THE ROLE OF GASTROINTESTINAL HORMONES IN THE PATHOLOGY OF THE DIGESTIVE SYSTEM.

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Annotation: The functioning of the digestive system, the conjugation of motility, secretion and absorption are regulated by a complex system of nervous and humoral mechanisms. There are three main mechanisms of regulation of the digestive apparatus: central reflex, humoral and local. Gastrointestinal hormones play an important role in the humoral regulation of digestive functions. These substances are produced by endocrine cells of the mucous membrane of the stomach, duodenum, pancreas and are peptides and amines. Gastrointestinal hormones are involved in the regulation of secretion, motility, absorption, trophism, release of other regulatory peptides, and also have general effects: changes in metabolism, activity of the cardiovascular and endocrine systems, and eating behavior.

Keywords: regulatory peptides, metabolism, endocrine systems.

Gastrointestinal (gi) hormones are chemical messengers that regulate intestinal and pancreatic function, including regulation of secretion, motility, absorption, digestion, and cell proliferation. These hormones are secreted by endocrine cells, which are widely distributed throughout the GI mucosa and pancreas. Although these hormones were initially described as solely endocrine products, subsequent studies have shown that they can act in an autocrine or paracrine fashion to affect cellular function. GI hormones may also serve as transmitting agents for nervous impulses discharged into blood vessels after nervous stimulation in a true neurocrine fashion. This review will focus on the trophic effects of GI hormones on nonneoplastic and neoplastic tissues. The presence of gastrin was postulated in 1906 by Edkins, but it was not until 1938 that Komarov prepared useful, histamine-free antral extracts of this gastric stimulant. Gastrin is now firmly established as the physiological regulator of gastric acid secretion and an important regulator of gastric mucosal cell proliferation. The major site of gastrin synthesis and secretion is the gastrin-containing cell (G cell) in the antropyloric mucosa (4–6). Other minor sites of gastrin production include the endocrine cells of the pancreas (7), pituitary (8), and extraantral G cells (9). Gastrin release is stimulated by food components, particularly aromatic amino acids and amine derivatives of amino acids, and is inhibited by luminal acid .

Among the multiple intracellular signaling pathways that mediate the proliferative effects of GPCRs, a family of related serine-threonine kinases, collectively known as ERKs or MAPKs, appear to play a central role (70). After phosphorylation by their immediate upstream MAPK kinase, members of the MAPK family translocate to the nucleus, where they phosphorylate transcription factors, thus regulating the expression of genes that control growth (71). Hormones act as ligands to eventually activate p42 and p44 MAPK (Fig. 2B) (72). The mechanism by which this occurs involves a complex interplay of several known nonreceptor kinases and receptor kinases. The ability of tyrosine kinase inhibitors to reduce the activation of MAPK by GPCR (73) and the rapid tyrosine phosphorylation of Shc (Src homology and collagen) after GPCR stimulation with the consequent formation of Shc-Grb2 (growth factor receptor-bound 2) complexes (74) provide evidence that tyrosine kinases link GPCRs to the Ras-MAPK pathway.

Additionally, GPCRs link to the Jun N-terminal kinase (JNK), p38 MAPK, and the big mitogen-activated kinase 1 or ERK5 pathways (Fig. 2C) (68). JNK, also termed stress-activated protein kinase (SAPK), is structurally related to MAPK, but the pathways used by GPCRs to

activate these kinases are different. Activated Rac and Cdc42 affect JNK activation through stimulation from free βγ-dimers and Gα12 and Gα13. Four p38 MAPKs have been described, p38 α (CSBP-1), p38 β, p38 γ (ERK6 or SAPK3), and p38δ (SAPK4). Yamauchi demonstrated that Gαq and βγ dimers activate p38α. Two nonreceptor tyrosine kinases, Btk and Src (77, 78), have been associated with this process. ERK5 can be activated by oxidative stress and plays a role in early gene expression. GPCRs can potently stimulate ERK5 through a mechanism that involves Gαq and Gα13, independent of Rho, Rac1, and Cdc42 (80, 81). Furthermore, ERK5 regulates early gene expression through the phosphorylation of the transcription factor, myocyte enhancer factor 2.

The molecular mechanisms through which GPCRs transduce signals are complex and likely involve multiple signal pathways. In addition, the signaling pathways are likely cell-specific, which may explain the diverse physiological functions controlled by GI hormones ranging from regulation of secretion, mobility, and, in some instances, growth depending upon the target tissue.

Human progastrin, the precursor of gastrin, consists of a 21-amino-acid signal peptide, a 37-amino-acid N-terminal extension, the gastrin-34 sequence, and a 9-amino-acid C-terminal extension . In antral G cells, progastrin is stored and processed into secretory granules, and N-terminal and C-terminal extensions are removed by prohormone convertases. The C-terminal basic amino acids are sequentially removed by a carboxypeptidase, which results in the formation of glycine-extended G34 (G34-gly). G34-gly is amidated by peptidyl glycine α-amidating monooxygenase to form G34-NH2 (G-34) or cleaved at internal lysine/lysine residues to form G-17-gly (referred to as G-gly) (10). Recent studies have demonstrated that the majority of G-17-NH2 (also known as gastrin or G-17) arises from the conversion of G34-NH2 to G-17-NH2, rather than by amidation of G-gly, suggesting that the conversion of G-gly to amidated G-17 is blocked and that G-gly is a terminal, secondary end-product of progastrin processing in normal antral G cells. This is contrasted by evidence in colon cancer cells that gastrin bypasses the processing machinery and is expressed in larger, unprocessed forms that are transported in secretory vesicles and continuously fused with the plasma membrane and released.

