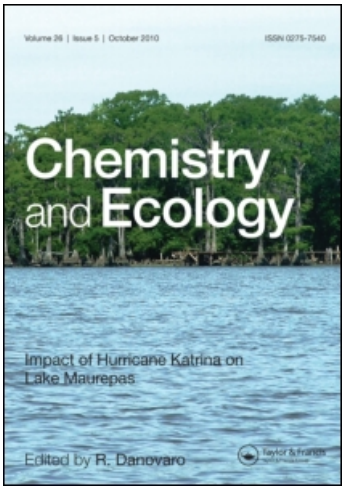

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Shell Disease of Brown Shrimp, *Crangon crangon* (L.), and Other Marine Crustacea from The Solway Firth

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The *Crangon crangon* (L.) population of the Solway Firth displays a mean incidence of shell disease of 13.2%, which is some 1.5 times more than any other crustacean species in this area. The condition is characterized by superficial pitting and cracking of the carapace leading to the formation of blackened erosions which are commoner in larger individuals and, in all, are lost at the moult. Visually identical damage appears within 4–5 days of mechanical abrasion of the exoskeleton, but is inhibited if the abraded animals are maintained in an antibiotic medium.

Disease incidence fluctuates throughout the year with minima in March and August and maxima during the winter months. Apparently the activity of the local commercial shrimp fishery is the primary cause of the high incidence of shell disease amongst these shrimp. Mechanical damage to the exoskeleton, incurred as a result of abrasion by fishing instruments, facilitates penetration of the epicuticle by chitinoclastic bacteria which then induce chitinolysis of the underlying calcified exoskeletal layers. The condition does not seem to cause a significant mortality of the shrimp population nor does it affect the value of the commercial catch.

INTRODUCTION

Shell disease, characterized by varying degrees of pitting and cracking of the outer calcified exoskeletal layers, has been reported in many crustacean species of several orders (Rosen, 1970). Some authors have also described penetration of the inner, non-calcified, exoskeleton and necrosis of the soft underlying tissue (Young and Pearce, 1975; Gopalan and Young, 1975). Gopalan and Young believed that this condition may be due to secondary invasion by pathogenic bacteria not involved in the primary induction of erosions. When damage is superficial, the affected individual may overcome the condition by moulting (Rosen, 1970, Gopalan and Young, 1975), but necrosis of underlying tissue may explain the observation by

Schlotfeldt (1972) that erosions of *Crangon crangon* were retained and enlarged after the moult.

Typically the edges of erosions are brown to black in colour. This could be due to the presence of chitinoclastic bacteria which have been shown to produce brown pigment in enrichment media (Zobell, 1946; Rosen, 1967), but is more probably part of the crustacean's natural response to tissue damage. Thus Lightner *et al.* (1977) correlated blackening of erosions with melanin deposition in areas of heavy haemocyte infiltration and Poinar *et al.* (1968) (in Nappi, 1975) showed that parasitic nematodes in larvae of the beetle genus *Diabrotica* become gradually surrounded by melanin produced by the transformation of cytoplasm released by autolyzed haemocytes.

The characteristic colouration has prompted the use of terms such as "pitted" shell disease (Hess, 1937), "burn spot" disease (Mann and Pieplow, 1938; Bakke, 1973), "corroded spot" disease (Dogel and Petrushevskii, 1957), black spot disease (Schlotfeldt, 1972) and black necrosis (Abbott, 1977) to describe what appear to be visually similar conditions. Whether all reported cases of exoskeletal erosions are due to a disease state resulting from a single cause has not been proven, however, and the etiology of shell disease remains confused.

Mann and Pieplow (1938) identified fungal hyphae in the exoskeleton of decapods suffering from "burn spot" disease. They succeeded in culturing the fungi and in infecting healthy crabs with pure culture. Egusa and Ueda (1972) isolated a *Fusarium* sp. from Kuruma prawn *Penaeus japonicus* infected with black gill disease and induced the condition in healthy prawns by intramuscular inoculation with fungal conidia. Despite these results it is generally believed that chitinoclastic bacteria are the primary causative agent of exoskeletal erosions in crustacea. These bacteria have been isolated from erosions in a variety of species (Hess, 1937; Rosen, 1967, 1970; Cook and Lofton, 1973; Baross, 1978; Malloy, 1978 and Chan, 1979), and while abundant in marine sediments (ZoBell and Rittenberg, 1938) occur as commensals on the integument of many marine crustaceans (Lear, 1963; Rosen, 1967). Rosen (1970) suggested that several organisms are responsible for inducing shell disease, but Cook and Lofton (1973) implicated the bacterium *Beneckea* Type I as the principal infective agent. Whether one or more species are involved the symptoms produced are essentially similar. Not all attempts to infect healthy crustacea with chitinoclasts have been successful and it would seem that the bacteria are facultative pathogens co-existing with their hosts under normal conditions and only inducing disease in stressful situations. Several authors regard mechanical damage to the epicuticle as an essential pre-requisite for the development of erosions. Malloy (1978) induced erosions in experimental lobsters with *Beneckea* bacteria only when the integument was damaged prior to inoculation. Chan

