Pharmacological evidence for the transport of glucose through a symporter in the rectal gland of *Squalus acanthias*

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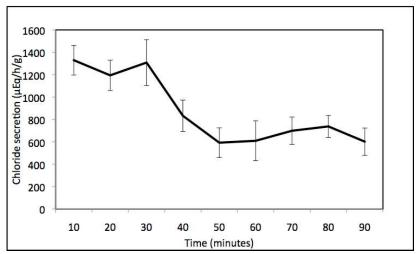
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Glucose is the fuel that provides energy to the cells of the rectal gland. Glucose requires a transporter to enter the cells. This report supports the notion that glucose enters the cell through such a transporter.

The secretion of chloride by the rectal gland of *S. acanthias* is fueled by glucose. Glucose enters the rectal gland cells via a sodium dependent glucose symporter that is SGLT1.^{1,3} The entry of glucose into the cell is prevented by phloridzin that inhibits glucose transport through glucose symporters.¹ a-methyl D-glucoside, an analog of glucose that is not metabolized, is also transported by these symporters and effectively competes with the transport of glucose.² To characterize further the mode of uptake of glucose into the rectal gland cells we used a-methyl D-glucoside to determine whether it blocked the uptake of glucose.

Isolated rectal glands of *S. acanthias* were each perfused through the single artery by gravity at 16° C and 40 mm Hg pressure with oxygenated shark Ringer's solution containing 5 mM glucose, and 5 x 10^{-4} M theophylline, in a single pass perfusion. Venous effluent and duct fluid were collected separately from PE-90 catheters placed in the vein and duct of the gland. Collections were made every ten minutes. After thirty minutes of perfusion with theophylline, the perfusate was changed to an experimental perfusate that also contained 20 mM α -methyl D-glucoside, and collections continued at ten-minute intervals. Reagents were purchased from Sigma-Aldrich. Chloride was measured using a Buchler-Cotlove chlorhidrometer. Chloride secretion was calculated from the chloride concentration in the duct fluid, the volume of the fluid, the collection time, and the weight of the gland and expressed as μ Eq per gram of gland per hour. Statistical analysis was done using ANOVA.

Figure 1. Effect of a-methyl D-glucoside on chloride secretion by the rectal gland of S. acanthias. α -methyl D-glucoside, 20 mM, given at the end of thirty minutes of control perfusion reduced the secretion of chloride. Mean values \pm SD from 5 experiments.



The results are shown in Figure 1. The addition of a-methyl D-glucoside to the perfusate of the glands after thirty minutes of control perfusion, reduced the secretion of chloride by about 50%. A reduction similar to that had been seen when glucose was removed from the perfusate, or the transport of glucose was inhibited with phloridzin.¹

These experiments provide additional support to the notion that the transport of glucose into the cells of the

rectal gland is mediated by a SGLT type symporter, in the case of the rectal gland SGLT1. This type of symporter, in contrast to the GLUT family, transports α-methyl D-glucoside, that competitively inhibits the transport of glucose, and is inhibited by phloridzin, both of which inhibit the glucose-dependent secretion of chloride by the rectal gland. In addition, the finding that inhibition of glucose transport with a-methyl Dglucoside has a similar effect as the removal of glucose suggests that only one type of transporter mediates the entry of glucose into the rectal gland cell.

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