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The Shell Disease Syndrome in Marine Crustaceans

U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
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The Shell Disease Syndrome in Marine Crustaceans

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ABSTRACT

The shell disease syndrome has been reported from many freshwater and marine crustaceans of economic importance. Signs of the disease syndrome include erosion and pitting of the exoskeleton, resulting from activities of chitin-destroying microorganisms--bacteria and fungi of several genera. Infection is usually limited to the exoskeleton, although underlying living tissues may be invaded by other opportunistic microbial pathogens.

Shell disease is usually relatively rare in natural populations, although a few epizootics have been reported. High prevalences occur in captive populations, especially in overcrowded holding facilities, probably because of greater likelihood of damage to the cuticle and the presence of other abnormal environmental conditions, including organic loading with consequent high bacterial populations.

Mortalities from shell disease have been observed, occasionally at high levels, in impounded populations. Destruction of gills and adhesions of the exoskeleton which prevent ecdysis have been considered to be responsible factors, as have secondary systemic infections which develop after perforation of the chitinous integument.

Shell disease has been observed in crustaceans from badly degraded coastal/estuarine waters, often associated with so-called "black gill" syndrome. Exoskeletal erosions are common in lobsters, crabs, and smaller crustaceans from areas where contaminated sediments occur, and the shell disease syndrome has been produced experimentally by exposure of normal animals to sediments from sewage sludge and dredge spoil disposal sites. Organic loading in degraded waters, with consequent increases in populations of heterotrophic bacteria--some of which may be facultatively pathogenic--may be a significant factor.

Shell erosion in Crustacea is, therefore, a particular problem in impoundments, in aquaculture facilities, and in degraded habitats. It is contagious, but its etiology is complex, involving chitinoclastic and other microorganisms and environmental stressors. It may contribute to mortality, principally by providing a route of entry for other facultative pathogens, but also by destroying the gills of crustacean hosts and by interfering with molting.

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CONTENTS

I. INTRODUCTION

Probably the most common and most widespread microbially induced disease condition in crustaceans is the presence of various kinds of exoskeletal erosions, due to the action of chitinoclastic bacteria and fungi. Microorganisms of this type are ubiquitous in the sea and are also common in freshwater (Benton, 1935; Zobell and Rittenberg, 1938; Hock, 1940, 1941; Lear, 1963; Hood and Meyers, 1974); they usually degrade the exoskeletons of dead animals, but some may affect living individuals as well. Over 30 species of chitin-destroying bacteria are known, of which half have been isolated from shells of crustaceans.

It should be emphasized at the outset that "shell disease" is really not a discrete disease entity, but instead can be best described as a "disease syndrome" (or a complex of disease syndromes) characterized by progressive exoskeletal erosion resulting from activity of chitinoclastic microorganisms--often affecting stressed crustaceans, or those with damaged exoskeletons.

Exoskeletal erosions have a variety of common names, including "shell disease" or "box burnt disease" in blue crabs, Callinectes sapidus, "shell disease" in lobsters, Homarus americanus, "rust disease" in king crabs, Paralithodes camtschatica, "brown spot," "black spot," or "burn spot" disease in shrimp and prawns, "black spot" disease in the European edible crab, Cancer pagurus, and "spot" disease in crayfish. The original names given to the disease condition in European crustaceans were "Brandfleckenkrankheit" (Mann and Pieplow, 1938) and "Happichische Fleckenkrankheit" (Schäperclaus, 1954).

For purposes of this report, information on the shell disease syndrome in marine crustaceans has been somewhat artificially divided into the following categories:

- Etiology;
- The disease in crustaceans from natural habitats;
- The disease in impounded crustacean populations;
- The disease in crustacean aquaculture; and
- The disease in crustaceans from degraded habitats

These categories are obviously not mutually exclusive, since distinctions between cultured and impounded populations may be blurred, and since differences between natural and degraded habitats may be matters of degree. Despite such shortcomings, the five subdivisions seem logical, since insights can be gained from separate considerations of each.

The literature on shell disease in crustaceans is extensive and is increasing; the first report dates back almost a century (Happich, 1900). Only a portion--but I hope a representative portion--of that literature has been summarized here (Tables 1 and 2). As can be seen in the "References" section, interest in the shell disease syndrome has shown a remarkable spurt in the past several years, principally because of reports of high prevalences in crustaceans from North American and European waters. In some instances, surveys were conducted in severely polluted estuarine/coastal areas, and a possible relationship between the disease condition and habitat degradation has been suggested--especially in the news media. In other studies, high prevalences have been found in feral and impounded populations. This document is an attempt to summarize available information about shell disease and to reach some conclusions about its etiology, environmental requirements, distribution, and effects.

II. ETIOLOGY OF THE SHELL DISEASE SYNDROME

The exoskeleton or cuticle of crustaceans is made up of a thin outer layer, the epicuticle, consisting of proteolipoidal material, including polyphenols, covering three inner chitinous layers--the exocuticle, which is pigmented and calcified, the calcified endocuticle, and the noncalcified endocuticle (Dennell, 1947, 1960). Chemically, chitin is a polysaccharide structurally similar to cellulose except that the repeating unit is derived from acetylglucosamine instead of glucose. The outer cuticular layer of the exoskeleton, the epicuticle, is biochemically inert, and shell erosion can occur when this layer is breached by abrasion, injury, or possibly enzymatic digestion, exposing the underlying chitinous layers to adequate numbers of chitin-destroying microorganisms (Stewart, 1980). Additionally, microbial invasion may occur through hypodermal ducts or setal pores (Fisher et al., 1978).

Because of the lipoidal nature of the epicuticle, microorganisms producing extracellular lipase may initiate lesions, even in the absence of abrasions. In one series of experiments, all microorganisms able to infect healthy nonabraded animals were lipolytic (Cipriani et al., 1980). The authors pointed out, however, that the exoenzymes chitinase and protease would be important in subsequent lesion development.

A variety of microorganisms has been identified from shell lesions, and it is probable that at least several kinds of bacteria and fungi may act independently, concurrently, sequentially, or synergistically in destroying chitin. In penaeid shrimp, for example, bacteria of the genera Vibrio, Aeromonas, Spirillum, and Flavobacterium and fungi of the genera Fusarium, Haliphthoros, and Atkinsiella have been isolated from erosive lesions (Lightner, 1988a). Chitin-degrading organisms

Table 1. Summary of information about the shell disease syndrome in marine crustaceans.

Geographic distribution	Known from many parts of the world, in crustacean populations from natural habitats, impoundments, aquaculture, and degraded habitats
Species affected	Reported most frequently from larger economically important species of Crustacea (lobsters, crabs, shrimp) but occurs in smaller species (such as amphipods and mysids) as well (Bogdanova, 1957)
Etiological agents	Heterotrophic microorganisms--bacteria and fungi--which have the ability to degrade chitin
Prevalences	Prevalences usually very low (< 2%) in natural populations; epizootic levels (10-90%) seen in impounded or cultured populations, or those from sewage sludge dumpsites (possibly other areas as well)
Temperature effects	Not well documented, but a direct relationship has been reported (McLeese and Wilder, 1964; Schlotfeldt, 1972)
Disease signs	Progressive erosion of exoskeleton, often beginning as brown focal lesions which increase in size and destroy extensive areas. May be accompanied in advanced cases by secondary infections of underlying living tissues

Table 1 (continued).

Disease effects	
(a) Physiological	Destruction of exoskeleton provides route of invasion for other facultative pathogens. Destruction of gill filaments reduces respiratory competence. Shell adhesions may prevent ecdysis
(b) Mortality	Reported mortalities of impounded lobsters and crabs have been attributed to gill damage, secondary infections, or failure of ecdysis
Preventive measures	Avoid overcrowding impounded populations and limit duration of impoundment. Provide adequate diet to cultured populations, limit densities in culture tanks, and reduce other forms of stress (poor water quality, hypoxia, temperature extremes)
Treatment	Oxolinic acid baths have been reported to be a successful control measure in freshwater prawn culture (El-Gamal et al., 1986). Furanace and oxytetracycline have been reported to be effective for shell disease control in penaeid shrimp culture (Lightner, 1984)

Table 2. Reported occurrences of shell disease in marine crustaceans of economic importance.

