Pilchard Herpesvirus in Australia 1995-1999

R.J. WHITTINGTON
Faculty of Veterinary Science, University of Sydney, Private Bag 3, Camden NSW 2570, Australia

J.B. JONES
Fisheries Department of Western Australia, Animal Health Laboratory, 3 Baron-Hay Court, South Perth, WA 6151, Australia

A.D. HYATT
CSIRO Livestock Industries, Australian Animal Health Laboratory, Locked Mailbag 24, Geelong, VIC 3220, Australia

ABSTRACT

Two epizootics have occurred in populations of the Australasian pilchard Sardinops sagax neopilchardus in waters of southern Australia. The first occurred between March and September 1995. It is thought to be the largest fish kill ever recorded and is also unique in the geographic extent of the mortalities. The economic loss attributed to the 1995 mortality event was in excess of A$ 12 million. The second occurred in 1998-1999 when approximately 60% of the total pilchard biomass in Southern and Western Australian waters was lost. After the 1998-1999 epizootic, two of the three pilchard fisheries of Western Australia were closed for a season and although the national economic impact has not been formally assessed, it exceeded A$ 15 million in Western Australia alone (Gaut, 2001). In 1995 mortalities occurred along more than 5000 km of the Australian coastline (Fig. 1) and also affected pilchards in New Zealand. The disease front spread from its origin in South Australia at about 30 km/day, often against prevailing currents and was not impeded by storm events. Thus it was not caused by planktonic toxins/pathogens. Likewise, there was no consistent association of the mortalities with environmental parameters such as temperature or salinity.

A feature of both epizootics was the wave-like nature of the mortalities, originating in South Australia and spreading to the east and west at a rate of 10 to 40 km per day. This provided an opportunity to mathematically model the disease (Murray *et al*., 2001a; 2001b). It was established that three parameters control the wave-like geographical spread: the rate of disease transmission, the length of the latent period (the period between becoming infected and becoming infective), and the diffusion coefficient. When examining model outputs against observed patterns of spread, a latent period of about 4 days, a very short infection period of about 1 day, a diffusion coefficient consistent with that of swimming pilchard schools, and an infection rate of over 90% were required. The model also revealed that the

Whittington, R.J., J.B. Jones and A.D. Hyatt. 2005. Pilchard herpesvirus in Australia 1995-1999. *In* P. Walker, R. Lester and M.G. Bondad-Reantaso (eds). Diseases in Asian Aquaculture V, pp. 137-140. Fish Health Section, Asian Fisheries Society, Manila.

epizootic was host density independent and that a single point source of infection was required in order to generate the observed wave. Multiple sources of infection, or the presence of vectors (such as seabirds) do not allow the wave to generate and the epizootic stalls.

Affected fish died within minutes of signs of respiratory failure (Fig. 2). To establish aetiology, fish were sampled before, during and after the advancing mortality front at Iluka in New South Wales and in Western Australian waters (Fig. 3). Relevant lesions were confined to gills and were unlike lesions associated with known gill pathogens or toxins in other species of fish (Whittington *et al*., 1997). Lesions were initially focal and inflammatory (Fig. 4b) but became locally extensive then generalised (Fig. 4c), with inflammation then being replaced by epithelial hypertrophy and hyperplasia over about 4 days. Lesions were invaded by bacteria and protozoa (Fig. 4d). The pathology in affected fish across the distribution of the disease was similar, suggesting a common aetiology.

A herpesvirus was the only factor consistently associated with lesions, including those in early stages of the disease (Fig. 5) (Hyatt *et al*., 1997). The herpesvirus has been detected by EM and PCR. The virus is now being characterised using molecular methods, but has never been grown in cell culture. At the time of the mortalities, attempts to grow the virus in standard fish cell lines were unsuccessful. Pilchard heart and liver cell lines were later developed by the Australian Animal Health Laboratory. Failure of the virus to grow in these new cell lines may be due to loss of viability after prolonged storage of tissues at -80°C.

The origin of the herpesvirus has never been determined, though suspicion has centred on either importation of large amounts (>10,000 t per annum) of frozen whole pilchards into South Australia for direct use as feed in tuna cages, or perhaps ballast water discharge of infected pilchard schools (Jones *et al*., 1997).

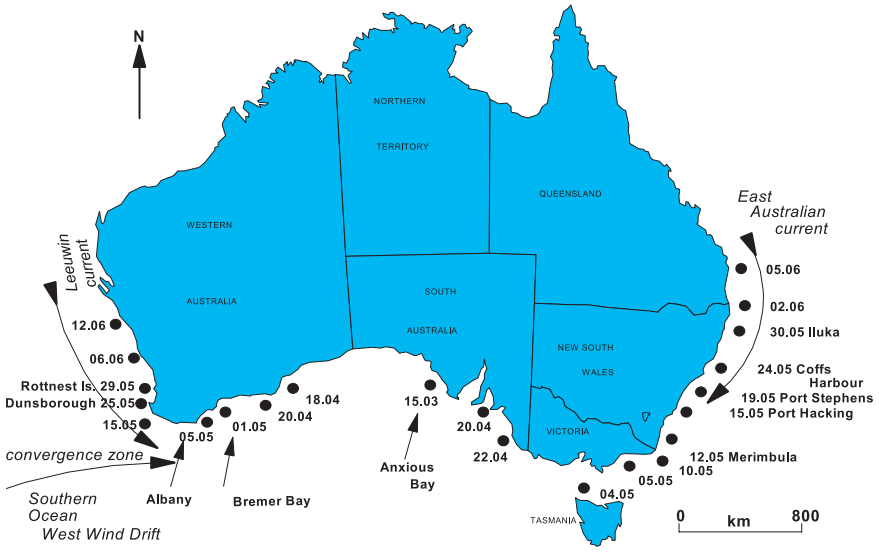

Figure 1. Spread of the pilchard epizootic from its origin in Anxious Bay in 1995. Dates (day.month) and locations of observed mortalities are indicated for easterly and westerly spread from the origin. Prevailing currents are shown by curved arrows.

