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Bonamia exitiosa epizootic in *Ostrea chilensis* from Foveaux Strait, southern New Zealand between 1986 and 1992

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Disease caused by the haplosporidian parasite, *Bonamia exitiosa*, swept through the dredge oyster (*Ostrea chilensis*) population of Foveaux Strait between 1986 and 1992, with consequent mortality reducing the population to 9% of the pre-disease level. Dead and dying oysters were first seen by fishers in far western Foveaux Strait in 1985 and more were found further east in 1986. Infection spread slowly through Foveaux Strait so the progress of the epizootic can be described from population surveys. A wave of infection radiated through the oyster population from the epicentre of infection in central western Foveaux Strait, and was followed by a wave of mortality. The epizootic ceased in oyster beds around the margins of oyster distribution in 1992. Infective particles released by diseased oysters spread through the water to infect other oysters directly. The epizootic broadly fitted a simple deterministic epizootic model and suggested that both diffusion and turbulent processes were important in transmission of infection. *Bonamia exitiosa* was also present in oysters at the end of an epizootic in 1964 and was probably the cause of that epizootic. Bonamiasis appears to be an endemic disease in Foveaux Strait. The high mortality in the 1986–1992 epizootic was like that caused by a newly introduced disease in an immunologically naïve population. We propose that other stressors have increased the susceptibility of oysters to this disease. Mechanical disturbance of oysters by increasingly intense dredging appears to be a major source of stress, as does the increasing scale of modification of benthic habitat by fishing. Recovery of the oyster population after the epizootic is closely linked to regeneration of habitat. The prognosis for the fishery could be improved by mitigating mechanical disturbance during dredging by use of lighter dredges and less damaging towing strategies, as well as pursuing rotational fishing strategies that allow benthic habitat to regenerate in undisturbed areas.

Keywords: benthic habitat, *Bonamia exitiosa*, effects of fishing, epizootic, Haplosporidia, *Ostrea chilensis*, oysters.

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Introduction

Dead or dying animals are not readily observed in the sea, so relatively few epizootics have been recorded in the marine environment. The only epizootics reported to date among marine invertebrates occurred in shallow water environments or in high profile fisheries in which dead or dying organisms were highly visible. The most extensive epizootic documented in marine invertebrates was the catastrophic mortality of the sea urchin *Diadema antillarum*, when a water-borne pathogen spread rapidly through the entire Caribbean (an area of 3.5×10^6 km^2) between January 1983 and January 1984 (Lessios *et al.*, 1984). The onset of disease was rapid, sea urchins dying within 4 days of infection, and mortality of local populations was almost 100% within 10 days (Hughes *et al.*, 1985). An equally catastrophic epizootic almost destroyed a major fishery for the Atlantic calico scallop (*Argopecten gibbus*) off Florida in 1989 (Moyer *et al.*, 1993). The acetosporan disease spread rapidly through the scallop population in the entire fishery area of 6.5×10^4 km^2 within 6 weeks. The scallop population began to rebuild, but was again reduced to very low density by a recurrence of disease in 1990 (Moyer *et al.*, 1993). Although these were widespread epizootics affecting entire populations, high mortalities have also been reported in localized areas in wild

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shellfisheries (e.g. scallops, Gulka *et al.*, 1983). In addition, epizootics have devastated stocks of cultivated oysters but in these events the transhipment of infected oyster stocks has greatly accelerated the spread of disease (Grizel *et al.*, 1986), so the rate of natural spread cannot be disentangled from the effects of anthropogenic activities.

The greatest number of epizootics is recorded in the terrestrial environment where their mortality is highly visible. Many epizootics are caused by diseases spread by either human activities (Anderson and May, 1991) or terrestrial animals (e.g. rabies in foxes, Murray *et al.*, 1986; Murray and Seaward, 1992). Terrestrial epidemiologists have developed a generalized epizootic model for directly transmitted diseases in which the rate of spread of infection and mortality and the intensity of mortality is predicted from the relationship between host-density, threshold of infection, incubation time, and the rate of spread of infective particles. The simple deterministic model predicts that as the disease spreads, the density distribution of non-infected hosts maintains an S-shaped profile that will become steeper the slower the disease spreads (Bailey, 1975). The density of infected hosts and the prevalence of infection have bell-shaped profiles with prevalence of infection peaking after most of the hosts have died (Figure 1). The profiles of these fronts are solutions to differential wave equations, so they have been termed travelling waves (Mollison, 1991). The model predicts that these waves travel at a constant speed and maintain the same profile throughout the epizootic (Mollison, 1991) (Figure 1). Murray *et al.* (2001, 2003) modelled the wave of infection of a herpesvirus epizootic, which caused massive mortalities in pilchard populations along 5000 km of the shelf of southern Australia in 1995 and 1998/1999, which demonstrated such fronts.

In the late 1980s an epizootic affected a population of the dredge oyster, *Ostrea chilensis*, in Foveaux Strait, southern New Zealand. Foveaux Strait separates Stewart Island and South Island, New Zealand (Figure 2), and has supported an important dredge fishery for oysters for more than 100 years (Cranfield, 1979; Cranfield *et al.*, 1999). The first signs of the disease were seen by fishers who found heavy mortality of oysters in localized areas in the far west of Foveaux Strait in 1985, and further east in central western Foveaux Strait in 1986 (Cranfield *et al.*, 1991). Histological examination of recruit-sized oysters (oysters greater than or equal to 58 mm in length) from this area revealed that infection by a haplosporidian microcell parasite *Bonamia* sp. (later named *Bonamia exitiosus* (Hine *et al.*, 2001), and renamed *B. exitiosa* (Berthe and Hine, 2003)) was the cause of the mortality (Dinamani *et al.*, 1987a, 1987b). The epizootic spread through Foveaux Strait between 1986 and 1992 and devastated the entire oyster population (Doonan *et al.*, 1994). Unlike the other major marine epizootics previously mentioned, *Bonamia* spread slowly enough to give us opportunity to investigate its effect on both individuals and the entire host population.

