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Shell Disease among Red Crabs Inhabiting Submarine Canyons of the New York Bight

**U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
National Marine Fisheries Service
Northeast Fisheries Center
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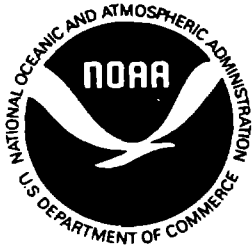
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Shell Disease among Red Crabs Inhabiting Submarine Canyons of the New York Bight

Randall R. Young

Waste Mgt. Inst., State Univ. of New York at Stony Brook, Stony Brook, NY 11794

U. S. DEPARTMENT OF COMMERCE

Robert A. Mosbacher, Secretary

National Oceanic and Atmospheric Administration

John A. Knauss, Administrator

National Marine Fisheries Service

James E. Douglas, Jr., Acting Assistant Administrator for Fisheries

Northeast Fisheries Center

Woods Hole, Massachusetts

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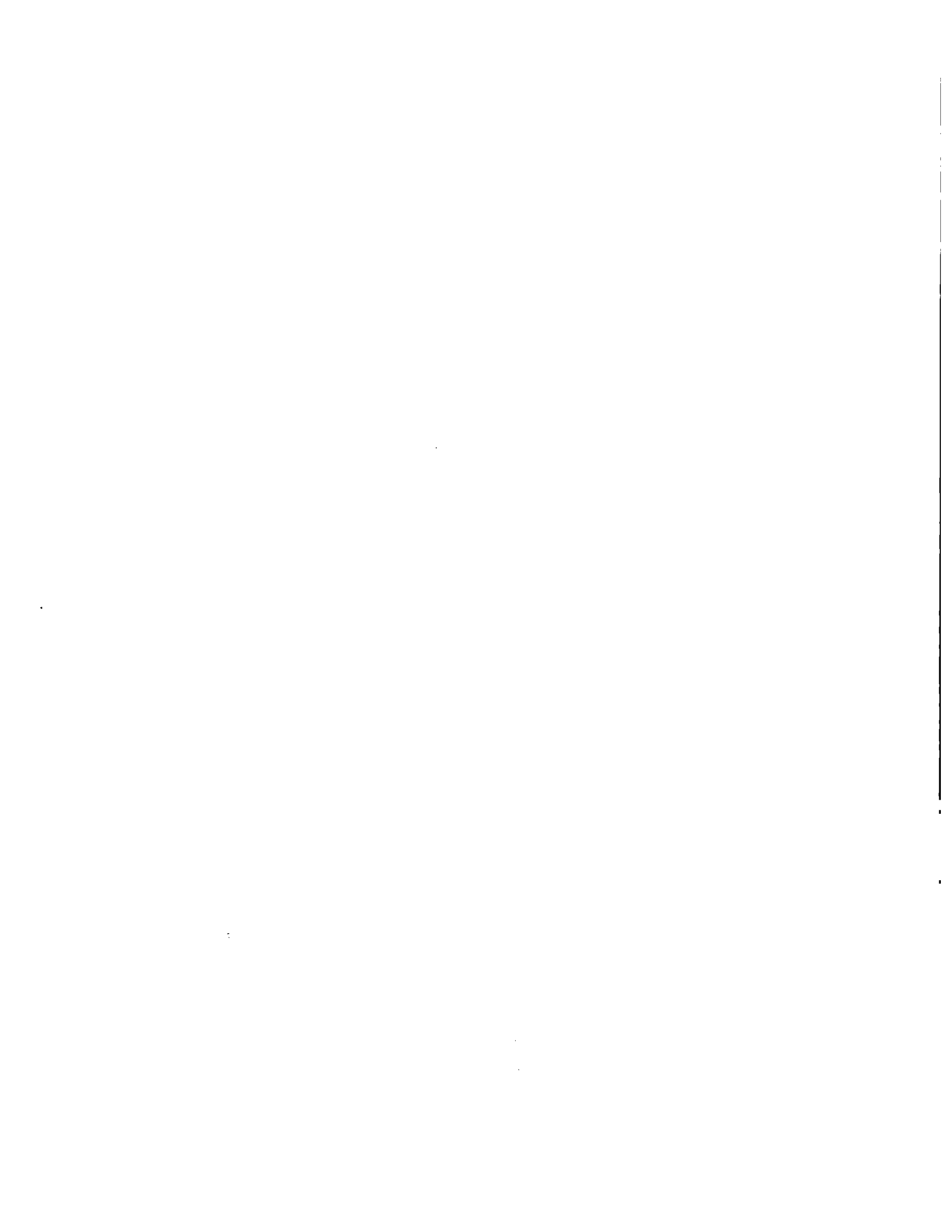


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ABSTRACT

This study was undertaken to assess the extent and severity of shell disease among New York Bight deepsea red crabs (*Geryon quinquedens*). The shell disease syndrome is a contagious condition which occurs among many crustacean species and habitats, and is caused by a variety of chitinoclastic bacteria and fungi.

The severity of shell disease among red crab samples collected from three submarine canyons was assessed by numerically rating each individual according to predetermined criteria. Some live specimens were maintained in aquaria aboard ship for subsequent bacterial culture experiments. Additional East Coast specimens dating back as far as 1884 and maintained in the Smithsonian Institution crustacean collection were also examined and rated in the same manner.

Crabs from the three canyons sampled, Hudson, Block, and Atlantis, had overall disease prevalences of 92, 92, and 86 percent, respectively. Of the Hudson Canyon specimens, 13 percent were rated as moderately to severely diseased. Thirty percent of the Block Canyon crabs and 19 percent of Atlantis Canyon crabs were moderately to severely diseased. Disease prevalences among Smithsonian Institution specimens ranged from 69 to 100 percent. The shell disease among the 1884 Hudson Canyon sample was significantly less severe than the modern samples.