Host	Geographic location	Common name of disease	Pathogens isolated	References
LOBSTERS				
American lobster (Homarus americanus)	Nova Scotia	Shell disease	Presumptive vibrios	Hess, 1937
"	Nova Scotia	"	Pseudomonas, Vibrio	Malloy, 1978
"	Maine	"	Not reported	Taylor, 1948, 1949; Sawyer & Taylor, 1949
"	Norway	"	Vibrio, Aeromonas, Pseudomonas	Roald et al., 1981
"	California (culture facility)	"	Not reported	Fisher et al., 1976
"	New York Bight	"	Not reported	Pearce, 1971; Young & Pearce, 1975
"	Massachusetts coast	"	Not reported	Estrella, 1984

Table 2 (continued).

Host	Geographic location	Common name of disease	Pathogens isolated	References
European lobster (*Homarus gammarus*)	U.K.	Fungal shell disease	*Fusarium* sp.	Alderman, 1987
"	Italy (aquaria)	"	*Fusarium* (*Raumularia branchialis*), (Fungi Imperfecti)	Sordi, 1958
"	Norway	Shell disease	*Vibrio*, *Aeromonas*, *Pseudomonas*	Roald et al., 1981
European "crawfish" (*Palinurus elephas*)	U.K.	Fungal shell disease	(Fungi Imperfecti)	Alderman, 1973
Spiny lobster (*Palinurus vulgaris*)	Italy	"	*Didymaria palinuri* (Fungi Imperfecti)	Sordi, 1958
"	Irish Sea	Burn spot	(Fungi Imperfecti)	Alderman, 1971, 1976
Spiny lobster (*Panulirus argus*)	Florida	Shell disease	Not reported	Robinson & Dimitriou, 1963
Spotted spiny lobster (*Panulirus guttatus*)	Florida	"	Not reported	Iversen & Beardsley, 1976

Table 2 (continued).

Host	Geographic location	Common name of disease	Pathogens isolated	References
CRABS				
Blue crab (*Callinectes sapidus*)	Louisiana, Mississippi	Shell disease	*Vibrio*, *Pseudomonas*	Overstreet & Cook, 1972; Cook & Lofton, 1973; Overstreet, 1978
"	Florida	"	Not reported	Iversen & Beardsley, 1976
"	South Carolina	"	Not reported	Sandifer & Eldridge, 1974
"	Chesapeake Bay	"	Chitino-clastic bacteria	Rosen, 1967, 1970
Shore crab (*Cancer pagurus*)	Southwest coast of Ireland; England	Black spot shell disease; shell necrosis; black necrosis; burn spot disease	Not reported	Schäperclaus, 1935; Gordon, 1966; Bakke, 1973; Ayres & Edwards, 1982
"	Norway	Shell disease	Not reported	Bakke, 1973
Rock crab (*Cancer irroratus*)	Northeast coast of U.S.	Shell blackening, carapace erosion	Not reported	Sawyer, 1982
Stone crab (*Menippe mercenaria*)	Florida	Shell disease	Not reported	Iversen & Beardsley, 1976

Table 2 (continued).

Host	Geographic location	Common name of disease	Pathogens isolated	References
King crab (*Paralithodes camtschatica*) and blue king crab (*Paralithodes platypus*)	Eastern North Pacific	Rust disease	Chitinoclastic bacteria	Bright et al., 1960[2]
Tanner crab (*Chionoecetes tanneri*)	Oregon coast (deep water)	Shell disease	*Photobacterium* sp.	Baross & Tester, 1975; Baross et al., 1978
Red crab (*Geryon quinquedens*)	Submarine canyons of New York Bight	"	Not reported	Young, 1988
Pacific crab (*Mursia gaudichaudii*)	California coast	Exoskeletal lesions resembling "burn spot disease" of European crabs	Not reported	Mearns, 1973
Dungeness crab (*Cancer magister*)	Yaquina Bay, Oregon	Exoskeletal lesions	*Vibrio anguillarum*	Baross et al., 1978
SHRIMP				
European brown shrimp (*Crangon crangon*)	Germany	Black spot disease	Not reported	Meixner, 1969a, b
"	U.K.	Black necrosis	Not reported	Schlotfeldt, 1972; Abbott, 1977

Table 2 (continued).

Host	Geographic location	Common name of disease	Pathogens isolated	References
Shrimp (*Crangon septemspinosa*)	Northeastern U.S. coast	Shell disease	Not reported	Gopalan & Young, 1975
Pacific white shrimp (*Penaeus occidentalis*); white shrimp (*Penaeus setiferus*)	Florida (in culture ponds)	"	Not reported	Cook & Lofton, 1973; Iversen & Beardsley, 1976
Penaeus spp.	Ubiquitous	Bacterial shell disease (brown spot)	*Vibrio*, *Aeromonas*, *Spirillum*, *Pseudomonas*, *Flavo-bacterium*	Cook & Lofton, 1973; Couch, 1978; Cipriani et al., 1980; Lightner, 1988a
Penaeus spp.	Ubiquitous in culture environments	Fungal shell disease (associated with a disease condition known as "black gill" in *Penaeus japonicus*) (Kuruma prawn)	*Fusarium* spp., especially *F. solani* (other fungi such as *Haliphthoros* sp. and *Atkinsiella dubia* may be isolated--but rarely)	Lightner, 1975, 1988a; Lightner et al., 1979b; Solangi & Lightner, 1976
English prawn (*Palaemon serratus*)	U.K., France	Brown spot disease	Chitino-clastic bacteria	Anderson & Conroy, 1968; Campillo, 1976

are a normal part of the aquatic environment, and they can be found on the body surfaces and in the shell pores of crustaceans. Some, such as Vibrio parahaemolyticus adsorb readily to chitin, where they may form microcolonies which degrade and utilize the substrate (Kaneko and Colwell, 1973, 1975). Chitinoclastic microorganisms may become opportunistic pathogens of stressed or damaged individuals, and may increase in numbers dramatically in aquaculture facilities or in polluted waters. Participating microorganisms have been shown to produce extracellular lipases, chitinases, and proteases (Lightner, 1988a).

In earlier studies with European freshwater crustaceans, several fungi (including Ramularia astaci, Septocylindrium eriocheir, and Didymaria cambari) were isolated from shell lesions, and were thought to be the primary etiological agents of shell disease--called "Brandfleckenkrankheit" (Mann and Pieplow, 1938; Unestam, 1973), whereas in North America, beginning with the studies of Hess (1937) on shell disease in lobsters, bacteria have been reported as the usual causes of the syndrome (Rosen, 1970; Amborski et al., 1975; Baross et al., 1978; Malloy, 1978). Fungi are not uncommon, however, in exoskeletal lesions of marine or freshwater crustaceans; they may be primary or secondary invaders (Alderman, 1973; Burns et al., 1979; Johnson, 1983; Alderman, 1987). Some recent publications on shell disease in Crustacea have discussed available information on the basis of two major subcategories: "fungal shell disease" and "bacterial shell disease" (Johnson, 1983; Alderman, 1987; Austin and Alderman, 1987). This may be a useful descriptive device, but the precise etiology of shell disease is in too many cases uncertain enough to discourage such an exclusive categorization (at least in this document). A succinct description of the participants in the shell disease syndrome was given by Cipriani et al. (1980). "The lesion [exoskeletal erosion] appears to be a microenvironment in which several microbial taxa, producing a variety of degradative exoenzymes, interact to create the disease signs."

One possible key to outbreaks of shell disease syndrome was pointed out by Fisher et al. (1976), Fisher et al. (1978), and Malloy (1978)--that maintenance and repair of the epicuticle was critical in reducing the occurrence of exoskeletal erosions, and that stressors such as inadequate nutrition, temperature extremes, or onset of ecdysis resulted in higher levels of the disease condition.

Another insight of possible importance is that some conditions referred to as "shell disease" might have a more direct relationship to environmental pollutants, and might not be related at all to effects of chitinoclastic microorganisms. As an example, recent use of insect growth regulators, a class of less persistent pesticides which includes diflubenzuron (commercial name, Dimlin), could result in contamination of

coastal/estuarine waters and damage to non-target crustaceans by interference with chitin synthesis. Evidence for this possibility has been provided by recent experimental studies. One of the reported effects of exposure of fiddler crabs, Uca pugilator, to diflubenzuron was blackened lesions on regenerating appendages--areas that were "soft to the touch" (Weis et al., 1987). The authors suggested that the lesions may reflect deficiency in chitin formation, but they did note a resemblance to "shell disease" as described from crabs and lobsters in the New York Bight by Young and Pearce (1975). Another experimental study, in which barnacles, Balanus eburneus, were exposed to diflubenzuron disclosed lesions in the form of "cuticular disruptions" in cast exoskeletons (Gulka et al., 1982). In some specimens both the exocuticle and the endocuticle were disrupted; cast exuvia of diflubenzuron-treated animals were consistently more fragile; and molting was frequently inhibited. Since in both these studies the lesions appeared very soon after molting, mechanisms other than chitin destruction by microorganisms were probably operative--mechanisms which might include interference by pollutants in the biochemistry of cuticle formation, or in energy transfers within the organism. It seems possible that other less specific pollutants could act in similar ways.

