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STATUS REPORT ON GAFFKEMIA IN LOBSTERS

### IN ATLANTIC CANADA

by

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#### SUMMARY

Gaffkemia was not detected in freshly captured lobsters examined during surveys conducted in 1973 and 1974 in the Canadian Maritime Provinces. A large number of lobsters were examined bacteriologically to determine whether the lobster pathogen Aerococcus viridans (var.) homari was part of the normal microflora of the lobsters external surfaces. None was detected on these surfaces, thus the pathogen is not part of the lobsters' normal external microflora.

Five outbreaks of gaffkemia in commercial units were recorded over the past several years. Some of these outbreaks could have been initiated within the holding units themselves with the epizootics perpetuated by the holding unit designs.

#### INTRODUCTION

The lobster disease, gaffkemia, has plagued the live lobster trade in the United States, Canada and Europe, probably since its inception. It is a constant hazard which strikes on an unpredictable basis; several disease free years are followed by serious outbreaks and then further disease free periods.

The disease was first recognized as a bacterial infection in 1947 by two American Scientists, Snieszko and Taylor. They isolated the causative agent during an epidemic in Maine pounds and showed that it was caused by the bacterium which Hitchner and Snieszko (1947) named Gaffkya homari. More recently the name of the bacterium was changed to Aerococcus viridans (variety) homari.

This disease is endemic to lobster areas and periodically produces highly significant economic losses. Normal lobsters infected even by very small numbers of the bacterium invariably develop a fatal infection in a time span directly dependent upon the temperature. The huge numbers of the pathogen present in the weakened or dead animals are released to the water during cannibalism and are available for infecting other lobsters in the unit. The pathogen enters the animal through minor breaks or wounds in the integument

which inevitably occur under the conditions of holding. The lobster does not acquire the disease through eating infected material since the acidity in the stomach is sufficient to kill the pathogen. Thus one infected animal introduced to a unit is sufficient for the initiation of an epidemic resulting in the death of most of the animals in the unit. Low water temperatures tend to prolong the development of the disease and thus temperature is usually the only real control mechanism in continuous use in holding operations (Stewart and Rabin, 1970).

An aspect of many tank units which contributes to a more rapid and universal spread of the disease throughout the system is the use of a common or cascading water system whereby all of the water introduced will pass through a series of tanks carrying the pathogen from one tank to another. Additionally, many tank units have their water intake close to the outfall thereby recycling at least a part of the water and ensuring complete exposure of all animals to the pathogen which otherwise might have been confined to one or several tanks.

There are several problems inherent in detecting *G. homari in vivo* in wild lobster populations. The first stems from the fact than an infected lobster will not feed after the development *in vivo* of large numbers of *G. homari* (Stewart, Zwicker, Arie and Horner, 1972) and becomes less and less active as the infection proceeds. The consequence of this is that methods of capture based upon the animals' interest in food or activity tend to exclude infected lobsters other than those in the very early stages of the infection. At 15°C, for example, infected lobsters eat only sparingly upon the second day of the infection and refuse food thereafter. Thus the only infected lobsters likely to be included in a fisherman's catch are those possessing too few *G. homari* in the hemolymph to be detected by microscopic examination of a hemolymph smear; 1 x 10<sup>6</sup> bacteria or more per ml of hemolymph are required before microscopic examination is at all useful. Thus the cultural method described earlier (Stewart *et al.*, 1966; Stewart, 1972) has been employed for our examinations and surveys.

In response to the ICES 1972 resolution requesting information on gaffkemia and also for our own needs we have run surveys in the maritimes region. In addition we have accumulated as much information as we could in outbreaks in commercial units.

### Surveys of Freshly Captured Lobsters

Our survey work has been directed toward lobsters captured in widely separated areas and immediately after the fisherman had landed them. The sampling areas, widely separated geographically, were located in Lobster Fishing Districts 3,5,6 and 8 (Fig. 1). Over 400 lobsters were examined bacteriologically during 1973 and 1974 during the May to September periods. Blood samples and shell surface smears were taken from each lobster and treated by the method outlined by Stewart et al., (1966) and Stewart (1972). The shell surfaces were investigated because it had been suggested that the pathogen was part of the normal microflora of lobster shell surfaces; the suggestion further speculated that the disease state would arise upon wounding of the animal thereby introducing A. viridans (var.) homari internally.

All of the lobsters examined were shown, by the diagnostic procedures used, to be free of A. viridans (var.) homari in contrast to previous surveys (Stewart et al., 1966). None of the lobsters carried the pathogen internally or on the shell surface. Thus it can be concluded that A. viridans (var.) homari is not

part of the normal microflora of the lobster's external surfaces.

