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CARTAS AL EDITOR

Study of the Inhibitory Effect of Serotonin on Sugar Intestinal Transport

The patterns of storage (1) and release of serotonin (4, 5) found in enterochromaffin cells of the intestinal mucosa suggest that local paracrine/neuroendocrine actions mediated by serotonin may be important modulators of intestinal functions (6, 7).

Recently published studies from our laboratory (2) have shown that serotonin produces a reduction of D-galactose absorption across rabbit jejunum. This effect seems to be located on the Na^+-dependent uptake across the mucosal border.

A number of experiments have presently been carried out in order to corroborate this inhibitory effect of serotonin on D-galactose Na^+-dependent intestinal transport.

Male New Zealand rabbits weighing 2.0-2.5 kg were maintained on a standard rabbit diet with free access to water. After killing by a blow on the head, the proximal jejunum (5 cm distal to the ligament of Treitz) was removed and rinsed free of intestinal contents with ice-cold Ringer's solution. The tissue was then stripped of its serosal and external muscle layers. The stripped mucosa was mounted as a flat sheet in Ussing-type chambers. The bathing solution on the mucosal and serosal surfaces of the tissue were maintained at 37 °C using a circulating water bath. Both solutions contained D-galactose at the same concentration. Four concentrations of D-galactose (4, 10, 20 and 50 mM) and two of serotonin (10^{-7} and 10^{-6} M) were assayed. Mucosal to serosal fluxes (Jms) were measured by placing ^{14}C-labelled galactose in the mucosal side. Samples were removed from the non radioactively labelled side at 20 min intervals for 80 min, after 60 min preincubation period. Only one sample was taken for counting from the radioactively labelled side and samples of the radioactive solution were counted using a liquid scintillation counter.

The D-galactose transport across the mucosal border of the intestine presents two components: a Na^+-dependent mechanism, which includes S1 and S2 saturable systems of transport (3), and a diffusive process.

Previous fluxes determinations were carried out at different D-galactose concentrations and with or without Na^+ in the medium to obtain the diffusive component.

Subtracting the amount of sugar absorbed in absence of Na^+ from that absorbed in its presence, allows the obtention of the uptake value via Na^+-dependent saturable transport.

The results obtained have been repre-

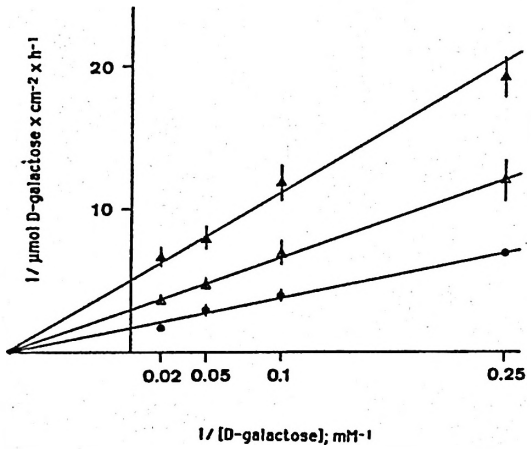

Fig. 1. *Lineweaver-Burk plot of the rate of Na^+-dependent D-galactose absorption measured under different conditions.*
● Control ([5HT] = 0 mM); △ [5HT] = 10^{-7} M; ▲ [5HT] = 10^{-6} M. Values have been corrected for the Na^+-independent transport.

sented as an apparent kinetic study (fig. 1), and show that serotonin diminishes maximal Jms but does not modify the «apparent Michaelis constant» Kt of the D-galactose Na^+-dependent intestinal transport.

The effect of serotonin on D-galactose intestinal transport could be caused by a local effect of the hormone on the intestine. Previous experiments carried out in our laboratory have shown that the effect of serotonin might be mediated by the binding of serotonin to its receptors located in the intestinal mucosa (2). The present study confirms that serotonin inhibits D-galactose intestinal absorption. This inhibition might be caused by the serotonin-receptor complex which would mainly modify the mucosal to serosal maximal influx mediated by the mucosal Na^+-dependent carrier of D-galactose, with no action on the affinity of D-galactose by this carrier. Further studies attempt to determine the physiological substances that might be responsible for the effects of serotonin on sugar intestinal transport.

Key words: Serotonin, Sugar transport, Intestine.

Palabras clave: Serotonina, Transporte de azúcares, Intestino.

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