A final cautionary note seems pertinent to this discussion of the etiology of shell disease. Crustaceans, particularly those that have just molted, are vulnerable to injuries from contacts with predators or other members of the species--injuries which may take the form of losses of spines or damage to larger portions of the carapace. Such mechanical losses may sometimes mimic the effects of shell disease (and may, of course, act as portals of entry for pathogens).

To summarize information about etiology, the shell disease syndrome may be the result of:

- Chitinolytic, lipolytic, and proteolytic activity of microorganisms--principally bacteria and fungi--acting alone or in combination on injured or intact cuticle;
- Interference with chitin synthesis, by inadequate nutrition or by biochemical effects of pollutants such as certain pesticides with specific activities; and
- Activity of stressors in the environment--especially high population densities, abnormal temperatures and salinities, presence of toxic chemicals, and hypoxia--acting to disturb metabolic processes and internal defense mechanisms of the host.

From the foregoing, it appears that damage to the epicuticle, or suppression of cuticular repair because of inadequate diet or other stressors, can, in the presence of high densities of chitinolytic microorganisms, enhance the development

of the shell disease syndrome--as was pointed out by Fisher et al. (1978) and Stewart (1980, 1984). Such a multifactorial etiology, and the probable existence of several forms of the shell disease syndrome, invite extensive discussion of the disease condition in various habitats. This approach will be followed in the following sections.

III. THE SHELL DISEASE SYNDROME IN CRUSTACEANS FROM NATURAL HABITATS

The shell disease syndrome occurs at low prevalences in lobsters, crabs, and shrimp sampled from natural habitats, although indications of occasional epizootics exist--principally in early reports of the condition in lobsters on the North American east coast.

"Shell disease" caused by chitinolytic bacteria was first described in American lobsters, Homarus americanus. Hess (1937) isolated chitin-degrading bacteria from live lobsters impounded at Yarmouth, Nova Scotia, but collected from various parts of the Canadian maritime provinces. This was the first report of attacks by such microorganisms on living Crustacea. The disease was characterized by a pitting and sculpturing of the exoskeleton (Fig. 1); although it was first seen in impounded lobsters, similar conditions were later observed in freshly caught lobsters from several widely separated Canadian fishing grounds. Initial lesions occurred on the walking legs, and were distinguished by white outer margins, from which the bacteria were most readily isolated. Hess found the disease to be relatively rare in natural populations but noted severe shell erosion and weakening of lobsters stored in pounds over the winter. Microorganisms isolated were able to decompose pure chitin in saline solution containing no other nitrogen or carbon source. None of Hess' isolates--nor, for that matter, isolates prepared in subsequent work with lobsters--was reported to reproduce the disease experimentally.

In later studies, significant mortalities of lobsters were reported to accompany the shell disease; Taylor (1948, 1949) found that 71% of infected captive lobsters died from the disease, but observed no correlation between mortality and intensity of external shell erosion. Acquisition of the disease by healthy lobsters placed in seawater tanks with infected individuals indicated direct transmission. The disease developed slowly, requiring at least three months before the advanced stages were reached. Progress of chitin destruction was reported to be directly temperature-dependent and new shell laid down after molting was not affected, except by reinfection (McLeese and Wilder, 1964; McLeese, 1965).

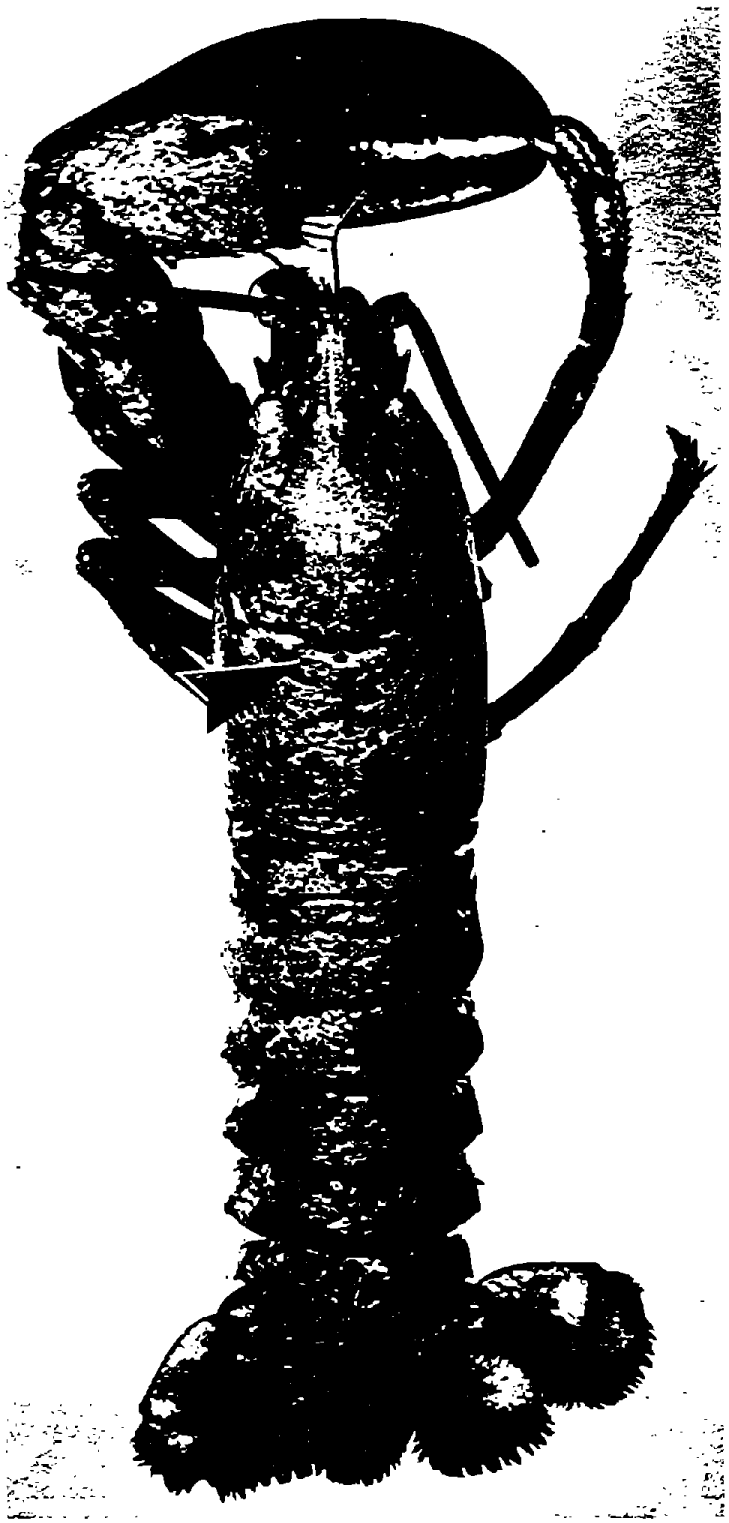

Fig. 1. Shell disease of lobsters caused by chitin-destroying bacteria.

Sawyer and Taylor (1949) observed that shell disease produced thickening or complete destruction of the chitinous layer of the gill filaments. No living gill tissue was attacked, but the authors postulated respiratory impairment as an important consequence of the disease. The infection appeared to be entirely external, confined to the exoskeleton; it did not invade living tissue nor was it transmitted internally. Sawyer and Taylor also reported the disease to be present on the Maine coast as well as in Canada, and considered it a potential threat to the lobster industry, in view of the ease of transmission and the observed mortalities of captive individuals. The method of infection of lobsters was unknown; lodging of bacteria in pores and ducts of the shell was proposed by Sawyer and Taylor as a route of invasion.