(1979) lightly abraded the exoskeleton of the freshwater shrimp *Atya bisulcata,* and, when the treated individuals were exposed to raw river water, or cultures of chitinoclasts, erosions developed. Abrasions immediately sealed with nail polish did not, however, develop erosions. Gopalan and Young (1975) reared healthy shrimp (*Crangon septemspinosa*) in natural and artificial, antibiotic seawater. After 6 weeks 50% of shrimp in natural sea water were infected with shell disease, but none of those in the antibiotic medium were. These experiments, together with those of Chan (1979), indicate an infectious etiology for shell disease. Rosen (1970) suggested that the lack of success in some bacterial inoculation experiments is due to the chemical structure of crustacean epicuticle which is composed of polyphenolic substances relatively inert to biochemical attack. If this barrier is breached in some way, e.g., by mechanical abrasion, the underlying calcified chitin is susceptible to digestion by chitinoclastic bacteria. If this hypothesis is correct, subtle changes in body chemistry may also influence individual susceptibility to shell disease. Lightner *et al.* (1977) induced multiple black lesions in the subcuticular tissues of the general body surface, walls of the oesophagus, stomach, hind gut, gills and gill cavity of the shrimp *Penaeus californiensis* fed on a diet low or deficient in L-ascorbic acid. Fisher *et al.* (1976) found that the greatest susceptibility of juvenile lobster *Homarus americanus* to chitinolytic bacteria occurred in animals fed on long term synthetic diets or fed on short term synthetic diets and treated with malachite green. Pollution has been implicated indirectly in some cases, thus Schlotfeldt (1972) studied *C. crangon* in the North Sea and recorded an incidence of 0.9%–8.9% apparently aggravated by the anionic detergent tetrapropylenebenzosulfonate. Young and Pearce (1975) observed significant numbers of crabs and lobsters suffering from shell disease on sewage sludge disposal grounds in New York Bight. They found that healthy lobsters kept for 6 weeks in aerated tanks containing sewage sludge developed identical disease symptoms while control specimens remained in good condition. Gopalan and Young (1975) demonstrated an apparent increase in incidence of shell disease amongst *Crangon septemspinosa* taken from moderately to severely polluted areas of the Atlantic coast of America when compared to non-polluted areas. The association is unclear, however, for Abbott and Perkins (1977) found high levels of black necrosis (mean 26.4%, range 8–54%) in *C. crangon* taken from the essentially unpolluted Solway Firth.

C. crangon is extremely abundant in the Solway Firth and supports an important local fishery. The whole catch is taken for human consumption, chiefly as potted shrimp. Apart from its economic significance the shrimp, because of its abundance, is a key species in the ecology of the Solway system being heavily preyed upon by many species of direct and indirect

