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Occurrence of Gas-Bubble Disease in Three Species of Bivalve Molluscs[1]

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Gas-bubble disease was observed in adult oysters and hard clams held in heated running sea water during the winter. Heating the cold sea water in closed heat exchangers caused it to become supersaturated with atmospheric gases. Exposure of the animals to this water caused the formation of gas-filled conchiolin blisters on the valves of the oysters. Bubbles of gas were observed in the gill filaments of the oysters and clams and in the mantle tissue of the oysters. Any method, such as the use of baffles or an aerated head tank, that reduces the dissolved gas concentration in the water will help prevent the disease.

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Nous avons remarqué que des huîtres adultes et des quahaugs gardées durant l'hiver dans l'eau de mer chauffée en circulation étaient atteintes de la maladie de la bulle de gaz. Le chauffage de l'eau de mer froide dans des échangeurs en circuit fermé l'a sursaturée de gaz atmosphériques. Au contact de cette eau, il s'est formé sur les valves des huîtres et des quahaugs des boursoufflures de conchioline remplies de gaz. Des bulles de gaz sont apparues dans les filaments branchiaux des huîtres et des quahaugs et dans le tissu du manteau des huîtres. Toute méthode, comme l'emploi de déflecteurs ou l'aération du réservoir de charge, capable de réduire la teneur en gaz de l'eau, contribuera à prévenir cette maladie.

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THIS note describes the occurrence of gas-bubble disease in three species of bivalve mollusc, *Crassostrea virginica*, *C. gigas*, and *Mercenaria mercenaria*. Recognition and prevention of the disease is of particular importance to mariculturists and researchers who maintain these animals in heated flowing sea water during the winter.

Occurrences of gas-bubble disease in fin fish have been well documented in fish hatcheries (Harvey and Smith 1961; Pauley and Nakatani 1967) and in nature (Renfro 1963). A similar disease has been reported in lobsters by Hughes (1968). Marsh and Gorham (1905) reported that molluscs are susceptable to the disease but did not describe their observations or identify the species involved.

We first observed the disease in the clams and oysters during the winter of 1971. In the oysters, the disease first appeared as crescent-shaped conchiolin blisters bordering the mantle. As the disease progressed, conchiolin deposits on both valves became very large causing the mantle and gills to recede. Bubbles of gas were observed in the gill filaments and in the outer layers of the mantle tissue of some individuals. In many animals deposits of mud were observed to collect inside the shell cavity. In severe cases the body of the oyster was reduced to a fraction of its original size as the shell cavity became filled with conchiolin blisters.

Only about 10% of the affected oysters died from the disease, but the survivors continued to be in poor condition until late spring. By June the diseased oysters had covered the conchiolin deposits with shell material, forming hard mud and gas-filled blisters that occupied a significant portion of the shell cavity (Fig. 1 and 2).

Adult hard clams (*M. mercenaria*) held under identical conditions appeared to be less severely affected by the disease than the oysters. Mortalities among the clams were rare. An obvious lightening of the color of the gills was the only gross symptom of the disease in clams. Microscopic examination of the affected gills showed the lack of color to be due to the presence in the gills of numerous trapped gas bubbles that prevented the free circulation of blood.

Analysis of two 10-cm^2 samples of gas collected by water displacement from conchiolin blisters and gas embolisms in the tissue of 15 diseased oysters showed the gas to consist of 91.2% nitrogen, 6.4% oxygen, 1.2% argon, and 0.5% carbon dioxide. Marsh and Gorham (1905) reported a similar composition of gas

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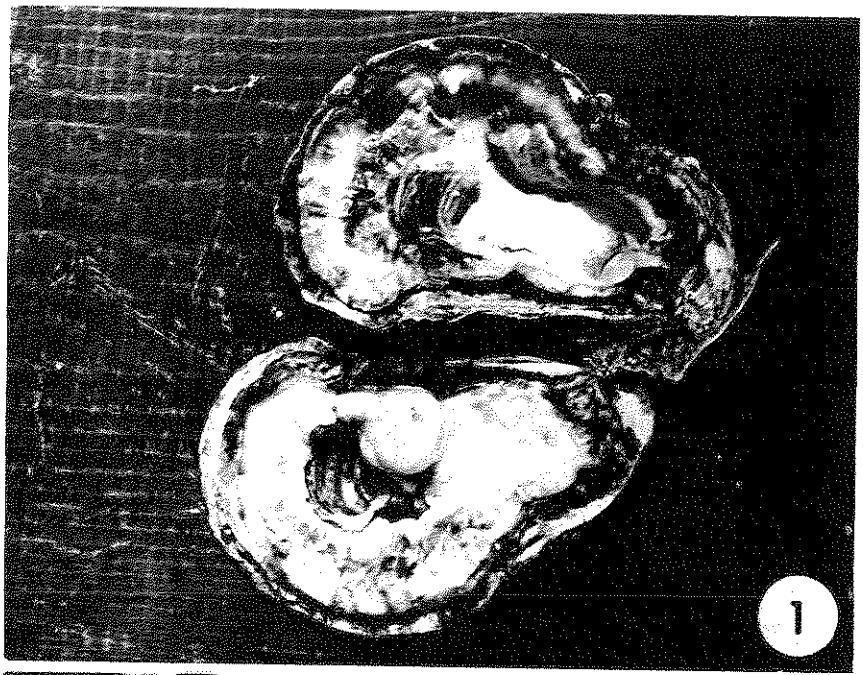

