samples were obtained from the portal vein inflow cannula and hepatic venous sinus outflow. O_2 content w determined using a polaragraphic O_2 electrode (Radiometer, Copenhagen) equipped with a constant temperatu cell maintained at 15°C (kindly provided by Dr. Patricio Silva). O_2 consumption was calculated using a solubility constant of 34 µl O_2 ml^{-1} Ringers buffer at 760 mm Hg and 15°C, and was expressed as µmoles O_2 min^{-1} g^{-1} liver after correction for the appropriate perfusate flow rate. As shown in Figure 1, O_2 consu

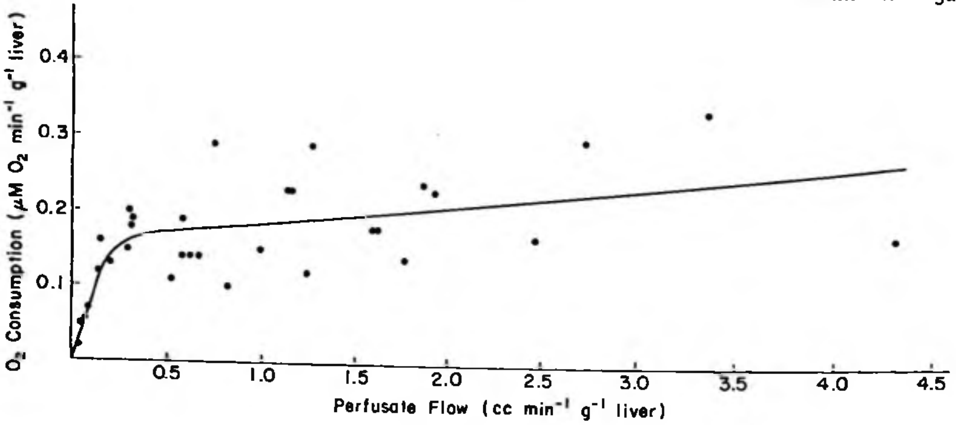

Figure 1

rose steadily as perfusate flow increased from 0.05-0.40 ml min^{-1} g^{-1} liver but at higher flow rates remai essentially constant. Although there was variation in the amount of O_2 consumed between individual experiments, O_2 consumption remained relatively constant within individual experiments over 4 hours. We conclu that perfusate pressures of 1.5-2.5 cm H_2O, which achieve perfusate flows greater than 0.40 ml min^{-1} g^{-1} liver, provide optimum O_2 delivery in this isolated perfused skate liver model.

BILE SECRETORY FUNCTION IN ISOLATED PERFUSED LIVER OF THE LITTLE SKATE, *Raja erinacea*. IV. STUDIES OF INTERCELLULAR JUNCTIONAL PERMEABILITY

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Previous studies in the isolated perfused skate liver (Bulletin, MDIBL, 1976) demonstrated high bile to plasma inulin ratios and also suggested that hydrostatic filtration may contribute to the formation of bile in this species, unlike the isolated perfused rat liver. These findings suggest that water and solu might enter bile in part by passage through intercellular junctional complexes at the bile canaliculus, a conclusion consistent with previous electron micrographs which suggested that these junctions were leaky (Bulletin, MDIBL, 1974). To define more clearly the permeability of these paracellular pathways, we util ionic lanthanum ($LaCl_3$) which previously has been used to demonstrate junctional complex permeability in epithelial tissues (Martinez-Palomo, Erlij and Bracho J. Cell Biol. 50:277, 1971). In four studies a w oxygenated Tris-buffer Elasmobranch Ringer's (NaCl 286 mM, urea 350 mM, $CaCl_2$ 5 mM, $MgCl_2 \cdot 6H_2O$ 3 mM, Tris buffer 3 mM, pH 7.40) containing 3-5 mM $LaCl_3$ was exchanged for the standard Elasmobranch Ringer's buffer with bicarbonate. After perfusion for 10 min at 2.5 cm H_2O pressure, the liver was fixed by perfusion wi 2.5% glutaraldehyde in 0.1 M cacodylate buffer. Sections of liver were examined by electron microscopy. shown by Figure 1, the electron dense lanthanum could occasionally be demonstrated within the junctional complex at the bile canaliculus as well as within the canalicular lumen. These findings provide more dir evidence for biliary permeability to water and electrolytes at the level of the junctional complex which separates the canalicular lumen from the lateral intercellular space. Paracellular pathways may be important in the formation of bile in this species.