The appearance of the shell disease ranged from very small black spots to large grey to black patches covering a substantial portion of the carapace. The disease was often found in a bilaterally symmetric arrangement. Two explanations are offered to explain how shell disease might spread in this manner: (1) the protective epicuticle covering corresponding body parts on each side of the animal are equally abraded by sediment, rocks, or other body parts, exposing the chitinous layers beneath to chitinoclastic microorganisms; and (2) these microorganisms may gain access to the underlying chitinous layers of the integument through pores which are arranged bilaterally.

Affected animals can rid themselves of shell disease by molting. There seems to be a positive correlation between animal size and disease severity. This may be because longer intermolt periods among older individuals allow more time for chitinoclastic organisms to spread through the integument of their host.

INTRODUCTION

SCOPE OF PROJECT

Shellfishermen working the waters off the continental shelf near the New York Bight have complained recently that an unreasonably high percentage of their crab and lobster catches was afflicted with shell disease which they attribute to the disposal of municipal sewage sludge at the 106-Mile Dumpsite. The New York Bight, shown in Figure 1, is the area extending from Cape May, New Jersey, to Montauk Point, Long Island, and seaward about 190 km to the continental shelf break. This study was undertaken to quantify the prevalence and severity of shell disease among deepsea red crabs, *Geryon quinquedens*, inhabiting this region and to begin a data base that can be used in further study of this phenomenon in relation to ocean waste disposal.

NATURE OF SHELL DISEASE

The condition has been known variously as spot disease, burned spot disease, rust disease, or, most commonly, shell disease. Rosen (1970) included an informative review on the subject in his study. It is characterized by "a progressive chitinolysis and necrosis of the exoskeleton of aquatic crustaceans." Although no single organism has been universally regarded as the causative agent, investigators have isolated a variety of chitinoclastic bacteria and

fungi in shell lesions of numerous crustacean species (Cook and Lofton 1973; Fisher *et al.* 1978; Murchelano 1982).

That more than one agent causes shell disease is further suggested by the fact that there are reports of the occurrence of the malady in a wide variety of environments. It has been found in habitats including bog ponds, lakes, rivers, estuaries, and oceanic littorals, and in climatic conditions ranging from ice-covered lakes in winter to semitropical estuaries in summer (Rosen 1970). Furthermore, two independent investigations have implicated the same three genera of chitinoclastic bacteria in shell-diseased crustaceans. Cook and Lofton (1973) isolated several species of bacteria belonging to the genera *Beneckea*, *Pseudomonas*, and *Vibrio* in lesions of *Penaeus* shrimp and blue crabs (*Callinectes sapidus*). Malloy (1978) found the same bacteria present among afflicted American lobsters (*Homarus americanus*). It should be noted that the species belonging to the genus *Beneckea* have since been included in the genus *Vibrio*. Other bacterial isolates implicated as being responsible for shell disease include those from the genera *Aeromonas*, *Spirillum*, and *Flavobacterium* (Lightner 1988).

Chitinoclastic bacteria of this type are ubiquitous in the marine environment. However, crustaceans are not defenseless against attack. Their integument is composed of four layers, only the bottom three of which are chitinous. The topmost layer, the epicuticle, is made up largely of lipoproteins rather than chitin (Warner 1977) and offers some protection from these pathogens.

The syndrome is generally restricted to the exoskeleton only, and tends to spread parallel to the shell rather than

into it (Rosen 1967) although Young and Pearce (1975) found that epidermal tissues of American lobster specimens were also necrotic. Regardless of the depth that the original lesions attain, however, they may provide an entry point for other organisms to invade the epidermal layer.

Many investigators have reported that the condition is definitely contagious. Both Rosen (1970) and Fisher (1988) have affirmed that shell disease is contagious among lobsters (*H. americanus* and *H. vulgaris*), Gopalan and Young (1975) demonstrated that it is communicable among seven-spine bay shrimp (*Crangon septemspinosa* Say), and Sindermann (1988) contended that it is infectious among blue crabs.

Mortalities in adults due directly to shell disease are infrequent (Rosen 1970; Fisher *et al.* 1978). Death may occur in extremely diseased individuals at ecdysis resulting from cohesion of the exoskeleton and the subskeleton at the sites of infection. Melanization, occurring due to a normal defensive response to block the progress of the infection, causes the shell layers to bind together, sometimes trapping the animal in its own exuvium. Infection of the gills may also increase mortality by reducing the effective respiratory surface area. Furthermore, shell disease may indirectly increase mortality by providing a portal of entry to other pathogens.

MATERIALS AND METHODS

Samples were collected aboard the NOAA ship *Albatross IV* on June 29 and 30, 1988, from the vicinities of three submarine canyons (Figure 1) along the continental shelf edge near the New York Bight. Table 1 gives the exact sampling locations and depths. Thirty-minute bottom trawls were made at 2-2.5 knots (3.7-4.6 km/hr) using a 3/4-size try-net apparatus. As many specimens as possible were examined immediately upon capture for signs of shell disease. A sketch of each animal was made, indicating the affected areas of the body, and a disease severity rating was assigned to each individual using the criteria listed in Table 2. The severity ratings range from one to five, with a "1" indicating that no shell disease was visible to the naked eye, and a "5" signifying a severely diseased individual. A zero rating was not used to describe individuals lacking apparent signs of shell disease because the initial stages of shell disease are visible only through a microscope, and a zero rating might give the erroneous impression that the animal is absolutely free of any shell disease. Carapace width, sex, and shell texture were also recorded. All crabs that could not be inspected promptly were bagged and placed in a deep freezer aboard ship for later examination. The total numbers of crabs examined from Hudson, Block, and Atlantis Canyons were 202, 77, and 110, respectively.