Subsequent long-term studies (1983-1988) of shell disease in lobsters from Massachusetts coastal waters by Estrella (1984 and pers. commun., 1988[1, 6]) have disclosed lower prevalences in cooler, more exposed, deeper water sites, as compared to inshore sampling locations in harbors or near sewage outfalls.

King crabs, Paralithodes camtschatica and P. platypus, from the eastern North Pacific, are occasionally affected by "rust disease," which seems to result from action of chitin-destroying bacteria on the exoskeleton. Bright et al. (1960)[2] described observations of rust disease in landed catches of king crabs from Kachemak Bay, Cook Inlet, Alaska, as well as experimental studies of the bacteria involved. The disease was characterized by progressive darkening and softening of the exoskeleton, particularly on the ventral surfaces. Underlying living tissues were unaffected. Natural infections reached 11% in larger older crabs in 1957 but were much lower overall in 1958 and 1959. Shell abrasions and injuries served as foci of the disease, which developed experimentally within two weeks. The disease was not carried over to the new exoskeleton after molting, but recently shed crabs were highly susceptible because the new shell was easily punctured or abraded. Chitin-destroying bacteria were isolated from infected crabs and cultured organisms produced the

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[1]Estrella, B. T. Massachusetts Division of Marine Fisheries, East Sandwich, Massachusetts.

[2]Data furnished from unpublished contract report, "King crab investigations of Cook Inlet, Alaska," by Donald B. Bright, Floyd E. Durham, and Jens W. Knudsen of the Allan Hancock Foundation, University of Southern California, Los Angeles, to U.S. Bureau of Commercial Fisheries Biological Laboratory, Auke Bay, Alaska, June 1960. (Cited in Sindermann and Rosenfield (1967) with permission (dated 1969) of Laboratory Director, U.S. Bureau of Commercial Fisheries Biological Laboratory, Auke Bay, Alaska.)

disease experimentally in normal crabs. Similar bacteria were also isolated from seawater in Kachemak Bay. The authors concluded that the disease would not affect the commercial fishery seriously unless the catch of crabs was substantially less than annual recruitment, since larger individuals, which do not molt annually, were more frequently infected. Since that early report by Bright et al., similar exoskeletal lesions have also been observed with prevalences up to 76% in tanner crabs (Chionoecetes tanneri) from deep water (500-2000 m) off the Pacific Northwest (Oregon) coast of United States (Baross and Tester, 1975; Baross et al., 1978). Chitinolytic bacteria, principally of the genus Photobacterium, were isolated from lesions consistently, and were found to be capable of digesting chitin rapidly at low temperatures (below 10°C).

A condition known as "Brandfleckenkrankheit" or "burn spot disease" occurs in European edible crabs, Cancer pagurus (Schäperclaus, 1935; Gordon, 1966; Bakke, 1973; Ayres and Edwards, 1982). Although it is thought by some to be of fungal etiology, others believe that it may be due to chitinovorous bacteria. Gross pathology consists of dark brown to black spots with red margins on the carapace and appendages, particularly the chelipeds. The chitin in the center of the spots becomes friable, and may be destroyed, exposing the underlying tissues. Gordon reported that in 1956 many diseased crabs from waters to the southwest of England and from the North Sea reached the market. A similar disease is known in European freshwater crayfish and river crabs, from which three fungi have been isolated and described (Mann and Pieplow, 1938; Mann, 1940).

Another exoskeletal disease condition, caused by chitin-destroying microorganisms, was described from blue crabs, Callinectes sapidus, taken in Chesapeake Bay (Maryland) by Rosen (1967, 1970). Chitinoclastic bacteria were isolated from characteristic brown depressed areas of chitin destruction. The disease was progressive and was evident in 3% of crabs examined. It is important to note, however, that the crabs had been crowded together in shedding tanks for an extended period. Shell disease has also been seen in blue crabs from Galveston Bay, Texas (Hopkins, pers. commun., 1968[3]), from the Mississippi coast (Overstreet, 1978), from Biscayne Bay, Florida (Iversen and Beardsley, 1976), from the South Carolina coast (Sandifer and Eldridge, 1974), and from the Pamlico River, North Carolina (Engel, pers. commun., 1988[4]) (Fig. 2). Studies of shell disease in blue crabs from the Gulf of Mexico, reported by Cook and

[3]Hopkins, S. H. Texas A&M University, College Station, Texas.

[4]Engel, D. W. National Marine Fisheries Service, Beaufort Laboratory, Beaufort, North Carolina.

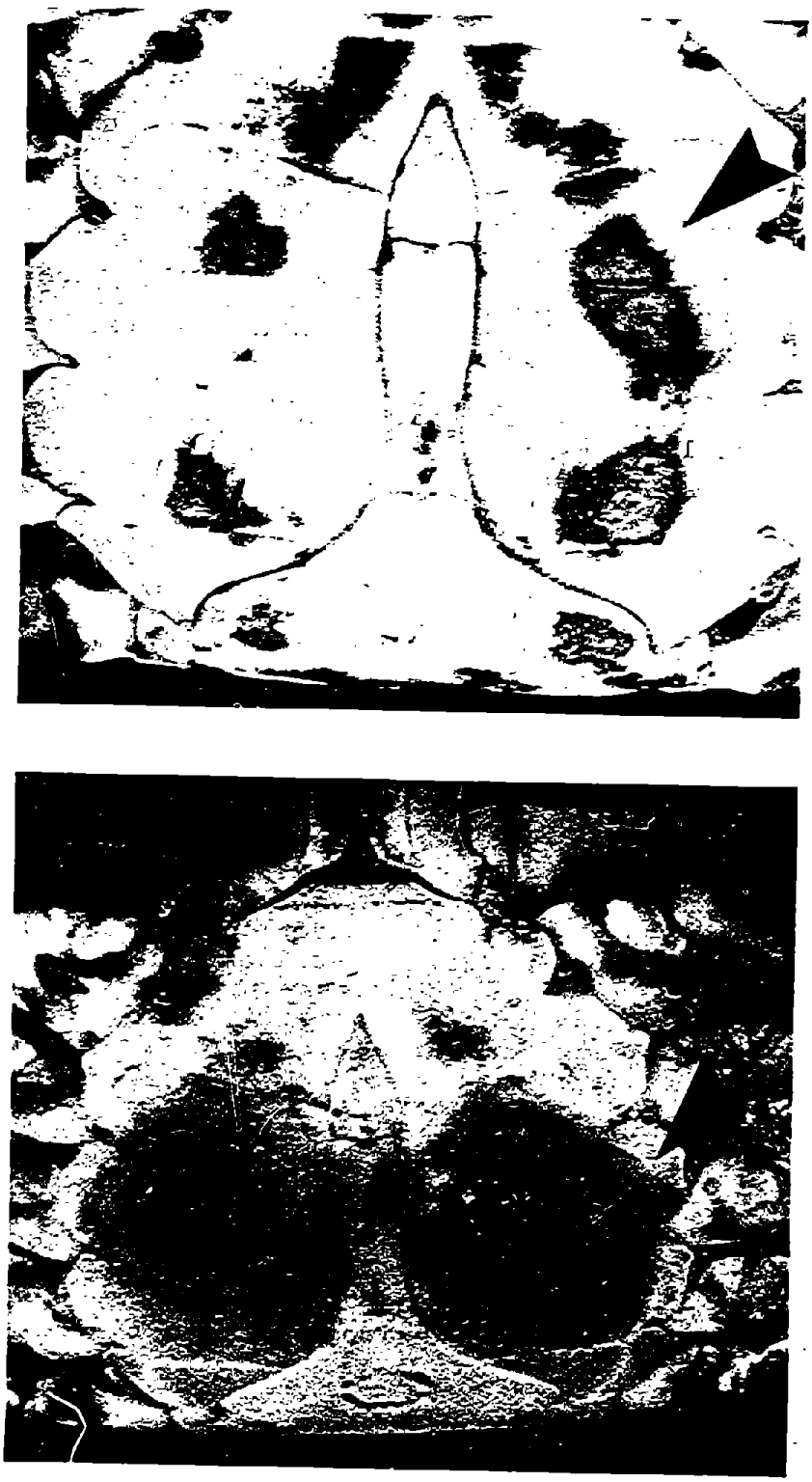

Fig. 2. Shell disease in the blue crab--early lesions (above) and advanced lesions (below). (Photographs supplied by R. M. Overstreet, Gulf Coast Research Laboratory, Ocean Springs, Mississippi).

