
ACTION ITEMS FROM MEETING No. 4 – 15 December 1998

ITEM	ACTION	ACTION BY	STATUS
1.	Additional sampling of dying pilchards	Dr Ward to coordinate with other groups, particularly AAHL	Item to be given the highest priority.
2.	Sampling requirements for ballast water	AQIS to assist if sampling required	Not an immediate priority.
3.	Non-destructive sampling of Australian gannets	Dr Ward to follow up with WA Fishery Authorities. Dr Keesing to coordinate required samples in SA waters	Agreed to that the issue of evaluating whether Australian gannets were a vector of the virus would be better addressed by incorporating controlled experiments into the already scheduled transmission tests. Also greed that opportunistic sampling of wild gannets would be undertaken when possible.
4.	Proposal to be prepared for Fisheries Research & Development Corporation for funding the preparation of a comprehensive report into the incident.	Dr Morgan to contact Mr Glen Hurry for a copy of the submission	The chair has contacted Mr Glenn Hurry of DAFF who agreed to provide a copy of the draft submission to FRDC
5.	Pathological examination of small fish, survivors and fish not yet impacted by the mortality event.	Dr Reuter to coordinate this work from WA and Vic. Dr Keesing to coordinate collection from SA.	Dr. Ward to coordinate further sampling and arrange for these samples to be made available to Dr. Reuter and Dr. Westbury.
6.	Herpes Virus DNA from New Zealand and from 1995 & 1998 pilchard mortality events would be sent to AAHL for genetic fingerprinting.	AAHL to incorporate this work into its research proposal.	Delays reported due to a breakdown of the laboratory's gene sequencer which is now back in operation.
7.	Pilchards from overseas countries would be	Dr Morgan to seek an update from DAFF	Dr Morgan to follow up overseas

ITEM	ACTION	ACTION BY	STATUS
	tested for the presence of Herpes Virus. In the meantime, overseas pilchards scientists would be contacted and requested to exam/collect gill samples during and pilchard mortality events.	and circulate.	scientists with DAFF.
8.	CCEAD Teleconference	Various research proposals to be re-drafted to not only incorporate CCEAD comments but also the modifications made at meeting with regard transmission test proposal	Dr Morgan to send consolidated, revised research program to CCEAD
9.	Cause of mortalities - pathology	PIRSA to continue funding the pathology, subject to the endorsement of the group	
10.	Cause of mortalities – transmission tests	Joint SARDI/WA proposal to be re-drafted to include issues of gannets and budget to be recaste to better reflect the contributions being made by State and industry and then forwarded to Dr Morgan	
11.	Development of Diagnostic tools	Dr Morgan to write to AAFA indicating the high priority within existing funding arrangements. Dr Westbury to re-caste the proposal into a FRDC format and forward to Dr Morgan for subsequent submission to FRDC after liaison with SAFRAB.	
12.	Epidemiology of the Mortalities	Dr Murray to re-draft the modeling proposal in accordance with CCEAD's comments	
13.	Samples for examination of pancreatic lesions	Dr Brian Jones to supply samples to AAHL in addition to examinations being undertaken in WA. Dr G Morgan to write to AQIS requesting their support.	

ITEM	ACTION	ACTION BY	STATUS
		Dr G Morgan to also contact Dr R Fletcher re monitoring in NSW waters to be co-ordinated through the group and to enquire as to availability of resources.	

