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Diseases and Defense Mechanisms of the Lobster, *Homarus americanus*

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Gaffkemia is a fatal bacterial disease which periodically causes heavy mortalities among American and European lobsters, *Homarus americanus* and *Homarus vulgaris,* respectively. The disease and the causative agent, *Aerococcus viridans* (var.) *homari* [formerly *Gaffkya homari;* see Stewart and Arie (1974)], were described originally by Snieszko and Taylor (1947) and Hitchner and Snieszko (1947). The septicemic nature of the disease and the original taxon assigned to the pathogen prompted the name ''gaffkemia'' (Roskam, 1957) for the disease. Severe losses from gaffkemia are experienced periodically in commercial lobster units. A brief account of the disease condensed from reviews by Stewart and Rabin (1970), Sindermann (1971), and Stewart (1975) follows.

The causative agent *A. viridans* (var.) *homari* is a gram-positive, catalase negative, beta hemolytic, tetrad-forming coccus which appears to possess no exoenzymes. This lack of exoenzymes and consequent lack of invasive powers results in the bacterium being transmitted only through breaks or ruptures in the integument of the host which permit entry to the hemolymph. The exposure to *A. viridans* (var.) *homari* must come simultaneously with or soon after integumental rupture for the pathogen to circumvent the lobster's rapid and effective wound healing mechanism. Transmission does not occur when the lobster ingests infected material since the acidity of the gastric fluid (pH 5.0) is lethal to *A. viridans* (var.) *homari.* Despite the pathogen's lack of invasive powers, the development of epizootics is aided by the aggressive behavior of lobsters, which provides wounds for entry, and by the fact that normal lobsters have no apparent resistance to the pathogen after entry. Small numbers of a virulent strain of *A. viridans* (var.) *homari,* consisting of 10 or less per kilogram of the lobster's weight, injected into the hemolymph are sufficient to ensure a fatal infection. Injection, however, of large numbers (6×10^8/kg) does not accelerate the infection sufficiently to produce a significant decrease in time to death. There are no external signs of the disease, with the exception that as the infection proceeds the lobsters become lethargic and progressively weaker until they die.

As the disease develops, the lobster suffers a massive decrease of circulating hemocytes, resulting in impairment of the clotting mechanism by the removal of the clot initiating factor contained in the hemocytes. The fibrinogen levels and other hemolymph proteins are not affected significantly. As a result of the loss of hemolymph clotting power, the risk of a fatal hemorrhage is introduced in the event of wounding. If the fatal hemorrhaging is avoided, death ultimately occurs apparently as a direct result of massive dysfunction of the hepatopancreas.

The intrinsic defense mechanisms of the lobster have been studied: Agglutinin, bactericidin, phenoloxidase, and phagocytic capacity. It appears that in the normal lobster none of these are effective against this pathogen (Cornick and Stewart, 1968, 1973; Paterson and Stewart, 1974; Paterson et al., 1976; Paterson[1]; and Stewart and Zwicker, 1972). Induction of a high degree of resistance has been obtained (Stewart and Arie, 1974) and appears to involve mainly changes in phagocytic capacity. A degree of specificity, dependent on the vaccine used, has been observed in the phagocytic response (Paterson et al., 1976; and Paterson, footnote 1).

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A New Bacterium (Presumptive *Vibrio* Species) Causing Ulcers in Flatfish

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Summer flounder, *Paralichthys dentatus,* captured in the wild and held for 10 days in a tank supplied with flowing seawater (about 25 ppt salinity) from Milford harbor developed ulcerative lesions of the tail and dorsal muscle. Lesions began as a white patch at the flexure point of the tail (where the caudal fin meets the body) and sometimes were accompanied by hemorrhagic necrosis at the tip of the caudal fin. Frank ulceration extending into the skeletal muscle could be seen within a few days when the epithelium sloughed off under the patches (Fig. 1). Frequently, bony rays of the entire caudal fin were exposed by progression of the ulcerative process either anteriorly from the tip of the fin or from the initial lesion caudally (Fig. 2).

First appearance of the ulcers in two summer flounder was followed within 1 week and progressively for 4 weeks by ulcers in other summer flounder in the same tank, as well as in winter flounder, *Pseudopleuronectes americanus,* and hogchokers, *Trinectes maculatus,* in an adjacent tank. Although the lesions resemble those caused by *Vibrio*

Figure 1.—Posterior portion of a summer flounder infected with the new *Vibrio* species. Ulcers are seen to extend deep within the dorsal muscle.