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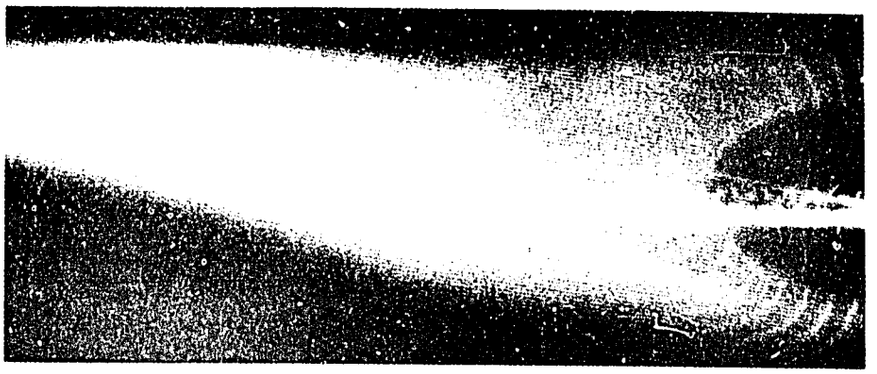

Curved spine of catfish shown in this radiograph is typical of the crooked back problem of fish deficient in vitamin C. Half-developed vertebrae shown were common in the vitamin deficient fish.

Catfish Need Vitamin C

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OVER FIFTY YEARS AGO ascorbic acid was recognized as the nutrient responsible for preventing scurvy in man and was given the title of vitamin C. All animals have a metabolic need for vitamin C, but it had been thought that only man, monkey, and guinea pig needed this vitamin in the diet. New research evidence now points to a need for vitamin C by catfish, disputing results of early research that indicated none was needed.

Since commercial catfish production has gained in prominence, many fish have been brought to the Cooperative Fish Disease Laboratory at Auburn showing disease symptoms but with no signs of infestation with pathogenic organisms. Affected fish all came from some type of modified culture environment where natural aquatic food was limited, and they had been intensively fed commercial type rations that were not supplemented with vitamin C.

The diseased fish usually showed either broken or crooked backs (lateral or vertical curvature of spine), white bands around the body just behind the dorsal fin, irregular surface swimming, tetany when handled, and poor growth and mortality, or a combination of these. Vitamin C deficiency was suspected as being responsible because none of these symptoms have been observed in Auburn research where catfish are grown in artificial cultures with diets supplemented with vitamin C. Neither have the symptoms appeared in catfish with access to significant amounts of pond organisms that are rich in ascorbic acid.

A study at Auburn University Agricultural Experiment Station measured response of channel catfish to vitamin C when grown from stocking to harvest size in a culture system with limited natural aquatic food and intensive feeding. Half of the 1,600 test fish got the following nutritionally adequate feed containing vitamin C:

Ingredient	*Per cent*
Corn	32.7
Herring meal	21.7
Peanut meal	20.3
Dehulled soybean meal	20.3
Dried corn fermentation solubles	6.7
Dicalcium phosphate	1.0
Vitamin mixture	.5
Vitamin C	.88

The other half of the fish got the same feed without the vitamin C.

The 1,600 fingerlings were stocked in four cages (39 x 39 x 39 in.) suspended in a 2.9-acre pond. Feed was allotted once daily, 6 days per week, at a rate beginning at 4% of fish weight and decreasing to 1.5% near end of the trial. Feed allotments were adjusted biweekly based on monthly samplings.

Approximately 25% of the fish from each cage were weighed monthly and inspected for signs of vitamin C deficiency. All fish were counted, weighed, and examined for gross signs of vitamin C deficiency at the end of the feeding period. Radiographs were made of sample fish from all treatments to study bone structure.

Based on growth response, survival, and incidence of deformed fish, the diet used was nutritionally adequate when supplemented with vitamin C. The fingerlings gained over 1 lb. in 180 days, with a favorable feed conversion ratio of 1.29 lb. feed per pound of weight gain. Without supplemental vitamin C, average gain was approximately 2/3 lb. with a feed conversion of 1.88.

Mortality was only 2% among fish fed the complete diet, but 22% for fish on the ration deficient in vitamin C. During the last 73 days of the feeding period, 8 to 12 dead fish per week were removed from cages of vitamin C deficient fish. Cause of deaths was attributed primarily to the bacterium *Aeromonas liquefaciens*.

Deformities were identified in 45% of the fish that got no vitamin C in the diet, as compared with only 4% with vitamin adequate feed. Most common deformity was a lateral curvature of the spinal column, usually about midlength of the fish. There was usually external swelling near the damaged area, often with a vertical white band 1/4 to 3/4 in. wide on the back.

Many fish also showed vertically curved spines, most commonly characterized by a large hump near and usually immediately behind the dorsal fin. Severely deformed fish had broken backs. Enlarged, spongy, or half-developed vertebrae were frequent. Hemorrhagic areas were common along the spinal column, particularly at point of injury. Many normal appearing fish had ruptured capillaries near the backbone.

Differences in fish weight and appearance between the two diets were not manifested until after the first 12 weeks of the feeding period, or until the fish weighed 1/5 to 1/3 lb. All fish fed actively until about the last 30 days of the test when the vitamin C deficient fish accepted feed more slowly.

The Auburn feeding trial is the first demonstration that rapidly growing channel catfish need ascorbic acid in feed to prevent extensive deformation of the spinal column, poor growth, and increased sensitivity to infection by bacteria. However, the "crooked back syndrome" may be caused by other factors, independently or through interaction with vitamin C deficiency. Deficiencies of tryptophan and vitamin E and exposure to high levels of various pesticides have caused similar abnormalities of fish.

Most commercial catfish feeds do not contain supplemental vitamin C, since there has been no previous indication of such need in the diet. Major feed ingredients like grains, oilseed meals, and animal byproducts contain only insignificant amounts of ascorbic acid, so commercial fish feed is generally lacking in this nutrient. Although fish probably get enough ascorbic acid from natural pond foods under low to moderate pond stocking density, the shift to intensive culture means that vitamin C is a necessary supplement for feeds.

Highlights of Agricultural Research, Vol. 20(2), Summer 1973. Agricultural Experiment Station, Auburn University. Auburn, Alabama 36830.