Journal of Shellfish Research, Vol. 17, No. 3, 805–811, 1998.

SHELL LESIONS IN NEW ZEALAND *HALIOTIS* SPP. (MOLLUSCA, GASTROPODA)

ROSEANNE M. GRINDLEY,[1] JONATHAN A. KEOGH,[1] AND CAROLYN S. FRIEDMAN[2]

[1]*Department of Marine Science*
PO Box 56
Dunedin, New Zealand
[2]*Fish Health Laboratory*
California Department of Fish and Game
c/o Bodega Marine Laboratory
PO Box 247
Bodega Bay, California 94923

ABSTRACT Shell lesions are reported in the three New Zealand species of *Haliotis* (*Haliotis iris* Martyn, 1784, *Haliotis australis* Gmelin, 1791, and *Haliotis virginea virginea* Gmelin, 1791). The lesions are described as blisters of conchiolin, and occasionally nacreous material, forming on the inside of the abalone shell near the apex. Twelve (16%) of 76 *H. iris*, 21 (38%) of 56 *H. australis*, and 5 (100%) of 5 *H. virginea virginea*, had lesions, some of which disrupted the adductor muscle scar. Specific histological stains confirmed the presence of fungal hyphae within lesions and the shell matrix of affected *H. iris* and *H. australis*. No pathological changes or evidence of fungal infection were observed in any of the soft tissues of the lesion-bearing animals examined. Lesions were most prevalent in populations in the Catlins region of South Otago and off the northeast coast of Stewart Island, although they were found throughout the southern New Zealand range of *H. iris*, including offshore islands. The mean shell length of lesion-bearing *H. iris* was significantly ($p < 0.05$) less than that of nonlesion-bearing *H. iris* at 4 of 11 locations sampled.

KEY WORDS: abalone, paua, *H. iris*, *H. australis*, *H. virginea virginea*, New Zealand, shell lesion, fungus

INTRODUCTION

The marine genus *Haliotis* (Mollusca: Gastropoda) is distributed through the temperate and tropical regions of the world, including New Zealand, where it is represented by three endemic species: *Haliotis iris* Martyn, 1784, *Haliotis australis* Gmelin, 1791, and *Haliotis virginea virginea* Gmelin, 1791 (Powell, 1979). Commonly called abalone, or paua, the largest and most abundant species, *H. iris*, is the basis of a substantial inshore fishery. Harvested abalone are often exported as a highly prized food, whereas the shells are used for jewelry and in the inlaying of furniture. They are also increasingly important as an aquaculture species, for both meat and pearl production.

Recently, it was observed that some individuals of *H. iris*, in the vicinity of Stewart Island (47°S and 168°E), had lesions on the inside of the shell. The lesions generally appeared as brown, jelly-like material resembling conchiolin. The lesions severely damaged the shell of affected individuals, with a resulting loss of earnings for both fishers and exporters. Furthermore, aquaculturalists reported that this condition could be fatal to animals kept in captivity (D. Langdon pers. comm.). The effects of this condition, and its prevalence and distribution in wild abalone populations, were unknown. A search of English language journals revealed no reports of similar shell lesions among the many other species of *Haliotis* found outside New Zealand. There are several reports of parasites that invade the shells of abalone. Crofts (1929) reported that *Haliotis tuberculata* Linnaeus shells were frequently damaged by boring bivalves such as *Lithodomus* sp. and *Pholadidea* sp. Further reports include a boring barnacle (Batham and Tomlinson 1965), a sponge (Clavier 1992), and parasitic worms (Cox 1962, Oakes and Fields 1994). In New Zealand, Sinclair (1963) and Sainsbury (1977) described various shell conditions in *H. iris* from the Wellington area and from Banks Peninsula and Peraki Bay areas, respectively. Both authors describe shell pathologies that appeared to share some characteristics with those described here.

This study provides a detailed description of the macroscopic and microscopic structure of the shells and soft tissues of affected and unaffected specimens of the three abalone species native to New Zealand. In addition, the geographic distribution and information on the prevalence of these lesions in wild populations of abalone in New Zealand are provided.

MATERIALS AND METHODS

Description of Lesions and Histology

A total of 76 *H. iris* (50.8–151.7 mm shell length), 56 *H. australis* (26.5–86.0 mm shell length), and 5 *H. virginea virginea* (47.2–63.8 mm shell length) were collected by divers (using SCUBA) from shallow subtidal populations at Little River, Stewart Island (46°53′S, 168°06′E), in February 1995 and from Jacks Bay in South Otago (46°30′S, 169°44′E), in December 1995. The shell was detached from the soft tissue of the animal, and the inside was examined for the presence of lesions. The size, location, and description of shell lesions were characterized from these lesion-bearing animals. Selected individuals were sampled for microscopic analysis, as given below. To determine the extent to which the adductor muscle attachment site was affected by the lesion, a random selection of 63 affected *H. iris* shells and 24 affected *H. australis* shells were set aside from the commercial catches sampled as described below.

