adults, and free-swimming late fetuses (5 inches, rostral tip to midvent). Fetuses survive removal from the uterus.

Adult thyroids apparently are not hyperactive during gestation, as shown by uptake values 16-72 hours after subcutaneous injection of 24-175 microcuries (0.08 - 0.23 percent, which is less than the rabbit). Unlike some vertebrates tested, maternal thyroid activity does not decrease during late gestation, but is at a level similar to nonpregnant and male animals. Stomach fluid shows high activity, higher than uterus, blood, bile and intestine. Thyroids of late fetuses in utero show some radioactivity above blood and background 24 hours after maternal injection, but eyes may be slightly more active. Highest activity is in stomach, less in yolk.

Radioactivity appears in thyroids of free-swimming fetuses (subcutaneous injection, 5-9 microcuries) within first 5 minutes, increases progressively during following hours (0.11-1.9 percent uptake), and amounts within minutes to as much, or later more than (2-10 times), that of maternal thyroid (weight approximately 230 mg). Thyroidal activity is generally 1.3-5 times that of blood after first few hours when compared with almost the entire fetal blood volume; stomach 1.2-4 times, intestine 1.4-2 times and yolksac yolk is much lower. Radioactivity of stomach is greater than intestine, uterine fluid and eyes in all cases, and may be higher than thyroid especially in first few hours. Abdominal yolk and eyes may show higher radioactivity than blood.

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The Excretion of Ammonia by the Gills of the Fresh-Water Catfish, *Ameiurus nebulosus*

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The excretion of ammonia by the gills of fish constitutes one major path of nitrogen excretion. This study was planned to determine whether this branchial process contributes to the maintenance of acid-base balance as does renal ammonia excretion in the frog, dog, and man. In fresh-water catfish (140 to 250 grams) the anus was ligated, and the urinary papilla was either ligated or catheterized for the collection of urine. The fish were placed in closed plastic containers with 400 ml distilled water through which oxygen was bubbled continuously. No ammonia escaped in the excurrent gas. The ammonia in the bath after 90 to 200 minutes is considered to have been excreted by the gills, assuming a negligible contribution from the skin and buccal mucosa.

Control branchial excretion of ammonia ranged between 15 and 70 μEq per 100 gm of fish per hour. When ammonia accumulated in the bath in successive periods, or when ammonium sulfate was added to the bath,

the rate of ammonia excretion decreased. Intraperitoneal injection of 1 mEq HCl produced a marked acidosis, but was followed by a reduction in the rate of ammonia excretion to 47-63 per cent of control values in 4 experiments. Alkalosis following intraperitoneal injection of 1.1 or 1.5 mEq $NaHCO_3$ was accompanied by an increase in ammonia excretion in 4 experiments. On the other hand, intraperitoneal injection of 0.6 or 1.0 mEq NH_4Cl produced a mild acidosis and increased ammonia excretion two- to six-fold over control periods in the 5 out of 6 fish which survived this procedure. In the latter experiments, 6 to 78 per cent of the injected ammonia was excreted via the gills within 2 hours, while the urine contained less than 4 per cent when collected after four hours. The final concentration of ammonia in the bath at the end of these experiments was two or three times its concentration in the blood. This occurred even when the blood was more acid than the bath, and on this account cannot be explained as simple diffusion of free ammonia. Excretion of ammonia against a gradient of 2 mEq per liter ammonium sulfate was still two-thirds as efficient after intraperitoneal administration of 25 mg iodoacetate or 1 mg 2, 4-dinitrophenol. These agents produced a mild acidosis as well, and if the acidosis was prevented by the simultaneous administration of 1 mEq $NaHCO_3$, no decrease in branchial ammonia excretion was seen.

These results indicate that branchial excretion of ammonia does not contribute to acid-base balance in the catfish. In the frog, dog, and man (and possibly the catfish, as well), in which urinary ammonia excretion is increased during acidosis, the acidotic state is accompanied by a decrease in the pH of the urine, favoring diffusion of free ammonia from the tubule cells and capture in the tubule lumen. In contrast to the kidneys, the gills of the catfish do not produce a local acid environment in acidosis, and the effect of acidosis is to decrease the gradient for free ammonia between the blood and the bath. This decrease in gradient diminishes ammonia output across the gills, in a fashion analogous to the effect of adding ammonium salts to the bath water.