JOINT PILCHARD SCIENTIFIC WORKING GROUP
ACTION ITEMS FROM MEETING No. 5 – 19 January 1999

ITEM	ACTION	ACTION BY	STATUS
1.	Additional sampling of dying pilchards	Dr Ward to coordinate with other groups, particularly AAHL	Ongoing
2.	Sampling requirements for ballast water	Gary Morgan to advise AQIS on gap in ballast water strategy on live active aquatic organisms – not able to exclude swimming live aquatic organisms in ballast water.	Pending
3.	Non-destructive sampling of Australian gannets	Dr Ward to follow up with WA Fishery Authorities. Dr Keesing to coordinate required samples in SA waters	Action now complete.
4.	Proposal to be prepared for Fisheries Research & Development Corporation for funding the preparation of a comprehensive report into the incident.	Vic Neverauskas to contact Stan Jarzinsky (AFFA) to finalise the proposal using FRDC format for research proposals.	Pending
5.	Pathological examination of small fish, survivors and fish not yet impacted by the mortality event.	Dr Ward to co-ordinate further sampling and arrange for these samples to be made available to Dr Reuter and Dr Westbury	Ongoing
6.	Herpes Virus DNA from New Zealand and from 1995 & 1998 pilchard mortality events would be sent to AAHL for genetic fingerprinting.	Dr. Westbury to provide progress report.	Pending
7.	Pilchards from overseas countries would be tested for the presence of Herpes Virus. In the meantime, overseas pilchards scientists would be contacted and requested to exam/collect gill	Dr G Morgan to action	Pending

ITEM	ACTION	ACTION BY	STATUS
	samples during and pilchard mortality events.		
8.	Development of Diagnostic tools	Dr Morgan to write to AFFA indicating the high priority within existing funding arrangements. Dr Westbury to re-caste the proposal into a FRDC format and forward to Dr Morgan for subsequent submission to FRDC after liaison with SAFRAB.	Action now completed.
9.	Epidemiology of the Mortalities	Dr Murray to re-draft the modeling proposal in accordance with CCEAD's comments	Action now completed.
10.	Funding research program.	CCEAD endorsement expected by 22nd January, 1999, then seek funding. Dr Morgan to write to AQIS and request support for program	FRDC and Biosecurity fund – chair to check access arrangements within PIRSA. It was noted that these funds need to be released ***as a matter of urgency.***
11.	Samples for examination of pancreatic lesions	Dr Brian Jones to proceed with an examination of pancreatic tissue of the formalin-fixed material from the 1995 pilchard kill	Ongoing
12.	Monitoring in NSW waters.	Chairman to contact Dr Rick Fletcher re monitoring in NSW and co-ordination through the group – also to enquire re availability of resources with NSW to facilitate this.	Ongoing.
13.	Tasmanian mortality (pilchards or sprat)	Dr. Ward to contact AAHL and/or Tasmania to submit samples for species identification	Pending
14.	ProMed Note	Dr. Bernoth to draft, obtain agreement and submit.	Pending
15.	Bulletins	Peter Christy to continue to provide twice	On-going

ITEM	ACTION	ACTION BY	STATUS
		weekly Bulletins	

JOINT PILCHARD SCIENTIFIC WORKING GROUP
ACTION ITEMS FROM MEETING No. 6 – 23 February 1999

ITEM	ACTION	ACTION BY	STATUS
1.	Additional sampling of dying pilchards	Dr Ward to coordinate with other groups, particularly AAHL	Ongoing
2.	Sampling requirements for ballast water	Gary Morgan to advise AQIS on gap in ballast water strategy on live active aquatic organisms – not able to exclude swimming live aquatic organisms in ballast water.	Pending
3.	Herpes Virus DNA from New Zealand and from 1995 & 1998 pilchard mortality events would be sent to AAHL for genetic fingerprinting.	Dr Morgan to write to AAHL stating that the sequencing of DNA from the current outbreak and the 1995 outbreak needs to be compared as a matter of urgency in order to determine whether the current outbreak is linked to the 1995 virus, or whether the 1998 virus is a 'new' virus.	Pending
4.	Pathological examination of small fish, survivors and fish not yet impacted by the mortality event.	Dr Ward to co-ordinate further sampling and arrange for these samples to be made available to Dr Reuter and Dr Westbury	Ongoing
5.	Pilchards from overseas countries would be tested for the presence of Herpes Virus. In the meantime, overseas pilchards scientists would be contacted and requested to exam/collect gill samples during and pilchard mortality events.	Dr G Morgan to action	Pending
6.	Funding research program.	Dr Morgan has discussed the Biosecurity Fund with PIRSA Corporate Finance. The PIRSA/SARDI account is to be consolidated and sent as one account.	pending
7.	Samples for examination of pancreatic lesions	Dr Brian Jones to proceed with an examination of pancreatic tissue of the formalin-fixed material from the 1995 pilchard kill	Ongoing
8.	Bulletins	Peter Christy to issue a weekly pilchard bulletin. Further information is available on the Fisheries WA web site at http://www.wa.gov.au/westfish/comm/broc/infobulletin/index.html	On-going
9.	The Chairman to write to AQIS and request	Dr Morgan reported that he had written to AQIS but was still awaiting	Ongoing