Several diseased specimens from each collection site were kept alive in fiberglass holding tanks for the duration of the cruise. A separate, covered tank was maintained for

● = Albatross IV samples

▲ = Smithsonian Institution samples

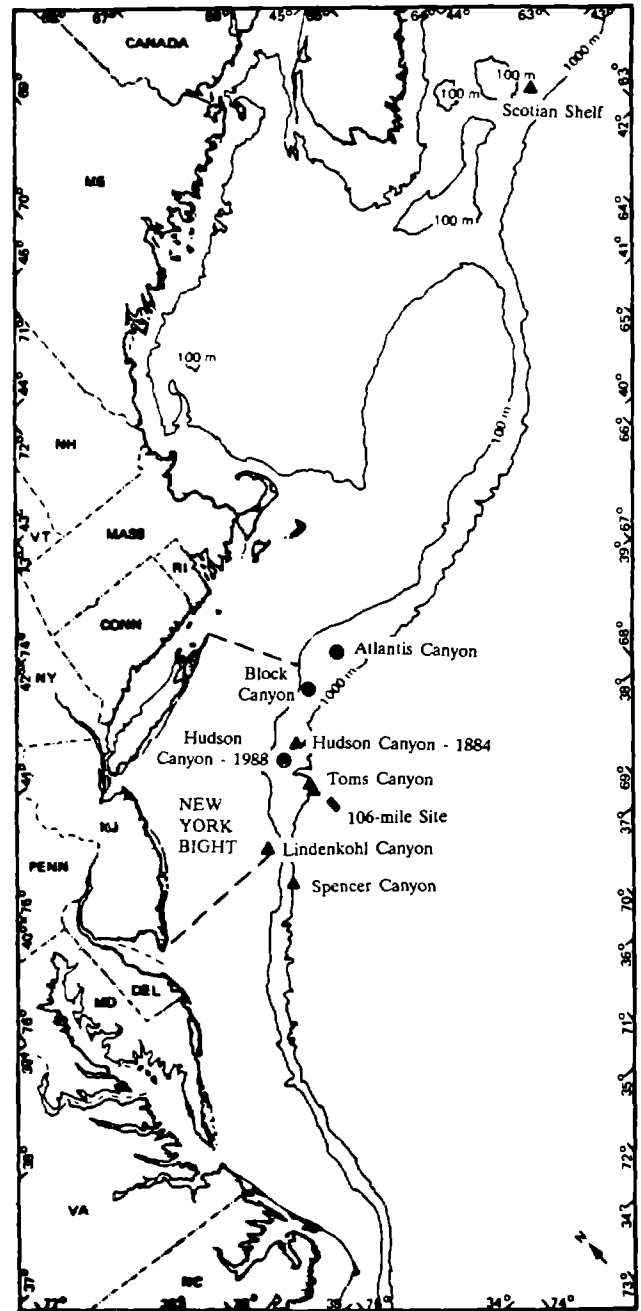


Figure 1. Location map (darkened circles denote sites of *Albatross IV* samples; darkened triangles denote sites of Smithsonian Institution samples).

each site to eliminate transmission of pathogens among members of different populations. To reduce stress to the animals, the water was aerated and the temperature kept between 15° and 20°C by floating sealed bags of ice on the

Table 1. Trawl locations and depth

| Date | Location | Latitude | Longitude | Average Depth (m) |
|---------|-----------------|----------|-----------|-------------------|
| 6-29-88 | Hudson Canyon | 39°28'N | 72°07'W | 450 |
| 6-30-88 | Block Canyon | 39°54'N | 71°19'W | 542 |
| 6-30-88 | Atlantis Canyon | 39°58'N | 70°14'W | 640 |

water surface. These specimens were delivered to the Marine Biological Laboratory in Woods Hole, Massachusetts, for pathological examinations and culture experiments.

Because this initial cruise did not afford the opportunity to collect samples from any other areas, additional specimens from the crustacean collection at the Smithsonian Institution in Washington, D.C., were subsequently examined. These specimens were inspected and rated in the same manner as described above. Forty-eight specimens collected near the Hudson Canyon (Figure 1) during the summer of 1884 aboard the *Albatross* were examined. Seven additional specimens collected from Toms Canyon to Spencer Canyon aboard the same vessel in the autumn of that year were also inspected. Two more recent small samples were also included in the investigation. The first of these was a sample of 13 collected in 1976 and 1977 near the Lindenkohl Canyon. The other sample consisted of five individuals caught south of Nova Scotia, Canada, in 1981 and 1985. Information concerning the collection of these specimens is contained in Table 3.

RESULTS

Although shell disease was present in each of the three populations sampled from the canyons, most of the individuals were only slightly affected. Comparatively few were moderately affected and fewer still showed signs of advanced disease. Disease prevalences in these samples are presented in Table 4 and the size distribution among the three sites is illustrated in Figure 2.

At the Hudson Canyon site, 92 percent (185/202) of the red crabs examined exhibited at least some degree of shell disease. This is displayed graphically in Figure 3. Of these, 78 percent were very slightly or slightly affected, 8 percent moderately affected, and 5 percent severely diseased. Of the individuals assigned a disease severity rating of "4" or "5," 85 percent had a carapace width of 9.0 cm or greater. Figure 4 indicates the percentage of individuals within given size classes belonging to each assigned severity rating. Bottom temperature near the Hudson Canyon station was 12°C.

