Hormones and Neurotransmitters

Hopefully, this summary covers most actions of hormones and transmitters mentioned in physiology lectures.

Hormone	Site of Secretion	Stimuli for Secretion	Actions
Gastrin	• G cells of the stomach	• Small peptides and amino acids • Distention of the stomach • Vagal stimulation: (GRP)	• ↑ Gastric secretion • Stimulates growth of **gastric mucosa** • Contracts LES • Relax ileocecal sphincter and pyloric sphincter • Stimulates GIT motility • Stimulate intestinal crypts secretions • Vasodilator
Cholecystokinin (CCK) Gastrin is from the same family = stimulate parietal cell	• I cells of the duodenum and jejunum	• Small peptides and amino acids • Fatty acids	• ↑ Pancreatic enzyme secretion • ↑ Pancreatic HCO3- secretion • Stimulates contraction of the gallbladder and relaxation of the sphincter of Oddi "cholagogues" • Stimulates growth of the **exocrine pancreas and gallbladder** • **Inhibits gastric emptying** (contracts pyloric sphincter) • Relax LES and Ileocecal sphincter • Stimulate intestinal crypts secretions • Stimulate intestinal motility • Vasodilator
Secretin باختصار: يعاكس gastrin، ويحفّز إفراز HCO3 من كل مكان	• S cells of the duodenum	• H+ in the duodenum • Fatty acids in the duodenum	• ↑↑↑ Pancreatic HCO3- secretion (potentiated by CCK and Ach) • ↑ Biliary HCO3- secretion • ↓ Gastric H+ secretion

<table>
<tr><td></td><td></td><td></td><td>Inhibits trophic effect of gastrin on gastric mucosa
Relax LES
Contracts pyloric sphincter and ileocecal sphincter
Inhibit intestinal motility
Stimulate Brunner’s glands (HCO3)
Stimulate intestinal crypts secretions
Vasodilator</td></tr>
<tr><td>GIP:
(Glucose-Dependent Insulinotropic Peptide)
Or (gastric-inhibitory-peptide)</td><td>K cells of the Duodenum and jejunum</td><td>Fatty acids
Amino acids
Oral glucose (faster release of insulin than IV)</td><td>↑ Insulin secretion from pancreatic β cells
↓ Gastric H+ secretion
Contract pyloric sphincter</td></tr>
<tr><td>Motilin</td><td>M cells of the duodenum and jejunum</td><td>Fat
Acid
Nerve</td><td>Stimulates:
Gastric motility & emptying
Intestinal motility
MMC during fasting</td></tr>
<tr><td rowspan="2">Others</td><td>Serotonin:
Stimulate Stomach & intestinal motility</td><td>Insulin:
Stimulate intestinal motility</td><td>Glucagon:
Inhibit intestinal motility
Contract ileocecal sphincter
Stimulate intestinal crypts secretions</td></tr>
<tr><td>Somatostatin:
Inhibits motility & secretion of stomach.
Reduce blood flow and thus limit absorption.</td><td colspan="2">VIP:
dilate intestinal capillaries (Vasodilator)
increase buffer action (releasing of water, electrolytes and mucus from crypts of Lieberkuhn)
Suppress muscle contraction (unlike Ach)
Relax LES</td></tr>
</table>

This is another way to put it:

Sphincters	Contract	Relax
Lower esophageal sphincter	Tonic contraction by vagus (between swallows) Gastrin (from stomach)	NO – VIP (from vagus- during swallowing) CCK – secretin (from upper small intestine)
Pyloric sphincter	Secretin – CCK – GIP	Gastrin - Gastric food volume
Ileocecal sphincter	Distension of cecum – Secretin – Glucagon – alpha-adrenergic – Ach	Gastrin – CCK – beta-adrenergic

Stomach			
Secretions		Emptying	
+	-	+	-
Vagus: • Ach • GRP = stimulate release of gastrin	Somatostatin	Gastric food volume	Enerogastric reflex: ENS – sympathetic – Vagus
Gastrin, = stimulate ECL cells	Secretin	Gastrin	CCK (most potent)
Histamine (from ECL cells)	GIP		Secretin
			GIP

Pancreatic secretions			
Acinar cells (enzymes)	Ductal cells (HCO3)		
CCK Ach	Secretin (major stimulant, potentiated by CCK and Ach) CCK & Ach		
Intestine			
SI motility	Absorption & secretion	Colon motility	
• Vagus • Sympathetic (inhibitory) • Motilin (Intestinal motility & MMC) • Villikinin (motility of villi) • Gastrin - CCK – Insulin - Serotonin	• Glucocorticoid = absorption of H2O & ions (SI & LI) • Somatostatin = H2O & ions absorption (ileum & colon) • Epinephrine = NaCl absorption (ileum) • Aldosterone = synthesis of Na+ channels (colon) • Catecholamine = Decrease intestinal secretion	+ ENS Ach & substance P	- ENS VIP & NO

Factors affecting bile secretion		
Cholertics (increasing choleresis)	Cholagogus (increasing discharge)	Blood flow
1. Bile acid concentration in portal vein (feedback mechanism) - 90% of bile secretion is determined by the rate of clearance of the reabsorbed bile acids in enterohepatic circulation - -ve feedback effect on release of CCK and synthesis of cholesterol	1. CCK: - presence of fat and protein in upper duodenum - Stimulates contraction of the gallbladder and relaxation of the sphincter of Oddi	Increased portal blood flow during digestion = increase bile secretion
2. Hormones: increase ductal secretion of HCO3, by: - secretin - CCK - gastrin - glucagon	2. Vagal stimulation: Stimulates contraction of the gallbladder and relaxation of the sphincter of Oddi	Congestion = increased intrahepatic vascular pressure = stop bile secretion
3. Vagal indirect stimulation : - increases gastric acid - thus increase CCK and Secretin which increase ductal secretion of HCO3	Vagal stimulation and secretin augment action of CCK on the gallbladder	

- Note: **Intestinal phase** = highest rate of gallbladder emptying

بالتوفيق للجميع

- لولوه الصغير