ITEM	ACTION	ACTION BY	STATUS
	their support for funding research program which has been submitted to FRDC.	a reply. Action	
10.	CCEAD endorsement of research program	Eva-Maria Bernoth will ask the CCEAD secretariat to provide a consolidated statement of support.	Pending
11.	Herpes Simplex Virus in humans	Dr Morgan will write to David Cunliffe of the SA Health Commission to inform him of this observation.	pending

JOINT PILCHARD SCIENTIFIC WORK GROUP
ACTION ITEMS FROM MEETING No. 7 – 30 March 1999

ITEM	ACTION	ACTION BY	STATUS
1.	Additional sampling of dying pilchards	Dr Ward to present a final report on the ageing of dead pilchards.	Pending
2.	Sampling requirements for ballast water.	Gary Morgan has written to AQIS regarding the ballast water issue but has yet to receive a reply.	Pending
3.	Herpes Virus DNA from New Zealand and from 1995 and 1998 pilchard mortality events to be sent to AAHL for genetic fingerprinting.	Dr Morgan to write to AAHL stating that the sequencing of DNA from the current outbreak and the 1995 outbreak needs to be compared as a matter of urgency in order to determine whether the current outbreak is linked to the 1995 virus, or whether the 1998 virus is a 'new' virus.	Pending
4.	Pathological examination of small fish, survivors and fish not yet impacted by the mortality event.	Dr Ward to ensure that samples are made available to Dr Reuter and to Dr Westbury.	Pending
5.	Pilchards from overseas countries to be tested for the presence of Herpes Virus. In the meantime, overseas pilchards scientists would be contact and requested to examine/collect gill samples during any pilchard mortality events.	Dr Morgan has contacted AAFA regarding facilitating this work.	Pending
6.	Funding research program	Dr Morgan has submitted a request to the SA Biosecurity fund for both re-imbursement of costs associated with the work involved in monitoring the pilchard kill and for funding of a component of the research program. A decision is expected within a week.	Pending
7.	Samples for examination of pancreatic lesions	Dr Brian Jones advised that pancreatic tissue had been collected and will be sent to AAHL	Ongoing
8.	Bulletins	Peter Christy to reduce frequency of pilchard bulletins to once every 2 weeks	Ongoing
9.	The Chairman to write to AQIS and request	Dr Morgan has written to AQIS and is still awaiting a reply	Pending

ITEM	ACTION	ACTION BY	STATUS
	their support for funding research program which has been submitted to FRDC		
10.	CCEAD endorsement of research program	Dr Eva-Maria Bernoth reported that CCEAD has provided formal endorsement of the research program. (See attachment to minutes)	Completed
11.	Herpes Simplex Virus in humans.	Dr Morgan reported that he had informed the SA Health Commission of the WA observation that there has been an apparent increase in the incidence of human Herpes simplex in Perth.	Completed
12.	An investigation of alternative virology laboratories to provide alternative services to develop pilchard gill cell cultures.	Dr Reuter to seek formal proposals from other laboratories to develop pilchard gill cell lines.	Ongoing
13.	A full written status report of research work carried out to date to be prepared.	**All Members** to prepare a comprehensive status report on research undertaken to date for presentation at the next meeting	Pending

