TABLE 1.—*Regressions of carapace width on carapace length for male king crabs from three different areas in Alaska*

Area	Number of measurements	Regression equations[1]	Correlation coefficient
Chiniak Bay	106	$y = -14.34 + 1.2623x$	0.9992**
SE Bering Sea	89	$y = -14.11 + 1.2698x$	0.9996**
Kachemak Bay	86	$y = -15.12 + 1.2714x$	0.9956**

[1] y equals length; x equals width; measurements in millimeters.
** $p < 0.001$.

measurements for each 1-millimeter increment in length.

To determine if data were suitable for constructing a length–width conversion, two steps were undertaken as follows: (1) The data were plotted and linear regression equations calculated for each area; and (2) Tests were made for the significance of the regressions and of the differences between area slopes.

RESULTS

Regression equations for male king crab length–width data from the three areas are given in Table 1. There were no significant differences (0.01 probability level) among area slopes.

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Gas-Bubble Mortality of Fishes in Galveston Bay, Texas[1]

Gas-bubble "disease" in fish may result when their external environment is supersaturated with dissolved atmospheric gases. More specifically, the sum of the individual tensions of the gases dissolved in the water must exceed the hydrostatic pressure (Duodoroff, 1957). Under these conditions the fish's body fluids become supersaturated, and excess gas is soon released internally in the form of bubbles. These bubbles then circulate with the blood and gradually accumulate at constrictions. Since there is a continuous supply of dissolved gas diffusing from the water into the fish, the process continues until blood circulation is stopped.

The disease is occasionally encountered in hatcheries and fish-culture stations which use waters with high concentrations of dissolved gases, and special measures are required to prevent mortalities in these situations (Dannevig and Dannevig, 1950; Harvey and Smith, 1961).

At least two natural mortalities of fish due to gas-bubble disease have been reported. Woodbury (1941) described a fish kill caused by gas-bubble disease in black crappies, other centrarchids, northern pike, walleyes, and cyprinids in Lake Waubesa, Wisconsin. He noted dissolved oxygen concentrations as high as 327 percent of saturation, resulting from a dense bloom of *Chlamydomonas*. The fishes affected were mostly large mature adults; few small or young fish died. Rukavina and Varenika (1956) reported death of "Californian and stream trouts" caused by gas-bubble disease in two shallow ponds at the springs of the River Bosna, Yugoslavia. The disease occurred during hot sunny weather in late July 1956, when oxygen production from photosynthetic activity was especially high. No dissolved oxygen values were reported.

MORTALITY IN GALVESTON BAY

On the morning of January 14, 1959, an estimated 300 spotted seatrout, *Cynoscion nebulosus*, were found floating in upper Gal-

[1] Contribution No. 175, U. S. Bureau of Commercial Fisheries Biological Laboratory, Galveston, Texas.