Square pieces of shell measuring approximately 1 cm^2 were excised from affected and unaffected animals with a small hand-held electric grinder. These samples were placed in Bouins solution (Humason 1979) in seawater for at least 14 days, embedded in Paraplast™, and sectioned transversely at a thickness of 7 μm by the techniques described by Humason (1979). Shell tissue sections were stained with periodic acid Schiff's reaction (PAS), Hotchkiss-McManus method (McManus 1948), and Grocott's adaptation of Gomori's methenamine-silver nitrate method (SS) for fungi

(Grocott 1955). Samples of shell were taken from 11 lesion-bearing and 11 nonlesion-bearing *H. iris* and 10 lesion-bearing and 14 nonlesion-bearing *H. australis.*

In order to examine the effect of lesions on soft tissues, we examined all major tissues including foot, digestive gland, kidney, gill, and mantle from 10 *H. iris* and 10 *H. australis* with and without lesions. For animals with lesions, a sample of tissue adjacent to lesions was also examined. Cross-sections (3–4 μm thick) of selected tissues were placed in Davidson's invertebrate fixative (Shaw and Battle 1957) for 24–48 h and processed for routine paraffin histology (Humason 1979). Deparaffinized sections were stained with Lillie-Mayer haematoxylin and eosin (Lillie 1965) and PAS and viewed by bright field microscopy.

Distribution

Samples of commercially caught *H. iris* (i.e., animals larger than 125 mm shell length) from 21 locations around the South Island and Stewart Island of New Zealand were sampled at a processing factory in Dunedin. One to three groups of 250 abalone were sampled from each geographic location, and the presence or absence of the condition was noted for each animal (7,500 *H. iris* were examined in total). Twelve affected *H. iris* shells were also collected from a commercial abalone processor on the Chatham Islands (44°53′S, 176°30′W). Fifteen specimens of *H. australis* were collected from the Snares Islands (48°00′S, 166°35′E), and five specimens of *H. virginea virginea* were collected from Campbell Island (52°30′S, 169°10′E).

Size

To ascertain if an association exists between the size of commercially caught abalone and the presence of shell lesions, we examined *H. iris* from selected locations throughout the South Island of New Zealand. At least 107 *H. iris* were randomly selected from each of the commercial catches, and the shell lengths of affected and unaffected animals were measured to the nearest 0.1 mm with calipers. Catches in which more than 10% of the measured abalone were affected with shell lesions were selected for analysis, resulting in a total of 1,679 abalone in 11 catches from 9 locations. Single-factor analyses of variance were used to compare the shell lengths of affected and unaffected *H. iris* from each catch.

To assess the size range of animals exhibiting lesions, 56 specimens of *H. iris* and 36 specimens of *H. australis,* ranging above and below the minimum legal size (125 and 80 mm, respectively), were collected from Little River, Stewart Island. The shell lengths were measured to the nearest 0.1 mm, and the shell was dissected from the soft tissues, as before, to examine for lesions. A Kolmogorov-Smirnov two-sample test was used to assess the relationship between animal size and presence of shell lesions.

RESULTS

Description of Lesions

Lesion prevalence in populations of *H. iris* at the Little River and Jacks Bay sites was 54 (n = 56) and 45% (n = 20), respectively, whereas in *H. australis,* the estimates were 47 (n = 36) and 50% (n = 20), respectively (Table 1). All *H. virginea virginea* sampled from Little River (n = 2) and from Jacks Bay (n = 3) were affected. In over 95% of all affected specimens examined, we observed no indication of the presence of lesions on the exterior of the shell. Although *Polydora* tubes were evident on the exterior of the shells, irrespective of the presence of lesions, none penetrated through the innermost layers of the shell. A few *H. iris* did have lesions that were evident externally, and these were characterized by the collapse and destruction of the shell around the apex region (Fig. 1). Usually, a shell lesion appeared as a golden-brown growth on the inside of the shell. The condition was always evident at the apical region and, in large lesions, extended from the apex to include the site where the adductor muscle attaches to the shell. Small lesions were not usually beneath the attachment site of the adductor muscle (Table 2). In 21 (16%) of *H. iris* and 15 (38%) of *H. australis* shells examined, the adductor muscle scar was disrupted by an extensive lesion (Table 3). Lesions ranged in size from 0.01 to 40 cm^2, were soft with a jelly-like matrix, often contained a gritty component, and had a pungent odour (Fig. 2).

Sometimes, a spherical nacreous blister encompassed the soft jelly-like lesion (Fig. 3). In a few cases where the exterior of the shell had collapsed, the nacreous layer completely covered the lesion and isolated it from the mantle cavity. The nacreous blisters ranged in size from 9 to 30 cm^2. Shell lesions, similar to those described above for *H. iris,* were also observed in *H. australis* and *H. virginea virginea* (Fig. 2).

TABLE 1.

Number of specimens of *H. iris, H. australis,* and *H. virginea virginea* examined from each of two locations (Little River and Jacks Bay) and the number (and percent) affected by shell lesions.