JOINT PILCHARD SCIENTIFIC WORK GROUP
ACTION ITEMS FROM MEETING No. 8 – 12 May 1999

ITEM	ACTION	ACTION BY	STATUS
1.	Analysis of dying pilchards.	Dr Tim Ward to present a final report on the age profile of dead pilchards.	Pending
2.	Sampling requirements for ballast water	Verbal discussions have confirmed AQIS support but formal notification still to be received.	Pending
3.	Herpes Virus DNA from New Zealand and from 1995 and 1998 pilchard mortality events to be sent to AAHL for genetic fingerprinting	Samples have been sent to AAHL. AAHL to commence DNA sequencing as soon as practicable.	On-going
4.	Pathological examination of small fish, survivors and fish not yet impacted by the mortality event	Dr Tim Ward to arrange for the collection of additional pilchards which are to be formalin fixed before forwarding to the VPS laboratory.	On-going
5.	Pilchards from overseas countries to be tested for the presence of Herpes Virus.	Glen Hurry has agreed to co-ordinate this process and will begin shortly.	On-going
6.	Funding research program	Dr Morgan noted that funding for the research program from the SA Biosecurity Fund has been approved in principle.	On-going
7.	Samples for examination of pancreatic lesions	Samples have been sent by WA Fisheries to AAHL but no results as yet.	On-going
8.	Bulletins	The group agreed that further releases were to be at the discretion of the Director of Fisheries SA	On-going
9.	The Chairman to write to AQIS and request their support for funding research program which has been submitted to FRDC	Verbal discussions have confirmed AQIS support but formal notification still to be received.	On-going
10.	Investigating the capability of alternative laboratories to develop pilchard gill cell cultures.	Proposals to be forwarded to Dr Reuter shortly.	On-going
11.	Written status reports of the pilchard mortality	Final reports were tabled by Department of Fisheries WA and AFFA.	Pending

ITEM	ACTION	ACTION BY	STATUS
	investigation.	SARDI, AAHL and CSIRO Hobart will table report next meeting.	

JOINT PILCHARD SCIENTIFIC WORK GROUP
ACTION ITEMS FROM MEETING No. 9 – 2 August 1999

ITEM	ACTION	ACTION BY	STATUS
1.	Sampling requirements for ballast water.	No correspondence has been received from AQIS yet formalising a verbal agreement supporting the need for ballast water sampling .	Pending
2.	Herpes Virus DNA from New Zealand and from 1995 and 1998 pilchard mortality events to be sent to AAHL for genetic fingerprinting	Two blocks of tissue from New Zealand are now in storage at AAHL. Sequencing of herpesvirus from these tissues will commence after the sequencing work on herpesvirus from the 1998 mortality has been finished.	On-going
3.	Pilchards from overseas countries to be tested for the presence of Herpes Virus.	Glenn Hurry has no further action to report at present**.**	On-going
4.	Funding research program	Dr Morgan is to verify that the SA Biosecurity Fund will cover the cost of the pilchard herpesvirus transmission trials conducted in Western Australia.	On-going
5.	Samples for examination of pancreatic lesions	AAHL has not completed the examination of the lesions in pilchard pancreatic tissues from the 1995 mortality event provided by WA Fisheries.	On-going
6.	Investigating the capability of alternative laboratories to develop pilchard gill cell cultures.	Dr Morgan to investigate sources of additional funding.	To be reported to the next meeting.
7	CCEAD final report	Dr Morgan to contact CCEAD to provide an update report on kills and research.	Pending.