Lofton (1973), disclosed the presence of several genera of chitinoclastic bacteria in the lesions. The disease was reproduced experimentally by damaging the exoskeleton and swabbing with cultured microorganisms isolated from natural infections.

In submarine canyons of the New York Bight, prevalences of shell disease in red crabs, Geryon quinquedens, averaged a surprising 90%, with 5 to 13% showing signs of advanced infections (Young, 1988). Most of the severely diseased crabs were found in the larger size ranges sampled.

Shell disease, often described as "brown spot disease," is also known in penaeid shrimp from the Gulf of Mexico (Cook and Lofton, 1973; Couch, 1978; Lightner, 1988a). Characterized by brown to black eroded areas on the body, appendages, or gills (Fig. 3), the shell lesions are progressive, with white margins and depressed centers. Disease effects may be eliminated at molting, unless the underlying tissues have been damaged by secondary infections, and resulting adhesions prevent withdrawal from the old exoskeleton. The secondary infections may also become systemic and fatal (Lightner, 1988a).

IV. THE SHELL DISEASE SYNDROME IN IMPOUNDED CRUSTACEAN POPULATIONS

The shell disease syndrome clearly exerts its most severe effects in impounded populations of crustaceans--especially lobsters and crabs. This is entirely logical since:

- The animals may have been damaged, even slightly, during capture and transport; such damage can provide an entry route for chitin-destroying microorganisms.
- The animals are usually overcrowded in most impoundment facilities (live cars, tanks, and pounds), providing an excellent opportunity for transmission of contagious diseases.
- Also, persistent territoriality and aggressive behavior under such crowded conditions lead to fighting and consequent damage to exoskeletons.
- Furthermore, water quality conditions (temperature, oxygen, and ammonia levels in particular) may be less than optimum, resulting in stress on the captive animals.

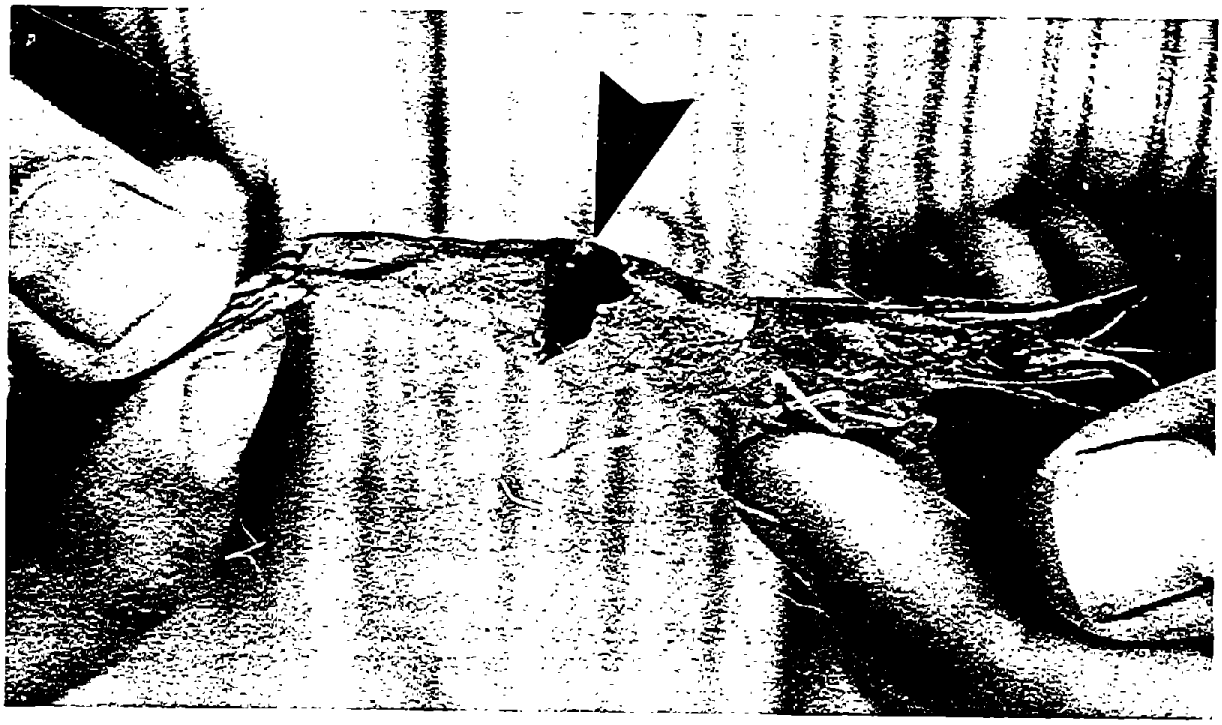

Fig. 3. Advanced bacterial shell disease lesion in a penaeid shrimp. (Photograph provided by D. V. Lightner, University of Arizona, Tucson, Arizona.)

- Some of the animals may have shed recently, rendering them more susceptible to the combined stresses of poor water quality and overcrowding--at a time of extreme physiological changes and high oxygen demand associated with ecdysis.

- Additionally, facultatively pathogenic bacterial populations, including chitinovorous forms, may increase rapidly in holding facilities, thereby providing a high level of infection pressure on already stressed animals.

All these factors, acting in concert, can result in high incidences of shell disease in captive populations. The aggressiveness of the chitinolytic microbial strains that develop, the temperature optima for such development, the extent of damage to the epicuticle, the degree of stress that the abnormal environment imposes on the animals, and the duration of impoundment--all can contribute to epizootic levels of shell disease under conditions of captivity.

It should be noted too that shell infections seen in impoundments may have existed before capture, or they may have been acquired subsequent to capture. The low prevalences of the disease normally seen in natural populations suggest, however, that most infections are acquired and develop after impoundment.[5] It is equally likely that bacteria with chitinolytic capabilities are part of the normal microbial flora present on the body surfaces of any crustacean, and as such may be introduced into holding facilities with their hosts. It should also be noted that mortality among impounded crustaceans may be high, especially in recently molted individuals, for a variety of reasons, only one of which is shell disease. For example, a systemic bacterial disease, "gaffkemia," caused by Aerococcus viridans var. homari, is well known as a cause of mass mortalities in captive lobster populations.

[5]It is interesting to note that shell disease of Nova Scotian lobsters, first described half a century ago by Hess (1937) is apparently still a problem in impoundments. In a recent (1988) communication from B. T. Estrella, high levels of the disease have been reported by Massachusetts lobster dealers in winter imports from coastal impoundments in southwestern Nova Scotia. Some possible factors contributing to high prevalences in those impounded populations include elevated organic loads in poorly flushed bays and coves, water temperatures higher than those of deeper coastal waters, and prolonged holding under crowded conditions.

V. THE SHELL DISEASE SYNDROME IN CRUSTACEAN AQUACULTURE

Among aquaculture populations, the shell disease syndrome has been observed and reported principally in penaeid and other shrimp, probably because they are the major farmed marine crustacean species on a worldwide basis--although experimental and pilot-scale culture of lobsters and crabs has also encountered problems with the disease syndrome.

Shell disease of possible bacterial etiology in English prawns, Palaemon serratus, was described briefly by Anderson and Conroy (1968). Labeled "brown spot disease" and characterized by erosion and destruction of the exoskeleton, the disease was particularly evident in crowded culture tanks. Although the etiology was undetermined, the authors observed an association between the occurrence of spots and the presence of myxobacteria. In a later study (Campillo, 1976), chitinoclastic bacteria were identified as the cause of "brown spot" of prawns. The disease condition was rare in wild-caught samples, but developed rapidly in captive populations. Mortalities were observed, and were attributed to combined effects of the bacteria and a ciliate, Ascophrys rodor, which also perforated the cuticle of the prawn (Campillo and Deroux, 1974).

Prevalences of brown spot disease in Gulf of Mexico shrimp (Penaeus aztecus and P. setiferus) were reported to be 2.5% in natural populations and 10.5% in an aquaculture facility (Cipriani et al., 1980). Even higher prevalences--up to 60%--were seen in temporary holding tanks, and some mortalities (2.8%) were attributed to the disease. The authors suggested, however, that reported high mortalities due to shell disease in aquaculture systems may actually be due to other factors, such as the presence of unrelated systemic microbial infections. Brown spot disease of penaeid shrimp is usually considered to be of bacterial etiology. However, invasion of the exoskeleton by the fungus Fusarium has been described in cultured shrimp (Lightner, 1981). Presence of the fungus invokes a strong host response--inflammation and melanization--which may circumscribe the infection but which leads to formation of adhesions that prevent molting and thus cause death (Solangi and Lightner, 1976).