Species	Location	No. of Specimens	Number Affected (%)
H. iris	Little River	56	30 (54%)
H. iris	Jacks Bay	20	9 (45%)
H. australis	Little River	36	17 (47%)
H. australis	Jacks Bay	20	10 (50%)
H. virginea virginea	Little River	2	2 (100%)
H. virginea virginea	Jacks Bay	3	3 (100%)

Histology of Shell and Soft Tissues

All unaffected *H. iris* and *H. australis* shells examined by light microscopy (n = 11 and n = 14, respectively) showed an organized, layered structure that consisted of alternating layers of cal-

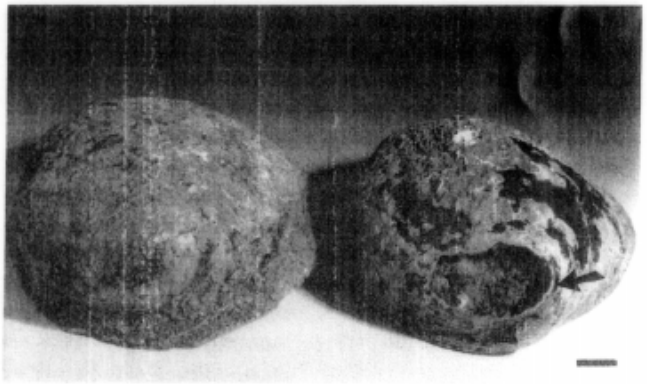

Figure 1. An affected *H. iris* (on the right) showing the external crumbling and caving-in of the apex of the shell (arrow). This occurs in some extreme cases of the condition. The *H. iris* on the left is unaffected by the condition. Scale bar, 1.5 cm.

TABLE 2.

The number of affected individuals of *H. iris* and *H. australis* and the relationship between the area of their shells with lesions and the area of their adductor muscle site exhibiting lesions.

Area of Shell Covered by Shell Lesion (%) in *H. iris* and *H. australis*	No. of Animals and Area of Adductor Muscle Scar Covered by Lesion (%)					
	None	+−10%	11–15%	15–20%	21–25%	26–50%
H. iris +−10%	16	—	—	—	—	—
11–25%	21	7	1	—	1	—
26–50%	4	4	2	2	1	1
51–75%	—	—	—	—	—	1
H. australis +−10%	4	1	—	—	—	—
11–25%	3	3	1	—	—	—
26–50%	1	4	1	1	1	—
51–75%	—	—	1	—	—	3

cium carbonate and protein (Fig. 4). In all affected *H. iris* and *H. australis* shells (n = 11 and n = 10, respectively), normal shell organization was disrupted and damaged. Gaps and necrotic tissue or conchiolin were frequently observed between shell layers, and the shell was thickened adjacent to the lesion (Fig. 5). The shells of all lesion-bearing animals contained hyphae-like structures that were generally straight, sparsely branched, and septate (Fig. 6). These structures were generally thin (1.20–2.38 μm in diameter), and no prochlamydospores were observed. Hyphae-like structures with apical swellings (4.8–7.2 × 7.2–15.0 μm) were observed in three specimens examined. These hyphal structures reacted positively to specific staining (PAS and SS) for fungi. Although similar hyphal structures were present in all of the affected shells of *H. iris* and *H. australis* that were examined, they were also sometimes found in nonlesion-bearing shells, either on the outside of the shell or, in a few instances, as a shallow penetration of the shell (Table 4).

Macroscopic and microscopic examination of the auricles, ventricles, right and left kidneys, hypobranchial glands, gonads, gills, adductor muscles, and mantle revealed no detectable differences in tissue architecture between animals with and those without lesions. Tissue sections taken from sites on the conical appendage directly adjacent to severe lesions revealed no pathological changes (e.g., no metaplasia or infiltration of hemocytes). No fungal hyphae were found in any of the soft tissues examined.

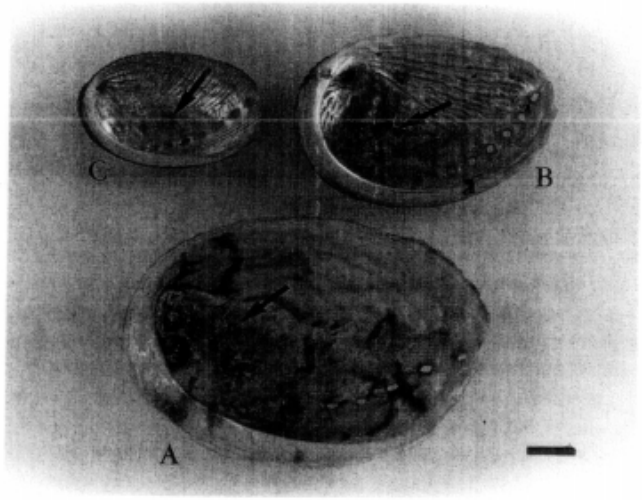

Figure 2. The interior shell surface of lesion-bearing *H. iris* (A), *H. australis* (B), and *H. virginea virginea* (C) shells. Lesions (arrows) are present on the inner surface. Scale bar, 1.5 cm.

Geographic Distribution

The 21 locations surveyed around the South Island and Stewart Island showed that shell lesions were found in *H. iris* throughout

TABLE 3.

Number of *H. iris* and *H. australis* that had a portion of the adductor muscle site disrupted and those that did not.

