

**A Review of the Outbreak of a Herpes-like Virus in the
Abalone Stocks of Western Zone Victoria and the
Lessons to be Learnt.**

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Executive Summary

As of March 2007 a herpes-like virus that escaped from an abalone farm near Port Fairy in May 2006 has spread to infect reefs over some 90 km of coastline in the Western Zone of Victoria's abalone fishery. To date it has infected reefs which formerly produced about 65% of that zone's long term catch about 160t of production per annum. On the farms it produced total mortalities of blacklip, greenlip and hybrid abalone. In the wild it has been observed infecting both blacklip and greenlip reefs and causing mortality rates of 40-95% so far.

There are three possible hypotheses for its genesis:

- it is a wide spread endemic native cultivated up to lethal concentrations in abalone farms,
- it is a restricted endemic that has been translocated by abalone farms into naïve populations, or
- it is an exotic species that has jumped from a closely related species or has been created in the farm system.

The first is the best case scenario. In that case the epidemic should have burned itself out in the wild relatively quickly as the natural resistance of the wild stocks asserted itself. The worse case scenario is the third scenario involving an exotic type of virus that has jumped between closely related species or has been created by hybridization in the hatchery. In this case there will be little natural resistance and the disease might spread through large regions of the Australian fishery potentially causing mortalities of >95% might occur over some years. The southern Californian stocks of abalone are showing little signs of recovery almost 2 decades after Withering Foot Syndrome caused >99% mortalities on natural abalone beds.

By assuming that the best case scenario is occurring and failing to manage against the risk that the worst case scenario might be occurring the Victorian Department of Primary Industry has potentially placed the entire Australian abalone industry in serious jeopardy.

A recent review of the situation by a group of international molluscan disease experts brought to Australia by the Western Abalone Divers Association of Victoria (WADA) highlighted the Australian lack of awareness that its small abalone farming sector (<\$10 million per annum; capitalized value <\$30 million) will inevitably continue to create disease challenges for the valuable wild stock fishery (\$250 million per annum; capitalized value \$2.5 billion). On probability some of the next disease challenges will have the potential to be worse than the current outbreak. The Australian abalone industry has little awareness of this bio-security risk which threatens this entire industry.

An immediate moratorium on changes and development in the abalone farming sector is recommended so that a thorough and rigorous Risk Assessment and Cost-Benefit analysis can be conducted to analyze the value and risk profile of the farm sector, within the context of the high value of the wild catch sector. Careful analysis might find that the abalone farming presents a larger risk to Australia's public good than its long term potential benefit can justify.

If the risk of abalone farming is accepted in Australian waters, a rigorous and actively enforced set of bio-security Standard Operating Procedures (SOPs) must be developed and implemented immediately for both farming and wild catch sectors, and for recreational fishing sector. The implementation, updating, and enforcing of these SOPs must be supported by adequate permanent funding by the government agencies promoting and regulating the development of the farm sector. A part of the SOPs should be minimum design standards for farms, regular disease monitoring and the power to demand instant de-stockings and destruction of stock in the event of disease outbreaks. Existing farms unable to comply with these rigorous minimum standards should be closed down immediately and compensated out of the industry. Immediate and pre-emptive de-stocking is the front-line action for disease containment. Issues of cash flow and compensation must not be allowed to cloud judgment when a disease outbreak occurs. A permanent fund must exist where by immediate compensation is available for farmers forced to destock because of disease.

The Abalone Council of Australia (ACA) must overcome previous problems of cohesion that have beset it and rise to face the nation wide challenge of getting Australia's management and research agencies to rationally and accurately assess the biosecurity risk created by the farm sector. The question must be seriously asked and analysed; given that Australia has the most valuable and sustainable abalone fishery in the world can we afford the risk of having a farm sector as well? If those agencies answer yes to this question, the ACA must hold them accountable for placing real rather than notional biosecurity controls in place which protects the wild stocks from future disease outbreaks on the farms, and compensates the wild sector for future failures.

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Introduction

This report and the accompanying literature review by Dr Rob Day were commissioned by the Abalone Council of Australia.

I am an experienced fisheries ecologist with experience with a wide range of fisheries around the Asia-pacific region, and with specific expertise with the Australian abalone industry. I have been consulting to the Victorian Western Zone Abalone Divers Association (WADA) since 2001 giving advice about their strategy of fine scale, reef by reef management. Since the initial outbreak of the abalone virus in the abalone farms of the Western Zone during 2005/2006 I have watched closely as the issue has unfolded. While I have no formal training as an epidemiologist I have followed the outbreak closely since its first outbreak, and recently helped convene and facilitate WADA's study tour by 5 international experts in molluscan diseases. As will be seen from the report the opinions advanced in this document are based heavily on what I learned from those experts during that week long tour in September 2006 and the reports they subsequently prepared for WADA.

Outline of Facts

As outlined by Hardy-Smith (2006b) during the months leading up to the outbreak (October – December 2005) a considerable amount of translocation of abalone occurred. Various translocation of wild caught abalone into SOM, CS and Great Southern Waters (GSW) as part of the FRDC family lines project which had the aim of establishing blood lines with a range of stable selected features, and between CS and Abonex for the purpose of commercial grow out of hatchery produced seed stock. Some mortalities were observed during these translocations but were not considered abnormal (Hardy-Smith (2006b)).

The first case of a new abalone disease was noticed in western Victoria at Coastal Seafarms (CS) near Portland in early December, 2005 when Paul Hardy-Smith was called in as a consultant to help the farm manage the outbreak. Transportation of abalone from CS to another pump ashore facility, Southern Ocean Mariculture (SOM) near Port Fairy, on 13 December 2005 apparently infected SOM with the disease. Similarly transport from CS to Abonex, an In-Sea farm in Westernport Bay, also resulted in infection during December 2005 (Hardy-Smith (2006b)).

An initial report of the outbreak was prepared by Hardy-Smith (2006a) in January 2006, as of that time large numbers of abalone were continuing to die at CS, a number of tanks had been de-stocked at SOM and this appeared to have arrested the outbreak, Abonex had removed all but a few survivors from their leases. At the time of the outbreak the farms were holding a mixture of blacklip and greenlip abalone together with a substantial proportion of blacklip – greenlip cross hybrids. The proportion of hybrids being farmed had been increasing over some years as they are favoured for their growth rates, appearance and their behaviour in the tanks.

Subsequently both CS and SOM were allowed to continue managing their respective outbreaks and marketing whatever product they could salvage through the local abalone processing facility at Port Fairy, Sou'west Seafoods. On 29 August 2006 Hardy-Smith (2006b) reported that mortalities had continued at CS until they totally de-stocked on 15 June. At SOM the disease had re-emerged on 17 March which had totally de-stocked by 15 May. A new infection had broken out on 24 April in a second farm in Westernport Bay, Kilcunda Abalone Farm (KAF) a barrel type grow out operation in close proximity to the Abonex farm.

On 2 May 2006 the disease was detected for the first time in the wild population in Taylors Bay the coastal lagoon from which the SOM pumps its water. On 9 June 2006 the two abalone zones (Reef Codes) adjacent to SOM (Burnetts & Water Tower Bay) were declared a Control Area prohibiting diving in them. Together these lagoons had provided 6.2% of the long term Western Zone yield of about 250t per annum. On 26 July the important reef code immediately to the west of the SOM, called the Craggs (another 7.25% of the annual Western Zone TAC), was also declared a Control Area because a few cases of the disease been detected there as well. On 1 August a DPI Vic bulletin was released notifying that the disease had also been detected in pockets all the way east past Port Fairy to Mills, extending the infected zone about 5 kms to the west, and 10-15 km to the east, from the original Control Area. On 15 August the Control Area was redefined to take in this larger area of affected coastline. In total about 25 km of productive bottom which had produced about 24% of long term Western Zone yields.

During August stock assessment surveys were repeated on long term population monitoring sites at the Craggs. They measured a 60% decline in population abundance since the previous year's surveys, the disease was still active in the area being surveyed. The survey divers could account for two-thirds of the population decline with freshly dead shells they collected in the transect lines. The survey divers also brought back graphic images of the dead and dying abalone losing muscle control, and sliding or falling off their home scars on the reef, as first their mouth and then their feet swelled. The divers noted the occurrence of the epidemic was extremely patchy with aggregations on different sides of the same large boulder being variously infected or apparently healthy. The survey divers also noted the heavy load of mucous streams being released into the water column by the dying abalone in areas where the epidemic was extremely active. They noted the normal fish seemed to be absent from these mucous loaded areas.