The shell disease syndrome is also common in cultured freshwater prawns, Macrobrachium rosenbergii, and has been implicated in hatchery mortalities (Aquacop, 1977; Martinez et al., 1982; El-Gamal et al., 1986). In the work of El-Gamal et al., the bacterium Aeromonas hydrophila was the most consistent isolate from areas of exoskeletal erosion in adults. Prominent burn spot lesions were reported to reduce the value of prawns marketed in Southeast Asia by 75%.

Cultured larval and postlarval lobsters, as well as adults, have been found to be susceptible to shell disease (Fisher et al., 1976; Fisher, 1977). Recent studies have frequently implicated chitinolytic Vibrio spp. as etiological agents of shell disease of lobsters although pseudomonads and aeromonads have also been isolated (Malloy, 1978). Mechanical damage to the epicuticle of the shell was considered by some investigators to be essential to invasion by chitinolytic organisms (Rosen, 1970; Fisher et al., 1978), although other routes are possible, such as synergistic activity of lipolytic, chitinolytic, and proteolytic bacteria (Baross and Tester, 1975). Additionally, in one recent study of shell disease in cultured Homarus gammarus in Europe, a fungus, Fusarium solani, was implicated (Alderman, 1981).

It seems that in cultured crustacean populations, as well as in other types of captive stocks, similar predisposing factors exist which lead to shell disease. The stresses of an abnormal environment, possible nutritional deficiencies, presence of chitinolytic microorganisms in a contained grow-out habitat, and high densities of animals, combine to produce conditions favorable to development of this and other infectious diseases.

VI. THE SHELL DISEASE SYNDROME IN CRUSTACEANS FROM DEGRADED HABITATS

The shell disease syndrome in crustaceans (also known as "exoskeletal disease" or "shell erosion") has been associated with badly degraded estuarine/coastal habitats in a number of studies and review papers (Rosenfield, 1976; Murchelano, 1982). The association is direct enough to consider the shell disease syndrome in some ways as the invertebrate analogue of fin erosion in fish (Sindermann, 1979).

Two studies of shell disease in lobsters--one of Homarus gammarus in the Oslofjord (Roald et al., 1981) and the other of H. americanus in the New York Bight (Young and Pearce, 1975)--have indicated possible association of shell disease with sewage sludge dumpsites. In the Norwegian study, 8 of 68 lobsters (12%) sampled in 1979 two miles from a former (1959-1975) sewage sludge dumpsite were affected, but no samples were obtained from control or reference sites. Chitinoclastic bacteria of the genera Vibrio, Aeromonas, and Pseudomonas were isolated from shell lesions, which occurred principally on the ventral sides of the large chelae. The authors suggested, but did not demonstrate, an association between sludge disposal and the disease. It is interesting, though not necessarily relevant, that three crabs, Cancer pagurus, with shell disease were found in unrelated sampling in the Oslofjord in 1972-1973 (Bakke, 1973).

A much more extensive and more definitive study of shell disease and its possible association with pollution in the New York Bight was reported by Pearce (1971) and Young and Pearce (1975). Lobsters (Homarus americanus) and rock crabs (Cancer irroratus) from grossly polluted areas of the New York Bight apex were found to be abnormal, with appendage and gill erosion a most common sign. Exoskeletal erosion occurred principally on the tips of the walking legs, ventral sides of chelipeds, exoskeletal spines, gill lamellae, and around areas of exoskeletal articulation where contaminated sediments could accumulate. Gills of crabs and lobsters sampled at the dumpsites were usually clogged with detritus, possessed a dark brown coating, contained localized thickenings, and displayed areas of erosion and necrosis. Similar disease signs were produced experimentally in animals held for six weeks in aquaria containing sediments from sewage sludge or contaminated dredge spoil disposal sites. Initial discrete areas of erosion became confluent, covering large areas of the exoskeleton, and often parts of appendages were lost. The chitinous covering of the gill filaments was also eroded, and often the underlying tissues became necrotic.[6]

Dead and moribund crabs and lobsters have been reported on several occasions by divers in the New York Bight apex, and dissolved oxygen concentrations near the bottom during the summer often approach zero (Pearce, 1972; Young, 1973). Low oxygen stress, when combined with gill fouling, erosion, and necrosis, could readily lead to mortality (Thomas, 1954).

In a related study, Gopalan and Young (1975) examined "shell disease" in the caridean shrimp, Crangon septemspinosa, an estuarine and coastal food chain organism common on the east coast of North America and important in the diets of bluefish, weakfish, flounders, sea bass, and other economic species.

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[6]Estrella, B. T. (pers. commun., October 1988) has provided some interesting new observations about shell disease in rock crabs (Cancer irroratus) from polluted sites. He pointed out that extensive erosion of the carapace often occurs over the gills, possibly because the relatively inefficient gill bailer system of the crabs allows accumulation of contaminated sediments and associated microorganisms in the gills, with the possibility of chitin destruction proceeding from the gill chamber upward. According to this hypothesis, lobsters, with a more efficient mechanism for water movement over the gills, would have fewer of these extensive carapace lesions over the gills (and this seemed to be the case in Estrella's extensive sampling in Massachusetts waters). Estrella also suggested other possible reasons for high prevalences and severity of shell disease in rock crabs--including behavioral effects related to burrowing in contaminated sediments, and effects of possible species differences in shell pore structure.

Examinations of samples of Crangon from the New York Bight apex disclosed high prevalences (up to 15%) of eroded appendages and blackened erosions of the exoskeleton. The disease condition was only rarely observed at other collecting sites (Beaufort, North Carolina and Woods Hole, Massachusetts). Histological examination of diseased specimens produced findings similar to those of Young and Pearce (1975) with crabs and lobsters. All layers of the exoskeleton were eroded; affected portions were brittle and easily fragmented; cracking and pitting of calcified layers occurred; and underlying tissues were often necrotic. Laboratory experiments using seawater from the highly polluted inner New York Bight resulted in appearance of the disease in 50% of individuals. Erosion was progressive, crippled individuals were cannibalized, and eroded segments of appendages did not regenerate after ecdysis. No disease signs developed in control animals held in artificial seawater.

A counterpart German study of the effects of industrial wastes on the brown shrimp, Crangon crangon (Schlotfeldt, 1972), disclosed high prevalences of so-called "black spot disease," with signs very similar to those seen in C. septemspinosa from the New York Bight. Juvenile and adult shrimps from the polluted Föhr Estuary had black areas of erosion on the carapace and appendages, with necrosis of underlying tissues, and, frequently, missing terminal segments of appendages. The disease condition varied in prevalence seasonally, with a peak of 8.9% in summer. Lesions persisted and worsened after ecdysis, and experimental exposure to detergent accelerated the course of the disease. Another study of Crangon crangon in the Burry Inlet, Wales (U.K.) disclosed a disease condition known as "black necrosis," with higher prevalences in larger shrimp (Abbott, 1977). Earlier, Meixner (1969a, b) had observed localized blackened areas, and injured appendages in Crangon taken in the German shrimp fishery, and higher mortality of affected individuals held in laboratory tanks.

A long-term study of the effects of waste discharges along the southern California coast (Southern California Coastal Water Research Project) has included examination of shell disease in crustaceans. In the early years of the project (1971 and 1972), an exoskeletal disease resembling "burn spot disease" was reported in the crabs Mursia gaudichaudii and Cancer sp. (Mearns, 1973). Samples of M. gaudichaudii collected in 1972 along the 150-m contour on the Palos Verdes shelf had 8% blackened exoskeletal lesions, while in similar depths in Santa Monica Bay the prevalence was 44%. According to Mearns, the disease seemed to be localized in populations of small crabs near the heads of submarine canyons, although small samples of larger crabs from inshore stations disclosed prevalences of up to 70%.

On the basis of these limited studies, it may be inferred that high prevalences of exoskeletal erosions in Crustacea have some association with degraded habitats, and particularly with sewage sludge dumpsites, although the condition may be found in samples from unpolluted sites as well. The inference suffers, however, from inadequate data from reference sites.