Species	Affected or Unaffected	Total Number	Attachment Site Not Affected	Attachment Site Affected
H. iris	Unaffected	67		
(n = 130)	Affected	63	42 (32%)	21 (16%)
H. australis	Unaffected	16		
(n = 40)	Affected	24	9 (23%)	15 (38%)

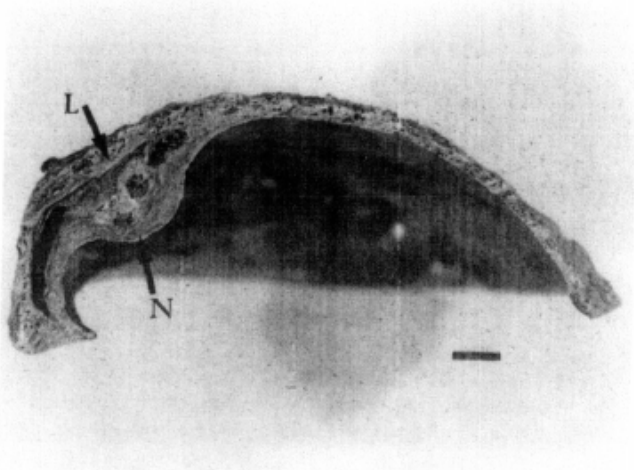

Figure 3. Transverse section of an affected *H. iris* shell. This shell has a lesion (L) completely covered by a nacreous layer (N). Note the thickening of the shell in the region of the lesion. Scale bar, 0.75 cm.

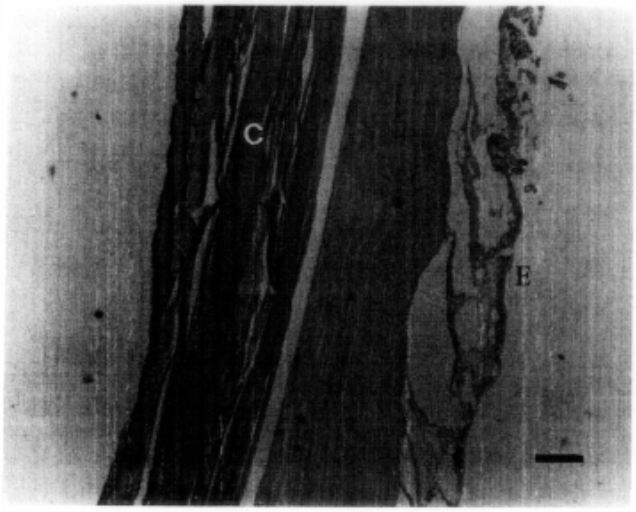

Figure 4. Transverse section of an unaffected *H. iris* shell stained with PAS. The exterior (E) of the shell and the alternating calcium carbonate (C) and protein layers are highlighted. Scale bar, 25 µm.

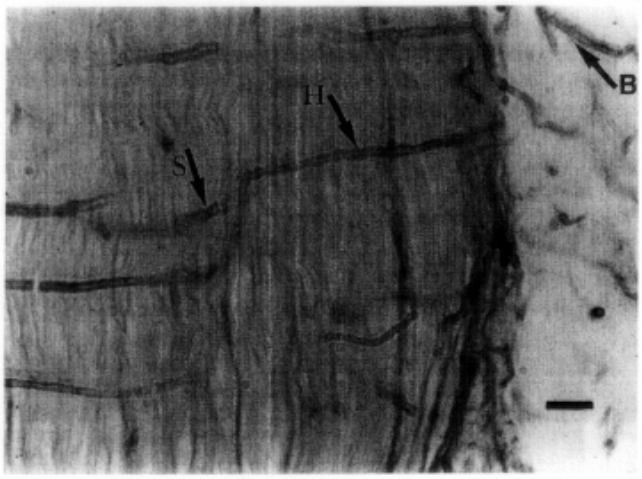

Figure 6. Transverse section of an affected *H. iris* shell stained with PAS. Fungal hyphae (H) may be seen throughout the shell matrix. The hyphae are sparsely branched (B) and separate (S). Scale bar, 10 µm.

the area sampled (Fig. 7). The prevalence of affected *H. iris* ranged from 0% at several sites, particularly in the Marlborough Sounds region, to a maximum of 70% at one location in the Catlins region. The two catches from the Breaksea/Dusky Sound location had lesion prevalences of 0.4 and 42.4%. Lesions were present in animals at all three of the off-shore islands sampled. Eight of the 15 *H. australis* from the Snares Islands were affected, and 1 of the 5 *H. virginea virginea* from Campbell Island was affected. Twelve affected shells of *H. iris* were collected from a Chatham Island fish processor.

Although the mean shell length of affected *H. iris* was less than the mean shell length of unaffected *H. iris* from all 19 catches sampled, differences were significant at only four locations: Cape Jackson, the northeast coast of Stewart Island, Port Adventure, and Coal River (Table 5). Animals sampled from Little River showed that lesion-bearing *H. iris* ranged in shell length from 64.3 to 147.0 mm, whereas lesion-bearing *H. australis* ranged in shell length from 44.8 to 86.0 mm (Figs. 8 and 9). No significant correlation was observed between shell length and the presence of shell lesions in *H. iris* ($p > 0.05$, $D_{max} = 0.3127$, $n = 56$) sampled from Little River (Fig. 8). However, we did observe a significant trend of increasing prevalence of shell mycosis to increasing shell length in *H. australis* (Fig. 9; $p < 0.05$, $D_{max} = 0.5202$, $n = 36$).