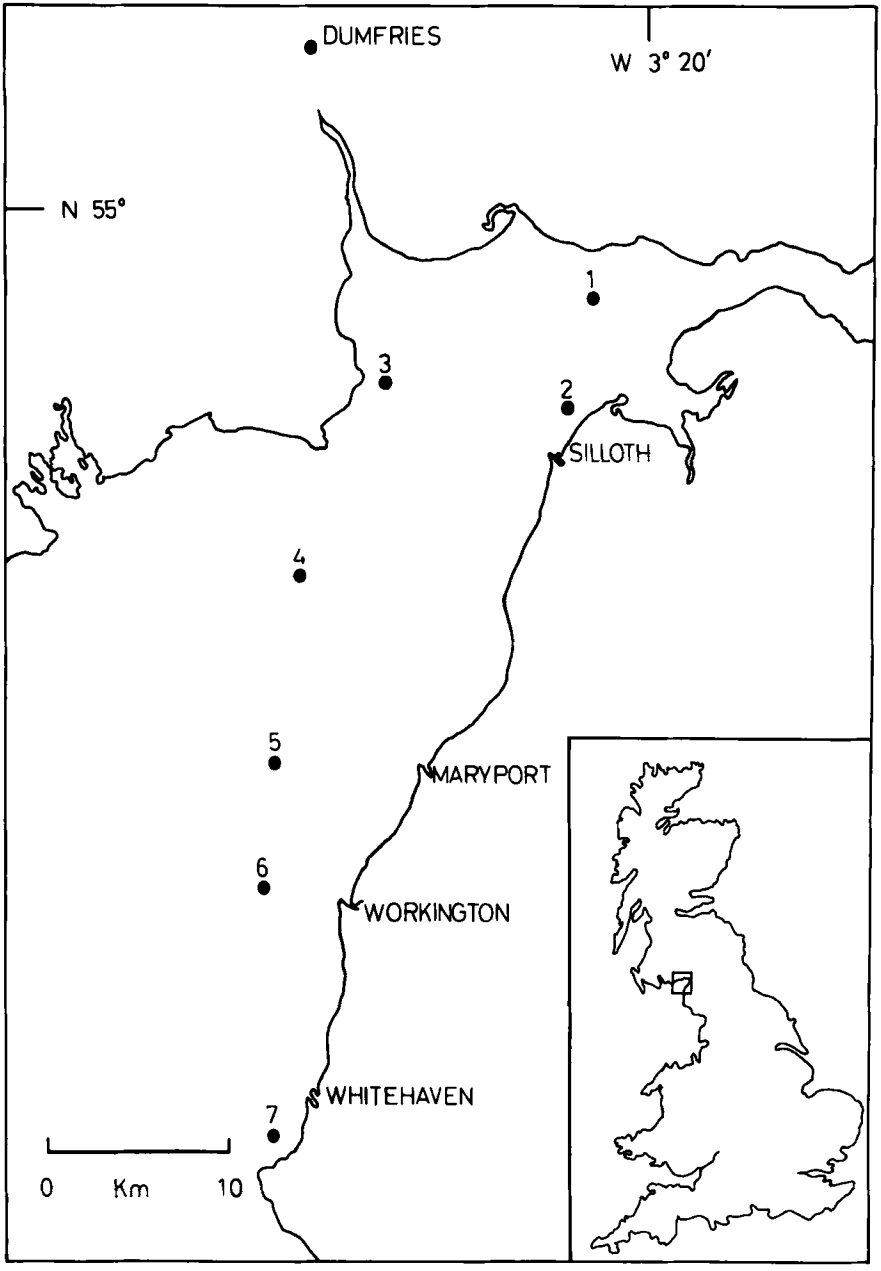

FIGURE 1 The Solway Firth showing the beam trawl sampling stations: 1. Powfoot; 2. Skinburness; 3. Borron; 4. Dumroof; 5. Robin Rigg; 6. Workington Bank; 7. Saltom Bay

commercial interest (Perkins *et al.*, 1963; Nottage and Perkins, 1978). The Kilcreggan laboratory has maintained a watching brief on the shrimp population since an intensive study of its biology was completed by Abbott and Perkins (1977). During this period the high incidence of shell disease, first noted by them, has been continued.

Shell disease is not invariably fatal though death does occur in some cases presumably as a result of the impairment of bodily funtions (Rosen, 1970). The presence of unsightly shell damage in species, such as crabs and lobsters, whose marketable value depends largely upon their visual appearance is obviously undesirable. In small species, such as shrimp, disfigurement may go unnoticed, but the possibility of a tainting effect remains. Fishermen on the Isle of Man term dirty, discoloured, edible crabs (*Cancer pagurus*) with broken claws and pitting of the carapace "Grannies". These individuals have a bitter taste and powerful purgative effect when eaten (Perkins, 1974). This paper presents the results of an investigation of shell disease in crustacea, particularly brown shrimp, taken from the Solway Firth.

METHODS

Samples were taken in a 2.4 m beam trawl. The ends of the beam were supported by steel shoes 36 cm high: the net, of courlene twine, had a stretched mesh of 22 mm and the foot rope (3.2 m) was fitted with 50 mm diameter rubber discus set 38 mm apart: a ticker chain was fitted also.

Weather permitting, hauls were made monthly during 1978–81 at selected sites in the Solway Firth (Figure 1). The coordinates at the beginning and end of each haul were recorded from a Decca (101) Navigator system installed abroad the towing vessel "Solway Protector". Every effort was made to trawl between the same Decca co-ordinates at each station in successive months (Table I).