From 16-21 September 2006 the affected abalone fishery stakeholder group, the Western Abalone Divers Association (WADA) organized an international review of the situation the core expertise involved in the review were the Drs and/or Profs:

- Mike Hine, New Zealand's senior marine veterinary consultant to Ministry of Agriculture Forests and Fisheries, also involved in controlling the early 1990s disease outbreak in the Western Australian pearl industry and implementation of bio-security protocols for that industry.

- Tristan Renault, a France based international expert on molluscan herpes through his work on the Herpes Virus carried in the genome of the Japanese Pacific Oyster.
- Caroline Friedman, An international expert on abalone disease outbreaks having studied the outbreak of two abalone diseases in California.
- Judith Handler, Senior aquaculture veterinary expert for Tasmanian DPIWE

I was invited to join the tour as the WADA scientific representative and facilitated an experts workshop on 20 September 2006.

The tour visited all the sites involved, and talked with those with first hand experience of the disease outbreak and its research to that date. On Monday 18th they met with the members of WADA in Port Fairy. On Tuesday 19th the various experts had the opportunity in the laboratory to look at samples first hand, and discovered for themselves that the herpes-like virus also appears to be around the mouth parts of the affected animals, from which some inferred a possible pathway into the ganglions. On Wednesday 20th I facilitated a small workshop with the four visiting experts, two senior Victorian DPI staff, four WADA executives, Ana Mouton Veterinary Expert for the South African Abalone Farming Industry brought in by Victorian DPI, Mark Crane and Serge Corbeil of AAHL (CSIRO) and Harry Gorfine abalone researcher with PIRVic.

Also present was Paul Hardy-Smith of Panaquatic Health Solutions P/L the consultant brought in by the farms in December 2005 to help them counter and subsequently used by Victorian DPI to document the outbreak (Hardy-Smith 2006a&b), and who has subsequently assisted in the drafting of Biosecurity Standard Operating Procedures (Gavine et al. 2007).

That workshop previewed the experts' findings and planned presentations for the half day Open Forum held on the topic the following day Thursday 21st by the Victorian Department. At the open forum on Thursday 21st almost all those present the previous day were involved giving presentations and/or on an expert panel answering questions, and much the same material was reviewed, more formally and superficially for an Australia wide audience of managers, industry and research.

The material presented and discussed during that tour, and the subsequent reports (Friedman & Renault 2007, Handler 2007, Moutton 2006) informs the opinions and recommendations advanced below.

In November 2006 the Control Area was extended eastward again as the disease became established in Kilarney, and The Cutting. During December 2006 the infection reached Warnambool at the eastern end of the Western Zone and was separated from the Central Zone of Victoria by just 4-5 km of sandy bottom. By this stage some 45km of coastline was infected comprising 42% of the zones long term production equivalent to about 105t per annum of production.

Stock abundance surveys were repeated through the zone during January – February 2007. At the Craggs a decline of about 79% was observed since the outbreak, declines of approximately 50% were measured at the Lighthouse Reef Code, 57% at Mills and Kilarney, 95% at the Cutting and 40% at Levy's. The surveys do produce relatively variable measurements under normal fishing, by comparison the surveys at Julia Percy Island which to date has not been affected by the virus, but has been fished relatively heavily, had declined by about 30%.

During February 2007 I dived the infected area over two days with one of WADA's top divers starting with the site of the original infections and working our way along to the Warnambool breakwater where the infection looked to be still actively infecting abalone making a DVD of what we saw. To the west of Warnambool we saw large amounts of dead shell and small clumps of re-aggregating survivors which at that stage appeared healthy, there was no active sign of the virus. Around Warnambool there was active signs of the virus; large amounts of intact moribund abalone, shiny empty shell, loose meats rolling around in the wash and a few surviving abalone, some still healthy others visibly sickening and dying. Most of the reefs we dived were blacklip reefs. We dived one formerly productive greenlip reef and it had clearly been heavily impacted, there were only a handful of survivors that we could find. We saw no evidence of the disease affecting any other species besides abalone.

At the Kilarney reef code, an area I have dived several times I observed what I visually estimated to be more like the 95% depletion measured by surveys at the adjacent reef code of the Cutting. In the other reef codes my visual impression was similar to the survey measurements. The variation between my visual estimation and the surveyed measurement at Kilarney may well reflect the patchy nature of the disease's impact within the same reef code as the survey site is somewhat inshore of the main commercially fished beds.

Also in February 2007 the disease was observed for the first time on Cape Nelson at Portland the source of another 23% (56t/annum) of the Western Zones long term catch. The disease was found first in a Reef Code called Killer Waves Killed which is favoured by recreational divers and fishers and at the bottom of a track down the cliff the locals call "Poacher's Stairs". There had been no commercial diver presence in the area for some months, and the areas to the east from Cape Grant to The Craggs remain uninfected as of the time of writing. So the inference is that this new infection has been transferred by recreational fishers or divers; either in diving gear or in abalone guts which local recreational fishers use to fish for sea-sweep. The disease is now working its way west around Cape Nelson and is spread over some 90km of coastline, only about 25 km of coast is contained within the Control Area.

As of the time of writing (late March 2007) the Victorian DPI had not moved to make any further extension of the Control Area despite repeated verbal and written pleas by the executive officer of WADA.

Expert Findings

Epidemiology (Patterns of Disease)

Inflammation of nerve ganglions (ganglioneuritis) was observed with light microscopy in affected abalone submitted from CS to Paul Hardy-Smith in January 2006.

Drs Mark Crane and Serge Corbeil of CSIRO's Australian Animal Health Laboratory (AAHL) in Geelong later used electron microscopy to visualize herpes-like viruses in inflamed neurons in the foot muscle. AAHL also conducted basic epidemiology trials demonstrating that the disease is transmittable by both the injection of fresh and frozen (-80°C) body fluids and via the water column. One hundred percent mortalities were observed within 5-6 days for all treatments while no mortality was observed amongst their controls. From these experiments they concluded that:

1. The virus is transmitted through the water column from sick abalone to the healthy abalone
2. The virus is highly pathogenic, killing abalone within a few days of infection
3. Injection of the virus in the abalone foot causes disease and mortality
4. The virus remains virulent and pathogenic after being frozen at -80°C

Drs Mark Crane and Serge Corbeil also conducted experiments into the effect of dilution of infective water on the mortality rates of the abalone. An initial experiment using water transported from an infect farm and tested at dilutions of 100%, 10%, 1%, 0.1% and 0.01% failed to produce any infections. From which they concluded:

1. There was no (or little) virus present in the water at the time of sampling (virus titres may fluctuate in a farm setting, particularly in flow-through systems), and/or
2. The transportation of the water to AAHL affected the viability of the virus present in the water, and/or
3. The virus in the water was present at a titre that was too low to cause a productive infection in the abalone

Subsequently the infected healthy abalone by placing them with with diseased animals (held in basket) until approximately 50% of them had died. They then took that water, as simulated farm water and exposed healthy abalone to various dilution levels (100%, 10%, 1%, 0.01%, 0.001%) for a 48 hour period. The 1-100% dilutions resulted in 100% mortality over the following 20 days. No mortality was observed with <1% dilutions. From this they concluded:

1. The virus remained infectious to animals even after a 1 in 100 dilution (although only one of the duplicate tanks was affected at this dilution compared with both tanks affected at 100% and 10% levels)
2. Even though the dilution factor at the outlet of the farm is greater than 1 in 100 and while dilution reduces infectious dose, it remains possible that wild mollusc species could become infected by virus released into the environment

Herpes and Herpes-Like Viruses

Viruses of the Family *Herpesviridae* have a range of characteristic features which can be identified visually using electron microscopes, these include:

- Hexagonal shaped outer coatings (capsids) 90 to 100nm in diameter
- Capsids which form in the nucleus of the hosts cells
- Electron dense cores originating as spiral of RNA (toroid)
- The virus buds into the cytoplasm of the host cell
- The virus acquires a semi-electron dense 'tegument'

However, a virus identified as having these characteristics is identified as being 'herpes-like' until its genetic material has been purified and sequenced. Only when its genetic sequence is shown to belong to the *Herpesviridae* Family can the virus be definitively identified as a herpes virus. In the case of ganglioneuritis from Western Victoria this latter stage of definitive identification is still proceeding and the virus can only be identified as being herpes-like. Never-the-less within this document I will proceed under the expectation that the final stage of the identification process will confirm that the virus belongs to the Family *Herpesviridae*.