The prevalence at the Block Canyon location (Figures 5 and 6) was 92 percent (71/77), with 62 percent rated a "2"

Table 2. Criteria for disease severity ratings

| Rating Number | Disease Severity | Criteria |
|---------------|------------------|-------------------------------------------------------------------------------------------|
| 1 | Imperceptible | Absence of visible spots |
| 2 | Very slight | <10 small spots or only small light grey patches |
| 3 | Slight | >10 small spots, but <10% of body blackened |
| 4 | Moderate | Large areas affected, 10-50% of body |
| 5 | Severe | Blackening over >50% of body; or open lesions present; or old, blackened amputation sites |

Table 3. Collection information for Smithsonian Institution specimens

| Catalog Number | Collection Date | Location | | Average Depth (m) |
|-----------------------------------|--------------------|----------|-----------|-------------------|
| | | Latitude | Longitude | |
| Hudson Canyon | | | | |
| 08000 | 7-23-1884 | 39°30'N | 71°50'W | 944 |
| Toms Canyon-Spencer Canyon | | | | |
| 14369 | 9-12-1884 | 38°36'N | 73°06'W | 1152 |
| 14370 | 9-13-1884 | 39°09'N | 72°03'W | 1481 |
| 14371 | 9-13-1884 | 39°12'N | 72°03'W | 1292 |
| Lindenkohl Canyon | | | | |
| 185424 | 6-20-1976 | 38°55'N | 73°00'W | 400 |
| 185433 | 9-12-1977 | | | |
| Scotian Shelf | | | | |
| Acc. No. 364842 | 7-15-1981 & 1-1985 | 42°50'N | 63°50'W | 550 |

Table 4. Prevalence of shell disease among red crabs from canyons adjacent to the New York Bight -- June 1988

| Severity Rating | Hudson Canyon | Block Canyon | Atlantis Canyon |
|-----------------|---------------|--------------|-----------------|
| 1 | 17 | 6 | 10 |
| 2 | 77 | 15 | 31 |
| 3 | 81 | 33 | 48 |
| 4 | 16 | 13 | 16 |
| 5 | 11 | 10 | 5 |
| Totals | 202 | 77 | 110 |

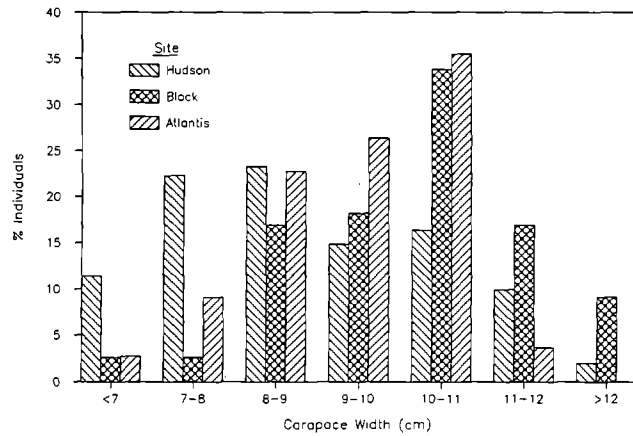


Figure 2. Size distribution of individuals from each canyon site.

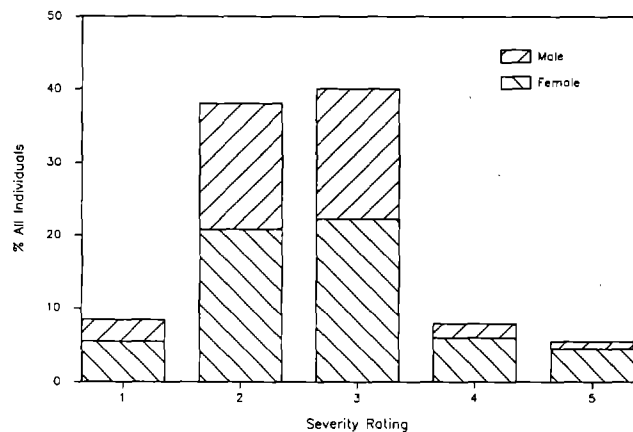


Figure 3. Shell disease prevalence and severity in Hudson Canyon.

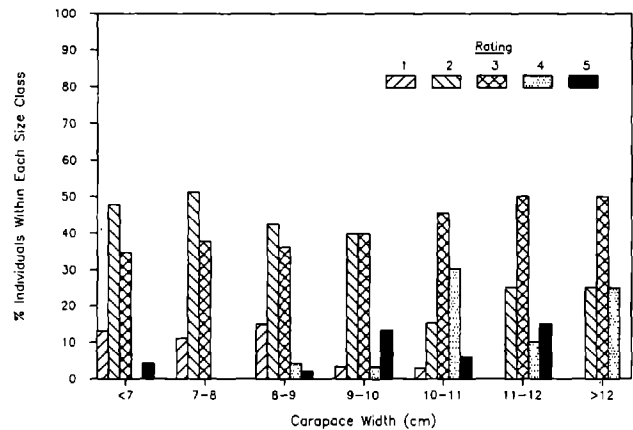


Figure 4. Size-percent frequency distribution in Hudson Canyon.

or "3", 17 percent assigned a "4" rating, and 13 percent given a "5" rating. All of the crabs rated moderately or severely affected measured 9.0 cm wide or more. Bottom temperature at this site was 6°C.

Ninety-one percent (100/110) of the Atlantis Canyon

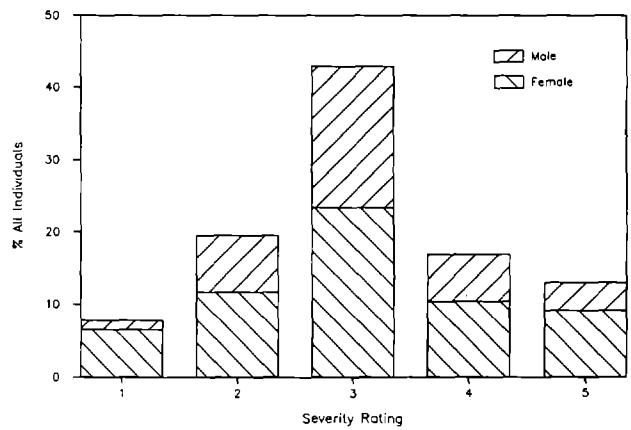


Figure 5. Shell disease prevalence and severity in Block Canyon.

specimens showed signs of shell disease (Figures 7 and 8). Seventy-two percent of the individuals received a "2" or "3" rating, 15 percent a "4", and 5 percent a "5" rating. Of the Atlantis population that was moderately or severely affected, 86 percent were 9.0 cm or larger. Bottom temperature at Atlantis Canyon was 5°C.