JOINT PILCHARD SCIENTIFIC WORK GROUP
ACTION ITEMS FROM MEETING No. 10 – 6 March 2000

ITEM	ACTION	ACTION BY	STATUS
1.	To co-ordinate with other states a consistent approach to estimating pilchard biomass. Further analysis of the proportions of the year classes that died in both mortality events, especially in WA.	Dr Keith Jones	
2.	To contact relevant predator researchers and provide progress report for the next meeting.	Ms Gaut	
3.	To get data on the impact on Australian salmon fisheries in WA.	Dr Brian Jones	
4.	To reapply to FRDC for grant for project to look at predator-prey interactions of Australian salmon and pilchards in SA.	*Dr Keith Jones*	
5.	To submit the report to FRDC through JPSWG	Dr. Morgan	
6.	To consolidate transmission reports from Dr Brian Jones into a working document.	Ms Gaut	
7	To make official declaration that the mortality event of 1998/99 is over.	Dr. Morgan	
8	To put together a final report for CCEAD.	Dr Morgan	
9	To put together a report on research progress for FRDC.	Dr Morgan	
10	To find out whether Australia has in the past imported pilchards from Japan; OMV – a salmon herpesvirus – is present in Japan and is apparently very similar to PHV.	Mr. Peebles	
Pending Action Items from Ninth Meeting			
2	Herpes Virus DNA from New Zealand and from 1995 and 1998 pilchard mortality events to be sent to AAHL for genetic fingerprinting – Dr Crane has reported that until such time as positive controls can be developed for the PHV PCR, then they will not be testing the New Zealand tissues.	Dr Crane	On hold
3	Pilchards from overseas countries to be tested for the presence of Herpes Virus.	Dr. Crane	On hold
4	Funding research program - WA to invoice SA (SA Biosecurity Fund) for the cost of the pilchard herpesvirus transmission trials conducted in Western Australia.	Dr. Brian Jones	Pending
6	Investigating the capability of alternative laboratories to develop pilchard gill cell cultures.	Dr. Crane	On hold

JOINT PILCHARD SCIENTIFIC WORK GROUP
ACTION ITEMS FROM MEETING No. 11 – 21 July 2000

ITEM	ACTION	ACTION BY	STATUS
1.	To suggest to SARDI that they co-ordinate with Fisheries in SA and WA to write a FRDC proposal for a project about pelagic ecosystem trophodynamics.	Mr Zacharin	
2.	To contact SARDI about getting copies of their two papers (MEPS and ICES).	Dr Bernoth	
3.	Mr Peebles to put together a proposal for a ballast water sampling project for submission to the BWRMG.	Mr Peebles	
4.	To write an endorsement on behalf of JPSWG of the modeling report for FRDC.	Dr Bernoth	
5.	To suggest a change of wording for the discussion and liaise with Dr Bernoth.	Mr Peebles	
6.	To notify Dr Sandy Murray of the decisions of the group regarding his report.	Dr Bernoth	
Pending Action Items from 9^{h} Meeting			
2.	Herpes Virus DNA from New Zealand and Australia from 1995 and from Australia from 1998 pilchard mortality events to be sent to AAHL for genetic fingerprinting – Dr Crane has reported that until such time as positive controls can be developed for the PHV PCR, then they will not be testing the New Zealand tissues.	Dr. Crane	On hold
3.	Pilchards from overseas countries to be tested for the presence of Herpes Virus.	Dr. Crane	On hold
4.	Funding research program - WA to invoice SA (SA Biosecurity Fund) for the cost of the pilchard herpesvirus transmission trials conducted in Western Australia. WA to send copy of invoice to SA.	Dr. Brian Jones	Pending
6.	Investigating the capability of alternative laboratories to develop pilchard gill cell cultures. – AAHL is waiting for pilchard heart and liver cell culture characterisation. It is not appropriate to develop gill cell lines until it is known whether the virus is viable.	Dr. Crane	On hold.

APPENDIX 9: LIST OF SOME INTERNATIONAL AGREEMENTS TO WHICH AUSTRALIA IS A SIGNATORY AND WHICH ARE RELEVANT TO AQUATIC ANIMAL HEALTH, QUARANTINE AND IMPORTS.

The International Aquatic Animal Health Code

The Code aims to facilitate trade in live aquatic animals and aquatic animal products by recommending minimum health requirements for imported products. The Code promotes health status zoning, pre-export testing, inspection and certification to reduce the risk of disease transfer through trade.

The Code was produced by the Fish Disease Commission of the Office International des Epizooties and was adopted in 1995. In 1997 a second edition was published and a third edition has just been endorsed, which will be on the internet and in print in October, 2000. The Code provides guidelines and recommendations including:

- general principles and practices for disease control in international trade
- specific import/export procedures
- internationally recognised lists of important diseases
- methodology for conducting import risk analyses
- the reporting of the disease status and disease control policies of Member countries.

Convention on Biological Diversity (CBD)

The CBD was created at the Rio "Earth Summit" in 1992 and entered into force in December 1993. The overall objectives of the Convention are the conservation of biological diversity, the sustainable use of its components and the fair and equitable sharing of the benefits arising out of the utilisation of genetic resources.