While little known in molluscs until the last decade, according to Le Deuff & Renault (1999) herpes viruses may be ubiquitous in this Family of animals and perhaps in virtually all other animal Families as well. So far amongst molluscs they have mainly been identified in bivalves but this is primarily a function of where the research effort has been applied.

They are renowned for lying latent for long periods within the genome of host species and individuals. They may even be transmitted latently down germ-lines, between generations via sperm and ova (vertical transmission), or in the fluids produced along with the sperm and ova (pseudo vertical transmission). Latent infections often erupt out of latency in response to some environmental stress. Hence the recent global outbreak of herpes in Pacific Oysters is thought to have been caused due by the latent virus being transported all around the world within the species' genome and then have been activated independently in each country by a period of widespread warmer than normal coastal sea temperatures. Trials with the Pacific Oyster Herpes Virus has shown that it is infective during the larval phase of the Pacific Oyster (*Crassostrea gigas*) and that it is particularly effective at cross-species infection of the larvae of other bivalve species (Le Deuff et al. 1994, Arzul et al 2001).

In Australia herpes-like viruses have only been seen before in bivalves (Hine & Thorne 1997, Handlinger 1999). However, a herpes-like virus has been decimating the Taiwanese and Chinese abalone culture industries over the last few years (Wang et al. 2004, Chang et al. 2005). The two viruses are visually (electron microscopy) and symptomatically extremely similar but current techniques are not capable of confirming if they are the same virus or not.

Techniques based on the immunological recognition of the viral DNA - Polymerase Chain Reaction (PCR) techniques need to be developed; both to determine the

relationship between the Australian and Taiwanese virus and to test whether or not the virus is present in latent form in some natural reservoirs. There is currently an FRDC funded project involving a CSIRO and DPI Vic partnership aimed at developing the necessary genetic tools. It is in the interest of all states to support the development of these basic techniques.

Herpes viruses are typically ancient species with large complex and stable genomes that have co-evolved closely with a single host species over many millions, or even hundreds of millions of years to ensure that they cause long term chronic diseases (i.e. human herpes), rather than pathogenic epidemics (large scale mortality) amongst their normal hosts. (Renault and Novoa 2004). Characteristically their life strategy is to infect a high proportion of a population (40-60%) and this achieved by evolving to keep their host alive rather than to cause mortality. Species of herpes are known to occasionally jump between closely related species and in this situation may cause high rates of mortality not seen in their normal host (Le Deuff et al. 1994, Richman et al. 1999, Arzul et al 2001). Because of their host specificity they are generally only able to jump between very similar species but the small change in species may produce lethal effects. Both these aspects of herpes behaviour are vividly illustrated by Richman et al. (1999) who diagnose the cause of the extremely high rates of mortality occurring in the fetuses and young calves of American and European zoo populations of elephants. In that situation a long history of the zoos housing Asian and African elephants together has resulted in the respective Asian and African herpes viruses jumping into the other species of elephant, resulting in a level of mortality which is preventing the zoos from establishing self sustaining captive breeding populations.

Source of the Outbreak

Why Bother Speculating on the Source and Timing?

Moutton (2006) argues that “it is tempting, but ultimately pointless, to speculate on outcomes resulting from decisions which were not taken”. But this is answered by Handler (2007) who points out that for the purpose of planning on-going “responses to future outbreaks in this or other locations” we need to review what has happened in detail. “The purpose of such an exercise should be not just to determine whether other actions are likely to have been more successful, but whether there were restraints that prevented such actions being taken that can be avoided in the future.”

Three Broad Possibilities

During the Wednesday 20th September 2006 workshop all the experts present agreed there are three sustainable hypotheses as to the source of this virus:

1. The virus is a wide-spread endemic which through stress and high density in aquaculture facilities has reached critical / epidemic infection rates and is now burning like spot fires through a population which should have some level of widespread resistance to it. This is the best case scenario as in this case the wide-

- spread resistance to the disease should begin asserting itself as viral loads are diluted back to the natural background level common in the wild.
2. The virus was a localized endemic which through translocation and culture has been introduced to naïve populations in western Victoria. In this scenario the Australian source stocks should have some level of resistance to this disease, but whether the resistant stocks will be extensive or extremely localized we can't know.
 3. The virus is some form of exotic virus which has jumped out of its natural host into the abalone stocks. Its source could be from overseas or Australia, but most likely from a closely related species. This is the most serious possibility as it implies that there will be little natural resistance to the disease anywhere in the Australian abalone stocks. In this worst case scenario we can only hope for some low level of natural resistance, and that exposed to the pressure of natural selection this herpes virus will rapidly attenuate back towards a more normal pattern of herpes epidemiology.

These three possibilities have since been re-iterated by Moutton (2006) and Gavin et al. (2007).

The visiting experts also made clear that it is unlikely we will ever know for certain exactly how and why this infection occurred. Most likely, even after PCR techniques have been developed and used to investigate the distribution of the virus in abalone and related species here and internationally, we will still be left weighting a sub-set of the various scenarios, and maintaining several prime explanations. Learning the lesson of this outbreak, and pre-empting the next disease challenge should not be delayed for want of greater research knowledge.

Competing Strands of Evidence and Logic

A Virus that Occurs Naturally in Wild Abalone Somewhere in Australia

Hardy-Smith (2006b) has conducted the authoritative review of the outbreak chronology and concluded 'that the index case in this disease outbreak is most likely to have occurred at CS' the Portland abalone farm and that the most likely source was "live wild abalone that were brought onto one of the farms at the start of the outbreak". In his interpretation of events he places a lot of emphasis on the start of the outbreak being mid-December.

As noted by Handler (2007) this conclusion is almost entirely based on Hardy-Smith's belief that the Geelong pump-ashore farm Great Southern Waters (GSW) was not exposed to the threat of disease, despite being a partner with CS and SOM in the FRDC family line projects, and having shared various translocated shipments of abalone. The evidence and logic assembled by Hardy-Smith (2006b) is that GSW which remained disease free shared shipments of wild stock abalone with CS and SOM up until 18 November 2005.. Consequently Hardy-Smith (2006b) concludes that the disease entered CS and SOM, after 18 November probably in early December from the South Australian wild stock abalone that went to CS and SOM alone.

While this possibility cannot be discounted, I agree with Handler (2007) that this interpretation of events unnecessarily limits our consideration of the full range of potential risks that were occurring at that time, and so denies us the chance to fully absorb all the lessons that need to be learnt from this event. This is a classic case where the old adage should be applied – the absence of proof is not proof of absence.

A well placed and reliable source who was working at GSW during that period, but who wishes to remain anonymous informed me during the Vic. DPI Open Forum on Thursday 21 September 2006 that Hardy-Smith's observation that GSW did not undergo a disease challenge in November, is only true in the broader sense; in that they never experienced any mass-mortalities. That source believes that the statement created a misleading impression for Hardy-Smith's analysis and that the Geelong facility did have a disease challenge involving farmed abalone that were transported from Tasmania on 10 November, 2005 'rehydrated' in a live holding recirculation system in Richmond, Melbourne and then shared between the three facilities (GSW, SOM, CS). That challenge however was only noticed for what it might have been in retrospect, because the GSW biosecurity protocols dealt with it routinely within their world best practice quarantine facility (Friedman & Renault 2007). When an unusually large number of containers containing triplets of translocated abalone began dying on the following days, the operator of the quarantine facility routinely euthanized everything in quarantine, there was no further infection and the possible relevance of the event only assumed importance in retrospect.

The point this raises here – is that there have been no protocols in place about routinely freezing and storing mortalities for thorough post-mortem health checks – if that had happened we could now go back and check whether these animals were infected by the virus.

In terms of possible source this observation opens up the possibility that the infection could have come from virtually any of the places that abalone held in the Richmond facility were transported from or any source linked to the Tasmanian farm.

An Exotic Source

This observation also raises the possibility of exposure to cross species infection in the recirculation system of the commercial live holding facility in Richmond where translocated abalone were routinely 'rehydrated'. That facility handled a wide range of live sea-food in-transit for export and import purposes. One species of particular relevance here because of its close relation to abalone and international source, that the facility is known to have been handling during that time is a Chilean limpet that was being imported for canning by Sou'west Seafoods in Port Fairy (Handler 2007).