Cultures of bacteria from the lesions of diseased crabs resulted in the identification of several species (Bullis *et al.* 1988), including *Vibrio alginolyticus*, *V. campbellii*, *V. fluvialis*, *Flavobacter meningosepticum*, *F. breve*, and *Escherichia coli*. Several fungi also isolated remain unidentified. Attempts to promote chitinolytic or lipolytic activity among these cultures have not been successful (Bullis, personal communication).

The prevalence and severity of shell disease among the Smithsonian Institution samples are presented in Table 5. Eighty-one (39/48) percent of the 1884 Hudson Canyon specimens showed at least some signs of shell disease. However, none of the crabs was assigned a severity rating greater than "3" (Figure 9). The 1884-Hudson Canyon crabs were significantly less severely diseased ($P < 0.01$) than were the modern Hudson Canyon specimens. In general, the disease severity increased with animal size (Figure 10). Ninety-three percent of the individuals assigned a "3" rating, the highest ascribed among this sample, were 9 cm or larger.

The Toms-Spencer sample had a 100 percent disease prevalence (7/7), with 57 percent slightly affected and 43 percent moderately to severely affected (Figure 11). The size-frequency distribution for this sample is given in Figure 12. All of the individuals in this sample had a carapace width of 9 cm or greater.

Sixty-nine percent (9/13) of the Lindenkuhl Canyon specimens exhibited shell disease (Figure 13). Of these, 54 percent fell into either the very slightly or the slightly affected categories. The other 15 percent were moderately affected. Figure 14 displays the size-frequency distribution for these specimens. All of the specimens rated moderately affected were larger than 9 cm.

At the Scotian Shelf site, 100 percent (5/5) of the crabs examined exhibited signs of shell disease (Figures 15 and 16). All of the crabs examined were slightly to moderately diseased.

DISCUSSION

APPEARANCE AND LOCATION OF DISEASE

A wide variation in the extent and appearance of shell disease was found among the red crab specimens examined. Among the *Albatross IV* samples, the appearance ranged from small black spots less than 1 mm in diameter to large patches of grey or black areas covering most of the carapace. Large grey to black patches were usually found in either the hepatic regions or the branchial regions of the carapace, but rarely in both. Scratches received from brushes with predators or rocks were always blackened. The points on the edge of the carapace as well as the spurs on the carpi were also commonly affected areas. Walking legs exhibited primarily small spots except at the joints, where larger areas of necrosis commonly occurred. Also, the sites of amputated legs seemed very susceptible to infection, with the entire basis often reduced entirely to very black necrotic tissue.

Contrary to earlier studies on blue crabs (Rosen 1967), early stages of the disease are most easily observed on the dorsal side of the red crab. The ventral side is only occasionally affected, with the most common occurrences being grey patches on the sub-hepatic or sub-branchial sections of the carapace and, less commonly, small black necrotic lesions on the abdomen or outer maxilliped.

The appearance of the crab sample collected in 1884 near the Hudson Canyon differed from any other set examined. Sixty-eight percent of the crabs either exhibited no visible sign of shell disease or were limited to only a few small spots. Those that had more than 10 spots almost always had them arranged in rows along the pores in their walking legs. Only one male specimen exhibited any of the patch-type blackening. Overall, this sample was by far the least affected and may be an example of an essentially non-impacted population.

The overall appearance of the other Smithsonian Institution specimens, including the second 1884 sample, resembled those taken aboard the *Albatross IV* in the summer of 1988.

SYMMETRY OF SHELL DISEASE

Very often, signs of shell erosion were found to be bilaterally symmetrical. This can sometimes be explained as regions receiving equal amounts of abrasion, thus equally wearing away the epicuticle and allowing infection by

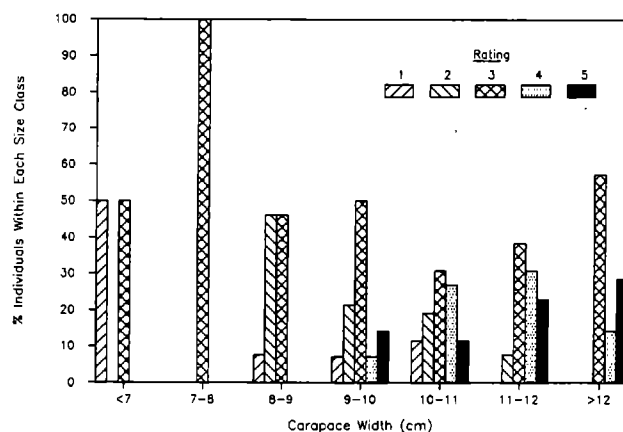


Figure 6. Size-percent frequency distribution in Block Canyon.

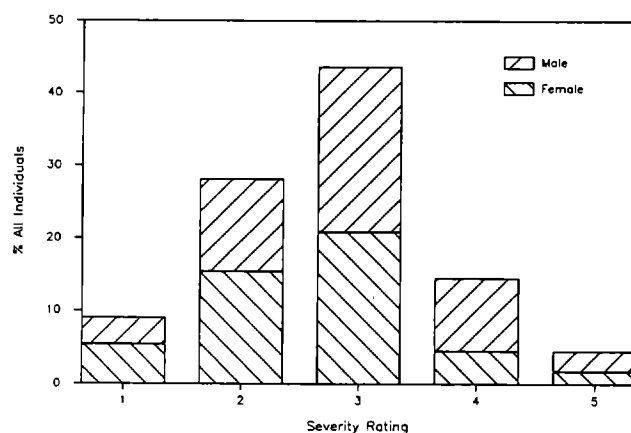


Figure 7. Shell disease prevalence and severity in Atlantis Canyon.