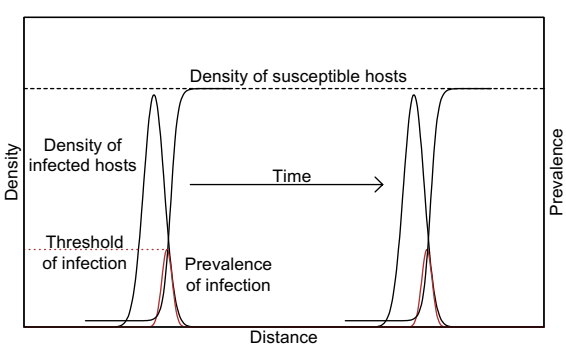

Figure 1. Hypothetical epizootic travelling in one dimension in which the critical threshold of infection is about one-third of the original density of susceptible hosts. The susceptible hosts have an "S" shaped front that moves in the direction of the arrow. The peak in the number of infected hosts trails just behind the susceptible host's front, and the peak of prevalence of infection is further back again.

Investigations during the epizootic established the life history of the parasite and the aetiology of the disease. The parasite spreads through infective particles released into the water from the gonads, kidneys, gills, and gut of diseased or dying oysters and from the breakdown of dead oysters in autumn (Hine, 1991a, 1991b). The critical threshold density of recruited oysters to trigger an epizootic was estimated to be ~ 1.26 oysters m^{-2} (Doonan *et al.*, 1999). Infected, dying recruit-sized oysters contain $\sim 5 \times 10^8$ infective particles (B. K. Diggles, unpublished results), and released particles are ingested by nearby oysters and enter the blood system from the gut (Hine and Jones, 1994). Half the released infective particles survive 48 h in seawater at 18°C (normal summer temperatures in Foveaux Strait) (Diggles and Hine, 2002). The 18-week 50% lethal dose of infective particles in recruited oysters was experimentally determined to be $\sim 1.1 \times 10^5$ (Diggles and Hine, 2002), 40% greater than the 50% lethal dose of *Bonamia ostreae* in *Ostrea edulis* (see Hervio *et al.*, 1995). Infective particles are phagocytosed by agranular haemocytes, but are able to resist lysis within the haemocyte (Hine, 1996). The parasite proliferates within the haemocytes, especially those invading the gonad and resorbing developing ovarian material in the late austral summer and autumn (Hine, 1991a; Hine and Jones, 1994; Hine, 1996), after the protandric oysters have spawned (Jeffs and Hickman, 2000). As infection intensifies, many parasites lyse their host haemocytes, and become extracellular, causing hyperplasia of the epithelia of mantle, kidneys, and to a lesser extent, the gills of the oyster. Finally, as basal membranes of moribund oysters break down, surface lesions develop and the oyster is unable to maintain turgor, cannot extend the gills and feed, and subsequently dies (Hine, 1996).

The prevalence of infection of oysters by *B. exitiosa* as well as the distribution of recently dead oysters and the numbers and distribution of living oysters in Foveaux Strait

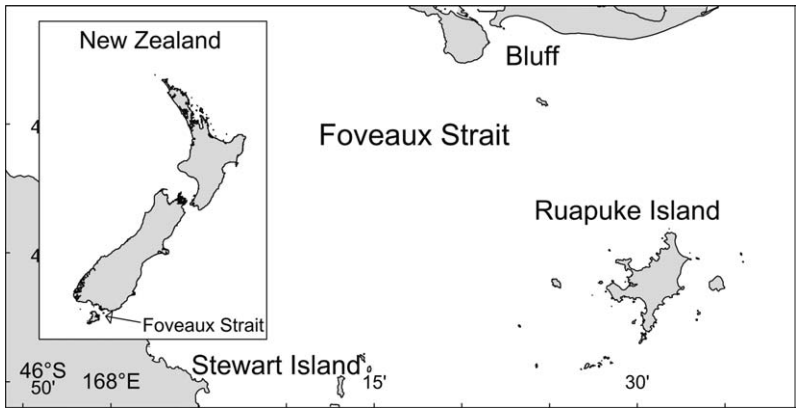

Figure 2. Location of Foveaux Strait within New Zealand.

was investigated between 1986 and 1995. We analyse these data and describe the rate of spread of disease in Foveaux Strait, the magnitude and distribution of mortality, and how it has affected oyster distribution. We compare the spread of this *Bonamia* epizootic with other marine epizootics and investigate how it fits the simple deterministic epizootic model. We describe the short-term recovery of the oyster population (prior to an even more recent epizootic), relate it to recovery from past mortality events, and discuss the implications for the fishery.

