The role of peripheral systems on hunger and satiety regulation

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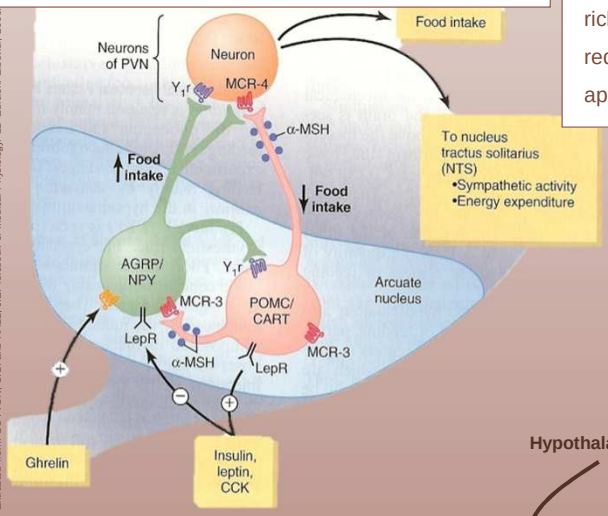
MAIN AIMS.

- A full view of food intake regulation and energy balance.
- To study hunger and satiety regulation by the hypothalamus.
- To learn about hormonal factors and peptides in the gastrointestinal tract involved in feeding regulation.
- To find out what role the hormonal factor of adipose tissue plays in the food intake regulation.

INTRODUCTION.

This regulation is controlled by hormonal signals from the adipose tissue, the nervous system, the endocrine system and the gastrointestinal system. These signals are integrated into the hypothalamus. In the arcuate nucleus many hormones converge that come from the adipose tissue and the gastrointestinal system to regulate both food intake and energy expenditure.

In the arcuate nucleus there are two neural types involve on the food intake regulation. One of them expressed proopiomelanocortin (POMC) which reduces food intake. The other type of neurons is rich in neuropeptide Y (NPY) and agouti-related protein (AgRP) which increases food intake and reduced energy ingestion. NPY and POMC neurons are targets of several hormones that regulate appetite, some of them are dealt with this work.



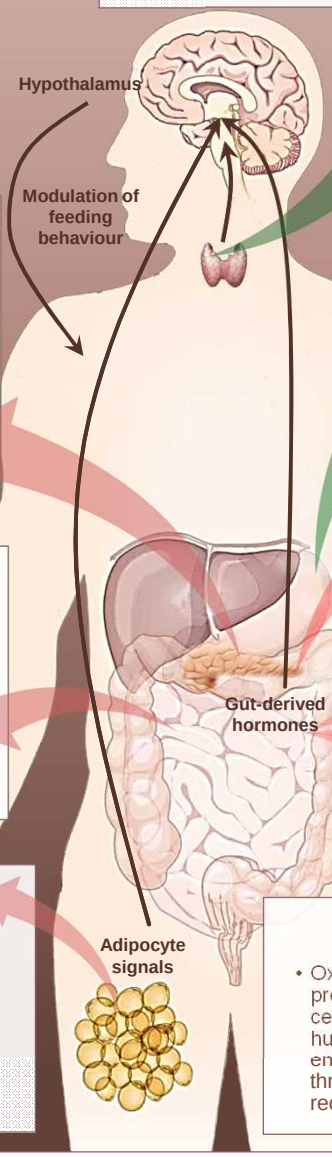
Extracted from: GUYTON, C.G. and HALL, J.E. Textbook of Medical Physiology. 11ª Edición. Elsevier, 2006.

Thyroid hormones

- These hormones are secreted by the thyroid glands. Thyroid hormones stimulate NPY neurons expression and POMC-neuron-inhibition. Thus, thyroid hormones stimulate food intake.

Ghrelin

- mRNA pro-ghrelin is expressed in the stomach cells. Pro-ghrelin is processed to ghrelin. The binding of the ghrelin with its receptors (GHSR) in the hypothalamus causes NPY/AgRP neuron activation. At the same time POMC neurons are inhibited. Thus, ghrelin stimulates food intake.
- Ghrelin increases weight gain through ghrelin receptors in the paraventricular nucleus of the hypothalamus.
- CCK, GLP-1 and leptin increase ghrelin levels. Insulin decreases ghrelin expression.



Insulin

- The pancreatic hormone is produced in the β-cells of the pancreatic islets of Langerhans.
- NPY/AgRP neurons are inhibited and POMC neurons are stimulated by insulin decreasing food intake and weight. The insulin actions are controlled by insulin receptors (IR) in the arcuate nucleus of the hypothalamus.
- An increase of adiposity causes a decreased insulin sensitivity.

PP

- The pancreatic polypeptide is released by islets of Langerhans in the pancreas.
- PP reduces hunger through Y4 receptors in the hypothalamus.

CCK

- CCK is released from the enteroendocrine cells of the duodenum and jejunum.
- The role of CCK is to stimulate satiety through CCK_A receptors present in vagal afferent fibers and circular muscle cells from the pyloric sphincter.
- The satiating effect of CCK is enhanced by leptin.

GLP-1

- Proglucagon undergoes post-translational processing resulting in GLP-1 in intestinal cells.
- GLP-1 decreases feeding and stimulates insulin expression in hyperglycemia state.
- The PYY and GLP-1 are co expressed after ingestion and act synergistically inducing satiety

Leptin

- Satiety and expenditure energy are stimulated by leptin. This hormone inhibits NPY/AgRP neurons and stimulates POMC neurons through leptin receptors in the arcuate nucleus.
- Insulin stimulates leptin expression. And leptin levels are decreased by thyroid hormones.
- GH receptors stimulate leptin production in white adiposity tissue.

OXM

- Oxintomodulina is produced by intestinal cells. OXM decreases hunger and increases energy expenditure through binding to GLP-1 receptors.

PYY

- Peptide YY is secreted by the intestinal cells in ilium.
- NPY/AgRP neurons are blocked and POMC neurons are stimulated by PYY across Y2 receptors in the hypothalamus. Thus, PYY inhibits food intake.
- PYY and GLP-1 have synergic effects.

Modified from *Nature Neuroscience*.

CONCLUSIONS.

Many factors are involved in hunger and satiety. Some of them act synergistically on the hypothalamus. The signals from different parts of the body provide information to the hypothalamus about the physiological state of the organism. In this way, the hypothalamus produces hunger or satiety feelings to maintain energy homeostasis of the body.

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