Tristan Renault the French oyster herpes expert regarded the pathogenicity (high mortality rates caused by the virus) to be of great interest. As discussed previously he notes that herpes viruses are normally closely evolved with a very specific host to cause chronic, rather than deadly disease and to be carried long term by a high proportion of the population. From his point of view the high mortality rates being observed in the wild

and farms suggested the virus could possibly have jumped into the naïve abalone from another species, and because of this the infectiousness and deadliness of the infection is extreme and not moderated correctly for the new species. If this has occurred the closely evolved species specific nature of herpes dictates that the original natural host species will be closely related in evolutionary terms to abalone.

The most likely candidate for a cross species jump include the Chilean limpet which shared the Richmond re-circulation system as discussed above. Another candidate could be the elephant snail (*Scutus antipodes*), which looks much like an abalone meat without a shell. These animals were apparently found in surprisingly high numbers living cryptically and unnoticed in the plumbing of the farms until the facilities were completely de-stocked and sterilized.

A third possible variation of the 'species jump' theory is suggested by the work of Richman et al. (1999) on elephants, and the fact that the farms involved have been moving towards farming substantial proportion of blacklip – greenlip cross hybrids.

Accepting, as the experts do, that both greenlip and blacklip abalone are likely to be carrying their own species of herpes, that these viruses are likely to be extremely similar and that they insert themselves into the same standard locations of the abalone genome, and considering the scale of hybridization that was being practiced by the farms in the lead up to the outbreak, there is a possibility that while hybridizing the two abalone species the two herpes viruses have also been hybridized. Under this scenario the exotic virus is a new hybrid species that is not correctly attenuated with any of the host species and this is causing its observed pathogenicity.

In all three of these latter scenarios the virus has jumped species within the farms or the Richmond recirculation facility and has then been transported around the farms, and the apparent correlation with the program of wild stock translocations highlighted by Hardy-Smith (2006a&b) is purely coincidental.

As highlighted by Friedman & Renault (2007) and Handler (2007) there have been no protocols in place for recording mortalities, deciding when a level of mortality is normal or above normal and there has been no routine health status reports conducted on mortalities (checking of the cause of death) or abalone entering the farms (checking for the presence of diseases). It is relatively normal epidemiology for diseases to start off slowly in a population with the infection of just a few animals, but gathering momentum as the proportion of sick animals slowly grows until finally like a wild fire the epidemic becomes unmistakable. This is exactly what was seen in the farms and in the wild. Outside Port Fairy and at Mills Reef the first individual sick animals were found some 6 weeks prior to the general outbreak being observed. With no strict protocols in place and no archiving of mortalities for post-mortems there is actually no way of saying the epidemic began in the farms in early December 2005. This is purely the time when the farm could no longer deny to themselves that something out of the ordinary was occurring and that it was time to refer to Dr Paul Hardy-Smith who has greater expertise with disease outbreaks than themselves.

There was a great deal of discussion about potential linkages with the Chinese herpes-like abalone virus and about the potential for transfer of the Taiwanese virus into Australia. In late 2004 a batch of abalone feed was imported from Taiwan which contained fishmeal, seaweed powder, soybean powder, gluten, vitamins and minerals. The feed caused stomach abnormalities and some mortalities and its use was apparently discontinued in early 2005. The disease experts involved in the WADA study tour discounted this as likely source of infection on the grounds of the, the heat treatment it would have received during importation, the time elapsed between its use and the current outbreak, and the different pathology observed. An Australian expert we consulted on the BSE outbreak (mad cow disease) in Britain and Japan stressed that heat treatment can produce highly variable results and believed that the food linkage has to date been dismissed to summarily. Samples of the feed were given to AAHL for testing with for the presence of the virus when the PCR technique is developed.

Given the volume of live abalone product being transported from Australia to Taiwan the experts were more inclined to expect that if the viruses are identical it will be because an Australian virus has been previously exported to Taiwan, and is only now being recognized in Australia. Mike Hine has since raised the possibility that because phylogenic studies suggest Australian and Taiwanese abalone share the same ancient lineage there is a chance both Australian and Taiwanese will naturally carry some identical viruses through the original infection occurring before the species diverged.

Another theory about the potential linkage between the Australian and Taiwanese herpes-like viruses can also be countenanced if the idea that a blacklip and greenlip herpes virus has been hybridized in the Australian farms. The extension of that idea would be that all abalone species are carrying very similar herpes viruses which have the potential to be hybridized. While I do not know what level of hybridization has been practiced in Taiwan and China, I have no reason to suspect that the pressures that induced Australian abalone farmers to scale up the practice, will not have also been at work in those countries. If in fact they have been hybridizing within their suite of very similar species and sub-species the Taiwanese and Chinese may have in fact given rise to their own version of lethal hybridized herpes virus and this possibility will exist where ever large scale abalone hybridization is practiced.

It will not be possible to distinguish between any of these various possibilities, until genetic recognition techniques (PCR) have been developed, and a range of species tested nationally and internationally for latent copies of the virus.

Managing Risk in the Face of Uncertainty

At the heart of risk assessment and management is the idea of identifying all possible outcomes and weighting them by both the likelihood that they will or are occurring, and their likely impact were they to occur. In this way devastating events that are considered highly unlikely may rightly receive greater attention than highly probable events that have only a small impact.

In the Hardy-Smith (2006a&b) and Moutton (2006) reports and the actions of the Victorian DPI authorities there has been a propensity to assume that this disease outbreak has its genesis in a translocation of a virus from the wild stocks into the farms. While this has to be entertained as a real possibility, it is also the best case scenario in terms of the likely long term impacts for the wild fishery. By not seriously considering the possibility that the worst case scenario might be occurring and moving immediately as soon as they were notified of the event in January 2006 three months before the virus escaped from the farms, the Victorian authorities have gravely compromised the sustainable future of the entire Australian abalone industry.

Likely Long Term Impact

This herpes-like disease clearly passes through phases of infections, small infections involving a scattered few individuals were detected initially in both the Craggs, and Mills Reef codes some weeks/months before full scale epidemics erupted. Some months after sweeping through each area the virus is leaving behind what appears to be some variable level of healthy survivors. The diving I undertook in February 2007 showed these survivors are re-aggregating in too much smaller but none-the-less normal looking aggregations. Typically a large crevice would be seen full of empty scars, 5-15 dead shells could be dug out of the sand at the bottom of the crevice showing what had happened to the original population, and clumped in one corner of the now empty crevice would be a little group of 2-5 abalone. A good sense of this is gained by viewing the WADA DVD that we made during these dives.

Data shown by Harry Gorfine (PIRVic) to the September 2006 study tour gave the results of transect surveys of emergent abalone which had recently been conducted at long term stock abundance survey sites at The Craggs. The results at that time detected a 60% drop off in abundance. Two thirds of which could be accounted for in the collection of the freshly dead shells within the transects.

The normal long term stock abundance surveys were repeated through the zone during January – February 2007. At the Craggs a decline of about 79% was observed since the outbreak, declines of approximately 50% were measured at the Lighthouse Reef Code, 57% at Mills and Kilarney, 95% at the Cutting and 40% at Levy's, an average impact to date of 62% across all age classes – including the pre-emergent juvenile age classes. These surveys do produce relatively variable measurements under normal fishing, by comparison the surveys at Julia Percy Island which to date has not been affected by the virus, but has been fished relatively heavily because of the circumstances, had declined by about 30%.

In February 2007 I swam over parts of the Kilarney reef code, an area I have dived several times and observed what I visually estimated to be more like the 95% depletion measured by surveys at the adjacent reef code of the Cutting. In the other reef codes my visual impression was similar to the survey measurements. The variation between my visual estimation and the surveyed measurement at Kilarney may well reflect the patchy

nature of the disease's impact within the same reef code. Based on the previous size structure of the emergent population at Kilarney, and its history of stable production, if the depletion I observed was characteristic of most of that reef code, rather than the depletion measured at the more inshore survey site, I estimate that 100-200t of biomass had been lost from that one reef code.

It is difficult to predict whether these measurements reflect what will eventually prove to be the full extent of the mortalities or whether the disease will remain active at lower levels on the infected reefs for some time to come. The virus could even become latent in surviving and apparently healthy animals, in this case it could erupt sporadically over long periods of time giving rise to further epidemics. In either case the disease might take years to eventually kill off of a large proportion of the infected populations. In California during the 1990s the Withering Foot Syndrome (WFS) took several years in each new location to produce >99% reductions in dense shallow water black abalone stocks (Friedman & Finley 2003; Miner et al. 2006).

