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The importance of the gastroduodenal axis in the control of body sodium homeostasis and blood pressure

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In order for normal sodium balance to occur, the amount of sodium that is ingested must equal the amount of sodium that is excreted, mainly in the urine. One mechanism by which sodium balance is regulated involves the sensing of ingested sodium by gastrin secreting cells (G-cells) in the stomach. An increase in intracellular sodium concentration in G-cells in the stomach, in conjunction with D1-like dopamine receptors, increases the transcription and secretion of gastrin. Of all the gut hormones released into the circulation, gastrin is the one that is taken up to the greatest extent by renal tubules. Gastrin transported into renal proximal tubules enhances the renal uptake of circulating L-3, 4-dihydroxyphenylalanine which is decarboxylated to form dopamine. Gastrin, via the cholecystokinin B receptor, and dopamine, via its five dopamine receptor subtypes (D₁R, D₂R, D₃R, D₄R, and D₅R), decrease renal tubular sodium reabsorption, in part, via inhibition of NHE3 and Na⁺, K⁺-ATPase activity, resulting in a natriuresis. Excess sodium is excreted and blood pressure remains in the normal range. However, in the presence of variants of G protein-coupled receptor kinase type 4, dopamine receptors (D₁R and D₃R) are desensitized, impairing their ability to inhibit renal tubular sodium transport. When excess sodium is retained and is not buffered in the interstitial space/lymph, blood pressure increases. Understanding the role of genes and gene-gene and protein-protein interaction in the regulation of renal function and blood pressure may lead to the tailoring of anti-hypertensive treatment based on genetic make-up (pharmacogenomics).

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