Area and methods

Hydrodynamic environment

Foveaux Strait separates South Island and Stewart Island, New Zealand (Figure 2). It is about 80 km long, 23−54 km wide, and depth shelves from 50 m in the west to 20 m in the east (Cullen, 1967). It is swept by tidal flows that may reach 120 cm $s^{-1}$and set to the east on the rising tide and to the west on the falling tide. Residual inflow of water into Foveaux Strait is 5.8 km d^{-1} to the northwest in the southeastern entrance and 5.8 km d^{-1} to the east in the western entrance. The residual outflow to the east in the northeastern entrance is 12.3 km d^{-1}, and results in water having a residence time of between 5 and 6 days in Foveaux Strait (Houtman, 1966). The strong linear flow develops helical circulations that give rise to the linearity of sedimentary bed forms and regenerating habitat on the seafloor of Foveaux Strait (Cranfield *et al.*, 2003). These hydrodynamic and topographic features will in turn affect the distribution and dispersion of plankton (e.g. Cranfield and Michael, 1989) and may affect infective particles released by diseased oysters.

Estimation of prevalence of infection by *Bonamia*

The prevalence of infection by *B. exitiosa* in Foveaux Strait was determined in samples of 50 recruited oysters dredged at stations selected close to those originally sampled as infection spread from the epicentre of infection, as well as from areas towards which, infection appeared to be spreading. Between 1986 and 1989, infection levels of *B. exitiosa* were determined from examination of histological sections (stained with haematoxylin and eosin). From 1990 onwards, infection levels were determined from examination of ∼20 stained (Merck Hemacolor™ system) imprints of oyster hearts, after this quicker method had been validated and calibrated against histological sections (Cranfield *et al.*, 1995). This method has since been validated against even more sensitive molecular methods of *in situ* hybridization (ISH) and PCR amplification of parasite DNA for diagnosing infection that showed that prevalence estimated from heart imprints of 25 oysters can underestimate the true infection rate by 10−20%, especially at low levels of infection (Diggles *et al.*, 2003). Infection was determined from examination of all the haemocytes in the field of view or in one heart imprint. If no *B. exitiosa* was found, all the imprints were examined. A total of 260 stations were sampled and 10 800 slides examined. The number of stations sampled in each survey is shown in Table 1. The percentage of the 50 oysters in the sample that were infected, is the prevalence figure recorded.

Estimation of distribution of density of living oysters, distribution of mortality and infection

The estimates of population density surfaces were made using kriging (Ripley, 1981; Cressie, 1991). Surfaces were fitted without trend, with an exponential covariance function $C(r) = \sigma^2 e^{-r/d}$ and distance parameter typically $d = 0.7$ nautical mile (determined from the visual inspection of semivariogram diagnostic plots). Models were fitted using log-transformed density estimates, and maps produced by back-transforming the kriged estimates. We assume no nugget effect, and hence employ the method as an exact interpolator, although the log-transformation introduces a bias into interpolated estimates. The hinge of

Table 1. Numbers of stations sampled by dredging between 1986 and 1995 and used in kriging prevalence of infection and distribution of living oysters, new clocks and all clocks in Figure 3.

Year	Prevalence stations	Oyster stations	Dredge used*	Area sampled (km^2)
1986/1987	47	94	Survey	~550
1990	71	293	Commercial	1 116
1992	45	255	Commercial	1 229
1993	No sampling	169	Commercial	875
1995	53	144	Commercial	680

*Survey dredge is a 1.25 m wide replica of a commercial dredge; commercial dredge is 3.35 m wide (both dredges are described in Doonan *et al.*, 1994).

oysters that have died from disease remains intact for up to three years (Cranfield *et al.*, 1991), so the density of valves that are still articulated, termed "clocks", indicates oyster mortality over that period. Clocks whose internal valve surfaces were clean and unfouled are from oysters that have died since the last period of fouling (the previous summer). They are termed "new clocks". Fouled clocks are termed "old clocks". Surface density estimates have been presented either as units of oysters m^{-2} computed from the number of oysters found (N), or as percentages from the relative density of new and old clocks or the estimated prevalence of infection by *B. exitiosa*, and used the estimated length of the tow (d), an assumed dredge efficiency of $q = 0.1656$ (Doonan *et al.*, 1994), and dredge width of W (either 1.25 m for surveys of 1986 and 1987, or 3.35 m for surveys 1990−1995), to calculate the absolute density as $N/(qWd)$.

Epizootic model

Although these kriged data of the two-dimensional spread of infection are informative, the number of stations sampling infection was not great enough to delineate clearly the waves of infection or mortality. These wave fronts are readily discernible and analysed in one-dimensional distributions. We therefore analyse density of living oysters, prevalence of infection, percentage new mortality and total mortality along the central axis of Foveaux Strait throughout the epizootic from the kriged data to estimate the rate of spread of infection and compare these data with the simple deterministic epizootic model (see Shigesada and Kawasaki, 1997).

Results

The prevalence of infection by *B. exitiosa* in 1986−1987 (Figure 3a) clearly defined the focus of infection from which the epizootic spread. Infection radiated out from this focus through the oyster population killing most of the oysters that were present within the epicentre of infection by 1990 (Figure 3b, e, f). The high numbers of clocks within the epicentre records the massive mortality of oysters that had occurred by 1990 (Figure 3k). By 1990, moderately dense populations of living oysters were confined to eastern Foveaux Strait and the northern, southern, and western boundaries of oyster distribution in central Foveaux Strait (Figure 3f), and the wave of infection and subsequent mortality had even reached the outer margins of these populations (Figure 3b, f). The wave of infection passed through these peripheral oyster populations and probably reached the boundary of oyster distribution in 1991. By 1992, patches of isolated infection remained among populations of oysters solely around the outer edge of Foveaux Strait (Figure 3c, g). Oyster mortality following infection of these outermost, less-dense oyster populations, was not as great as it had been at the epicentre of infection (compare Figure 3e, g).