Assuming that the disease continues as it is running west around Cape Nelson, but that somehow it is prevented from infecting Cape Grant and Julia Percy Island to the east, and Cape Bridgewater and Discovery Bay to the west 65% of the Western Zone long term production, some 160t of annual catch has now been infected.

If the disease's impact is limited to the current areas and the average 62% die-offs recorded so far by the PIRVIC surveys. WADA will be faced with two choices lock catches in permanently at approximately 40% of previous levels or attempt to manage a recovery back to pre-virus levels over some 15-20 years by accepting 20-30% of previous yields over that type of time span.

But even this is best case scenario. In California large changes have been observed in the encrusting communities on the bottom which has made the habitat unsuitable for abalone and abalone settlement (Miner et al. 2006). The grazing of dense abalone populations encourages the growth of the pink encrusting coralline algae which are the preferred settlement sites for abalone larvae. In the Western Zone of Victoria it is already obvious that the unused attachment sites of the abalone are being overgrown by filamentous algae which no doubt will give way to the same succession of longer lived sessile invertebrates and macro-algae. The long term change away from a heavily grazed crustose algal community may well delay or even prevent any hoped for recovery in these stocks. In California where the population decline has been extreme and the habitat has changed very little recovery is being seen after 15-20 years.

The loss of production will obviously also depress incomes and license values over these time frames by <60%. Should the die-offs observed continue as has been observed in California and populations reduced <95% over several years, the commercial fishery in these areas will be many decades recovering any productivity, if it recovers at all, and a capital loss of \$50-60 million will be incurred by Western Zone quota values. None of these scenarios encompasses the likelihood that the disease will continue spreading to new reefs in which case losses could become much higher.

There is also no way of knowing how much further this disease is going to spread through the Australian abalone industry. The disease has so far shown itself to be deadly for both black and greenlip, and their hybrid progeny. The virus is also showing itself extremely effective at spreading via the water column in the wind driven alongshore drift. In transmitting itself from the Port Fairy reefs to Mill's Reef it showed itself capable of jumping 8-10 kms of sandy bottom. With the transmission to Portland we also now have the first evidence that humans can carry the virus from place to place, most worrying back towards the west against the prevailing west wind drift. This makes it likely that it will now continue spreading both naturally east into at least the Victorian Central Zone, and eventually (perhaps more slowly) in human mediated jumps along the tourist trail west into South Australia. It would only need a slightly larger range of infectivity to that already displayed, and/or assistance from human mediated transport, for it to island hop across Bass Strait to Tasmania.

The long term outcome of this outbreak will be largely determined by which of the three epidemiological scenarios, outlined previously, is occurring.

The first of the three broad scenarios for this disease outlined previously; is that the virus is a widespread endemic which was been 'hot-housed up' to lethal concentrations by aquaculture, would have been likely to have the least impact on the wild stocks. Under this scenario the wild stocks should have resistance, it is the toxic concentrations developed in the farm and released into dense wild stocks that has created the 'hot-burning' epidemic. When the viral load is dispersed by the open flows of the ocean, the natural resistance of the wild stocks should exert itself and the disease resort to its normal latent and chronic nature. The disease should burn itself out and everything will return to normal.

This is the scenario favored by Hardy-Smith (2006a& b) and Anna Moutton (2006) in her report of the Viral Study tour. It is also the scenario that Victorian DPI have chosen to assume is occurring, and, to date, the only one they have chosen to manage. Unfortunately, as Handler (2007) points out, there is no evidence to support the notion that this scenario is happening. Moutton (2006) recycled the original Victorian DPI belief that the outbreak was not having a significant impact on the wild stocks as evidence that the wild populations were not naïve and therefore had had previous exposure to the virus, but one need only look at the DVD WADA had us make in February 2007 to see that this belief and the theories based on it are entirely fallacious being based on wishful thinking rather than fact. The longer the outbreak persists and the further it spreads the more foolish and risky this thinking looks.

The third group of scenarios which involve the introduction or creation of an exotic new species of herpes either by cross species jumping, or hybridization, are the potentially most devastating scenarios. Under these scenarios little if any natural resistance will exist in Australian abalone. Unlike humans abalone apparently do not have immune systems which are capable of developing recognition of a new disease. Instead it is to be hoped that some percentage of the current population will have a level of natural resistance and

that these resistant individuals will be left in significant numbers and be capable of rebuilding resistant populations quickly enough to prevent the long term habitat that could close them out of area semi-permanently. The other possibility that can be hoped for in this suite of scenarios is that virus itself will attenuate towards being more like a normal herpes virus which causes chronic disease rather than mass mortalities. Now that the virus is in the wild we should expect natural selection to be favouring the spread of strains which keep their hosts alive the longest and over time this should drive the virus to evolve a less destructive epidemiology. Unfortunately herpes are large, complex and stable so that its evolution is not expected to be rapid.

The second scenario is that this is a virus that naturally occurs in some corner of the Australian abalone stock and that it was translocated into naïve farm and wild populations in Victoria. Under this scenario there is hope that around the source of the virus the abalone populations will have naturally high levels of immunity. This scenario offers some intermediate level of hope, depending on whether the area in which natural resistance occurs is large or small. If it is a very localized area this scenario becomes virtually identical to the third group of scenarios, if large more like the first scenario. Only time and events will tell.

It is not being melodramatic to consider the possibility that over 2-10 years this virus could spread to virtually all parts of the Australian abalone industry and put at jeopardy an income from wild caught abalone valued at beach prices around \$250 million/annum and a capitalized value of around \$2.5 billion. Whether this doomsday scenario will occur, or to what degree, and with what speed it happens, really depends upon which of the three scenarios for the origin of the virus is correct.

Bio-security

In this section I turn from analyzing what has occurred and is occurring to what lessons should we be learning from the outbreak and how we should be trying to change previous practices that created the opportunity for this outbreak to occur. I start with more generalized bigger picture comments and finish with some detailed comments which I have focused around the draft Biosecurity Control Measure for Abalone Herpes-Like Virus: A Code of Practice (Gavine et al. 2007) which has recently been prepared and circulated by Victorian DPI for comment. My aim here is to provide both general and specific comments that members of the ACA can be using to influence policy reviews that will flow from this outbreak.

The international experts who participated in the WADA study tour agreed unanimously that abalone mariculture inevitably posed a disease risk to the wild stocks of a species and that the best that could be achieved was an active management of that risk.

While abalone farming continues in Australia there will always be some level of risk. The only hope is to minimize that risk to the greatest possible extent. They emphasized this first point by warning Australians not to become too focused on this particular outbreak

because there would be more to come and in all probability some of them will be more serious than the current disease. Their literal comment was; “Stay focused on outbreaks two, three, four, five etc.....”

They were genuinely surprised with the lack of awareness about the inevitability of the mariculture industry being challenged by disease and this would unavoidably have major impacts for wild cultivation. They commented on the fact that this lack of awareness extended from farm operators and farm investors, through abalone divers and quota holders, and most worryingly the management and research agencies who have fostered and supported the fledgling farm sector.

They were extremely critical of the general lack of an implemented and effective Standard Operating Procedures (SOPs) mandating the action to be taken immediately under such a bio-security incident.

A glaring but only indicative example of the lack of bio-security awareness has been the FRDC Family Lines project, which had as a central aim translocating broodstock from all over Australia so that selective breeding could occur, and in time stable blood lines which could be used for breeding without further recourse to wild brood stocks. The aim of the project – broodstock sources independent of the disease risk of wild abalone is important as a means of reducing long term bio-security risks, but the experts stressed that this was always going to be one of the industry’s most high risk bio-security enterprises ever undertaken by the farming sector. The project apparently had strong protocols in place ensuring the integrity of the family lines produced, but to date we have seen no evidence that it had any protocols concerning bio-security.

It is noteworthy that the SOM and CSF were leaders in that project. The experts rated their farms as positively third-world and expressed their surprise that such primitive conditions existed in Australia. Also noteworthy and indicative of the poor standards in practice is that one of the activities being undertaken; collaborating farms sharing animals from each translocation was a particular effective way of maximizing disease risk. The risk adverse strategy would have been to hold each translocation in GSW which had effective quarantine facilities, breed them in that facility and only share their proven disease-free progeny.

Assuming that further disease challenges would inevitably be created by the continuation of the abalone farming industry the experts emphasized the need to start rigorously assessing the risk of this activity, and implementing and enforcing SOPs based on the risk profile of the Australian farm and wild sectors. Much of the Wednesday 10th September 2006 Workshop was detailing standards that should be part of any SOP for an abalone farming industry, these details have since been documented in the formal reports to WADA prepared by Friedman & Renault (2007) and Handler (2007). To my mind it is indicative of the Victorian DPI attitude displayed through this outbreak that the Biosecurity Control Measures document prepared by Gavine et al. (2007) has been prepared with reference to this body of material.