Figure 5. Transverse section of an affected *H. iris* shell stained with PAS. The shell layers are disrupted, broken, and considerably thickened. The magnification and orientation are as for Figure 8, with the exterior (E) of the shell being marked. Scale bar, 25 µm.

DISCUSSION

The lesions described in this study of the New Zealand species of *Haliotis* were rarely visible on the exterior of the shell (Fig. 1) but were always evident on the inside of the shell. Unlike many damaged mollusc shells (Vermeij 1993), lesion-bearing *H. iris* and *H. australis* collected from the wild always exhibited damage around the apex, the oldest part of the shell. In specimens with extensive damage, lesions appeared to initiate at the apex and from there expand toward, and eventually under, the site of adductor muscle attachment. Small lesions (*circa* 0.01 cm^2) were jelly like in consistency, whereas larger lesions, which often covered considerable proportions of the inner shell (*circa* 40 cm^2 in area; Figs. 2 and 3), including the adductor muscle scar (Table 2), were hard in consistency, caused shell thickening (Fig. 5), and may have calcified.

The presence of fungal hyphae associated with all of the affected shells was confirmed with PAS and SS for fungi (Grocott

TABLE 4.

Number of lesion-bearing and nonlesion-bearing *H. iris* and *H. australis* in which hyphae were either absent, present on the shell surface only, or embedded in the shell or lesion.

	No. of Animals			
	No Visible Lesions			Lesions
Species	No Hyphae	Surface Hyphae Only	Hyphae Within Shell	Hyphae Within Lesion
H. iris	4	3	4	11
H. australis	8	4	2	10

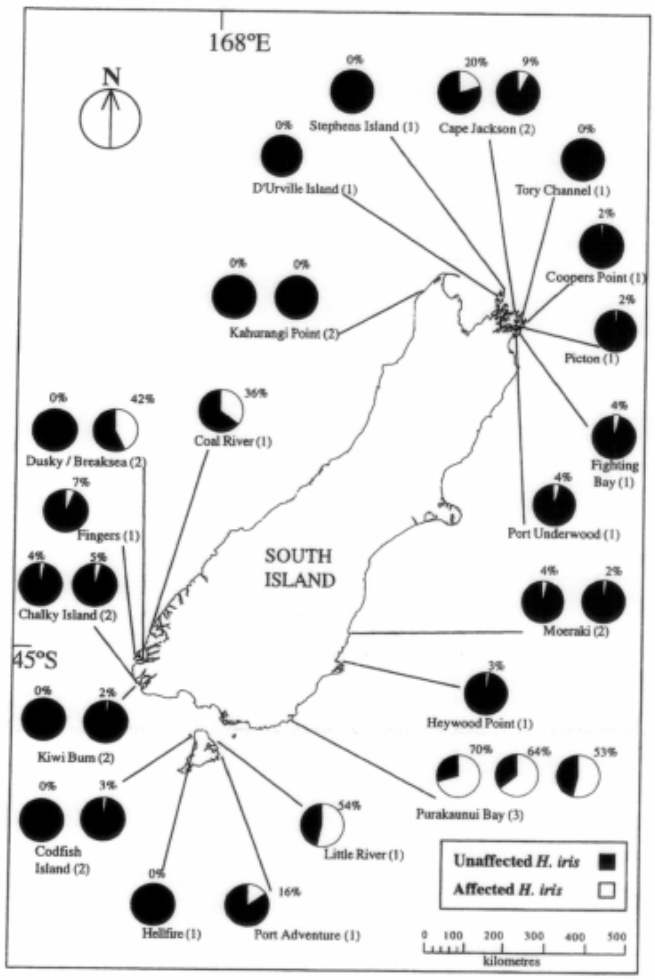

Figure 7. The South Island of New Zealand showing the locations of commercial catches of *H. iris* sampled for the presence of shell lesions. The percentage of affected *H. iris* from each catch is given and the number of catches sampled at each location is indicated in parentheses after the location name.

1955; Fig. 6). Although fungal hyphae were also found in a few shell samples taken from nonlesion-bearing shells (Table 4), they were only found in the surface layers of the shell, which suggests that they may represent an early stage in the development of lesions. Microscopic examination of stained tissue sections from visceral, pedal, mantle, and adductor muscle tissues of several lesion-bearing animals appeared normal (Bevelander 1988) and did not contain fungal hyphae. These lesions were thus confined to the shell of the animal.

Sinclair (1963) described a number of irregularities occurring in shells of *H. iris* from the Wellington district that share some macroscopic characteristics with the lesions we describe. Although the site of some lesions described by Sinclair (1963) is similar to those described here (i.e., posterior to the adductor muscle scar), the tubercules were described as similar in structure to nacreous pearls. No mention was made of a brown-colored, soft, jelly-like substance or of the potential for the adductor muscle to become detached from the shell. Sainsbury (1977) also briefly described blisters in *Haliotis* from Banks Peninsula, which in some cases resulted in the animal losing its shell. These blisters were attributed

TABLE 5.