The prevalence of infection was high (generally greater than 40%) within the wave of infection, and disease generally killed more than 80% of the oysters as the wave of infection passed through an oyster bed over a period of two to three years (compare Figure 3e, f, g and j, k, l). Ultimately more than 91% of the total oyster population was destroyed (Doonan *et al.*, 1994). After the passage of the epizootic, infection by *B. exitiosa* was difficult to detect among the surviving oysters using heart imprint techniques (see Figure 3d), and often all imprints of every oyster in the sample were examined before one or two haemocytes infected by *B. exitiosa* were found (Cranfield *et al.*, 1995). As predicted, the percentage of all clocks decreased over the three years following the peak of infection and mortality (Figure 3j−n).

By 1986−1987, mortality from *B. exitiosa* had reduced oyster density considerably around the epicentre of infection. By 1990, oyster density had been greatly reduced in a much larger area around this, as well as in oyster populations that occurred beyond it. By 1992, oyster density had been greatly reduced even in the peripheral oyster populations. The changes in numbers of living oysters, percentage of infection, new mortality, and all clocks along the east/west axis of Foveaux Strait between 1986−1987 and 1995 (Figure 4a−e) shows the wave of infection and subsequent mortality spreading to oyster populations both to the east and to the west. Infection was followed by mortality, then the accumulation of clocks and the gradual disappearance of clocks over the three years it took for the shell ligament to break down. These data allow us to measure approximate rates of spread of the disease along the axis. The wave of infection and the wave of new mortality progressed 15.4 km to the west between 1987 and 1990 (Figure 4a, b) suggesting infection spread at 5 km $year^{-1}$. Spread to the east may have been slower at 12.4 km over the same period, suggesting infection spread at 4 km $year^{-1}$ in that direction.

The fishery was closed between 1993 and 1996 (Cranfield *et al.*, 1993, 1996) to allow the oyster population

