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*Letter to the Editor*

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# Can Hormones Regulating Appetite be a Major Factor Contributing to Obesity?

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Obesity is a growing problem throughout the whole world; it is a complex condition that causes much suffering. There are many factors associated with obesity. Those factors include; individual genetics, increased food intake, and/or a lack of physical exercise. In addition hormonal balance plays a very critical role in weight control. There are a number of established hormonal disorders that is associated with obesity. The most common syndromes include Cushing's syndrome and Hypothyroidism.[1,2]

Other hormones play an important role in the pathology of obesity includes:

## GUT HORMONES

The Gastrointestinal – pancreatic complex is a source of important regulatory peptides. Most of Gastrointestinal tract hormones; increase satiety and decrease food intake, with exception of ghrelin that has the opposite effects.[3,4]

### Ghrelin

It is 28-amino acid peptide produced from the fundus of the stomach and upper intestines; is involved in both the long-term regulation of body weight and the short-term regulation of postprandial satiety. Ghrelin is best described as an appetite-stimulatory signal that acts on the brain. Also ghrelin regulates the release of growth hormone from the pituitary gland. It affects appetite is by binding to Growth Hormone Secretagogue Receptors (GHS-R), which are present in the hypothalamus and pituitary. This increases the intracellular $Ca^{2+}$ concentration through inositol 1, 4, 5-trisphosphate signal transduction, resulting in release of Growth Hormone (GH). Since GH is involved in many metabolic processes.

Fasting ghrelin level increases prior to food intake and is rapidly suppressed after eating. The secretion of ghrelin in stomach is stimulated by the combination of several factors; neural (vagus), mechanical (distension), and hormonal (insulin) secretion. Moreover, obese people have lower fasting ghrelin levels and reduced post-prandial suppression when compared to non-obese people. They also have low levels of GH, which could have significant effects on how the body process food and store nutrients. Moreover, Hyerinsulinemia and insulin resistance in obese people are associated with ghrelin suppression.

### Cholecystokinin (CCK)

It is a secreted by the mucosal cells in the duodenum, as well as by neurons in the brain, following consumption of a meal especially fat. The mechanism of action of CCK includes stimulation of gastric acid, gallbladder and pancreatic secretion, modulation of gastrointestinal motility and suppression of energy intake. It plays a role in the regulation of appetite and energy intake, It induces post-prandial satiety.

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### Glucagon Like Peptide (GLP)-1

It is a product of proglucagon cleavage; it is released from the L-cells of the gastrointestinal tract post-prandially in proportion to amount of the calories ingested. (GLP)-1 enhances the insulin response to ingested food, slows gastric emptying and inhibits glucagon response in a glucose dependent manner. In addition, it promotes satiety.

### Peptide YY or Peptide Tyrosine Tyrosine

It is a 36-amino acid peptide; It is released from the L-cells of the gastrointestinal tract together with (GLP)-1 post-prandially in proportion to amount of the calories ingested. It induces satiety and reduces the amount of food intake; it also inhibits gastrointestinal motility and reduces gastric and intestinal secretions. Depressed production is associated with obesity.

### Oxyntomodulin

It is co-secreted with GLP-1 and Peptide YY from the intestinal L-cells in response to food ingestion. It induces post-prandial satiety.

### Gastric Inhibitory Polypeptide (GIP)

A 42-amino acid polypeptide secreted by enteroendocrine K cells, from the duodenum and jejunum after the ingestion of a meal containing glucose or fat. It potentiates glucose induced insulin secretion; Moreover, GIP stimulates islet growth, proliferation of β-cells and reduces β-cell apoptosis. GIP increases the adipose tissue volume indirectly by potentiating fatty acid synthesis, and adipocyte fat deposition.GIP operates as an anabolic hormone in the bone.

### Insulin

It is a peptide hormone that is secreted from the beta cells of the pancreas. Insulin concentration in plasma increases rapidly after eating and decreases with starvation.

Plasma insulin levels are most sensitive to changes in blood glucose concentrations. Insulin binds to its receptor to initiate GLUT-4 in order to allow glucose to enter the cell either for energy production or storage. In addition, Plasma insulin concentrations depend on peripheral insulin sensitivity, which is related to both total body fat stores and fat distribution, with visceral fat being a key determinant.

## PERIPHERAL REGULATORS OF APPETITE- ADIPOSE TISSUE HORMONES

### Adiponectin

It is a 244-amino acid protein secreted exclusively from adipose tissue. Its circulating levels are up to 1,000-fold higher than other circulating hormones such as leptin and insulin. It activates AMP-activated protein kinase (AMPK) in peripheral tissues, mediating increased insulin sensitivity.[5] The central effects of adiponectin, through 2 major adiponectin receptors, AdipoR1 and AdipoR2, both are abundantly expressed in the Arcuate hypothalamus (ARH). They act by controlling AMPK activity and food intake. Adiponectin is considered a starvation hormone: during starvation, high adiponectin levels stimulate central and peripheral AMPK leading to increased food intake and decreased energy expenditure.

After a meal, adiponectin level decreases with a concomitant decrease in AMPK activity leading to satiety and an increase in energy expenditure.

### Leptin

It is a peptide hormone with numerous actions, including influences on energy homeostasis and neuroendocrine and immune function. It is released predominantly from adipocytes but also at lower levels from gastric epithelial cells and the placenta. In the hypothalamus, leptin has an opposite effect: it decreases AMPK activity, thus reducing the appetite centrally and increasing the peripheral energy expenditure.

Circulating leptin concentration is positively correlated with adipose tissue mass, but food restriction results in decreased plasma leptin, which is associated with increased hunger, this condition can be reversed by refeeding or insulin. Also absence of leptin, due to a mutation in the *Ob* gene has profound effects on body weight. It leads to hyperphagia and obesity.

## CONCLUSION

The etiology of obesity is complex and multifactorial, one of the important factors is the regulation of food intake; there are a number of gastrointestinal hormones, which act additively in inducing satiety and achieving efficient nutrient absorption.

This coupled with incomplete understanding of all factors responsible of appetite regulation and prevention of obesity. GIT hormones act through a short term effect; peptides as GLP-1 decrease hypothalamic AMPK activity leading to reduction in food intake, while ghrelin leads to AMPK activation and increased food intake. On the other hand, long term effect produced through peripheral adipose tissue hormones as leptin and adiponectin.

This means that single treatment approach, may be inefficient to control weight loss and may explain the failure of some morbidly obese patients to achieve a successful weight loss.

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