Location, mean shell length, and number of unaffected and affected *H. iris* sampled, together with estimates of significant differences (bold indicates $p < 0.01$) between the shell length of unaffected and affected animals at each site.

Location	Mean Length (Unaffected)	Mean Length (Affected)	p Value
Cape Jackson (Marlborough)	130.2 (n = 100)	129.4 (n = 18)	0.5396
Cape Jackson (Marlborough)	129.7 (n = 104)	127.5 (n = 57)	**0.0004**
Fighting Bay (Marlborough)	131.1 (n = 95)	130.7 (n = 12)	0.9568
Northeast Stewart Island	152.5 (n = 105)	147.5 (n = 82)	**0.0029**
Port Adventure (Stewart Island)	145.1 (n = 128)	137.9 (n = 40)	**0.0008**
Dusky/Breaksea (Fiordland)	132.4 (n = 91)	132.0 (n = 54)	0.6858
Coal River (Fiordland)	143.8 (n = 101)	138.7 (n = 89)	**0.0004**
Fingers Peninsula (Fiordland)	133.2 (n = 134)	133.1 (n = 17)	0.9251
Purakaunui Bay (South East Otago)	134.9 (n = 70)	134.0 (n = 82)	0.3637
Purakaunui Bay (South East Otago)	133.1 (n = 64)	132.1 (n = 88)	0.2350
Catlins (South East Otago)	134.4 (n = 45)	133.2 (n = 103)	0.1900

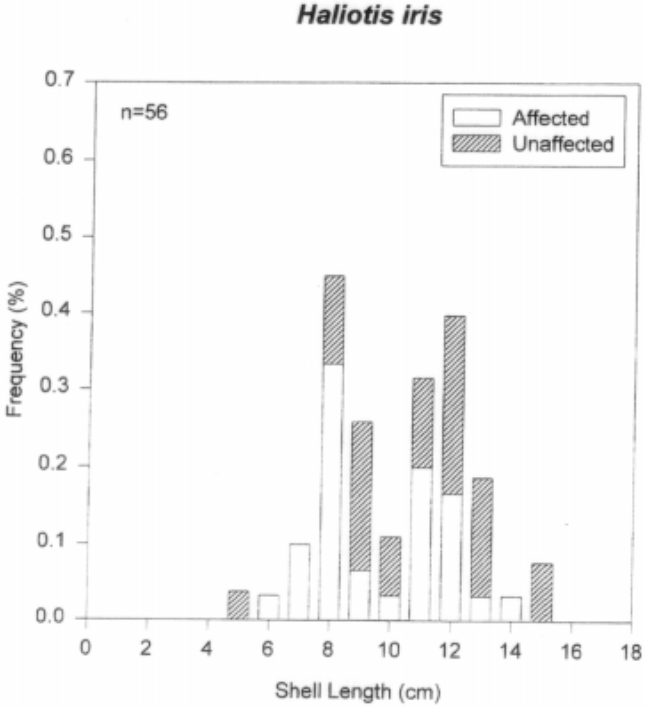

Figure 8. Histogram showing the percent frequency of affected and unaffected *H. iris* in different size classes collected from Little River.

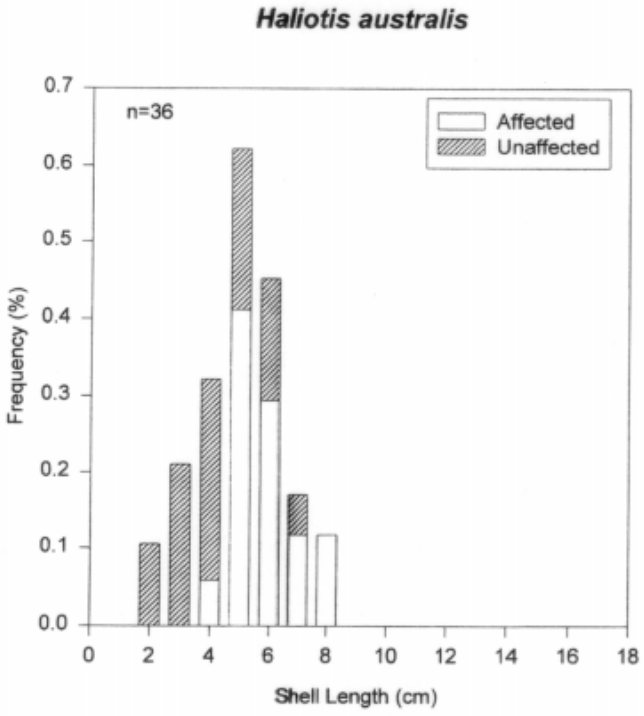

Figure 9. Histogram showing the percent frequency of affected and unaffected *H. australis* in different size classes collected from Little River.

to irritation by boring organisms and/or sand grains lodged in the mantle cavity. Although many of the abalone in our study were infested with *Polydora* sp., no penetration into the nacreous layer was observed. In addition, we did not observe any evidence of sand within the shell cavities of affected or unaffected animals. The lesions we describe appear to be composed of conchiolin and fungal hyphae. In an earlier study, we isolated pure cultures of a fungus from shell lesions of New Zealand abalone (Friedman et al. 1997). On the basis of the limited information provided by Sinclair (1963) and Sainsbury (1977), the lesions we describe differ from those they report.