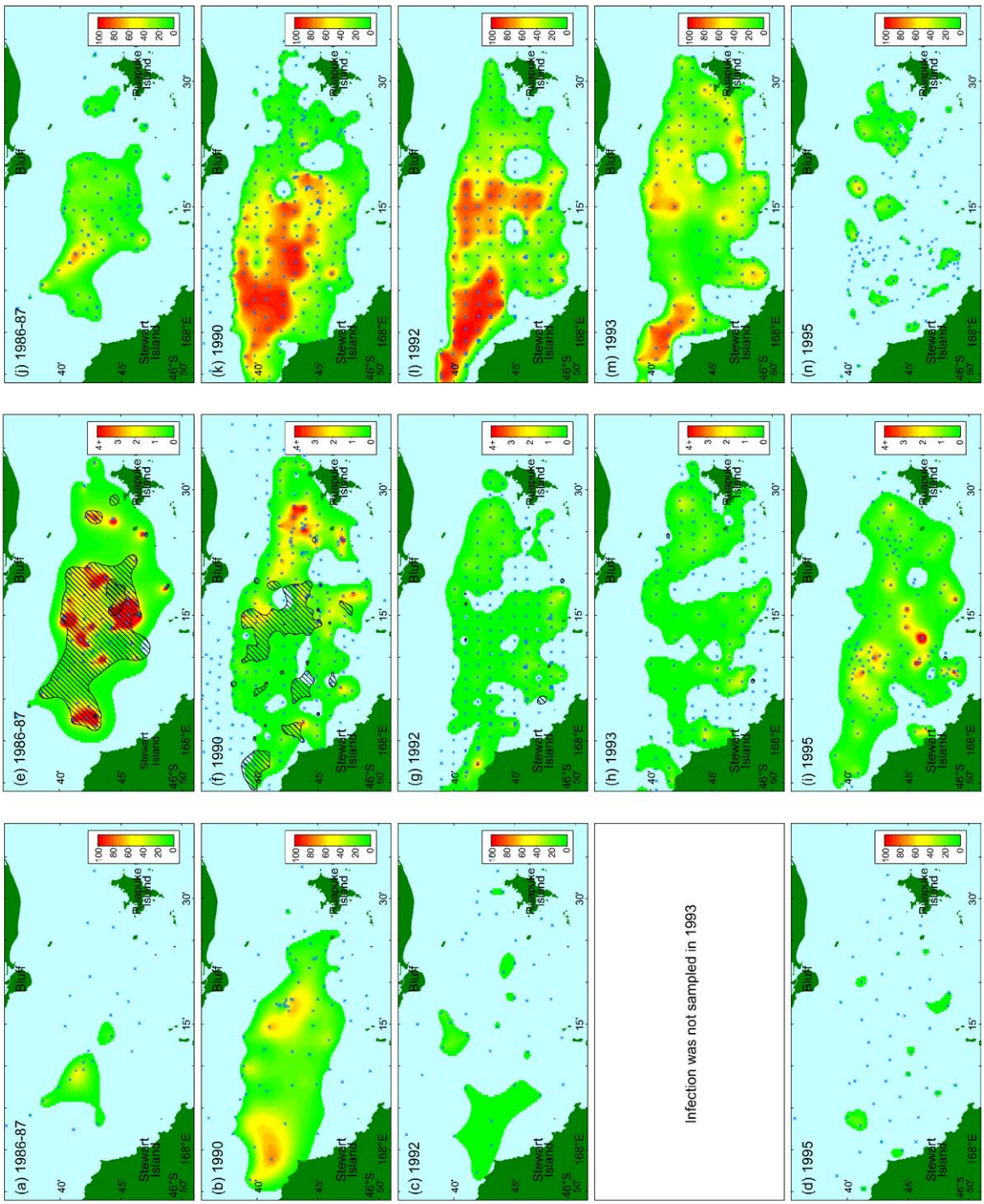

Figure 3. Kriged two-dimensional plots of prevalence of infection, density of living oysters and dead oysters estimated from sampling in Foveaux Strait between 1986 and 1995. The number of stations that were sampled each year is shown in Table 1 and their positions marked on Figure 3 by blue crosses. Figure 3a–d, distribution of prevalence of infection by *B. exitiosa* in Foveaux Strait 1986–1995 (prevalence of infection was not sampled in 1993). Figure 3e–i density of live oysters (oysters m^{-2}) between 1986 and 1995, hatched area shows extent of new mortality (5% new clocks) estimated from ratio of new clocks to sum of all clocks and live oysters. Figure 3j–n percentage of all clocks (ratio of clocks to sum of clocks and living oysters). Figure 3 shows how infection spread through Foveaux Strait between 1986 and 1992, shows how new mortality followed infection and how disease reduced the distribution of the oyster population up to 1992, how and where oyster density began rebuilding in 1993 and 1995, and how the distribution of clocks (new and old clocks) recorded the distribution of oyster mortality from bonamiasis.

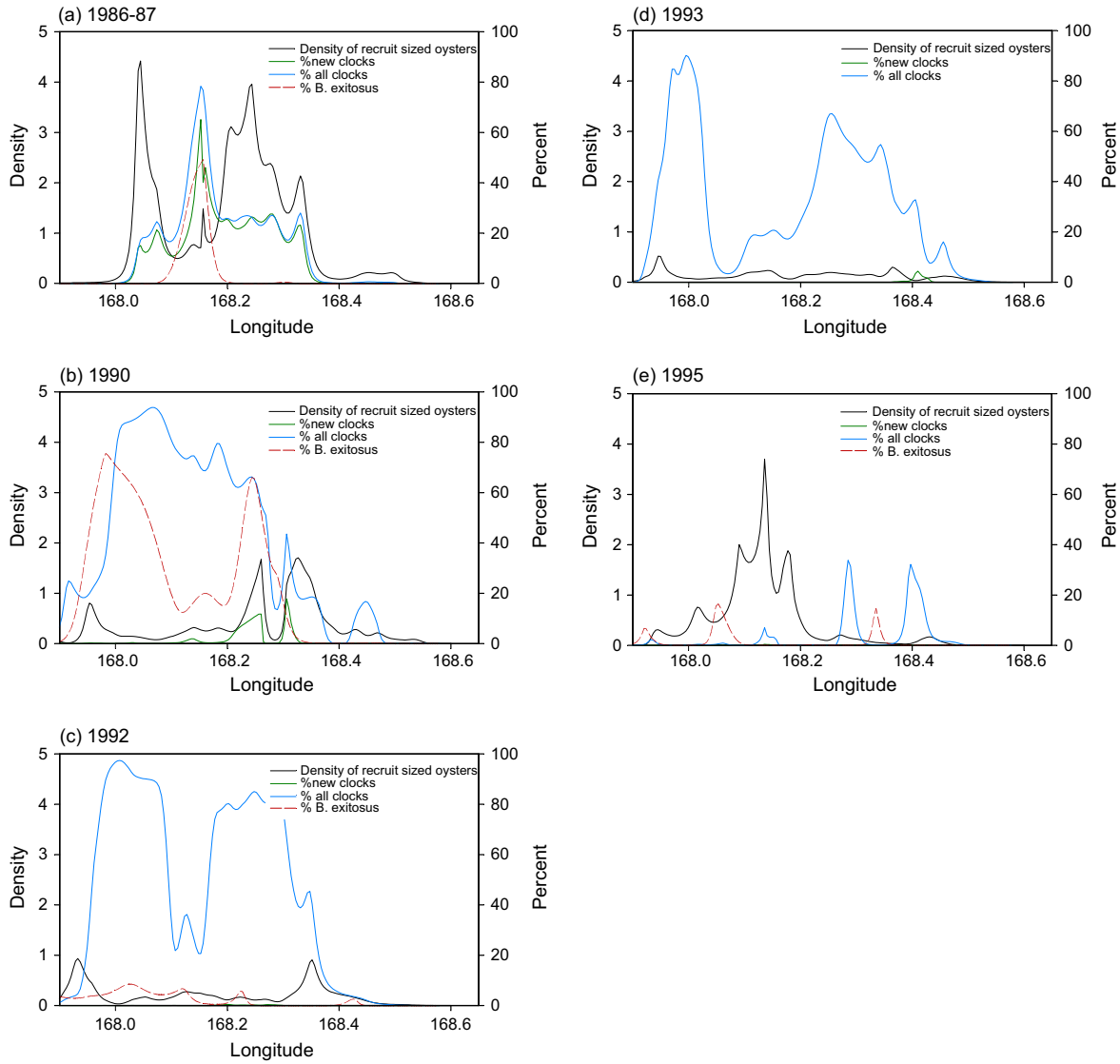

Figure 4. One-dimensional plot of the spread of the epizootic along the central axis of Foveaux Strait, showing the fronts of infection and mortality, moving through the susceptible oyster population.

to rebuild in the absence of dredging. Oyster density began rebuilding in southern and southwestern areas in 1993 (Figure 3h) and more widely in 1995, even including the epicentre of infection in which oysters had been almost completely removed by disease during the epizootic (Figures 3i and 4e).