TABLE I

Mean Decca Coordinates for the beam trawl stations in the Solway Firth

Powfoot	D33.7G, E58.8P to D33.9G, E58.0P
Skinburness	D35.4G, E55.7P to D35.7G, C54.7P
Dumroof Bank	C42.8G, D53.0P to C42.8G, D56.5P
Borron Point	D41.8G, C76.8P to D41.0G, C77.6P
Robin Rigg	D31.3G, C71.5P to D30.8G, C73.8P
Workington Bank	D38.0G, C53.3P to D38.4G, C54.6P
Saltom Bay	E35.8G, B69.6P to E36.0G, B71.0P

Additional shrimp samples were collected in 1979 and 1980, on an occasional basis, from a commercial fisherman's catch. These trawls were concentrated in the major fishing area, viz., the channels around Skinburness, Silloth and Dumroof Bank.

All samples were preserved in 10% formalin seawater immediately after capture. Subsamples of 100 shrimp were examined under binocular microscope at various magnifications. Each animal was sexed and the carapace length, i.e. distance between the posterior margin of the eye socket and the posteriodorsal margin of the carapace along the dorsal mid line, was measured, to the nearest mm upwards, using a calibrated eyepiece. Incidence, location and severity of shell disease was recorded. Similar analysis was performed on all other crustacean species.

Blocks of diseased integument were dehydrated in absolute alcohol, embedded in parafin wax, sectioned at 10 μm and stained with haematoxylin and eosin for histological examination. Most shrimp were sectioned whole without prior decalcification. Decalcification when required was performed in a solution of 2% EDTA and 4% Na salt of EDTA in 70% alcohol until a rubber-like texture was acquired.

For experimental work, hauls of 5 minutes duration were taken to minimize stress and injury. Inactive or visibly damaged shrimp were rejected and healthy looking individuals were placed in 25 l polythene bins with snap on lids. Each bin contained 20 l of sea water and 20–25 shrimp depending on size. They were transported by road from the Solway Firth and motion of the van was sufficient to promote adequate oxygenation via natural gas exchange at the water surface. No temperature control was used in transport, but collections were made in early spring when ambient temperatures were low. In the laboratory shrimp were subject to a 14 day acclimation period at 20°C. Each shrimp was isolated from its neighbours by enclosure in a perforated, cubical, tupperware container of side 10 cm. Ten such containers in a large grey polythene tank containing 15 l of seawater constituted one experimental system.

Throughout acclimation and experimentation tanks were cleaned weekly and shrimp fed freely on chopped ox liver removed 2–3 h after presentation. Aeration was provided by mains operated electric pump.

Mechanical abrasion of the carapace was achieved by the use of a sterilized scalpel blade to remove small portions of epicuticle. Abraded shrimp, together with controls, were subjected to different experimental regimes, thus,

i) Maintenance in natural sea water

ii) Maintenance in artificial sea water (New Tropic Marin synthetic marine salt)

iii) Maintenance in natural sea water + 10 ppm Streptomycin sulphate

iv) Maintenance in natural sea water + 5 ppm Sodium alkyl benzene sulphonate.

RESULTS

The mean incidence of shell disease in brown shrimp from the Solway Firth during the period January 1978—January 1981 was 13.2% (range 5–26%). Disease levels varied throughout the year, but were similar in magnitude in each year studied. Results for comparable months have been combined in Figure 2 showing disease minima in March and August with maxima in late autumn/winter. It was impossible to discern seasonal variations in disease incidence amongst other crustacean species, examined on an irregular basis, but levels were always lower than those found in the brown shrimp. The shore crab, *Carcinus maenas,* was the most severely affected with a mean disease incidence of 8.7%.

Amongst brown shrimp there was no significant difference in disease incidence between the sexes or between ovigerous and non-ovigerous females. When results for both sexes were combined a significant difference (Chi = 70.5 $p < 0.001$) did exist between size groupings (Figure 3). This result