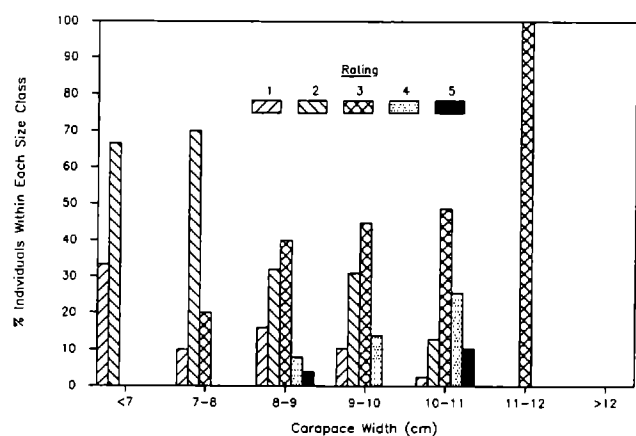


Figure 8. Size-percent frequency distribution in Atlantis Canyon.

chitinoclastic organisms. For example, necrosis of the shell tissue is sometimes observed where the ventral side of the coxae rub along the sea floor. More commonly, body parts of the animal itself which rub together were affected. For

Table 5. Prevalence of shell disease among Smithsonian Institution red crab specimens

| Severity Rating | Hudson | Toms-Spencer | Lindenkohl | Scotia |
|-----------------|-----------|--------------|------------|----------|
| 1 | 9 | 0 | 4 | 0 |
| 2 | 24 | 0 | 4 | 0 |
| 3 | 15 | 4 | 3 | 2 |
| 4 | 0 | 1 | 2 | 3 |
| 5 | 0 | 2 | 0 | 0 |
| Totals | 48 | 7 | 13 | 5 |

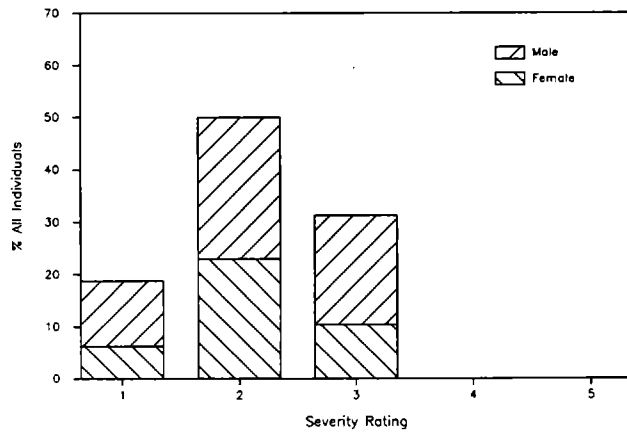


Figure 9. Shell disease prevalence and severity in Hudson Canyon, 1884.

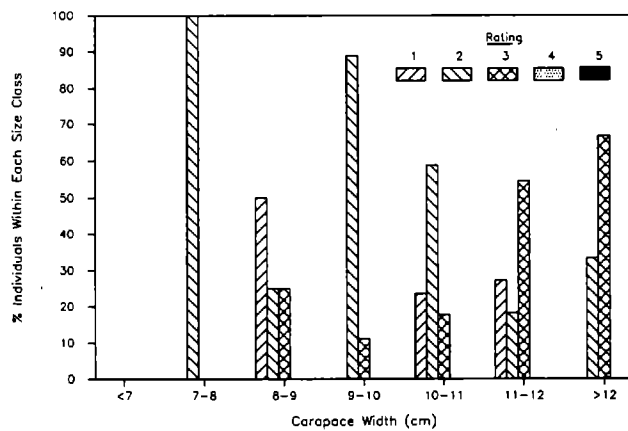


Figure 10. Size-percent frequency distribution in Hudson Canyon, 1884.

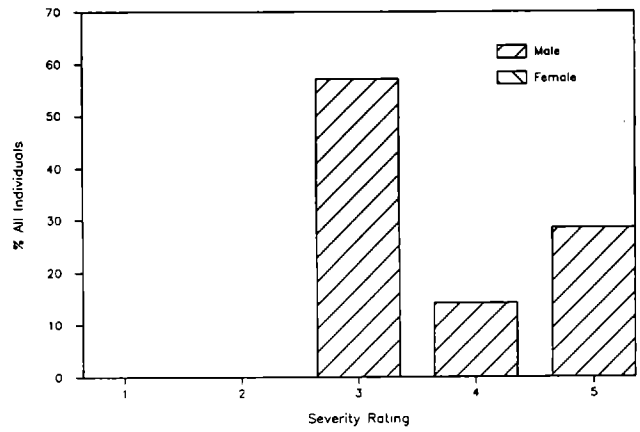


Figure 11. Shell disease prevalence and severity from Toms to Spencer Canyon, 1884.

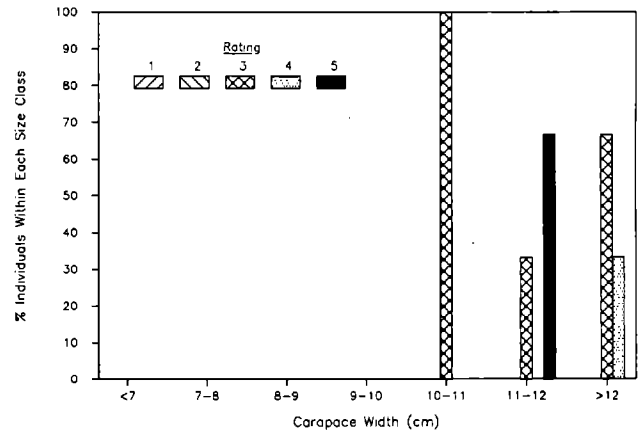


Figure 12. Size-percent frequency distribution from Toms to Spencer Canyon, 1884.