In broad principle they highlighted:

The need for Australian policy makers to acknowledge the disease risk posed by abalone farming to a valuable wild sector resource, and to put into place appropriate and effective risk management strategies that seek to strategically target the highest risk activities which are:

- The high density and low water flow conditions relative to natural populations increases the prime drivers of infection rates and fosters disease virility.
- Translocation of animals and diseases from differing locations (within state, Inter-state and Inter-national) always run the risk of transferring un-identified diseases between resistant and naïve populations.
- The close co-habitation of closely related species within crowded aquaculture conditions increases the risk and impact of chronic or latent diseases in one species jumping into another species and causing epidemics. In particular re-circulation systems handling a wide range of species, especially stressed individuals in transit to market, present a major risk of this occurring. Other species living openly or cryptically through hatchery systems also present this risk i.e. elephant snails in the plumbing.
- Contact between farmed and wild animals, via water flows, escapees, brood-stock translocations or disposal of offal and mortalities. A major bio-security issue on all farms is controlling the movement of abalone through the plumbing system. In all the Pump-On farms visited escapee abalone had established populations throughout the system including in the settlement ponds, which during disease challenges became infective reservoirs of the disease rather than entrapping sinks.
- Farms should not be placed near natural beds of abalone and in this regard the practice of In-Sea farming needs especially stringent consideration.

The major issue for this industry is the issue of contaminated water flows. The risk of disease outbreaks impacting wild stocks would be almost eliminated if the flow of water between farm and wild stock could be terminated. Given the central need to maintain water flows of the magnitude of 500-1000 l/sec this does not seem in anyway feasible while abalone farming continues.

There are however ways of managing the broader profile of risks associated with these discharges by simultaneously managing risks at multiple points of the process. By rigorously managing risks at multiple points across the risk profile the final risk of an outbreak can potentially be kept at very low levels.

For example the risk of another outbreak from a land based farm can be managed by:

Reduce the Risk of disease Entering Farms

By regulating for separate quarantine facilities, to receive all incoming animals before they enter production areas. These quarantine facilities should have separate water supplies, separate staff who park in different areas to other staff and enter via different approaches and who use separate equipment and facilities.

Before being released from quarantine all abalone should be subject to complete and thorough health examinations and all mortalities should be subject to post mortem health examinations and archiving.

Reduce the risk of epidemics developing in the production side of farms:

Farm Design

- Designing farms to have multiple separate units of production with separated water supplies, feeding arrangements, equipment and staff.
- Securing all the stock and preventing them from moving around the farms by; using air-gaps to prevent crawl back up stream through the plumbing, and grids to prevent them crawling down stream into other tanks, drainage channels and settlement ponds.
- Piping waste water into drains rather than have them splashing down and potentially creating infectious aerosols.
- Cement rather than dirt floors so that all surfaces can be washed down.

Rigorous Protocols

- Mortalities should be subject to post mortem health examinations and archiving.
- Mortality rates should be monitored carefully so that long term 'normal averages' can be determined and so slightly raised levels detected.
- Each tank should have its own cleaning brushes rather than carrying brushes between tanks and production areas.
- Staff not walking through and across tanks but using paths.
- Controlling access to farms and full biosecurity briefings for all people entering farms

Reduce the risk of epidemics entering the wild:

Public Policy & Protocols

- No farms to be situated within 10 km of natural abalone beds.
- Immediate destruction of stock upon the outbreak of a disease

Farm Design

- Small high risk water flows from quarantine facilities to be fully contained and subject to sterilization before release.
- Sufficient volume in settlement ponds to provide for residency time of 4-5 days rather than <1 day which seems to be relatively normal practice.

Implemented together in this way a suite of risk management actions like this could be used to reduce the overall risk associated with outflow issues. However, importantly in this situation all measures require implementation together to manage the overall risk. Typically in this situation special pleading of economic hardship and practicality leads to the reduction in one or more standards which inevitably weakens the broader multi-faceted strategy to a point of being meaningless.

Unfortunately the options for managing the risk with regard to In-sea Farms are much more limited with separation by distance being the principal means. Certified disease free

status of abalone entering these facilities and rigorous monitoring and post-mortem checking of mortalities would also help. However, In-Sea Farms are inevitably going to be stocked with animals from hatcheries and so will always present a major risk of introducing disease into the wild. From what we are observing with this disease one suspects separations of at least 50-100 km would be needed to have any degree of certainty that an outbreak from an In-Sea farm of significant size could not possibly be transmitted into wild stocks. It is difficult to imagine anywhere on Australia's southern coastline where this type of separation from natural stocks exists, thus there may well be a good case for deciding that this small and struggling sector is not worth the risk it creates.

The final point the visiting experts were agreed on was that the farm industry must have some form of self-financing insurance scheme which anticipates the future need of rapid de-stocking and which is designed to make compensation for compulsory de-stocking automatically available. A forced immediate de-stocking of the 4 farms involved any time between December 2005 and February 2006 would have eliminated the risk of the disease jumping into the wild. Such action would mean that the disease would now be fully contained, and income and capital losses, would have been restricted to the farm sector responsible for the outbreak. The experts repeatedly stated their belief that it is only with an automatic compensation systems that government decision making processes can remain unencumbered from special interest pleading and remain capable of the rapid decisions needed to pre-empt disease outbreaks and order compulsory de-stocking.

Specific Comment on Gavine et al. (2007)

General Comments

In general this is a shoddy misleading document which reflects the Victorian DPI's lack of interest, knowledge and concern for the wild sector of the Australian abalone fishery.

Indicative of this is the statement on page 1 that:

“The Victorian Department of Primary Industries (DPI) also took steps to restrict entry into marine areas that were known to be infected with the virus. The area around Port Fairy in south-west Victoria was declared a Control Area and fishing and diving activities restricted to minimize the risk of human activity to unaffected abalone populations elsewhere in the State.”

This statement is followed by similar statements on page 7 on the same issue – this time with regard to the Code of Practice for Recreational Divers and Fishermen. In this place it is followed by the statement:

“At the time of writing, the Closure extended from the high water mark 1km west of the Craggs car park to the Killarney Beach car park.”

Noting that this document was released for comment in March 2007, the point here is that Warnambool approximately 10 km east of the closed area has been infected since December 2006 and Cape Nelson some 40km west of the closed area has been infected since February 2007. Despite repeated requests from the WADA executive Victorian DPI has refused to extend the control area to include these new areas of infection.

Of particular concern is that the heavily infected Warnambool breakwater is outside the Control Area. When I was there in February 2007 I witnessed snorkelers parking in the carpark and walking 10m to dive straight into infected areas, and boat loads of scuba divers in small rubber dinghies boating out just 100-200m from the boat ramp, around the breakwater to dive in infected areas.

This document is riddled with similar misleading examples. The cynical and deliberately misleading nature of this document is illustrated by a statement on page 1:

“Origin: It is not known whether the virus is exotic or endemic to Australian waters, but experts strongly suspect that it is endemic as no probable linkages with an exotic source has been identified.”

In fact only the experts employed by the farm sector and Victorian DPI (Hardy-Smith and Moutton) share this view. The broader group of international experts involved in the WADA study tour all believed that various ‘exotic’ sources should not be excluded at this time (Friedman & Renault 2007, Handler 2007, Personal Communication Dr Mike Hine, Investigation and Diagnostic Centre, Biosecurity New Zealand, Ministry of Agriculture and Forestry).

This aspect of the document is further illustrated by the statement on page 7 with regard to the Code of Practice for Recreational Divers and Fishermen:

“These two protocols were released in December 2006, supported by a comprehensive community engagement campaign undertaken by DPI Victoria.”

The ‘comprehensive’ nature of this campaign is illustrated by the signage placed at the Port Fairy boat ramp – a small A4 sized sign, placed so that it is effectively hidden to everyone using the boat ramp by a rubbish bin. Unfortunately this example is typical of their campaign.

The ad-hoc and poorly advised nature of the document is illustrated by the following statement on page 7:

“The recreational harvest of abalone is considered to be low compared with the commercial catch and this sector was not specifically considered in the risk assessment process for this project.”