Figure 2. Pilchards, central coast, New South Wales, 1995.

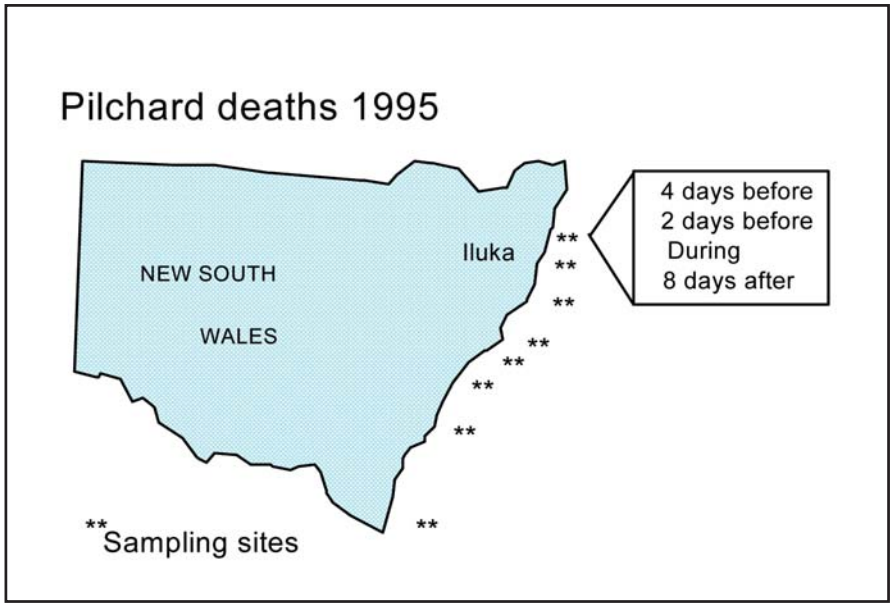

Figure 3. Sampling sites for pathological examinations.

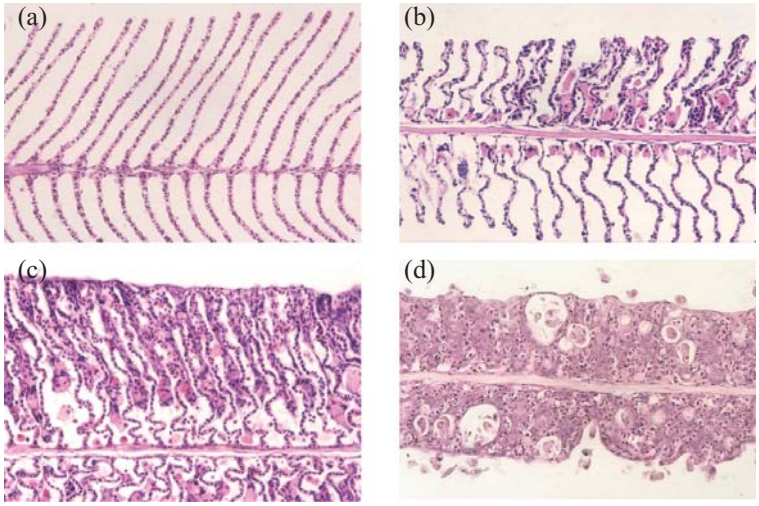

Figure 4. (a) Normal gill; (b) gill 4 days before mortality, with focal inflammation; (c) gill during mortality event with generalised inflammation and epithelial hyperplasia; (d) gill during mortality event with severe generalised inflammation and epithelial hyperplasia.

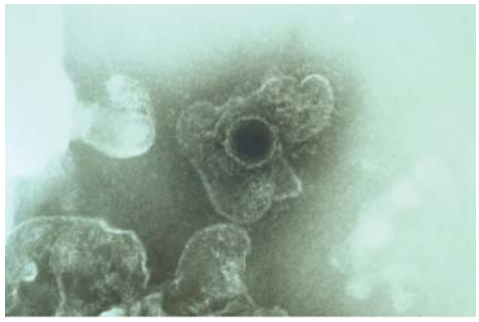

Figure 5. Electron micrograph of negatively stained enveloped capsid from gill of affected pilchard.

REFERENCES

Gaut, A. C. 2001. Pilchard (*Sardinops sagax*) mortality events in Australia and related world events. FRDC Project No. 99/227. Fisheries Research and Development Corporation, Canberra.

Hyatt, A. D., Hine, P. M., Jones, J. B., Whittington, R. J., Kearns, C., Wise, T. G., Crane, M. S. and 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. Diseases of Aquatic Organisms 28, 17-29.

Jones, J. B., Hyatt, A. D., Hine, P. M., J., W. R., Griffin, D. A. and Bax, N. J. 1997. Australasian pilchard mortalities. World Journal of Microbiology and Biotechnology 13, 383-392.

Murray, A. G., O'Callaghan, M. and Jones, B. 2001a. A model of transmission of a viral epidemic among schools within a shoal of pilchards. Ecological Modelling 144, 245-251.

Murray, A. G., O'Callaghan, M. and Jones, B. 2001b. Simple models of massive epidemics of herpesvirus in Australian (and New Zealand) pilchards. Environment International 27, 243-248.

Whittington, R. J., Jones, J. B., Hine, P. M. and Hyatt, A. D. 1997. Epizootic mortality in the pilchard Sardinops sagax *neopilchardus* in Australia and New Zealand in 1995. I. Pathology and epizootiology. Diseases of Aquatic Organisms 28, 1-16.