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centrations of this nitrogenous compound. Teleosts, on the other hand, have very low plasma and tissue concentrations of TMAO. In a teleost (the salmon) Benoit (1945) has shown that TMAO is exogenous in origin: TMAO was found in tissue only after food containing TMAO was fed. No data are available for the origin of TMAO in elasmobranchs.

A preliminary experiment was done to determine the constancy of the TMAO in the dogfish plasma. Five freshly caught dogfish in good condition were placed in a live car. Small blood samples were drawn at weekly intervals. It is presumed that no food was available to these fish in captivity. Of the 5 dogfish, one survived 14 days; one, 28 days; two, 34 days; and one was still alive at the end of the summer, 41 days after capture. All fish showed gross evidence of weight loss, but no accurate measurements of weight changes were made. There was no marked change in the plasma (TMAO) during captivity. The surviving fish maintained a mean plasma concentration of  $76 \pm 4 \mu\text{Mol/ml}$ . This compares well with a mean plasma (TMAO) of  $74 \pm 2 \mu\text{Mol/ml}$ . in 23 freshly caught dogfish.

It should be noted that if the kidney is the only route of TMAO excretion in the dogfish, and assuming a maximum loss of 10% of the total amount of TMAO filtered, a maximum of 20-25% of the estimated total TMAO in the dogfish could have been lost. The muscle of dogfish has been reported (Benoit) to contain  $140 \mu\text{Mol}$ . TMAO/Gm. and thus might serve as a reservoir for TMAO lost from the plasma during this period of starvation.

This study was carried out in a laboratory maintained by the New York University College of Medicine.

### **The Effect of Phlorozin on the Oxidative Metabolism of Certain Fish Kidneys**

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Studies have previously shown that phlorizin profoundly depresses the oxidative metabolism of rat and guinea pig homogenates. This compound, however, did not effect oxidative metabolism of kidney homogenates of flounder, goosfish and dogfish.

### **The Secretion of Hypertonic Salt Solutions In Marine Birds** Knut Schmidt-Nielsen, C. Barker Jørgensen and Humio Osaki Duke University

It has often been suggested that marine birds must drink sea water in order to cover their normal needs for water. This problem was investigated, using young cormorants as experimental animals.

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When nestlings and young adult birds in captivity were fed on fresh fish, the water content of the food was much higher than necessary for the renal excretion of the salts of the food, as well as for nitrogen (uric acid) excretion. Birds feeding on marine fish would therefore seem to be independent of drinking water, and ingestion of sea water would impair an otherwise favorable water and salt balance.

However, during experimental salt loads imposed orally or by infusion of hypertonic NaCl solution, it was found that cormorants react to an osmotic load in an unexpected way. Under such load they secrete a highly hypertonic fluid from the nose, the secretion dripping out from the internal nares and collecting at the tip of the beak, from which the bird shakes the drops with a sudden jerk of the head.

The concentration (500-600 mN NaCl) and the rate of secretion (up to 0.15 ml/min in a 1.5 kg bird) are so high that with continuous secretion the entire NaCl content of the body could be eliminated in roughly 10 hours. The secretion contains practically only sodium and chloride in nearly equivalent amounts.

The production of the nasal secretion is stimulated not only by NaCl, but also by a non-electrolytic osmotic load (sucrose). The secretion obtained in response to such stimulation is similar in composition to that obtained by stimulation with NaCl, indicating that the mechanism responds to general osmotic conditions, rather than specifically to NaCl concentration.

The physiology of this mechanism as well as the anatomy of the nasal region are being further investigated.

### Transpulmonary Passage of Leukemia Cells

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Experiments were designed to determine if cells, capable of producing leukemia, could pass immediately through the pulmonary circulation. The answer to this question is necessary to an understanding of the pathogenesis of leukemic infiltration. The transplantable B W 5147 lymphatic leukemia was used in A K R mice. Leukemia cells were injected intravenously and simultaneously the aortic blood was collected. This aortic blood was then injected intravenously into a second normal animal. If the second animal developed leukemia, this would indicate that cells capable of producing leukemia had passed through the lungs of the first animal. The mice used were 2 months old, and the second animals were sacrificed 1 month after the intravenous injection of aortic blood.

In one set of experiments the test cells were derived from a subcutaneous implant of B W 5147, and about 2 million leucocytes were used in each intravenous injection. In 6 out of ten such experiments, leukemia cells passed immediately through the lungs. In another set of experiments, diluted leukemia blood was used. The total number of leucocytes in each