Under the CBD, each contracting party (over 150 countries) is required to:

- Identify processes and categories of activities which have or are likely to have significant adverse impacts on the conservation and sustainable use of biological diversity;
- Prevent the introduction of, control or eradicate those alien species which threaten ecosystems, habitats or species;
- Develop or maintain necessary legislation and/or other regulatory provisions for the protection of threatened species and populations;
- Where a significant adverse effect on biological diversity has been determined pursuant to Article 7, regulate or manage the relevant processes and categories of activities;
- Promote national arrangements for emergency responses to activities, or events, whether caused naturally or otherwise, which present a grave and imminent danger to biological diversity and encourage international cooperation to supplement such national efforts and, where appropriate and agreed by the States or regional economic integration organisations concerned, to establish joint contingency plans.

The National Task Force on Imported Fish and Fish Products (DPIE, 1996) describes in detail the interaction of Australia's environmental and quarantine legislation with CBD rights and obligations.

Convention on International Trade in Endangered Species (CITES)
CITES was signed in 1973 and seeks to preserve the wide variety of wild fauna and flora through international cooperation, with regard to protection of certain species of wild fauna and flora against over-exploitation through international trade. Under the convention, Australia has a number of responsibilities, including:

- Parties to the Convention shall not allow trade in specimens of species included in Appendix I, II or III under CITES except in accordance with the provisions of the present Convention;
- The import of any specimen included in Appendix I shall require the prior grant and presentation of an import permit;
- The Parties to the Convention shall take appropriate measures to enforce the provision of the present Convention and to prohibit trade in specimens in violation thereof.

These shall include measures:

- To penalise trade in, or possession of, such specimens, or both; and
- To provide for the confiscation or return to the State of export of such specimens;
- As far as possible, the Parties shall ensure that specimens shall pass through any formalities required for trade with a minimum of delay.

Table Appendix 9.Other international agreements which affect Australia.

Convention on Wetlands of International Importance
United Nations Convention on the Law of the Sea
Convention for the Protection of the Natural Resources and Environment of the South Pacific

APPENDIX 10: DETAILED METHODS OF WA TRANSMISSION TRIALS UNDERTAKEN AT BREMER BAY, BY WA FISHERIES

1. A supply of locally caught live pilchards was collected at Bremer Bay and held within a sea cage prior to the start of the trial work. A second lot of pilchards were transferred to a 12,000 litre recirculation system set up within a local factory. The pilchards housed within both of these facilities were kept for a period of three weeks prior to the beginning of the trial work during which time they were acclimatised to a commercial aquaculture feed.
2. When confirmed reports began of pilchard deaths near Esperance, a team from the FHL sampled a range of moribund, sick and healthy pilchards at this locality with the aim of collecting viable virus. The gills from these fish were removed and immediately placed into viral transport media. Approximately 10kg of infected pilchards were collected in this way and the gills transported to Perth for further processing at the FHL.
3. Gill material was processed by homogenisation using a pre-cooled (-20°C) sterile mortar and pestle and then added to phosphate buffered saline (pH 7.4) with 10% fetal calf serum was added. The suspension was then clarified by centrifuging at 2000*g* for 15 minutes. A second clarifying step was taken by centrifuging at 10,000*g* for 30 minutes in a type TI 45 rotor. Samples of suspension were then examined by negative contrast electron microscopy to confirm the presence of viral particles. Virus was then pelleted at 110,000*g* for one hour to concentrate into a smaller volume suitable to prepare saturated feed. A sample of the concentrated virus suspension was subjected to sucrose density gradient centrifugation in an attempt to estimate numbers of intact herpes-like viruses that might be present in the suspension.
4. The trial set-up consisted of 6 x 660 litre tanks, each with independent filtration and aeration facilities. Three identical replicate tanks were used for the treatment whilst the remaining three were used as control tanks. The treatment and control tanks were contained within the same shed but separated by a 4m high plastic curtain in order to avoid aerosol contamination. At the start of the trial period approximately 20-30 of the pilchards were placed in each tank giving a minimum of 60 pilchards within the treatment group and a further 60 within the control tanks.
5. All six tanks were monitored twice daily for dissolved oxygen, pH, temperature and salinity to ensure that environmental parameters remained constant between treatment and control tanks. Nitrite and ammonia content was measured once daily and in the event of poor water quality, 1/3 of the water from each tank was replenished by natural sea water, or, if there was a risk of contamination with PHV from this source, from the reservoir tank (capacity 12,000 litres). Feeding within each tank occurred twice daily using a commercially available Black Breem feed to which the pilchards had been adapted.
6. On day one of the transmission trial (January 14, 1999) feed was coated with viral particles by mixing 3.3 grams of the PHV suspension (virus plus transport media) with 3.3 grams of pelleted feed. Uninfected feed was produced by mixing 3.3 grams of feed with 3.3 grams of virus-free transport media. Thus a total of 6.6 grams of each appropriate feed mixture was fed to pilchards in control and treatment tanks on six occasions over a two-day period. Feeding occurred on three occasions on each of the two days and was separated by a period of one hour. On all occasions the pilchards immediately consumed the feed with no residual feed remaining in the tanks.