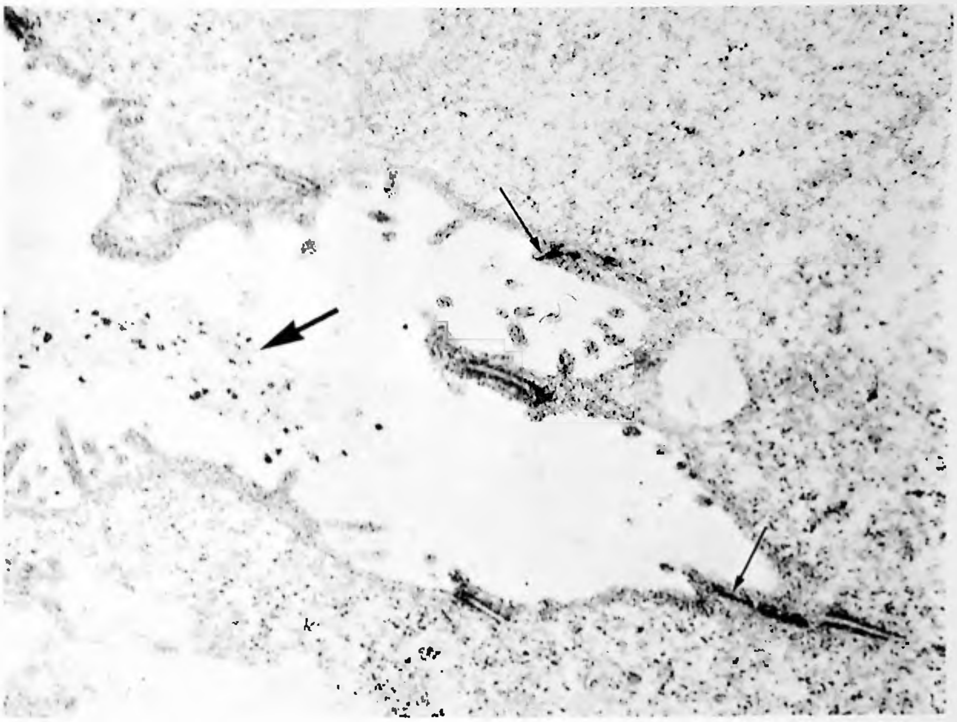

Figure 1. Representative Electromicrograph (X 25,000) of a skate liver canaliculus cut obliquely. Arrows show electron dense material (lanthanum) in both the junctional complexes (small arrows) and the canalicular lumen (large arrow). Stained with uranyl acetate and lead citrate.

MECHANISM OF CHLORIDE TRANSPORT IN THE RECTAL GLAND OF *Squalus acanthias*: IONIC SELECTIVITY

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The rectal gland of the spiny dogfish, *Squalus acanthias*, secretes chloride against an electrochemical gradient. It has been postulated that the active movement of chloride into the rectal gland cell is linked with the passive movement of sodium down its electrochemical gradient across the basolateral membrane. The gradient for sodium is maintained by the continued activity of Na-K-ATPase. Experimental observations using an isolated perfused rectal gland preparation have confirmed that the transepithelial movement of chloride in the rectal gland depends on the presence of sodium outside the cell (Bull. MDIBL 16:93-96, 1976) suggesting that the entry of chloride into the cell is coupled to that of sodium. We have also shown that ouabain 10^{-4} M blocks the transepithelial movement of chloride, providing evidence that Na-K-ATPase is energizing the process (Bull. MDIBL 15:69-71, 1975). If sodium and chloride enter the cell via a tightly linked carrier mechanism, the operation of this carrier would require chloride, and the secretion of both sodium and chloride will depend on the selectivity of the carrier mechanism for either ion. The experiments reported here were designed to explore these questions using the isolated perfused rectal gland. In addition, we sought to define further the participation of Na-K-ATPase in the process of chloride transport by inhibiting its activity while avoiding the use of ouabain.