Particularly in the light of the infection at Cape Nelson starting at the main point of recreational access one would think it obvious that in this sort of situation risk has nothing to do with the quantity of product being handled, but rather the number of operators involved and the awareness and motivation of those operators. All of which would place the recreational sector as a far greater source of risk than commercial operators – particularly when DPI has declined to extend the Control Area to include such points of easy access as the Warnambool breakwater.

Basic errors in fact contained in the document are illustrated by this statement on page 11:

“Around 50% of the total exported (2,032 tonnes) was exported as fresh, chilled or frozen product and the remainder (1,972 tonnes) was exported in cans.”

One would suspect that in this context the fact that a major part of the Australian catch is being exported live is relatively pertinent information.

With such misleading and partisan statements in the preamble and salted all the way through the document there can be no faith at all that this document is genuinely aimed at addressing this major issue for the Victorian and Australian abalone industry, and this sentiment is borne out by an examination of its content.

Standard Operating Procedure 4: Biosecurity protocol for abalone aquaculture farms

Of particular concern for the Australian abalone industry is SOP 4 which is specified by the section on the Code of Practice for the Aquaculture Sector as one of two primary SOP's for farming sector. This SOP exemplifies the continuing attitude of Victorian DPI in its concern to protect and foster the farm sector at great cost to the wild sector. It is all about protecting the farm sector from itself and has no regard to protecting the wild sector from the farm sector.

This is basically a cosmetic exercise to justify a business as usual approach. Notably absent are any measures that go to the heart of the issue – fundamental flaws in the ad-hoc design of these facilities. Aspects of basic best practice design and practice which were highlighted by the WADA's team of international experts, but which are sadly lacking in this flawed document are:

- Separation of abalone farms from wild abalone beds by 5-10 kms
- Effluent water retention times in settlement ponds of 4-6 days.
- Compartmentalised units of production so that complete self contained units of production can be rapidly destroyed to contain disease outbreaks
- Cement flooring rather than dirt floors so that all surfaces can be routinely washed down and disinfected.
- Piping for water flowing out of tanks, rather than open spillways and cut-off piping upto 1m above ground level, to prevent the creation of aerosols

- Grates to prevent downstream escaping of farmed abalone
- Routine diagnostic health status checks on farmed and quarantined abalone rather than subjective observations.

Of specific concern are:

SOP 4.1 Health management of stock an abalone farms

SOP 4.1 para 3. “Where moribund or dead abalone are observed with clinical signs of disease that causes suspicions of the presence of the herpes-like virus samples should be submitted for diagnostic testing

And

SOP 4.1 para 4. “Where herpes-like virus is not suspected, moribund and dead abalone should be immediately removed from the culture unit and disposed of according to the Disposal of Mortalities protocol (SOP 4.6)”

As highlighted by WADA’s panel of international experts subjective evaluation of mortalities will inevitably lead to the beginning of epidemics being over looked. If the disease is some form of exotic it could be changing quite rapidly as it adapts to its new environment, the WADA made DVD of February 2007 suggests the clinical signs of the dying abalone are already changing with less swelling around the foot and mouth parts than was seen 6 months ago. Even with the best will and application in the world looking for the expected will lead to the unexpected being overlooked.

There needs to be mandated post-mortem health checks and archiving of mortalities, along with rigorous recording, monitoring and evaluation of mortality rates so that slight increases can be immediately detected and investigated. This need is only referred to in loose generic terms at the end of SOP 4.1 with no direction or standards laid down as to what should be recorded, how average mortality rates should be recorded and how the significance of small increases might be determined.

SOP 4.3.1 Routine Cleaning of Culture Units

Para 3. “Culture units should remain empty for as long as possible between batches of stock.”

Friedman and Renault (2007) suggested that culture units should be held dry between batches – it is not clear that this is what SOP 4.3.1 means and a minmum time should be stated rather than the subjective “as long as possible”.

SOP 4.3.2 Period Cleaning of Pipework, wastewater channels and settlement ponds.

Para 1. “ All land-based pipes and wastewater channels must be cleaned regularly to limit the formation of biofilms and the accumulation of organic matter. During cleaning, the pipe work should be drained and external

fouling removed. Ideally, a “pigging system” should be used as well as flushing lines with a disinfectant (e.g. chlorine solution).”

A “pigging system” is a foam projectile which is forced along a line using compressed air. They are highly effective at removing biofilms and organic matter. There is a strong possibility that the current outbreak in the wild began when the intake pipe at SOM was “pigged” and diseased farm abalone were flushed out into the wild. No consideration is given in this document to this risk for the wild sector. It appears entirely acceptable to these authors for the farm sector to flush its fouling out into the wild.

SOP 4.3.4 Disinfection of influent water

Para 1. It has not been possible to quantify the risk of water coming onto a farm carrying viable virus of establishing infection in the abalone populations on that farm. Disinfection of influent water may be required were a farm to be repeatedly infected with the virus”

Indicative of the tenor of this document there is no corresponding section on disinfecting or containing effluent water where a farm is repeatedly infected with a disease. Likewise these protocols provide no standards for residency times in settlement ponds

This exemplifies the Victorian DPI attitude throughout this outbreak, there is and has been a one eyed view that the only possibility worth considering or managing involves a virus coming from the wild into the farm. No consideration is given to the reverse situation. Which it should be emphasized is the only occurrence known to have occurred i.e. in early May 2006 a virus escaped from a farm site into the wild.

This attitude is further illustrated by SOP 4.5 – my italics added.

SOP 4.5 Prevention of escapes

“Abalone are often found in the pipework and settlement ponds on farms. Uncontrolled abalone present two major risks to the *abalone farmer*:

- Abalone in pipeworks can re-enter the farm and spread infection to healthy stock. It is therefore recommended that there is an air-gap between the tank and drain to prevent re-entry of escaped abalone.
- There is an increased risk that abalone in settlement tanks can escape to the wild or interact with wild stock. *This can represent a reservoir of disease that can re-infect stock on a farm.* Settlement tanks should be regularly cleaned out to ensure no abalone populations are established.”

Friedman and Renault (2007) and Handlinger (2007) note that air-gaps are only effective in prevent abalone moving upstream through piping – thus they will only be effective in protecting the farm from abalone moving back into systems from the wild, settlement

ponds, drains and downstream tanks and will provide no protection to the wild from farm animals. Grates are needed to prevent the downstream escape of animals.

Regularly cleaning of settlement ponds will be ineffective because it can only be done by divers who inevitably will miss some proportion of the population. To be effective the ponds would need to be drained and left dry for several days, which would require the farm to be closed down for some days or having its effluent pumped directly into the sea in the interim. This is a motherhood statement which has no hope of being effectively followed.

From a wild sector point of view the only effective action here is the one not mentioned – grates. Presumably they are not mentioned because while they will be effective they will require constant maintenance by farms to prevent blockages which could result in flows backing up and flooding together with loss of downstream flows to other tanks i.e. risk to the wild sector is acceptable, extra work and difficulty for the farm sector is not acceptable.

Concluding Discussions

Potential Long Term Impact

The best case scenario favored by Hardy-Smith (2006a& b), Moutton (2006) and which the Victorian DPI have chosen to assume is occurring, and the only scenario they have chosen to manage suggests the long term impact of this outbreak should be localized and relatively short lived. Unfortunately, as Handlinger (2007) points out and the DVD produced by WADA in February 2007 graphical illustrates there is no evidence to support this blind optimism.

While we may never know for certain what gave rise to this outbreak the growing scale of the epidemic argues that something closer to one of the worst case scenarios is unfolding. Under these circumstance it is not being melodramatic to consider the possibility that over the next 2-10 years this virus could spread to virtually all parts of the Australian abalone industry and put at jeopardy an annual income from wild caught of around \$250 million/annum and a capitalized value of around \$2.5 billion. Whether this doomsday scenario will occur, or to what degree, and with what speed it happens, depends principally upon the extent and speed with which the virus attenuates towards being a chronic rather than pathogenic infection, and the level of resistance that might exist naturally around southern Australia.

At the heart of risk assessment and management is the idea of identifying all possible outcomes and weighting them by both the likelihood that they will or are occurring, and their likely impact were they to occur. In this way devastating events that are considered highly unlikely may rightly receive greater attention than highly probable events that have only a small impact. The Victorian DPI authorities and their consultants have from the outset insisted in the face of all evidence that the best case scenario has been occurring. This behaviour flies in the face of the precepts of professional risk management. By not seriously considering the possibility that the worst case scenario

might be occurring and moving immediately to close down the farms at the centre of the outbreak in December 2005 / January 2006 three months before the virus escaped from the farms, the Victorian authorities have potentially have compromised the sustainable future of the entire Australian abalone industry.