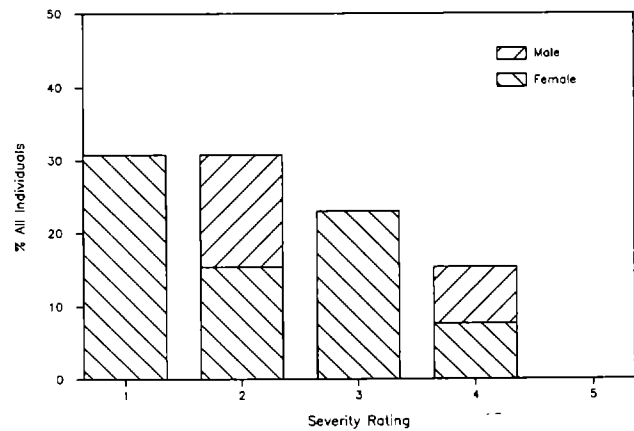


Figure 13. Shell disease prevalence and severity in Lindenkohl Canyon.

instance, where the merus of the first walking leg comes into contact with the cheliped, or where the merus of the cheliped rubs against the subhepatic region of the carapace, grey or black areas were often found. Less often, small lesions were observed at the point on the cheliped propodus where the carpus spur sometimes comes into contact.

Other examples of bilateral symmetry cannot be explained by abrasion of the epicuticle. Areas of the branchial or hepatic regions, or the points along the edge of the carapace, are often symmetrically blackened, but are not continually in contact with the sea floor or other body parts. Small spots are also common on the carapace where the branchial lobe meets the cervical groove. All of these examples suggest a second means of infection in addition to the abrasion of the epicuticle. One possibility previously discussed (Fisher *et al.* 1978) is that chitinolytic microorganisms may invade the chitinous layers of the exoskeleton through hypodermal ducts or setal pores.

Shell erosion was not, however, always bilaterally symmetrical. The infection of some nonsymmetric areas of the exoskeleton can easily be explained by abrasion or partial crushing of the shell, thus allowing access to the inner layers by chitinoclastic organisms. Other regions that had been affected nonsymmetrically had either become infected before their bilateral counterpart or the epicuticular defense was breached by another route. The chitinoclastic microorganisms may gain access to the chitinous layers with the aid of lipolytic bacteria. Non-chitinolytic bacteria which exhibited lipolytic properties were consistently isolated from shell lesions of the West Coast's true Tanner crab (*Chionoecetes tanneri*) (Fisher *et al.* 1978).

In general, bacterial genera isolated from the shell lesions agreed with several previous studies in which *Vibrio* and *Flavobacter* species were isolated. The other species identified, *E. coli*, was limited to only one specimen. As this species does not thrive under marine conditions, its presence is probably an anomaly that can be attributed to the less than ideal sampling conditions aboard ship (Bullis, personal communication).

These observations all lend support to the often raised supposition that shell disease may be initiated by more than one means and more than one pathogen (Rosen 1970; Murchelano 1982; Malloy 1978; Lightner 1988).

MOLTING AND SIZE CONSIDERATIONS

Crustaceans suffering from shell disease may overcome the affliction by achieving ecdysis, thus forming a new, uninfected shell (McLeese 1965; Rosen 1970). A comparison between hard-shelled (Figure 17) and soft-shelled (Figure 18) *Albatross IV* specimens clearly demonstrates the red crab's ability to rid itself of a diseased exoskeleton and start fresh with a new one. Nearly all of the soft-shelled individuals examined were either totally free of signs of shell disease or only very slightly affected, with only a few small spots visible. The few exceptions were

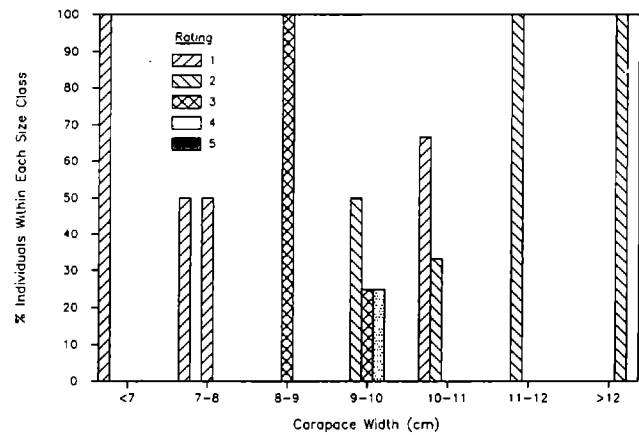


Figure 14. Size-percent frequency distribution in Lindenkolh Canyon.

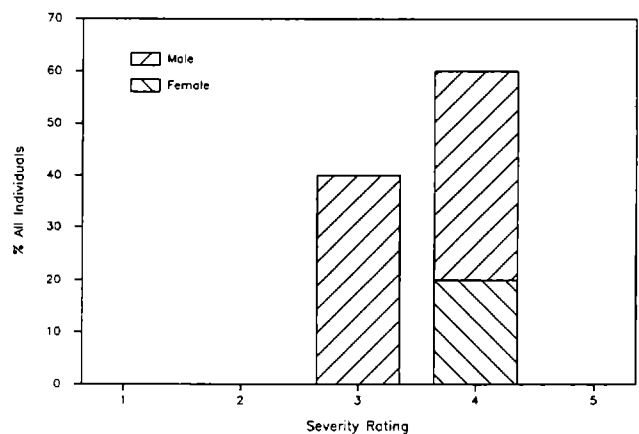


Figure 15. Shell disease prevalence and severity from the Scotian Shelf.

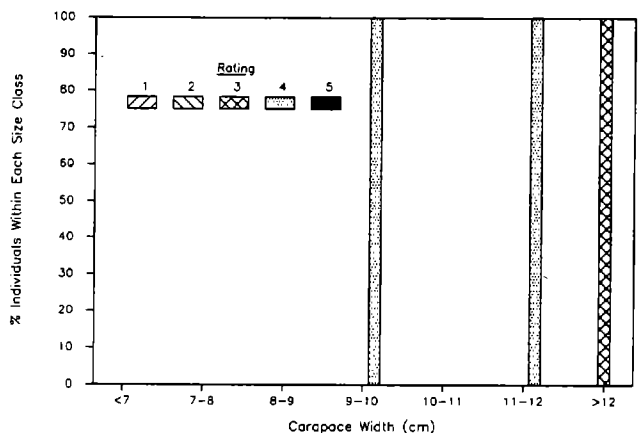


Figure 16. Size-percent frequency distribution from the Scotian Shelf.

almost all individuals which had suffered physical damage to the new shell, such as a crushed or torn area, which allowed entry of the disease-causing agents into the chitinous layers of the shell. This is not surprising, as newly molted individuals are vulnerable to damage without the protection of a hardened shell.