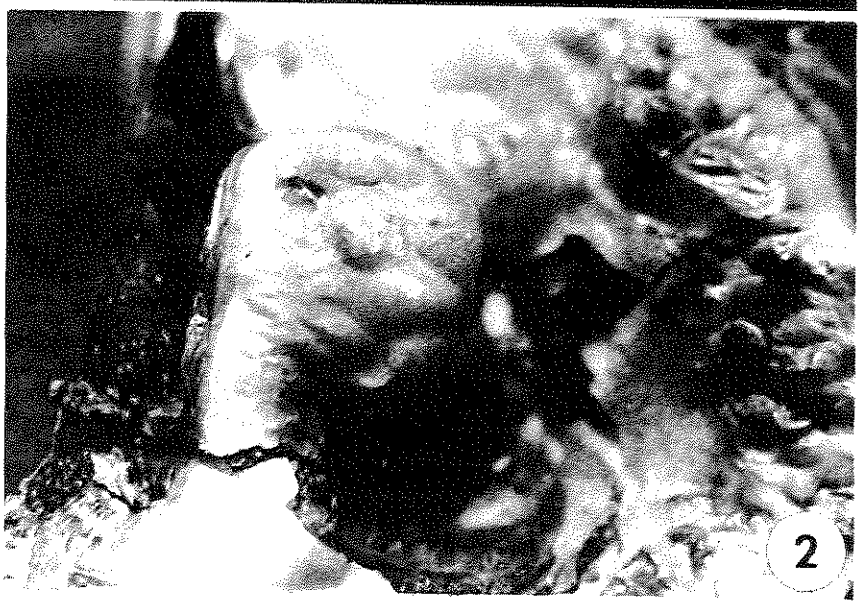

FIG. 1. Valves of an adult oyster, *C. virginica*, showing the effects of gas-bubble disease. The oyster survived the disease and appeared to be recovering.

FIG. 2. Close-up of a large shell blister on the right valve of the diseased oyster. The gas-filled conchiolin blisters had been covered with shell material when these pictures were taken.

taken from fish and lobsters suffering from gas-bubble disease.

As in fin fish (Harvey and Smith 1961; Renfro 1963), histological examination of diseased oysters revealed no pathogenic organism to which the symptoms of the disease could be attributed. The condition results when the animals' external environment is supersaturated with atmospheric gases.

Air forced into solution by suction through leaks in the intake side of a pump can supersaturate the water and cause gas-bubble disease (Marsh and Gorham 1905). Our water systems were carefully checked, but no leaks were found.

During the winter months, occasional failures in our heat exchangers subjected the molluscs to rapid and extreme temperature changes. Failure of the heaters caused the water temperature to drop from 20 C to 2–3 C in a few minutes. When the heaters were again in service, an influx of hot water quickly raised the temperature to 20 C. An experiment was conducted to determine whether this rapid increase in temperature and the resultant reduction in the gas-carrying capacity of the molluscs' body fluids was the cause of gas-bubble disease. Fifty adult oysters, obtained from local waters in February, were divided into five experimental groups and were subjected to the following treatments: 1) immediate initial introduction into 20 C water but without further temperature shock; 2) immediate initial introduction into 20 C water with seven weekly temperature shocks consisting of rapid cooling to 1–6 C, 6 hr of exposure to the cold water, and finally, rapid warming to 20 C; 3) gradual initial introduction into 20 C water with no subsequent temperature shock treatments; 4) gradual initial introduction into 20 C water with temperature shock treatments identical to those in treatment two; 5) maintenance in flowing water at the ambient temperature of 1–6 C with no temperature shock (control). Dissolved oxygen concentrations in the heated and ambient temperature sea water were monitored during the experiment. Oxygen concentrations as high as 129% of saturation were recorded in the 20 C water.

At the end of 8 weeks, examination of the surviving oysters for symptoms of the disease showed no significant relation between the temperature shock treatment and the incidence of disease. Sixty percent of the 20 oysters that were subjected to a weekly temperature shock (groups 2 and 4) showed some symptom of the disease. However, the same symptoms appeared in 75% of the oysters that had been held in water at a constant 20 C (groups 1 and 3). None of the control animals showed any symptoms of the disease.

We concluded that rapid temperature change was not the cause of gas-bubble disease in our brood stock animals. Rather, our observations indicate that the disease was caused by the long-term exposure of the animals to sea water that was supersaturated with atmospheric gases.

It is apparent that gas-bubble disease caused by the use of heated flowing sea water could be a hazard to many mariculture endeavors if preventive measures are not taken. We found that the excess gas could be driven off by allowing the water to cascade over a series of baffles or by strongly aerating it in a head tank before it entered the holding tanks. These precautions prevented occurrence of the disease in clams and oysters that were held in heated sea water during the winter.

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