



## Hormonal control of gastrointestinal motility

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Many hormones or peptides found in the gastrointestinal tract and endocrine pancreas are known to influence the motor function of gastrointestinal tract including the esophagus, stomach, small and large intestines, gallbladder and sphincter of Oddi. The peptides released from these organs that are shown to influence the motility include secretin, gastrin, cholecystokinin, somatostatin, glucagon, gut glucagon-like peptide-1 (GLP-1), motilin, neurotensin, peptide YY, pancreatic polypeptide, and several neuropeptides including vasoactive intestinal polypeptide (VIP), opioids, substance P, galanin, calcitonin gene related peptide and bombesin or gastrin releasing peptide. Only a handful of these peptides are shown to exert their actions in physiological conditions.

To be qualified as a hormone, a given peptide must meet the following criteria: (1) In response to a physiological stimulant such as a meal, it must increase in the circulation. (2) Parenteral administration of a peptide in an amount that mimicks the circulating peptide level can produce a predictable biological response, and (3) The biologic action by an endogenous peptide on a target organ or tissue is completely or profoundly suppressed by either a specific receptor antagonist or immunoneutralization of the circulating peptide or hormone with specific antibody. If the action of a peptide is mediated via either paracrine or neurocrine pathway, a circulatory hormone or peptide concentration may be too low to be detected by radioimmunoassay, whereas its local tissue concentration is very high. Similarly, a specific polyclonal or monoclonal antibody or receptor antagonist blocks predicted biologic action.

The peptides that meet these 3 criteria are: Cholecystokinin, secretin, PYY, motilin, and possibly, somatostatin, neurotensin and

gut glucagon peptide-1.

### CHOLECYSTOKININ (CCK)

Four physiologic actions have been identified: (1) Contraction of the gallbladder mediated via local release of acetylcholine, (2) Relaxation of the sphincter of Oddi mediated by CCK-induced release of VIP, (3) Inhibition of gastric emptying mediated via capsaicin-sensitive sensory vagal afferent pathway, to induce local release of VIP in the fundus and contraction of pyloric sphincter, and (4) Increased contraction of the distal colon probably mediated via local release of acetylcholine.

### SECRETIN

This hormone inhibits gastric emptying which appears to be mediated by capsaicin-sensitive sensory vagal afferent pathway. Although it has been shown to inhibit the motility of duodenum and the remaining small intestine when given intravenously, it has not been proven whether its action is physiological. Peptide YY (PYY): Although PYY containing endocrine cells (L-cells) are found throughout the entire intestinal and colonic mucosa, they are found in high density in the distal ileum and colon. The peptide released mainly by fat digests, inhibits gastric emptying and prolongs transit time of small intestine and colon. PYY is probably the major hormone participating in ileal brake mechanism. In addition, if given intravenously, it prolongs duration of cycle of interdigestive migrating motor complex of the stomach and duodenum.

### MOTILIN

It has been convincingly shown that motilin triggers phase 3 activities of gastric antrum and upper small intestine in interdigestive period. Although it stimulates gastric and upper intestinal motility to enhance gastric emptying, it is yet to be decisively determined whether or not its action is physiological.

### CANDIDATE HORMONES

Although somatostatin, neurotensin and glucagon-like peptide-1 influence the motility of gastrointestinal tract, these peptides have not fulfilled the criteria for their hormonal status. Somatostatin was shown to inhibit and inhibit ileal motility. In addition, it modulates human interdigestive motility of small intestine by shortening the interdigestive motility cycle when it is given intravenously in a physiological dose. It also stimulates esophageal body contraction, and decreases lower esophageal sphincter pressure (LESP). Neurotensin decreases LESP and delays gastric emptying and intestinal transit time, but it stimulates colonic motility. Glucagon-like Peptide-1 released by fatty acid from the distal ileum and colon, delays gastric emptying. Thus it appears to participate in the ileal brake mecha-

nism to inhibit small intestinal motility.

## PHYSIOLOGICAL SIGNIFICANCE

In response to ingestion of a meal, classic gut hormones such as gastrin, secretin, CCK, PYY are released into the circulation as acid gastric chyme enters the duodenal lumen. The latter 3 hormones play important roles on the control of gastrointestinal motility and transit. It is apparent that duodenal brake for control of gastric motility and emptying is induced mainly by secretin and CCK, and ileal brake mechanism for gastric emptying and intestinal transit is controlled by PYY and probably by GLP-1. Based on recent observa-

tions that the action of both CCK and secretin on gastric emptying is mediated via capsaicin sensitive vagal afferent pathway, the hormonal action on the stomach motility requires interaction with both peripheral and central nervous system, "gut-brain interaction". Thus the gut hormones released in physiological conditions can not exert their actions without the neuronal participation.

It is also apparent that the "enterogastric" proposed by Kosaka and Lim in Beijing, China in 1930, basing on their historical experiment carried out in dogs, turns out to be those peptides released by fat digests from the intestine that regulates gastrointestinal motility. Secretin, CCK and PYY are some of those peptides.

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