Discussion

Effects of disease on distribution of oysters in Foveaux Strait

This epizootic commenced at a clear focal point (approximately 46°41.5′S 168°9′E), with infection spreading radially. It destroyed oyster populations as it progressed through Foveaux Strait, and it ended only when infection had spread to the limits of oyster distribution. The surveys clearly show direct changes in distribution of oysters attributable to mortality, even showing some areas that the epizootic had reduced oysters below densities that were detectable (e.g. at the focus of infection, compare Figure 3e and h). They also show changes in distribution of oysters, as areas left behind the wave of infection had negligible living oysters and were carpeted almost entirely by clocks after the epizootic had passed (Figure 3k, l, and m). Changes in the density of oysters were also reflected in the reduction of catch per unit effort (cpue, measured in sacks

per hour) in the oyster fishery (Cranfield *et al.*, 1991). During the epizootic, fishers tended to maintain catch rates by fishing uninfected populations ahead of the wave of mortality, and exploited oyster populations progressively closer to the periphery of oyster distribution as mortality spread. In spite of these strategies, mean cpue of the fishery declined from 13.8 sacks per hour to 3.4 sacks per hour, and total catch fell from 115 000 sacks to 5821 sacks between 1984 and 1992, reflecting the scale of disease mortality (Cranfield *et al.*, 1991, 1993).

Catch per unit effort data recorded in the oyster fishery since 1948 show similar changes in cpue and catch in the past that also probably reflect disease mortality. Mean cpue declined from 10.5 sacks per hour to 5.8 sacks per hour, and total catch decreased from 123 000 sacks to 76 000 sacks between 1960 and 1963 (Stead, 1971a). Mean cpue declined from 16 sacks per hour to 13.5 sacks per hour, and total catch decreased from 95 000 sacks to 75 000 sacks between 1949 and 1950 (Stead, 1971a). These changes are similar in their onset but smaller in size than the changes seen during the 1986–1992 epizootic. The decline in 1960–1963 reflects the reduction in oyster density by oyster mortality that may have been locally as high as 50% in eastern Foveaux Strait (Stead, 1971b). At that time, the mortality was attributed by Howell (1967) to infestation of oysters by the sporocyst stage of the trematode, *Bucephalus longicornutus*. Infection reached a mean prevalence of 20% in 1963 and 14% in 1964, and thereafter became very low or undetectable (Howell, 1967). *Bonamia exitiosa* has since been found in histological preparations of oysters from 1964 (Hine and Jones, 1994). The parasite was abundant in preparations of the ten oysters examined, so prevalence and intensity of infection (see Cranfield *et al.* (1995); Diggles *et al.* (2003) for definitions) were high in 1964 (P.M. Hine, pers. comm.). Hine (1996) concluded that the mortality between 1960 and 1963 had stemmed from an undiagnosed outbreak of infection by *B. exitiosa*. The effects of this mortality are shown by changes in the distribution of oysters in eastern Foveaux Strait between 1962 and 1975, with the almost complete disappearance of oysters from the Ruapuke oyster bed and reduction of density in others (Cranfield *et al.*, 1999). Oysters had been reduced below commercial densities on Ruapuke bed by 1960, and it was not dredged thereafter. In the absence of fishing mortality, disease mortality in the 1960–1963 epizootic is the most likely cause of demise of the oyster population of this bed between the surveys of 1962 and 1975 (Cranfield *et al.*, 1999).

The catch rate and total catch of the oyster fishery also declined between 1949 and 1950 (Stead, 1971b) coinciding with the collapse of the fishery on the East Bed (see Cranfield *et al.*, 1999). Fishers then reported that the collapse followed oysters on this bed dying during 1948 and 1949 (although fishing mortality appeared to have also contributed to the collapse), resulting in massive accumulations of clocks like those found in the 1986–1992 epizootic (e.g. Figure 3k, l). The earliest survey of oyster beds (in 1905) mapped oyster beds (Hunter, 1906); however, the map is not precise or detailed enough to estimate subsequent changes in oyster distribution that could be related to epizootics. The survey, however, does record the abundance of clocks and their relationship to fishing: "Large quantities of dead shells are to be found on most of the beds, more so on those that have been worked continuously where the current does not run so strong." Hunter (1906) recorded that dead shells were abundant on four specific oyster beds. Such localized mortality in recent times has all been caused by bonamiasis and it is not unreasonable to attribute the mortality of all these earlier events (especially the 1960–1964 event in which *B. exitiosa* was directly implicated) to bonamiasis.

These results suggest an escalating scale of area of mortality from small localized patches of oysters in the early 1900s, to entire oyster beds by the 1950s and 1960s, and the entire fishery by the late 1980s, as well as to an increasing frequency of occurrence over this period.

How does the epizootic fit the generalized epizootic model?

The one-dimensional progression of the epizootic along the central axis of Foveaux Strait (Figure 4a–e) suggests that it broadly fits the epizootic model. The front of infected oysters has an "S" shape, and the peak in the prevalence comes after most oysters had died as the model predicts. The slow spread of the disease (4–5 km year^{-1}) has resulted in the steep profile of the disease front. The epizootic model predicted the infection front will move at constant speed and maintain the same profile throughout the epizootic as found in the pilchard herpesvirus epidemic in Australia (Murray *et al.*, 2001, 2003). The strong residual flow to the east and the discontinuous distribution of oysters along the central axis of Foveaux Strait may have contributed to the difference in speed of travel of fronts to the east and to the west, as well as apparent differences between years. On the other hand, similar differences in the speed between easterly and westerly direction of infection fronts of herpesvirus epidemics in Australian pilchard as well as differences in speed of fronts between years, were not related to the easterly current movement or to differences in population density of pilchard. The differences were explicable solely by changes in the diffusion coefficient (Murray *et al.*, 2003). Pilchard swimming behaviour and mixing rates of schools differed between western and eastern Australia, and the much wider foraging range of fish in the poor feeding conditions of 1995 compared with 1998/1999, resulted in different diffusion coefficients (Murray *et al.*, 2003). In Foveaux Strait, movement of water both east and west in one tidal cycle is greater than the spread of *Bonamia* infection over an entire year, so movement of disease particles in the current-swept water column is unlikely to have contributed to the