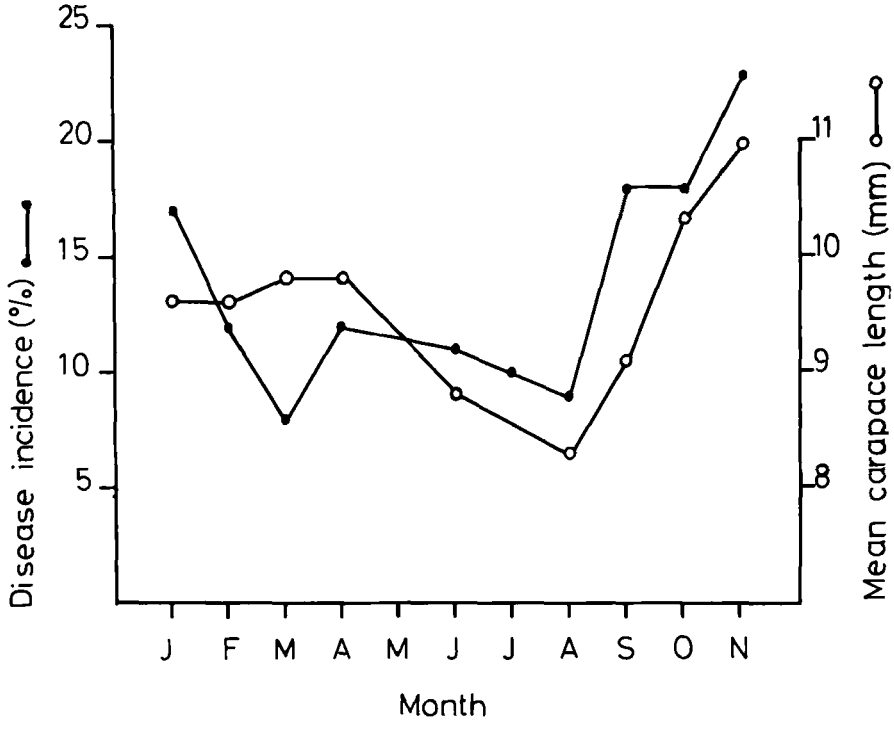

FIGURE 2 Seasonal variation in mean size incidence of shell disease amongst brown shrimp taken from the Solway Firth 1978–81

was due to a greater level of infection amongst larger animals particularly those ≥ 9 mm carapace length. In shrimp ≤ 8 mm carapace length infection was lower than expected if all size classes are affected equally.

In gross appearance shell disease, in all crustacea examined, was characterized by blackened, eroded areas of exoskeleton (Figure 4). All parts of the body could be affected and damage varied from small pitted areas to widespread sloughing of the cuticle. Microscopic examination of prepared sections cut through affected areas revealed that damage was confined to the outer exoskeletal layers. Penetration of the exoskeleton and damage to the underlying tissues was never observed. In Figure 5, a section through the eroded carapace of *Crangon,* the exoskeletal basement membrane is intact although it became detached in preparation, to the left of the picture. In the central area the outer exoskeletal layers have blackened and sloughed away. The damaged area is eroded, thus the term erosion is used in preference to any other throughout this paper to describe the visual symptoms of shell disease.

No trace of secondary infection, bacterial or fungal, was found in any of the diseased animals examined. Damaged shrimp maintained in the laboratory lost all trace of disease at the moult producing new unaffected externa. Previously damaged areas did not appear prone to reinfection and several shrimp overcame visually severe erosions in this way (Figure 6).

Superficial scraping of the carapace with a sterilized scalpel blade produced extensive erosions in 8 of 10 shrimp so treated within 4–7 days. The induced erosions were identical in gross and microscopic appearance to naturally occurring ones and were similarly lost at the moult. Abrasion of

TABLE II

The incidence of shell disease amongst crustacea taken from the Solway Firth during 1978–1981

Species	Number examined	% Incidence of shell disease
Amphipods	400	3.5
Mysids	51	0
Crangon crangon	7880	13.2
Crangon allmani	496	7.7
Pandalus montagui	62	4.8
Eupagurus bernhardus	57	0
Hyas araneus	6	0
Corystes cassivelaunus	609	5.6
Carcinus maenas	69	8.7
Macropipus holsatus	300	4.3

10 shrimp which were then maintained in sea water containing 10 ppm streptomycin sulphate produced no erosions although 3 individuals did develop black pigmented patches in the abraded area. Only 3 of 10 abraded shrimp maintained in artificial sea water developed erosions within 7 days, although after 14 days 6 were affected. The anionic detergent sodium benzene sulphonate had no apparent effect on erosion development. Of 8 abraded shrimp maintained in sea water containing 5 ppm sodium benzene sulphonate 6 developed erosions identical in appearance and severity to those in detergent free media. Shrimp in detergent solution lost all trace of damage at the moult. A control series of 16 shrimp tested in conjunction with the above investigations produced no erosions. No deaths were recorded in any of these experiments.