Source of the Outbreak

Rushing in where scientific angels would no doubt fear to tread, I conjecture that this outbreak will in time be confirmed to be a herpes virus, and that all species of abalone will be found to carry their own species of herpes which through close evolution with their hosts normally cause chronic rather than pathogenic infections. I further conjecture that these viruses are normally transmitted through the mucous trails that abalone lay down as they move around the reef surfaces. The virus probably attacks through the membranes of the mouth parts, traveling via the nervous system to the surface of the foot where the mucous laid down to lubricate movement is produced. In the current outbreak the virus is apparently over stimulating the mucous production mechanisms and the floating mucous filaments produced seem to providing the virus with a long distance infective ability. The mucous strands are presumably entrained by the wind driven along shore currents, eventually settling onto distant reef surfaces where they are picked up by grazing, or trapped inadvertently by abalone trapping drift algae with their feet.

I believe the most plausible explanation for the outbreak is that as the farms have increased the proportion of blacklip – greenlip cross hybrids they have been producing for farming that have inadvertently hybridized a blacklip herpes with a greenlip herpes to produce a virus, that like species crossing herpes, is now causes pathogenic rather chronic infections in blacklip, greenlip and hybrid abalone. I expect in time it will also prove to be pathogenic to the other Australian abalone species as well.

Lessons to be Learned and Judgements to be Made

As documented by the literature review prepared by Dr Robert Day of Melbourne University and which accompanies this document the mariculture industry around the world, like the terrestrial farm industry is continually throwing up disease challenges. As emphasized by the international experts WADA brought to Australia to review this current outbreak; *inevitably this will be only the first disease challenge to be thrown at the Australian abalone industry by abalone farming.*

It is clear that everyone involved with the Australian abalone industry, wild stock or farmed, researcher, managers or producer, has under estimated the disease risk created by developing an abalone farming sector.

It is time to stop the business as usual approach and question how the abalone industry of Australia is being managed in terms of maximizing its long term public benefit and minimizing the risks to its future. This should begin with a rigorous and transparent risk assessment process which places the risks created by abalone farming industry into the context of its value relative to the wild stocks, and the potential for long term damage to the wild resource from diseases introduced by aquaculture. Representatives of the wild

sector should be at the core of this process as they have most to lose if it fails to effectively do its job.

Without pre-judging the issue but cognizant of:

- The existing and long-term value of the wild sector of the abalone industry
- Australia's proud and almost unique record of sustainably managing its abalone fishery
- Australia's strategic advantage being its systems of good governance which have put it at the forefront of sustainable fisheries management.
- The history of the development of successful aquaculture for prawns, salmon, blue-fin tuna and pearls which has seen the devaluation of formerly high priced products, and
- The movement of those aquaculture industries away from developed countries with costly inputs, towards developing countries with lower priced inputs.

It may well be better public policy to halt the development of abalone farming in Australia and compensate current participants out of the industry, as a means of reducing the future risk to this valuable community asset.

The Future of Biosecurity Protocols in the Abalone Industry

On the subject of developing Standard Operating Procedures for Biosecurity a comment made by Anna Moutton (2006), the South African Abalone Mariculture industry's veterinarian expert in her report on the Viral Study Tour should be noted:

“The Department of Primary Industries is using this opportunity to develop and implement guidelines on good bio-security practices in the abalone industry. Practically, these guidelines cannot be enforced and the continued commitment of all in the abalone industry will be required for success.”

In other words SOPs are not worth the paper they are written on unless the industry is actively engaged, and regular enforcement of the SOPs occurs. The economic imperative of struggling operators will always be to deal with issues cheaply regardless of un-enforced protocols.

Recommendations

I recommend an immediate moratorium on changes and development in the Farm Sector while a rigorous Risk Assessment is conducted analyzing the value and risk profile of the abalone farming industry in the light of the high value of the Tasmanian wild catch sector. The question needs to be openly discussed; in the long term does the Australia economy and the Australian public stand to gain or lose by continuing to have an abalone farming sector?

If it is decided that abalone farming should be risked in Australian waters, a rigorous and actively enforced set of Bio-security SOPs should be developed and implemented

immediately for both the farm and wild-catch sectors. The implementation, updating, and enforcing of these SOPs must be attached to a permanent financial mechanisms which ensure realistic levels of long term funding. Without this basis of permanent government support for the implementation, ongoing education and long term enforcement there will effectively be no SOP in place in the long term. As a part of such a strategy there should be a program of regular disease monitoring and the power to demand instant de-stockings and destructions, accompanied by compensation mechanisms so that political decision making is not perverted by economic considerations for single operators. Furthermore, the cost of these programs and mechanism, and the risk that they will fail should be explicitly incorporated in the risk assessment of the industry.

If abalone farming is going to be accepted as a permanent part of the Australian abalone industry government needs to establish, implement and enforce a 'best practice' standard of design.

Under this 'best practice' design abalone farms should be required to establish fully isolated high security quarantine facilities to hold all animals entering a farm. Complete sterilization of the small flows of 'high risk' water from the quarantine system must be mandatory. Zero discharge from the production side of farms is also the preferred option for managing bio-security risks to the wild stocks, but if this cannot be achieved politically, the retention of production water for at least 4-5 days in settlement ponds before release must be the minimum design standard, along with the separation of farm discharge from wild stocks by at least 10 km.

On the production side farms need to be compartmentalized so that disease outbreaks can be contained through the immediate destruction of complete self-contained units of production. Air-locks and grids to ensure no uncontrolled movement of abalone occurs either upstream or downstream should also be mandatory for all production systems. Cement flooring in farms which can be washed and sterilized, rather than the common dirt flooring, and elimination of open outflow systems which create splash and the potential for infective aerosols to be created should also be made mandatory parts of farm design. Inlet and outlet piping must be designed so that they can be 'pigged' from the outside into the farm rather than discharging their potentially infectious effluent into the wild.

In-sea farms and reseeded are always going to be high risk activities that risk transporting undetected diseases into the wild. The risk of In-Sea Farming can in theory be controlled by significant spatial separation, although the scientific basis for setting 'safe' separation will always be questionable, while the question exists as to whether sufficient separation is in reality going to exist anywhere. Given that reseeded is targeted at former commercial reefs there will never be any prospect of minimizing the risk posed by this activity by separating it from natural stocks. Re-seeding has been shown to have been an important disease vector in the Californian abalone stocks. It should not be allowed in the future in Australia.

Immediate de-stocking is the front-line action for disease containment and would have prevented this current outbreak. Issues of cash flow and compensation must not be allowed to cloud judgment when an outbreak occurs. A permanent fund should exist where by immediate compensation is available for farmers forced to de-stock because of disease. This could take the form of an insurance fund paid into by growers wishing to operate in the industry.

The strictest controls on translocation should be in place, the routes of translocations should be vetted, and holding of translocated abalone in quarantine made mandatory. In vetting transport routes systems shared by other abalone or other live species must be avoided i.e. brood stock must be transported independently of live holding facilities used to hold a range of species in transit to markets.

Many of the existing abalone farms have been sited close to significant beds of wild abalone, and/or have grown and been developed through trial and error rather than by 'best practice' design. Particularly because they are likely to continue to be sub-economic, persisting by cutting corners as sucking in regular infusions of new shareholders funds, they will be unable to undertake the re-design and/or relocation required to make them compliant with 'best practice' design. These operations are likely to be long term sources of high risk for the wild abalone stocks of Australia. The ACA should make a concerted effort to make the State Agencies explicitly acknowledge this fact and compensate these operators out of existence. The old business as usual approach of having them go through successive bankruptcies, re-issuing of shares and refloats as new commercial entities will be a long term recipe for disaster for the wild catch sector of the industry. Likewise the production of documents such as Gavine (2007) which seek to put the best face on this business as usual approach and to whitewash the real gravity of the issues.

The ACA as the national body of the Australian abalone industry is ideally placed to pursue these issues as a national agenda and this is what the situation requires. In the past the abalone industry has had difficulty taking up a uniformed national approach because of its regional nature and state based management. Now is the time for change and this is the issue which requires change. As we may well be about to witness in overly graphic detail, epidemics know no political boundaries. In the case of biosecurity we are all threatened by the weakest links in the chain, as this Victorian situation is demonstrating to us now.

This really is a case of:

“never send to know for whom the bell tolls; it tolls for thee” – John Donne

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