7. In order to avoid cross contamination of the treatment and control groups, the control tanks were fed prior to the mixing of the feed and fresh gloves were worn when mixing feed and feeding the pilchards. Operators rinsed all equipment with a disinfectant ('Vircon') at the end of each feeding session.
8. All tanks and fish were monitored for a total of 23 days (including 2-day infection period). The number of deaths within each tank was recorded four times daily and any dead pilchards were sampled immediately. One set of gill arches was removed with half placed in glutaraldehyde fixative and the other half frozen. The remaining gill arches together with the head and organs were fixed in 10% seawater buffered formalin. All samples were individually identified and the time of death and tank number recorded.
9. On day 13 of the trial a sub-sample of eight fish were removed from the treatment tanks and samples collected.
10. At the end of the 23 day period 20 fish from each of the control and treatment tanks were killed and processed according to the protocol described in point 8. Residual pilchards (14 treatment and 15 control) were kept within the tanks and monitored for a further 21 days.
11. Gills from treatment and control fish were examined for lesions using histology. Detection of PHV within this tissue was to be done using PCR at AAHL.

APPENDIX 11: FAECAL AND BLOOD SAMPLES TAKEN IN WA IN 1998

Identification	Sample	Species	Date	Location
1.SW	Serum	Shearwater	9/03/1999	Breaksea Island: Albany
2.SW	"	"		"
3.GWP	"	Great-winged petrel	"	"
4.GWP	"	"	"	"
5.GWP	"	"	"	"
6.GWP	"	"	"	"
7.GWP	"	"	"	"
8.GWP	"	"	"	"
10.SW	"	Shearwater	"	"
11.GWP	"	Great-winged petrel	"	"
12.GWP	"	"	"	"
13.GWP	"	"	"	"
14.GWP	"	"	"	"
15.SW	"	Shearwater	"	"
16.GWP	"	Great-winged petrel	"	"
17.GWP	"	"	"	"
19.SW	"	Shearwater	"	"
20.SW	"	"	"	"
21.SW	"	"	"	"
1.BB	Faeces	Crested terns	26/01/1999	Bremer Bay Beach
2.BB	"	"	28/01/1999	"
3.BB	"	"	1/02/1999	"
4.BB	"	"	2/02/1999	"
5.BB	"	"	5/02/1999	"
DB1	"	"	"	Dillon Bay Loc 1
DB2	"	"	"	Dillon Bay Loc 2
Pl1	"	Pelican	"	Pallynup Inlet
CR1	"	Crested terns	"	Cape Reche
A	"	Pacific gull	21/01/1999	Whicham Island
B	"	BF Cormorant	23/01/1999	Kimberly Island
C	"	Silver gull	24/01/1999	Hood Island
D	"	"	"	"
E	"	Crested terns	26/01/1999	Investigator Island
F	"	Silver gull	24/01/1999	Hood Island
G	"	Crested terns	26/01/1999	Investigator Island