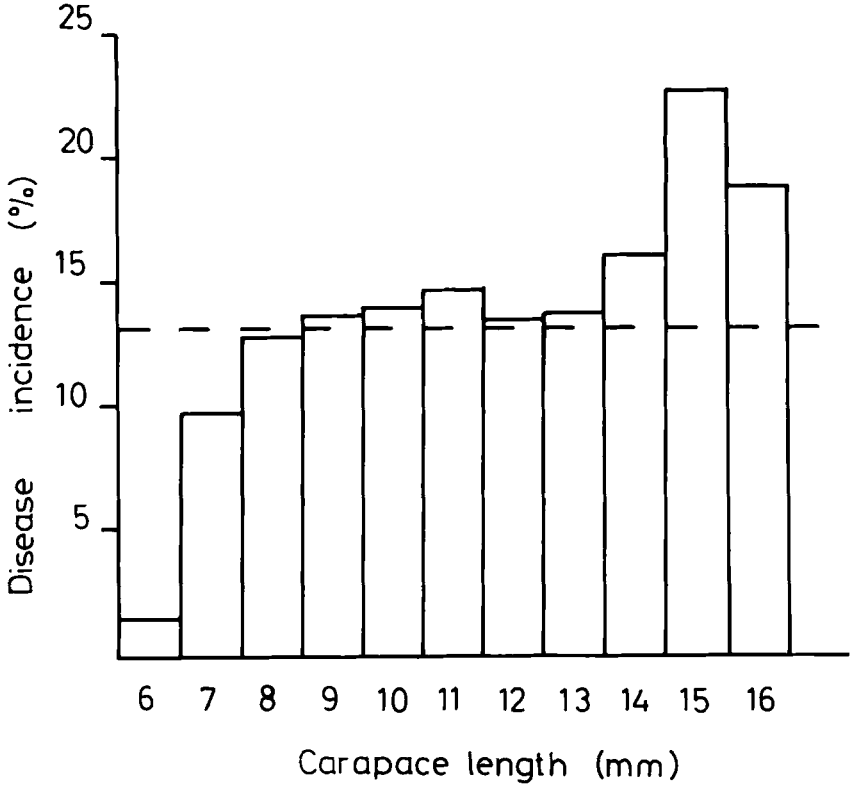

FIGURE 3 Variation in the incidence of shell disease amongst different size groupings of brown shrimp (both sexes combined) broken line indicates population mean incidence of 13.2%

FIGURE 4 Typical shell disease of brown shrimp *Crangon crangon* from the Solway Firth

FIGURE 5 Section through eroded carapace of brown shrimp

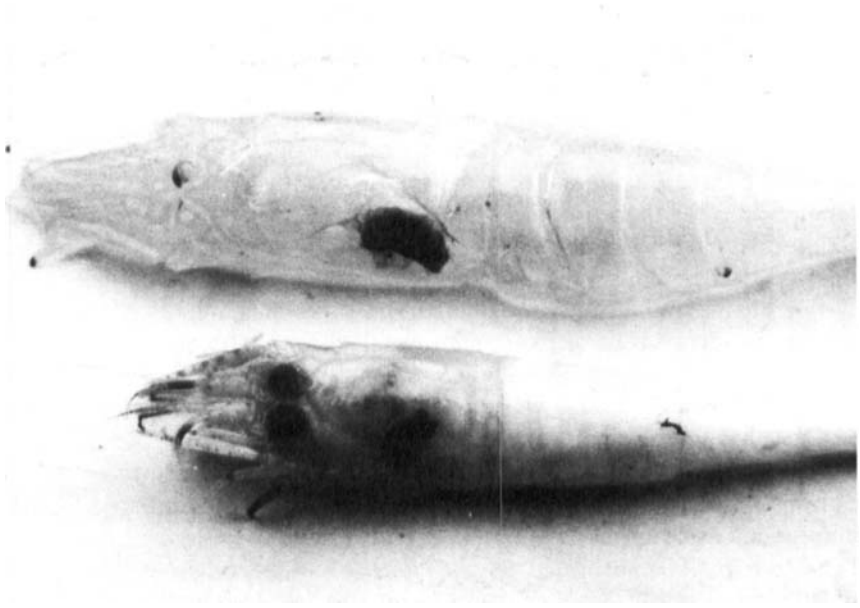

FIGURE 6 Induced lesion in brown shrimp and shrimp cast showing the loss of all damage at the moult

DISCUSSION

The shell disease affecting *Crangon crangon* and other crustacea in the Solway Firth appears, from gross and microscopic examination to be very similar to the condition described by a variety of authors and reviewed by Rosen (1970). It is characterized by chitinolysis and erosion of the exoskeleton which typically assumes a blackened colouration in the affected area. Three abraded shrimp developed pigmented patches in the treated area, but no erosion, when maintained in an antibiotic medium suggesting that this colouration is indicative of a bodily reaction to tissue damage. Chan (1979), observing a similar phenomenon, ascribed it to melanization of haemocytes aggregated in the damaged area.