## Commercial Unit Examinations

Five outbreaks have been recorded in commercial units from 1972 onward.

# (1) July 21, 1972. Lobster District #3 Holding Unit:

Severe losses were recorded prior to this date, i.e., of the order of 10% per day.

Twenty-seven percent of the 62 lobsters sampled were shown to be infected with the pathogen. The pathogen was present also in the water supply and was being recycled throughout the unit.

# (2) September 14, 1972. Lobster District #1, Pound and Tank Unit:

Severe losses had been experienced prior to this date and a sample of 20 lobsters (10 from the tanks and 10 from the pound) were sent to us for examination:-

9 of the 10 lobsters from the tank units were infected;

1 of the 10 lobsters from the pound was infected.

# (3) July 6, 1973. Lobster District #7 Holding Unit:

Three hundred lobsters were examined and 88% of the lobsters were infected, many in the last stages of the disease.

# (4) August 30, 1973. Lobster District #1 Holding Unit:

Severe losses had been experienced prior to this date.

One hundred and four lobsters were sampled from tank and pound units. Forty-five percent of the lobsters in the tank unit were infected. The pounds varied from a 5 to 20% infection rate.

## (5) September 19, 1974. Lobster District #8 Holding Unit:

Four hundred lobsters were examined; approximately 75% were shown to be infected, many in the last stages of infection.

It should be pointed out that the various holding units, to a great extent, draw upon the whole of the Maritimes provinces and Maine for their supply of lobsters. Consequently, an outbreak at any one unit can rarely be attributed necessarily to the lobsters from the local area.

#### DISCUSSION

Lobsters freshly captured in the Maritime provinces have shown no incidence of gaffkemia, either internally or externally, upon testing at the landing sites over the 1973 and 1974 seasons. Sufficient numbers were examined to discount completely the suggestion that the bacterial pathogen A. viridans (var.) homari is a part of the normal microflora of the lobster's external surfaces. If, however, lobsters are taken from heavily contaminated areas such as a pound or tank unit currently experiencing a gaffkemia epizootic then it is to be expected that A. viridans (var.) homari will be found both internally and on external surfaces.

The scattered outbreaks of gaffkemia in commercial holding units during periods when large samples of freshly captured lobsters from representative areas do not show any incidence of the disease is interesting. We do not examine all lobsters and representative sampling of course cannot ensure the complete absence of a disease; it takes only one infected animal to initiate an epizootic. In some instances, however, it is certain that much of the problem resides in the holding units with the epizootic initiated and perpetuated on site.

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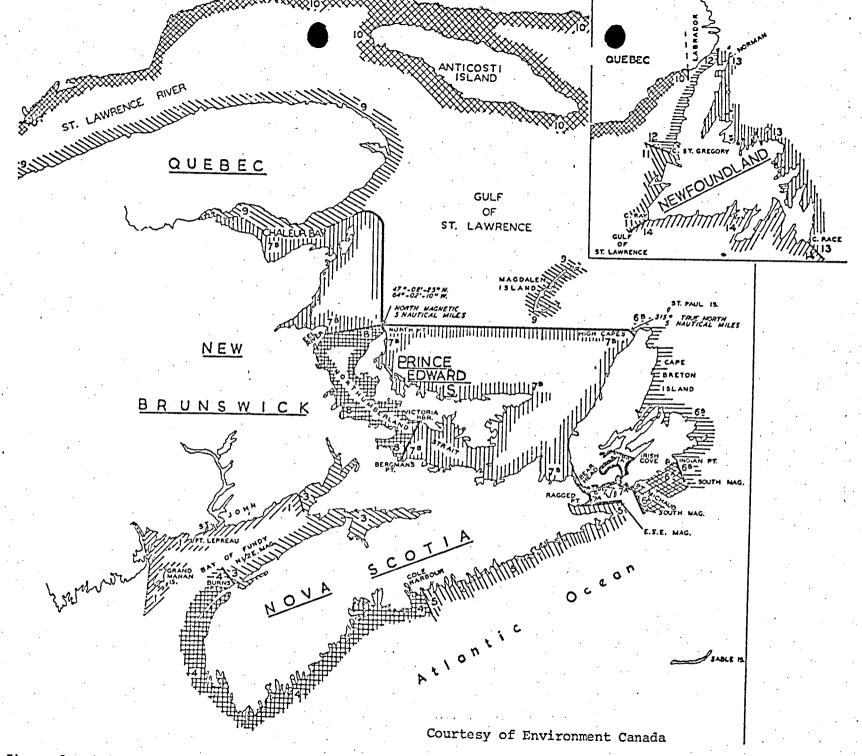


Figure 1. Lobster Fishing Districts