diffusion coefficient. The diffusion coefficient of *Bonamia* in Foveaux Strait will probably depend on hydrodynamic conditions adjacent to the seafloor. These conditions will be greatly modified by benthic habitat. As benthic habitat occupied by oysters is more common and denser in western Foveaux Strait than eastern Foveaux Strait (Cranfield *et al.*, 1999, 2003), the diffusion coefficient could be higher in the east than in the west and account for the different rate of spread in these directions.

Fronts in epizootics are well defined where diffusion processes are important in the transmission of disease. The shape of the curve of the likelihood of the transmission of disease over the distance between infected and uninfected hosts (the contact distribution) then tends to follow a normal distribution (Mollison, 1991; Shigesada and Kawasaki, 1997). Oysters prior to the outbreak were distributed through Foveaux Strait in a series of small dense patches that were frequently separated by 500 m to several kilometres of seafloor barren of oysters (Allen, 1979; Allen and Cranfield, 1979), and diffusion processes close to the seafloor are likely to have been important in the spread of disease over <1 m scale of distance separating oysters within dense patches.

The patchy distribution of infection ahead of the main infection front as well as the patchiness of new mortality along the eastern axis in 1987 and 1990 (Figure 4a, b), shows that the advancing front of infection was not always clearly defined. Infection could move ahead of the front, leaving some uninfected populations of oysters behind it. These populations had not been infected initially; nevertheless they appeared to have been mostly destroyed by the disease by the time the epizootic had run its course (Figures 4c, 4d, 3g). Where turbulence and other processes are important in the spread of a disease, the tail of the contact distribution exceeds the exponential distribution and approaches the chi-square distribution. The contact distribution of airborne diseases frequently has this form (Mollison, 1991), and during the spread of an epizootic with this type of contact distribution, infection will tend to "jump" past the front to create patches of infection ahead of it (Shigesada and Kawasaki, 1997). Within the strong tidal flow of Foveaux Strait, turbulent processes could have been important in the spread of the epizootic over the distances separating dense patches of oysters (probably in the range of 500 m to 5 km). The pattern of spread of infection found here suggests that turbulence as well diffusion close to the seafloor has been important in distributing infective particles, and that the contact distribution is more extreme than the exponential distribution.

The epizootic model predicts that disease mortality will be higher and spread of disease will be faster when the density of the oysters is high, although the modelling of Murray *et al.* (2003) predicts this effect will be much less than the effect of changes in the diffusion coefficient. Estimates of the size of the oyster population and oyster density have only been available since 1962, with the first precise dredge survey of Foveaux Strait. Since then, however, surveys have shown population size, area of the fishery, and density of oysters has decreased (Cranfield *et al.*, 1999; Annala *et al.*, 2002; authors' unpublished results), probably continuing a process of serial depletion that started when the fishery commenced (Cranfield *et al.*, 1999). The escalating mortality, greater spread, and increasing frequency of the epizootics between 1960 and the present, when oyster density has been greatly reduced, therefore suggest that other stressors have increased the susceptibility of oysters to disease, so epizootics were triggered at lower oyster densities. If the earlier epizootics were real events, then these stressors were less apparent in the early days of the fishery, as these epizootics occurred amongst populations of oysters with much higher densities, yet they remained very small-scale events that were highly localized.

Speed of epizootic compared with other marine epizootics

The slow spread of bonamiasis in Foveaux Strait contrasts with the spread of other marine epizootics. The herpesvirus infection of Australian pilchard in 1995 spread eastwards at 36 km d^{-1} and westwards at 22 km d^{-1}, and a further outbreak in 1998/1999 spread eastwards at 20 km d^{-1} and westwards at 10.7 km d^{-1}, the rapid rate being explicable by fish movements (Murray *et al.*, 2003). Among sedentary marine invertebrates, the spread of directly transmitted, diseases is dependent on transport by water and the rate of spread is much slower. Thus, estimates of the local spread of the epizootic among *Diadema antillarum* in the Caribbean ranged from $\sim$7 km $week^{-1}$ (Bak *et al.*, 1984) to $\sim$25 km $week^{-1}$ (Hughes *et al.*, 1985), and the speed of spread through the entire Caribbean was $\sim$53 km $week^{-1}$ (from data in Lessios *et al.*, 1984). The speed of spread of the epizootic among *Argopecten gibbus* ($\sim$36 km $week^{-1}$; estimated from data in Moyer *et al.*, 1993), is in the same range as the diademnid epizootic, but both diseases spread at a rate that was nearly two orders of magnitude faster than bonamiasis in Foveaux Strait. Although bonamiasis caused by *Bonamia ostreae* appears to have spread very rapidly through populations of *Ostrea edulis* in Europe, this pathogen was introduced in infected oysters (*O. edulis*) re-imported from the west coast of USA (Elston *et al.*, 1986), and was then spread widely in further transhipments of infected oysters (Balouet *et al.*, 1983; van Banning, 1991; Hudson and Hill, 1991; McArdle *et al.*, 1991) rather than by natural dispersion of infective particles over any significant distance. The slow spread of the *Bonamia* epizootic in Foveaux Strait probably reflects the long-standing relatively stable host/parasite relationship between *O. chilensis* and *B. exitiosa* (Hine, 1996). This has resulted in oysters being able to sustain higher doses before the lethal level is reached, compared with the host/parasite relationships with new or introduced pathogens (e.g. *B. ostreae*

and *O. edulis* and *Diadema* and bacteria). Notably, the 50% lethal dose of *B. exitiosa* particles in *O. chilensis* was 40% greater than *B. ostreae* particles in *O. edulis*.