Gastrin is synthesized by G-cells located in the mucous membrane of the antrum of the stomach (in the middle zone of the pyloric glands) and in the crypts, villi, Brunner glands of the duodenum. In the catabolism of gastrin, the small intestine and kidneys play a significant role, and the liver plays a much less important role in the degradation of natural gastrin. Along with the main type of action of gastrin on the secretory activity of the stomach - by direct stimulation of the parietal and chief cells after binding to their receptors - in recent years, the effect of gastrin mediated by the central nervous system on the functions of the stomach has been discussed.

Almost all researchers do not doubt the prevailing role of the endocrine type of the mechanism of action of gastrin, i.e. direct influence of gastrin synthesized by G-cells and entered into the blood on target tissues (stomach, pancreas). An increase in intragastric pH is a physiological stimulus for gastrin incretion. Gastrin and its synthetic pentapeptide (pentagastrin, reproducing essentially all the effects of antral hormone) significantly increase the functional activity of the mass of parietal and chief cells of the fundic mucosa, cause an increase in the debit of hydrochloric acid and pepsin, depending on the rate of incretion of the endogenous hormone or the dose of gastrin (pentagastrin) administered from the outside . Since the blood supply to the gastric mucosa largely ensures its functional activity, it should be noted that not only in animal experiments, but also in human studies, the regular increase in blood flow in the fundic part of the gastric mucosa with pent gastrin should be noted. Gastrin enhances the flow of prostaglandin E2

into the gastric juice both in animals and in humans after the administration of gastrin or pent gastrin.

This fact complements the information about the trophic effect of gastrin on the gastric mucosa. Gastrin and pent gastrin increase the tone of the lower esophageal sphincter, enhancing the barrier function of this barrier to gastroesophageal reflux. The trophic effect of gastrin on the exocrine tissue of the pancreas has been shown. With intravenous administration of gastrin and pent gastrin to animals and humans, a significant increase in the concentration and flow rate of pancreatic bicarbonates and enzymes is noted. According to the Dnipropetrovsk Research Institute of Gastroenterology, gastrin and pent gastrin have analgesic and antiasthenic morphine-like effects in diseases of the digestive system, lasting from 5 hours to 2-3 days after intravenous, intramuscular, intranasal or sublingual administration of the drug.

With hypergastrinemia develops: a tumor of the islets of Langerhans of the pancreas; a sharp increase in the secretion of hydrochloric acid by the stomach; diarrhea (due to the formation of an acidic environment in the duodenum, unfavorable for the action of pancreatic and intestinal enzymes; inhibitory effect of gastrin on the absorption of water and salts in the small intestine; gastric metaplasia in the mucous membrane of the small intestine; multiple gastroduodenal ulcers, often accompanied by hemorrhages, perforation, penetration into neighboring organs; Violation of gastrin incretion is noted in chronic gastritis, chronic duodenitis, gastric and duodenal ulcers, dumping syndrome and some other diseases of the gastrointestinal tract.

Somatostatin was isolated and characterized from ovine hypothalamic tissue during a search for a GH-releasing factor. Since the identification and purification of somatostation-14, precursor forms of greater molecular weight, including somatostatin-28, with somatostatin-14 making up the C terminus, and larger precursor forms of 120 or more amino acids have been identified . All of these peptides exert biological activity but differ in their relative potency. Somatostatin has been detected in the nerves and cell bodies of the central and peripheral nervous systems, including the autonomic nervous system of the GI tract and the endocrine-like D cells of the pancreatic islets and mucosa of the stomach and intestine (3). More than 90% of the somatostatin immunoreactivity in the human gut is located within the mucosal endocrine D cells (58). In addition, somatostatin is located in the nerves of the myenteric plexus. Somatostatin in the pancreas is located in the D cells at the periphery of the islets closely associated with the α-cells .

Somatostatin is a regulatory-inhibitory peptide, which, in contrast to BBS/GRP, may be considered as the universal endocrine off-switch. Somatostatin inhibits the release of GH and somatomedin C and all known GI hormones (3). Somatostatin also inhibits gastric acid secretion and motility, intestinal absorption, and pancreatic bicarbonate and enzyme secretion, and selectively decreases splanchnic and portal blood flow . In addition, somatostatin can inhibit the growth of normal and neoplastic tissues .

Back in 1928, Ivy (Ivy) and Oldberg (Oldberg) designated the term "cholecystokinin" extractable from the intestinal mucosa hormonal factor that causes contraction of the gallbladder. Fifteen years later, Harper and Raper reported that an extract from the small intestine mucosa stimulated the secretion of pancreatic enzymes and named the hormone pancreozymin responsible for this effect. Classical studies on the purification of cholecystokinin and pancreozymin preparations carried out in 1964 (Jorpes, Mutt) revealed their structural identity: this led to the designation "cholecystokinin-pancreozymin". The hormone is found in the endocrine I-cells of the duodenal, jejunal and, to a much lesser extent, ileal mucosa, and is naturally detected in the brain.

Its molecule consists of 33 amino acids. The leading effects of HCP are a powerful increase in gallbladder motility and a significant stimulation of pancreatic secretion of enzymes. The relaxation of the sphincter of Oddi, synchronous with the contraction of the gallbladder, after the introduction of CCP intravenously or intraduodenal administration of implementers of endogenous CCP incretion (fatty and peptide components of food, as well as bile acids) contributes to the flow of bile into the duodenum. HCP-stimulated pancreatic enzymes are also secreted there, creating optimal conditions for the breakdown of food Without affecting the pancreatic release of bicarbonates by itself, HCP potentiates (albeit moderately) the specific stimulating effect of secretin on this process.

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