Exp Biol Med (Maywood). 2006 Mar;231(3):277-81. doi: 10.1177/153537020623100306.

Sodium–iodide symporter mediates iodide secretion in rat gastric mucosa in vitro

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PMID: 16514173 DOI: 10.1177/153537020623100306

Abstract

In vivo studies on rats have demonstrated that considerable amounts of iodide are transported from the bloodstream into the gastric lumen. The mechanisms for and functional significance of this transport are poorly understood. Active (driven by Na(+)/K(+)-ATPase) iodide transport into thyroid follicular cells is mediated by the sodium-iodide symporter (NIS), which is also abundantly expressed in gastric mucosa. We aimed to further investigate the iodide transport in gastric mucosa and the possible role of NIS in this transport process. Iodide transport in rat gastric mucosa was studied in vitro in an Ussing chamber system using (125)I as a marker. The system allows measurements in both directions over a mucosal specimen. A considerable transport of iodide (from the serosal to the mucosal side) was established across the gastric mucosa, whereas in the opposite direction (mucosa to serosa), iodide transport was negligible. Sodium perchlorate (NaClO(4)), a competitive inhibitor of NIS, and ouabain, an inhibitor of the Na(+)/K(+)-ATPase, both attenuated gastric iodide transport from the serosal to the mucosal side. To investigate a possible neuroendocrine regulation of the iodide transport identified to occur from the serosal to the mucosal side of the stomach, thyroid-stimulating hormone (TSH), thyrotropin-releasing hormone (TRH), vasoactive intestinal peptide (VIP), histamine, or nitric oxide donor S-nitroso-N-acetyl-D,L-penicillamine (SNAP) was added. None of these substances influenced the iodide transport. We conclude that iodide is actively transported into the gastric lumen and that this transport is at least partly mediated by NIS. Additional investigations are needed to understand the regulation and significance of this transport.

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