Recovery of the oyster population

Oyster density appeared to have started rebuilding in southern areas of Foveaux Strait in 1993 (Figure 3h) and continued to do so in 1995 (Figure 3i). By 1995, oyster density had also rebuilt at the original focus of infection and other areas on which the epizootic had killed all oysters (compare Figure 3g, h, and i). Benthic habitat was either regenerating on the seafloor areas on which oyster density rebuilt, or these areas were close to areas that could provide propagules that could expedite regeneration (Cranfield *et al.*, 2004). Regeneration of benthic habitat was closely linked to the rebuilding of oyster density (Cranfield *et al.*, 2001, 2004).

Resistance of oysters to infection

Hine (1996) considered that the annual pattern of infection of *Ostrea chilensis* by *Bonamia exitiosa*, and the close relationship between the life history of *B. exitiosa* and the seasonal cycle in the physiology of *Ostrea chilensis*, were indicative of the long-term relationship between host and pathogen (unlike that of the introduced pathogen *Bonamia ostreae* and *Ostrea edulis* in Europe). Furthermore, *B. exitiosa* was present in oysters in 1964, as far back as historical samples of tissues let us investigate, and was probably the cause of earlier epizootics as well. This evidence points to *B. exitiosa* being endemic in Foveaux Strait oysters. If this is true, the oyster population of Foveaux Strait would be well adjusted to co-exist with the disease agent. Nevertheless, the mortality in the 1986−1992 epizootic was as high as if the disease agent had infected an immunologically naïve population. Hine (1996) considered that effects of fishing on benthic habitat might have stressed oysters, substantially lowering their resistance to the disease. Over the period of these escalating epizootics (1906−1992), dredging for oysters has progressively modified the benthic habitat of Foveaux Strait, suggesting that there may be some interaction between disease outbreaks and the benthic habitat (Cranfield *et al.*, 1999). The 1986−1992 epizootic commenced in oyster populations that had been fished intensively for years (so benthic habitat was likely to have been highly modified) and ceased in populations around the margin of oyster distribution that had been little fished up to that time and where bycatch indicated that benthic habitat was little modified (Cranfield *et al.*, 1999). Similar escalating disease mortality in oysters in Chesapeake Bay has been attributed to modification of oyster habitat by fishing (Rothschild *et al.*, 1994), and the environmental stress caused by this modification has been directly implicated in increasing acetosporan disease levels of oysters in experiments (Lenihan *et al.*, 1999).

Dredging itself is a stressor of *Ostrea edulis* infected by *Bonamia ostreae*, van Banning (1991) having found dredging of one of two comparably infected oyster beds in the Netherlands resulted in a much higher level of infection than on the undredged bed. Dredging for *O. chilensis* in Foveaux Strait not only modifies benthic habitat, but also subjects oysters to a high level of mechanical disturbance that could make them equally vulnerable to infection by *B. exitiosa*. Each oyster vessel tows two 530 kg double-bit double ring bag dredges in a circular path (to stay on the same position), remaining fishing the same position until the catch rate is no longer commercially acceptable. Over recent years the value of oysters has risen, so very low densities of oysters are commercially acceptable now and fishing pressure has intensified accordingly. We have no precise data on actual fishing positions of individual vessels, but Cranfield *et al.*, (1999) estimated that in 1998, fishers could have dredged the same seafloor 12−15 times a season; fishing could be twice as intense in 2003. Commercial oyster dredges capture only one-fifth of the recruited oysters on the seafloor (Doonan *et al.*, 1994), suggesting that the remaining four-fifths are overridden or swept aside by the dredge (and presumably disturbed). Undersized oysters are sorted by hand on the deck of the vessel and quickly returned to the sea, so a large proportion of oysters of all sizes are subjected to disturbance each season. Hine *et al.* (2002) tested handling stress that was probably similar to that encountered in dredging by stirring oysters in tanks 4 times a day for 14 days in a laboratory experiment. They found significantly elevated levels of *B. exitiosa* infection and subsequent mortality among oysters treated this way.

Implications for the fishery

Good recruitment of oysters has contributed to the rapid recovery of the fishery in some areas after epizootics. Benthic habitat has also been found to regenerate rapidly in some areas when fishing ceased and management strategies such as rotational fishing and habitat enhancement could accelerate the recovery (Cranfield *et al.*, 2001; Cranfield and Michael, 2002) and mitigate outbreaks of disease. If the mechanical disturbance of dredging stresses oysters and predisposes them to infection by *B. exitiosa*, development of lighter dredges and less damaging fishing strategies should reduce the chance of disease outbreaks by lowering disturbance. Lighter dredges would also reduce the modification of habitat, probably further reduce the chance of disease outbreaks, and accelerate recovery of the oyster population by enhancing recruitment. The oyster fishery could have a better chance of recovery and a disease-free future if such strategies were to be developed. The oyster industry could establish the benefit of such strategies by testing them experimentally on the scale of the fishery (Cranfield *et al.*, 2001; Cranfield and Michael, 2002).

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