The shell disease syndrome in crustaceans from polluted habitats is intimately associated with another general disease sign or syndrome--the so-called "black gill syndrome," characterized by sediment accumulation between gill lamellae, accompanied by darkening of filaments. Substrate is provided for microbial growth, and the syndrome is further characterized by chitin deterioration and gill tissue necrosis (Sawyer et al., 1979).

A recent study of shell disease and black gill disease in lobsters from Massachusetts waters showed similar trends for both diseases, with highest prevalences (up to 50%) in samples from the most polluted sites--particularly Boston Harbor and Buzzards Bay (Estrella, 1984). Prevalences in more exposed, deeper water sites were generally low (Cape Ann, Cape Cod Bay, outer Cape Cod, and Eastern Shore). Mortalities were not observed, but population impacts were postulated, based on increased vulnerability to hypoxia of lobsters with fouled or necrotic gills.

Black gill disease was also examined in rock crabs, Cancer irroratus, from the Atlantic coast of North America (Sawyer, 1982; Bodammer and Sawyer, 1981; Sawyer et al., 1979, 1983, 1984). High prevalences were associated with sewage sludge and/or dredge material dumping areas where environmental degradation was worst. Prevalences of black gill disease were as high as 30% in some samples from the New York Bight apex. The etiology of the disease is clearly complex, but it involves the presence of black silt between lamellae and the presence of epibiotic and epicommensal fouling organisms. Foci of black discoloration were found to be areas in which several adjacent lamellae were dead and necrotic, with accompanying melanization of gill tissue (Sawyer et al., 1983). An association of the black gill condition with shell erosion was stated clearly by Sawyer (1982): "Specimens with gills judged to be 100% black often had external blackening and carapace erosion. Black gills often were melanized extensively with necrosis of gill cuticle or entire gill filaments."

It should be noted, though, that black gills may also be prominent gross signs of systemic infections, nutritional deficiencies, or the presence of toxic compounds in the environment and in the tissues. Phagocytes, fixed and motile, may form pigmented aggregates near the tips of gill filaments, as the terminal step in removal of killed pathogens, dead cells, or toxic chemicals, since these deposition areas will be lost at

ecdysis (Figs. 4 and 5). The black gill condition has been reported in feral, cultivated, and experimental shrimp infected by bacterial and fungal pathogens, and from crustaceans exposed experimentally to heavy metals, biocides, petroleum, petroleum derivatives, and other contaminants (Egusa and Ueda, 1972; Rinaldo and Yevich, 1974; Lightner et al., 1975, 1977, 1979; Fontaine et al., 1975; Couch, 1977; Sindermann, 1977; Bian and Egusa, 1981; Doughtie and Rao, 1983a, b; Lightner, 1988b). Thus, the black gill syndrome is a common and nonspecific pathological manifestation, with complex etiology, and is not unique to polluted habitats; in this sense it is similar to the shell disease syndrome. A recent attempt was made to distinguish between the "black gill syndrome," an exogenous gill discoloration seen in crabs and lobsters, which seems related to sewage sludge-contaminated habitats, and a "black spot" gill condition, characterized by discrete localized foci of melanin deposition, forming blackened nodules in filaments or lamellae in response to disease or injury (Sawyer, 1982)--but a broad-scale evaluative study of the black gill condition in Crustacea from various environments is clearly needed.

It might be well at this point to emphasize that the "black gill syndrome" seems to be part of an important hemocyte-mediated defensive response of Crustacea to chemical or physical trauma, or to pathogen invasion. It results in sequestering and subsequent removal during ecdysis of toxic substances or microbial invaders. It also results in truncated gill filaments, with consequent reduction in gas and ion exchanges.

The essence of the black gill syndrome seems to be the formation of apical cellular plugs consisting of hemocytes, formed in response to gill trauma or systemic microbial infection. Phagocytized material is aggregated in gill apices. Gill blackening has been reported as a consequence of protozoan and fungal infection (Couch, 1978; Lightner et al., 1975) and of exposure to chemical contaminants (Couch, 1977; Nimmo et al., 1977; Doughtie and Rao, 1983a, b). In one study (Doughtie and Rao, 1983b), several defensive responses to toxin exposure were proposed. Included were:

(1) Formation of a hemocyte plug in the apices of gill lamellae;

(2) Phagocytosis of cellular debris by circulating granular hemocytes;

(3) Phagocytosis of cellular debris by fixed hepatopancreatic cells;

(4) Encapsulation by circulating hemocytes of degenerated tissues resulting from action of toxins;

Fig. 4. Juvenile cultivated shrimp, Penaeus stylirostris, with conspicuous black gills. (Photograph provided by D. V. Lightner, University of Arizona, Tucson, Arizona.)

Fig. 5. Gills of juvenile shrimp, Penaeus californiensis, with severe black gill condition. (Photograph provided by D. V. Lightner, University of Arizona, Tucson, Arizona.)

(5) Darkening of hemolymph in gill lamellae and in areas where degenerative changes were occurring; and

(6) Attainment and maintenance of "heterostasis"--an abnormal level of equilibrium in the continued presence of a toxic stressor--promoted by enhanced detoxifying activity of the hepatopancreas and excretory activity of the antennal gland.

These responses may be important to survival of crustaceans in degraded habitats, and are often associated with cuticular erosions, especially of the gills.

VII. DISCUSSION AND CONCLUSIONS

The shell disease syndrome has been reported from many crustaceans, including a number of the larger economically important species of crabs, shrimp, and lobsters. The disease condition appears to be a stress and injury-related phenomenon, characterized by degradation of the chitinous exoskeleton by lipolytic, chitinolytic, and proteolytic exoenzymes of microorganisms acting independently or in concert. Bacteria are most frequently described as principal pathogens, but fungi have been implicated as well. Bacterial infections seem to predominate in marine crustaceans, whereas fungal infections are often reported in freshwater forms.

The shell disease syndrome occurs, usually at low levels, in natural populations, but its effects may be enhanced and prevalences increased in crustacean populations under stress from environmental degradation, or in populations in which individuals are crowded abnormally—in impoundment facilities or in aquaculture grow-out tanks.

Effects of the disease may be enhanced by (1) overcrowding or handling, which increases the likelihood of cuticular abrasions, (2) stresses from abnormal environments, and (3) high organic loading of containment waters, which contributes to multiplication of heterotrophic microbial populations--some of which may be facultatively pathogenic to crustacean hosts.

The disease is contagious and progressive, but effects are usually confined to erosion of the exoskeleton, except when underlying living tissues are exposed and invaded by other opportunistic microorganisms. In some species, ecdysis can terminate infections (although reinfections may occur). The rate of exoskeletal destruction is probably variable with species or strains of pathogens, nutritional state of the host, density of pathogen populations, and other environmental factors, such as temperature.

Mortalities due to the shell disease syndrome have been reported. In lobsters, death may be the consequence of progressive erosion and destruction of gill membranes with resultant reduced oxygen uptake, especially in hypoxic situations (near sewage sludge dumpsites, for example). In shrimp, failure to complete ecdysis because of shell adhesions has been identified as a causal factor. In these and other species, death may also result from secondary infections, after the exoskeletal barrier has been breached, especially in the presence of high populations of facultative pathogens. Additionally, it is quite likely that severely affected animals would be more vulnerable to predators.

Among the larger economically important Crustacea, shell disease has been found to be more prevalent in older individuals, that molt infrequently or not at all--since the exoskeleton would be available for longer periods to the activity of chitin-destroying microorganisms, and since the likelihood of minor damage (which provides entry routes for such microorganisms) would be increased. In some heavily exploited populations, shell disease prevalences may have an inverse relationship to fishing mortality. An intensive fishery (as exists, for example, for lobsters and king crabs) removes more of the larger, slower growing, more heavily diseased individuals from the fished stocks, resulting in reduced prevalences. This relationship was first suggested by Bright et al. (1960)[2] for king crabs of the North Pacific, and has been supported by findings of Ayres and Edwards (1982), who sampled a developing fishery for European edible crabs (Cancer pagurus) off the southwest coast of Ireland. Rejection rates in newly exploited areas, because of shell disease, were between 5 and 7%, and most of the crabs rejected were larger older animals which had not molted for some time. In other areas, which had been exploited for longer periods of time, rejection rates because of shell disease were usually less than 1%, and few younger crabs with advanced lesions were observed. Advanced shell disease in younger animals may be an indication of abnormal environmental conditions or nutritional deficiencies--such as those which may prevail in aquaculture facilities. Additional support for the observation that shell disease is more prevalent in larger older individuals has been provided by Estrella (pers. commun., October 1988[6]) from results of a five-year survey of lobster populations off the Massachusetts coast. So, in natural habitats and in unfished populations the disease can be described as "an infirmity of advancing age." However, in abnormal habitats--impoundments, culture facilities, or polluted waters--shell disease can become a stress-related condition resulting from impaired metabolism, with effects on chitin synthesis and possibly other defense mechanisms, which are not necessarily age-related.