Records of fungus-like structures invading the calcareous parts of marine animals, particularly molluscs (e.g., Stirrup 1872), date back more than 100 y (Kohlmeyer 1969). Johnson and Anderson (1962) reported hyphae and chlamydospores of a fungus that showed affinities to *Endogone* (Phycomycetes) in a shell of *Anomia,* and Najdenova and Zakhaleva (1992) described fungal shell diseases in oysters (*Ostrea edulis* Linne) and mussels (*Mytilus galloprovincialis* Lamarck) from the Black Sea. Korringa (1951) reported a shell disease that resulted in conchiolin warts on the inner surfaces of the shells of *O. edulis* and that was later attributed to the fungus *Ostracoblabe implexa.*

The possible role of the fungus reported here in the development of shell lesions in New Zealand *Haliotis* awaits further elucidation as does the extent, if any, to which the presence of the fungus is facilitated by the presence of worms and/or other irritants, such as sand grains. Although it is clear that these lesions are restricted to the shell, as was also apparently the case with those described by both Sinclair (1963) and Sainsbury (1977), it is possible that the presence of the lesion and associated fungus may affect these molluscs in ways not yet documented. Friedman et al. (1997) did not observe an association between size of lesion and serum protein levels, sex, or condition index. However, a significant relationship was observed between number of circulating hemocytes and degree of shell mycosis in *H. australis* and was thought to represent a sign of stress or response to the shell disease. Thus, further examination of physiological parameters and reproductive development of abalone with and without shell lesions is warranted.

Some lesion-bearing *H. iris,* collected from the wild and held in captivity, have been reported to have very loosely attached shells (Langdon pers. comm.). The existence of shells in which the adductor muscle attachment site was disrupted by a lesion (Table 3) led us to conclude that, in such extreme cases, the abalone may lose its shell, as has been indicated by Sainsbury (1977). Thus, populations with affected animals may experience elevated mortality relative to those that are free of the shell lesions described in this study. Other shells exhibiting similar lesions showed evidence of having isolated the lesion beneath nacreous blisters (Fig. 3). This suggests that some animals have the potential to deposit shell layers over a lesion, thereby possibly containing it and preventing the shell from being lost.

Lesion-bearing *H. iris* were widespread along the South and Stewart Island coasts, with prevalences in individual populations that ranged from 0 (e.g., Stephens Island) to 70% (e.g., Purakanui Bay) (Fig. 7). Samples from adjacent populations (e.g., Dusky/Breaksea) also showed remarkably different levels of incidence of shell lesions (Fig. 7). The presence of shell lesions in all species examined, from several of the off-shore islands where human habitation is low, suggests that they occur naturally. Furthermore, abalone divers assert that this condition has existed for at least 25 y (Grindley 1997), supporting the contention that lesions are a well-established feature of New Zealand *Haliotis.* Until more is known about how such lesions are caused, it will remain problematic to explain the extent of these variations.

Regardless of the ultimate cause, the available evidence does not support the contention that the three lesion-bearing species of *Haliotis* in New Zealand have different etiological agents. In the two instances where specimens of *H. iris, H. australis,* and *H. virginea virginea* from the same site were examined, all three species were found to bear lesions (Table 1). In addition, morphologically indistinguishable fungi were isolated from early shell lesions of both *H. iris* and *H. australis* (Friedman et al. 1997). The existence of shell lesions in *H. iris* and *H. australis* confirms that they can occur in two (*Paua* and *Padollus*) of the three subgenera within *Haliotis* (Lee and Vacquier 1995). Without a more detailed examination of other marine invertebrate species, both in New Zealand and elsewhere, it is premature to comment on the host specificity of the lesions that we describe.

The mean shell length of affected abalone, from all sites combined, was significantly ($p = 0.006$) smaller than that of unaffected animals (Table 5). More detailed analysis (Table 5) suggested that at only 4 of the 11 sites examined were shell lengths of affected and unaffected animals significantly different. Although there are many possible reasons for these differences, it is of note that in all four cases, the shell lengths of affected animals were less than the shell lengths of unaffected animals (Table 5). This suggests that either growth and/or survival may be impaired by this condition, at least at some locations. Although the age of affected animals has not been estimated in this study, the large size range of *H. iris* and *H. australis* exhibiting advanced lesions (Figs. 8 and 9) suggests that lesions may be acquired early in life. This hypothesis is supported by Figure 9, in which the frequency of shell

lesions increased with increasing shell length. Determining the effect of shell lesions on growth, reproduction, and survival of affected individuals relative to those without lesions and clarifying the possible role of epizootics in the development of these lesions will be important areas for future research.