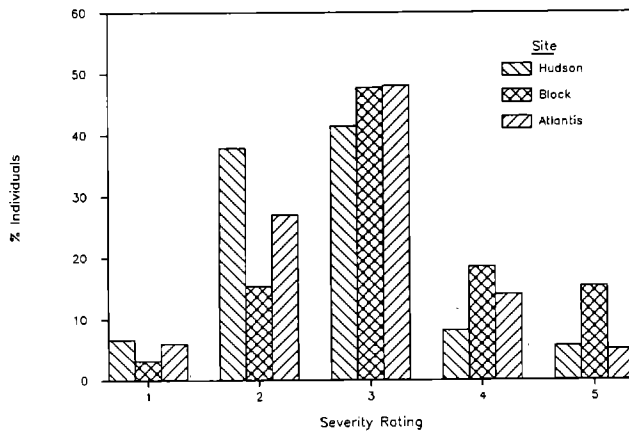


Figure 17. Disease prevalence and severity among hard-shelled crabs.

There seems to be a direct correlation between animal size and shell disease severity. Of the modern samples, the Hudson Canyon population was comprised of the smallest individuals on average, and also had the lowest mean severity rating. Block Canyon, on the other hand, had the highest percentage of larger individuals and also the highest prevalence of "4" and "5" ratings. Upon examination of the "3"-rated individuals in Figure 10, even the 1884 Hudson Canyon reference specimens show a trend of increasing disease severity among larger sized animals. This correlation is probably related to molting, since the frequency of molting decreases among larger individuals (Warner 1977). Longer intermolt periods allow more time for the chitino-clastic microorganisms to spread throughout the shell of their host.

CONCLUSION

The occurrence of shell erosion among crustaceans has been shown to be affected by many environmental factors. High population density greatly enhances the spread of the condition (McLeese and Wilder 1964; Rosen 1967). The rate at which the disease progresses is dependent upon temperature (Rosen 1970; Sindermann 1988); progression below 1°-2°C being slow and above 4°-5°C more rapid. A higher prevalence of shell disease has been shown to occur in individuals fed an inadequate diet (Fisher *et al.* 1976). Also, lobsters and crabs exposed in aquaria to sewage sludge and dredge spoils by Young and Pearce (1975) exhibited signs of shell disease. They also suggest that fouling or low oxygen levels may contribute additional, synergistic stresses to the animals. An overall degradation of the environment may possibly furnish some common chitino-clastic microorganisms the potential to become pathogenic by reducing the abilities of crustaceans to ward off infection (Rosen 1967; Young and Pearce 1975).

It is not possible to ascertain from the data provided by this study alone whether there is any connection between the ocean disposal activities at the 106-Mile Dumpsite and the incidence of shell disease among red crabs inhabiting

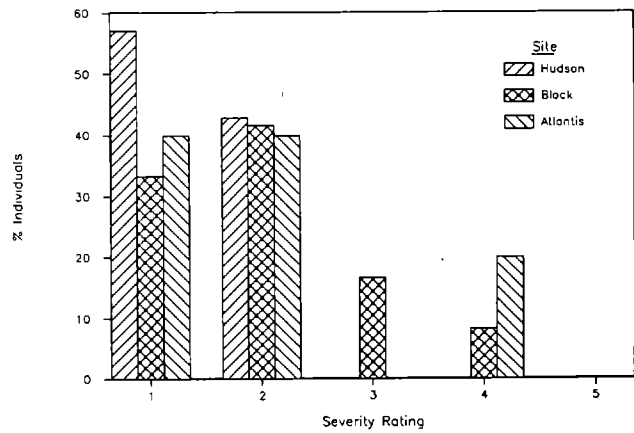


Figure 18. Disease prevalence and severity among soft-shelled crabs.

submarine canyons along the continental slope. Further studies are needed to answer questions concerning prevalences of shell disease in red crabs from other areas, the identification of the pathogens responsible for the disease, population densities of the crabs, the degree of infectiousness of the syndrome, temperature trends and their implications, and sediment characteristics. Also, further analysis of sewage sludge transport may determine whether any sludge even reaches the submarine canyons northeast of the 106-Mile Dumpsite.

However, the prevailing presence of severe shell disease among individuals captured from other areas and times suggests that the dumping of sewage sludge does not alone promote the condition. While the 1884 autumn sample collected from Toms Canyon to Spencer Canyon is unfortunately small, it nevertheless indicates the presence of shell disease near the vicinity of the present deep-water dumpsite years before the dumping of sewage sludge was initiated. The fact that these crabs were collected only a few months after the 1884 Hudson Canyon specimens suggests that either they were part of a separate population, that there are distinct seasonal differences in the prevalence of the disease, or that shell disease among red crabs may spread rapidly under certain conditions. Furthermore, the more recent specimens caught south of Nova Scotia indicate that shell disease occurs in other, distant waters as well.

A better understanding of the cause or causes of shell disease is essential. The disease may increase adult mortality enough to reduce the stock size, thus reducing the size of crab landings. In addition, although the condition may affect only the exoskeleton of crustaceans, leaving the edible tissue completely safe to consume, it nevertheless detracts from the visual appeal of the animal. Because successful marketing of both crabs and lobsters is dependent upon being able to visually attract buyers, there is the potential for a severe economic impact on the shellfisheries industry. Although red crabs are most commonly marketed as a processed meat product rather than whole, consumers may nonetheless turn away from them and other seafood products that they perceive to be unsafe.

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