Microscopic examination revealed that chitinolysis was confined to the outer integumental layers and did not penetrate the exoskeleton. Diseased shrimp maintained in the laboratory lost all trace of damage at the moult and were apparently not prone to reinfection. These results are consistent with those described by Rosen (1970), but conflict with the findings of Schlotfeldt (1972) suggesting that the latter was observing either a different

pathological condition or general shell disease complicated by secondary infection(s).

There is a marked tendency for smaller shrimp to be less heavily affected by shell disease than larger individuals thus a low disease incidence is concurrent with minimum carapace length in August, the month of maxium juvenile recruitment (Figure 2). Nursery areas around Borron and Powfoot Channel, where the population is predominantly juvenile, also exhibit lower levels of disease than the spawning grounds at Dumroof, Robin Rigg and Skinburness with combined means of 8.5 and 14.0% respectively. Consistent with their more rapid growth rate juvenile shrimp moult more frequently than adults and if shell disease is superficial and lost at the moult a lesser incidence is to be expected in small shrimp. Disease incidence was the same in both sexes, even though the females live longer and grow larger. Equal incidence of disease was also found in ovigerous and non-ovigerous females despite the smaller size and more frequent moult of the latter.

The incidence of shell disease amongst other marine crustacea in the Solway was lower than in the shrimp (Table II), and small species, e.g. amphipods and mysids, exhibited very low levels of disease (3.5 and 0% respectively). The shrimp *Crangon allmani* (carapace length usually not more than 11 mm) is smaller than *C. crangon*, is infrequently encountered in the inner Solway, but seasonally abundant in the outer area. Disease incidence in this species is just over half that recorded for *C. crangon* and still markedly less than the incidence observed in that proportion of the *C. crangon* population, i.e. ≤ 11 mm carapace length, which is of similar size.

Abbott and Perkins (1977) suggested that mechanical abrasion of the integument as a result of damage incurred by fishing instruments might explain the high incidence of shell disease in Solway brown shrimp. No experimental evidence was offered to support this contention, but the present study has shown that erosions can be readily induced in the exoskeleton of shrimp by mechanical abrasion although the antibiotic inhibition of chitinolysis and slow development of erosions in artificial seawater indicate the involvement of bacteria. Chan (1979) ascribed a high winter incidence of shell disease in Hawaiin freshwater shrimp, *Atya bisulcata,* to the abrasive action of high suspended solid loads in the rivers at this time producing exoskeletal damage susceptible to bacterial infection. Beam trawling for shrimp in the Solway is chiefly undertaken at low tide when the animals are concentrated in the channels of the inner area. Damage caused will be most severe at such times and enhanced by riddling of the catch to remove, and return to the sea, undersize individuals and bycatch. The smallest sizes pass through net and riddle most easily and this may explain the lesser incidence of disease amongst smaller size groupings. *Crangon allmani* is infrequently found on the brown shrimp fishing

grounds and is consequently rarely exposed to this form of damage. Although much fishing is undertaken in the outer area it is for benthic fish species using otter trawls of relatively large mesh size compared to the shrimp nets used in the inner area. Small crustacea will pass through these nets easily. Even larger species, such as swimming crab (*Macropipus holsatus*) and masked crab (*Corystes cassivelaunus*), while abundant on the otter trawling grounds are rarely included in the catch. These two species display shell disease incidence of 4.3 and 5.6% respectively. Most of the *Corystes* affected were male and damage was mainly confined to the chelae suggesting it may have resulted from fighting. The wounds observed were identical in nature to those reported from the fiddler crab (*Uca burgesii Holthuis) by Jones (1980) and ascribed to this cause. Carcinus maenas* is the only other crustacean species commonly taken in shrimp trawl catches. This species exhibits the second highest incidence of shell disease (8.7%) in the Solway, it was also the only truly intertidal species examined. This habitat is notably more severe than the sublittoral and its inhabitants are subject to a greater variety of stress, abrasion and general damage. A population of *Carcinus* confined in a metal tank on the beach at Garelochhead, Firth of Clyde, exhibited a higher incidence of shell disease (82.2%) than those taken from the open shore (41.4% $\lambda^2 = 82.7$ $p = 0.001$) (Perkins, unpublished data). The only difference in these two situations was in density of animals present and amount of shelter available.