ACKNOWLEDGMENTS

We thank Dr. Graeme Bremner for drawing these shell lesions to our attention and for incisive criticism of early drafts of the article. It is a pleasure to also acknowledge the sustained and able assistance of Dick Langdon in sharing his hard-won knowledge and in obtaining and shipping live specimens. We are very grateful to Ian Kennedy and Peter Brett of Paua Supplies Ltd., who made it possible for us to mount a shed sampling programme. The work was fostered materially by the Marine Science and Aquaculture Research Centre and by a Summer Research Bursary (RMG) from the Division of Sciences, both part of the University of Otago. The Histology and Photographic Units at the University of Otago Medical School provided much needed assistance with the silver staining and photography, respectively. Two anonymous referees, Jim Moore, and Vic Chow made valuable contributions to an earlier draft for which we are grateful. Finally, we acknowledge our debt to Dr. Jenny Dugan, who facilitated the furtherance of this research.

LITERATURE CITED

Alderman, D. J. & E. B. G. Jones. 1967. Shell disease of *Ostrea edulis* L. *Nature.* 216:797–798.

Batham, E. J. & J. T. Tomlinson. 1965. On *Cryptophialus melampyges* Berndt, a small boring barnacle of the order Acrothoracica abundant in some New Zealand molluscs. *Trans. Roy. Soc. N. Z. Zool.* 7:141–154.

Bevelander, G. 1988. Abalone Gross and Fine Structure. The Boxwood Press, Pacific Grove, CA. 80 pp.

Clavier, J. 1992. Infestation of *Haliotis tuberculata* shells by *Cliona celata* and *Polydora* species. pp. 16–20. *In:* S. A. Guzman del Proo, M. J. Tegner, and S. A. Shepherd (eds.). Abalone of the World. Biology, Fisheries and Culture Supplementary Papers. Proceedings of the First International Symposium on Abalone in La Paz, Mexico. November 21–25, 1989.

Cox, K. W. 1962. California Abalones, Family Haliotidae. *Calif. Dept. Fish Game Fish. Bull.* 118:1–133.

Crofts, D. R. 1929. *Haliotis. Liverpool Mar. Biol. Comm. Mem.* 29:2–166.

Friedman, C. S., R. M. Grindley & J. A. Keogh. 1997. Isolation of a fungus from shell lesions of New Zealand abalone, *Haliotis iris* Martyn and *H. australis* Gmelin. *Molluscan Res.* 18:313–324.

Grindley, R. M. 1997. Shell lesions in New Zealand *Haliotis.* MSc Thesis. University of Otago, Dunedin, New Zealand. 70 pp.

Grocott, R. G. 1955. Stain for fungi in tissue sections and smears, using Gomori's methenamine-silver nitrate method. *Am. J. Clin. Pathol* 25: 975–979.

Humason, G. L. 1979. Animal Tissue Techniques. 4th ed. W. H. Freeman and Company, San Francisco. 661 pp.

Johnson, T. W., Jr. & W. R. Anderson. 1962. A fungus in *Anomia simplex* shell. *J. Elisha Mitchell Sci. Soc.* 78:43–47.

Kohlmeyer, J. 1969. The role of marine fungi in the penetration of calcareous substances. *Am. Zool.* 9:741–746.

Korringa, P. 1951. Investigations on shell-disease in the oyster, *Ostrea edulis* L. *Rapports et Procès-Verbaux des Réunions, Conseil Permanent International pour l'Exploration de la Mer* 128:50–54.

Lee, Y.-H. & V. D. Vacquier. 1995. Evolution and systematics in Haliotidae (Mollusca: Gastropoda): inferences from DNA sequences of sperm lysin. *Mar. Biol.* 124:267–278.

Lillie, R. D. 1965. Histopathologic Technique and Practical Histochemistry. 3rd ed. McGraw-Hill, New York. 715 pp.

McManus, J. F. A. 1948. Histological and histochemical uses of periodic acid. *Stain Technol.* 23:99–108.

Najdenova, N. N. & V. A. Zakhaleva. 1992. Fungal shell diseases of Black Sea Mollusks. *Gidrobiologicheskii Zhurnal (Kiev) Hydrobiology Journal* 28:78–81.

Oakes, F. & R. Fields. 1994. Infestation of *Haliotis rufescens* shells by a sabellid polychaete. Conference Abstract from the Second International Abalone Symposium held in Hobart, Tasmania. February 7–11, 1994.

Powell, A. W. B. 1979. New Zealand Mollusca. Collins, Auckland. 500 pp.

Sainsbury, K. J. 1977. Population Dynamics and Fishery Management of the Abalone, *Haliotis iris.* Ph.D. Thesis. University of Canterbury, Christchurch, New Zealand.

Shaw, B. L. & H. I. Battle. 1957. The gross and microscopic anatomy of the digestive tract of the oyster *Crassostrea virginea* (Gmelin). *Can. J. Zool.* 35:325–347.

Sinclair, M. 1963. Studies on the paua, *Haliotis iris* Martyn, in the Wellington district, 1945–46. *Zool. Publ. Victoria Univ. Wellington.* 35:1–16.

Stirrup, M. 1872. On shells of Mollusca showing so-called fungoid growths. *Proc. Literary Philosoph. Soc. Manchester.* 11:137–138.

Vermeij, G. J. 1993. A Natural History of Shells. Princeton University Press, Princeton, NJ. 207 pp.