HUNGER HORMONES: THE KEY PLAYERS

Hormones That Tell Your Body You’re Satiated

Cholecystokinin (CCK) is secreted by the cells that line the duodenum (the first segment of the small intestine) when they detect the presence of fat. This causes the release of digestive enzymes from the pancreas and bile from the gallbladder. Increased levels of CCK signal to the stomach to slow down digestion so that the small intestine can digest the fats. CCK is also a neuropeptide, like neuropeptide Y (NPY), the essential neurotransmitter in regulating hunger, and affects the neurons in the brain to signal satiety. This is the most immediate hunger-suppressing signal and is the reason that eating fat with your meals is so important.

Oxyntomodulin is released in response to protein and carbohydrates in the stomach and signals a change in energy status to the brain. Oxyntomodulin enhances digestion by delaying gastric emptying and decreasing gastric-acid secretion.

Peptide YY (PYY) is released by cells that line the jejunum, the ileum (the next two segments of the small intestine), and the colon in response to eating and is especially sensitive to protein. PYY signals to the gallbladder to stop secreting bile and to the pancreas to stop producing digestive enzymes. PYY is important in increasing the efficiency of digestion and nutrient absorption after meals by slowing down gastric emptying, slowing down digestion, and increasing water and electrolyte absorption in the colon. PYY inhibits NPY receptors in the hypothalamus, thereby turning off hunger signals.

Glucagon-like peptide-1 (GLP-1) is secreted in the ileum in response to carbohydrate, protein, and fat. It rapidly enters the circulation and is one of the fastest and shortest-lived satiety signals. It inhibits acid secretion and gastric emptying in the stomach, increases insulin secretion, and decreases glucagon secretion. GLP-1 decreases hunger signals by reducing the amount of NPY.

Leptin plays a key role in regulating energy intake and expenditure, including appetite and metabolism. Leptin is released by adipocytes (fat cells) and by the cells that line the stomach, so it signals both that the body has been fed and that there is sufficient stored energy. This appetite inhibition is long-term, in contrast to the quick suppression of hunger by CCK and the slower suppression of hunger between meals mediated by PYY. Leptin both rapidly inhibits NPY production and deactivates NPY neurons in the brain to signal that the body has had enough to eat, producing a feeling of satiety. It is also one of the most important adipose-derived hormones.

Adiponectin is secreted from adipose tissue into the bloodstream, where it signals decreased gluconeogenesis (the conversion of fats and proteins into glucose for energy), increased glucose uptake, lipid catabolism (breaking down of fats), triglyceride clearance (storage of fats), increased insulin sensitivity, and control of energy metabolism. Adiponectin acts directly on NPY neurons in a similar way as leptin, but its effects are on top of the actions of leptin.
Hormones That Tell Your Body You’re Hungry

Ghrelin is considered the main hunger hormone and the counterpart of leptin. It is secreted by the cells that line the stomach when the stomach is empty and also by the pancreas when it detects low blood sugar. Also, the liver secretes ghrelin when its glycogen storage runs low (and glucagon is high). When ghrelin is released into the circulation, it activates NPY neurons to stimulate appetite. Increased levels of ghrelin bring on the sensation of hunger. Ghrelin is a potent stimulator of growth hormone (GH) secretion and regulates nutrient storage, thereby linking nutrient partitioning with growth and repair processes. Ghrelin activates several anti-inflammatory pathways in the body and promotes cell regeneration, especially within the gastrointestinal tract. Ghrelin regulates glucose homeostasis through a direct action on the pancreatic islet cells (the cells that secrete insulin). It is also important for memory function and gastrointestinal motility.

Cortisol is well known as the master stress hormone, but it has key roles in regulating metabolism and hunger. Cortisol levels determine whether the body uses glycogen stores (stored carbohydrate) or triglyceride stores (stored fat) for energy. Cortisol can also stimulate gluconeogenesis, the process of converting amino acids (proteins) and lipids (fats) into glucose in the liver. It is believed that cortisol affects appetite by acting on NPY neurons in the brain and affects NPY and leptin levels. Cortisol seems to have a particular effect on the desire to eat foods high in fat and sugar. This is why stress management (which really means controlling any factor that might mess with your natural cortisol levels) is so important.

Glucagon is a hormone secreted by the pancreas when it detects low blood-glucose levels (typically between meals, but also as part of the “sugar crash” after eating something high in carbohydrates). Glucagon tells the liver to convert stored glycogen into glucose, which is released into the bloodstream, a process known as glycogenolysis. When glycogen stores are low, high glucagon levels drive gluconeogenesis. Increased glucagon amplifies the hunger sensation.

Insulin is secreted by the pancreas in response to high blood-glucose levels. It causes cells in the liver, muscle, and fat tissue to take up glucose (and fatty acids in the case of adipocytes) from the blood, storing it as glycogen. While insulin is released as a result of eating carbohydrates, it paradoxically increases hunger as opposed to decreasing it. (Insulin signals satiety only when secreted in moderate amounts and in conjunction with elevated blood-glucose levels.) This is caused by direct action on the NPY neurons and is the reason that eating a high-carb meal is not as satiating as eating a meal that includes fats and proteins. It also explains why we feel hungry again so soon after a sugary snack.