There is a suggestion from the literature on shell disease that chitin-destroying marine microorganisms may vary in ability to invade and degrade the exoskeletons of crustaceans. Some recent reports of shell disease in larger species such as lobsters and crabs suggest rapid invasion, with destruction of significant areas of the chitinous shell, while other reports suggest a more dispersed, slower-acting, generalized erosion. The difference can be seen most clearly in lobsters. Samples from Maine and the maritime provinces may contain individuals in which the shell is extensively but superficially eroded, creating a rough pitted surface; some samples from the middle Atlantic states, on the other hand, may exhibit large but often discrete lesions, with loss of spines and disappearance of extensive portions of the carapace overlying the gills or of the cuticle of the chelae. These differences in activity and effects may be related to geographic or other variability in the kinds and numbers of chitinoclastic microorganisms present. Some may act more aggressively than others, depending on their temperature optima, their exoenzyme competencies, and probably on the nature and condition of host defenses. Other factors, such as organic loading of habitats, may also contribute to the differences seen.

Growth of chitinoclastic bacteria may vary with temperature, salinity, and hydrostatic pressure--so, for example, some (such as Vibrio parahaemolyticus) may prosper in estuarine waters and some may not; some (such as Photobacterium sp.) may flourish in deeper colder waters whereas others may not. Additionally, seasonal population maxima have been reported for some chitinoclastic Vibrio species, coinciding with decline in zooplankton populations (Kaneko and Colwell, 1978).

The pathogenic role of chitinolytic microorganisms has often been described as limited to destruction of the exoskeleton and not the underlying tissues (except to provide a portal of entry to other microbial invaders). This invasive process should be reexamined, particularly in light of reports on fungal shell disease by Forster and Wickins (1972) and Alderman (1987) indicating that some forms of the disease syndrome--at least those in which fungi have been implicated--may involve invasion of underlying living tissues. The earlier report by Forster and Wickins mentioned the presence of fungal hyphae in the tissues, and the recent report by Alderman described "erosive lesions...penetrating deeply into underlying tissues" and noted that "brown nodules of melanized wound-reaction tissue become deposited below the lesions." It is not clear from Alderman's report whether the penetrating lesions were caused by the fungal pathogen or by secondary microbial invaders, but this is clearly a matter of concern.

To reinforce these findings of potential or actual tissue invasion, an aberrant form of shell disease in snow crabs, Chionoecetes bairdi, from the North Pacific, caused by the ascomycete fungus Trichomaris invadens, has been shown recently to be a severe pathogen. Formerly thought to be only a cause of exoskeletal erosion (Van Hyning and Scarborough, 1973), the fungus was subsequently found to invade epithelial, connective, and other tissues of the host (Hibbits et al., 1981). Such invasion was thought to prevent molting and to cause mortalities (Sparks, 1982). There is a suggestion in these reports that at least some of the fungi involved in shell disease may also be capable of invading and destroying living tissue as well.

The statement by Alderman (1987) about the deposition below shell lesions of brown nodules of melanized wound-reaction tissue indicates a form of host response to destruction of the integument, and suggests an active process to contain the invading microorganisms. It is interesting that an earlier publication on shell lesions in lobsters from the New York Bight (Young and Pearce, 1975) described host responses in the form of cellular clots in the eroded pits of the exoskeleton. A similar response was described in penaeid shrimp exposed to experimental infection with chitinoclastic Vibrio spp. (Pylant and Leong, 1978). Their study disclosed a three-phase reaction:

(1) An attempt by the traumatized animal to slough the old integument;

(2) Migration of hemocytes to the site of infection, where they form a melanized defensive clot or plug, presumably to prevent further infection; and

(3) Deposition of chitin to the already existing exoskeleton, possibly in a further attempt to protect underlying tissues against bacterial infection.

More studies of host responses to shell disease, particularly among the larger Crustacea, should contribute to full understanding of the disease process (see, for example, Johnson, 1976).

The shell disease syndrome has been observed in many freshwater and marine species of Crustacea and under many conditions, both natural and artificial. Actual shell erosion seems to involve activity of chitinoclastic bacteria and fungi, sometimes with subsequent secondary infection of underlying tissue by other facultative pathogens. Initial preparation of the exoskeletal substrate by mechanical, chemical, or microbial action probably is significant; thus high bacterial populations and the presence of contaminant chemicals in polluted environments, as well as extensive detrital and epibiotic fouling of gills, could combine to make the disease syndrome a common phenomenon and a significant mortality factor in crustaceans inhabiting degraded environments.

Some evidence exists for an association of the shell disease syndrome and habitat degradation. Prevalences have been found to be high in samples from polluted sites; prevalences show trends similar to those of the black gill syndrome, which has a statistical association with extent of pollution; and experimental exposures of crustaceans to contaminated sediments, heavy metals, biocides, petroleum, and petroleum derivatives can result in the appearance of the black gill syndrome, often accompanied by shell disease. It is important to note, however, the cautionary words of Ayres and Edwards (1982), pointing out that dumpsites are rarely fished intensively, and hence would have more larger and older animals which molt less frequently, and hence, could have higher prevalences of shell disease. (This avoidance phenomenon exhibited by European crab fishermen, as described by Ayres and Edwards, may not, however, apply to the lobster fishery on the northeast coast of United States, where the vicinities of domestic sewage outfalls or sewage sludge dumpsites can be favored locations for traps.) It is also important to keep in mind the observation of Sawyer (1982) and Sawyer et al. (1985) that season, stage of the molt cycle, and differential migrations may have profound effects on prevalences of the black gill condition (and, by analogy, on prevalences of shell disease).

The shell disease syndrome exemplifies a condition at the boundary between infectious and noninfectious disease processes. This is the area where environmental stressors and facultative microorganisms exert their impacts; where high bacterial populations in eutrophic waters interact with exposed, or injured, or chemically modified surface layers; where normally harmless epibiotic fouling organisms can assume pathogenic roles; and where nonspecific lesions such as exoskeletal erosions can occur in epizootic proportions. Prevalence of the shell disease syndrome in Crustacea may well prove to be an excellent indicator of abnormal environmental conditions, and a measure of stress on individuals. Ingredients to be considered are presence of toxic chemicals, large populations of facultatively pathogenic microorganisms, inadequate nutrition, extremes of salinity and temperature, and existence of anoxic or hypoxic conditions.

The appearance of shell erosion may therefore be the consequence of a disturbed balance between processes of chitin maintenance and repair and the activities of chitinoclastic microorganisms--this disturbance created by either natural or man-made environmental changes. Critical to an understanding of the relationship are environmental, genetic, and immunological factors which may either promote repair or, conversely, enhance exoskeletal degradation. Also essential to full understanding of the shell disease syndrome are further experimental studies, particularly those concerned with identification of specific microorganisms capable of pathogenesis, experimental manipulation of predisposing environmental variables, and the immunologic responses of hosts to cuticular disruption.

Shell disease, then, represents a stage in the dynamic relationship of one group of chitin synthesizing animals--the crustaceans--with chitin-utilizing microorganisms. An uneasy balance is maintained by the metabolic processes associated with new cuticle formation at ecdysis, and subsequent cuticular maintenance and repair--and the growth, metabolism, and reproduction of microbes capable of degrading chitin. The balance may be disturbed by environmental changes that reduce the crustacean's capabilities (metabolic and defensive) to maintain an intact cuticle, or that encourage multiplication of the facultative heterotrophic microorganisms with chitinolytic capacities. Such disturbances can lead to destructive changes in the crustacean exoskeleton that are labeled "shell disease."

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(continued from inside front cover)

47. MARMAP Surveys of the Continental Shelf from Cape Hatteras, North Carolina, to Cape Sable, Nova Scotia (1977-1984). Atlas No. 2. Annual Distribution Patterns of Fish Larvae. By Wallace W. Morse, Michael P. Fahay, and Wallace G. Smith. May 1987. viii + 215 p., 27 figs., 2 tables. NTIS Access. No. PB87-232831/AS.

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