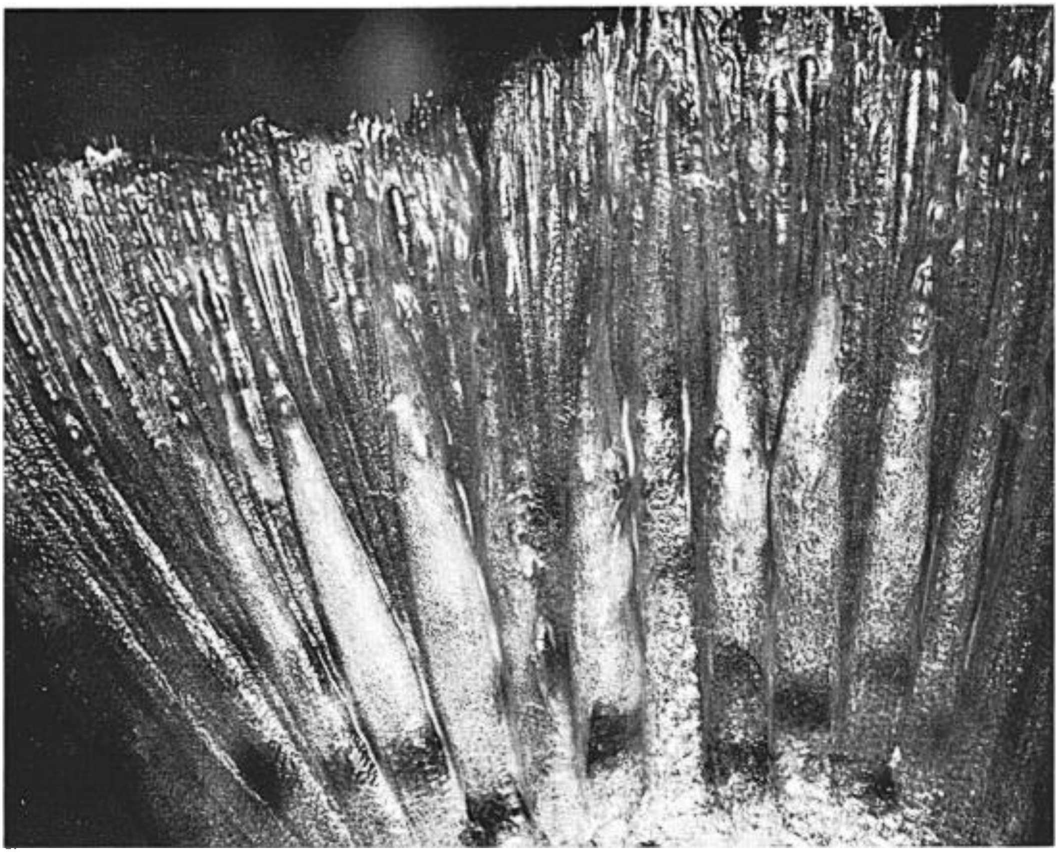

FIGURE 1.—Caudal fin of seatrout showing gas bubbles.

veston Bay. The dead seatrout were nearly all large. A sample of 23 had a mean standard length of 50 centimeters and a mean weight of 4.2 pounds. Other dead fishes noted were postlarval largescale menhaden, *Brevoortia patronus*; bay anchovies, *Anchoa mitchilli*; juvenile Atlantic croakers, *Micropogon undulatus*; small eels, probably *Myrophis punctatus*; and a longnose gar, *Lepisosteus osseus*. The fish had apparently died during the previous night, as rigor mortis had not yet occurred. They were being pushed toward shore by wind and wave action.

Although no external or internal physical injuries were evident, unusual features of the dead seatrout were the presence of excessive mucus on the body, bright red gills, distended swim bladders, and blisters containing gas in various parts of the body. These blisters were present within the membranes connecting fin rays (Figure 1) and occasionally under the skin on the sides of the body. Microscopic examination of the gills revealed most of the branchial arterioles to be completely filled with gas bubbles, which entirely displaced blood in the gill vascular system. Occlusion of blood circulation was presumed to be the primary cause of death.

This fish kill was observed in a portion of Galveston Bay which at times receives pollutants from the Houston Ship Channel, yet extensive chemical analyses for harmful toxicants all proved negative. Also, test fishes placed in samples of the water from the kill area lived for several months without apparent ill effects. Inspection of the area showed that water in this part of the bay was very turbid and colored rust-brown as a result of a dense plankton bloom. The predominant plankters, present in approximately equal numbers, were identified as a euglenoid, *Eutreptia* (*marina*), and two dinoflagellates, *Prorocentrum cutel-*

lum and *Gymnodinium (symplex)*. Dissolved oxygen concentrations remained near 250 percent of saturation throughout the day.

Prevailing climatic and hydrographic conditions favored the development and maintenance of very high dissolved-oxygen concentrations and, perhaps, supersaturation with nitrogen and other gases as well. For several days prior to the kill there was little cloud cover, and the resulting high insolation promoted intense photosynthesis. Winds were generally light and variable, barometric pressure was high but decreasing, and water temperatures were low but increasing. Under these conditions the body fluids of the fishes took on oxygen (or a mixture of atmospheric gases) in excess of their saturation values, and the surplus came out of solution as bubbles. The bubbles then accumulated, forming larger bubbles and obstructing circulation.

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