When a diseased state exists amongst a population exposed to waste effluents it is very tempting to propose that the condition is causally related to pollution of the environment. The connection between chemical contamination and the induction or potentiation of disease is an entirely logical one. Laboratory studies have demonstrated the induction of viral disease (Friend and Trainer, 1970), potentiation of epidermal ulcers (Pippy and Hare, 1969; Rødsaether *et al.*, 1975), structural change (Couch *et al.*, 1977) and carcinogensis (Ishio *et al.*, 1971; Fries and Tripp, 1976). Conversely, Abbott and Perkins (1977) proposed the possibility of an antibiotic effect of, for example, low concentrations of heavy metals reducing disease incidence. Field studies are few and sometimes confused and/or contradictory, thus the maximum incidence of shell disease in *C. crangon* observed by Schlotfeldt (1972), in the course of an industrial waste investigation in the North Sea, i.e. 8.9% was only slightly greater than the minimum found by Abbott and Perkins (1977) for the same species in the Solway Firth. Gopalan and Young found an incidence of 7–34% of shell disease in *Crangon septemspinosa* taken from areas, off the American Atlantic coast, which have been described as moderately to severely polluted. Few affected individuals were encountered in allegedly non-polluted habitats, but only 248 shrimp from non-polluted areas were

examined, while single samples from the other stations were collected at different times over a full 12 months making direct comparison impossible.

The only significant imput of sewage and industrial effluents to the Solway Firth occurs along the industrialized English coast between Maryport and Whitehaven. The mean size and incidence of shell disease amongst brown shrimp taken from the spawning grounds and the industrialized area of Saltom Bay are compared in Table III for the 4 months in which samples were taken concurrently in both areas during 1980. There is a very close similarity in disease incidence at sites both near to and far removed from industrial inputs. Solway shrimp are very mobile, however, and have been shown to undertake pronounced seasonal migration (Abbott and Perkins, 1977). These results might indicate a greater degree of mobility than previously imagined.

TABLE III

Comparison of body size and incidence of shell disease amongst brown shrimp taken from the spawning gravels of Robin Rigg and Dumroof Bank and the industrialized coast of the Solway Firth in 1980

	Spawning grounds		Industralized coast*	
Month	Mean carapace lenght (mm)	% Incidence of shell disease	Mean carapace length (mm)	% Incidence of shell disease
February	10.4	17	9.1	19
April	9.7	18	9.7	16
October	9.2	16	9.4	19
November	8.3	21	9.0	16
Mean	9.4	18	9.3	17.5

* Ismay and Perkins (1981)

No evidence was found to suggest that any effluent input into the Solway was an active agent in the induction of crustacean shell disease. Low levels of disease amongst species abundant on the industrialized coast and the observed seasonal variation in disease incidence in the most intensively studied species, brown shrimp, both suggest another cause. Seasonal variation in disease incidence amongst brown shrimp is difficult to explain in terms of fluctuating physico-chemical conditions. Maximum disease incidence does occur in winter, however, when temperature and, consequently, frequency of moulting are both at a minimum. The Solway is a vertically homogeneous estuary well mixed as far upstream as Silloth (Perkins *et al.*, 1963) and no other explanations in terms of physico-chemical gradients are obviously discernible.

This study has shown that chitinolysis of the shrimp exoskeleton, even in very large erosions, is superficial and that all damage is lost at the moult. This process coupled with a 3–4 month respite from fishing pressure might be expected to produce a minimum in the incidence of erosions amongst the shrimp population at the beginning of the fishing season if damage caused by fishing gear is the primary mechanism in erosion development. This is precisely what happens in the Solway. Fishing ceases in winter when the weather is worst and shrimp are dispersed seawards. Disease incidence at this time is high, but falls away to a minimum at the start of the fishery in March.

Some of the evidence presented above is circumstantial, but, the whole, viewed in relation to other investigations in this field, constitutes a reasonable basis for proposing that the high incidence of shell disease in Solway brown shrimp is caused primarily by the activity of an intensive fishery for the species. While it would be interesting to establish firmly the nature and identity of the causative organisms the condition does not seem to be of sufficient significance to warrant more detailed study. Even the most extensive damage observed was only superficial and lost at the moult. No evidence was found to support the supposition that the condition is a significant cause of mortality or a factor adversely affecting the appearance or palability of the shrimp catch.

This investigation illustrates the need for detailed study of a situation before any causal relationship between effluent input and disease incidence is proposed. In the present case the high incidence of shell disease amongst shrimp might well be indicative of the general well being of the Solway system. If the environment were polluted shrimp, a notably sensitive species, would presumably not be so abundant. A fishery, if viable, would not be so intensive and a diminished incidence of shell disease would result if the major cause is damage done by fishing instruments.

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