Class: C1

The diagram illustrates the hunger-satiety circuit within the brain, detailing the intricate interactions among various brain regions and hormones that regulate appetite and satiety.

Hunger and satiety hormones, such as leptin, which is released by fat cells in proportion to fat stores, and ghrelin, which is released by empty gut, influence the arcuate nucleus (ARC) in the hypothalamus. The ARC integrates circulating nutrients like glucose and hormones like leptin and insulin. These inputs covey vital information about the current state of the body, such as energy stores and nutrient availability. Specifically, leptin excites satiety-producing neurons, while hunger-producing neurons inhibits it. Leptin inhibits hunger-producing neurons, while ghrelin excites it.

The paraventricular hypothalamic nucleus (PVH) in the hypothalamus plays a pivotal role in metabolism and other autonomic functions. Within the PVH, melanocortin neurons serve as a critical component. Satiety-producing neurons excites the receptor of melanocortin neurons, while hunger-producing neurons inhibits it. The reason why melanocortin neurons is called the satiety switch is that its high activity causes satiety while its low activity causes hunger.

Nestled in the brainstem, the parabrachial nucleus (PBN) serves as a crucial relay station to higher-order brain areas and receives digestive input from the gut. Melanocortin neurons excites “Holy Grail” neurons which is a general term for the neurons connected to appetite among tens of thousands of unmapped neurons.

“Holy Grail” neurons transmit the information to subcortical structures which are involved in emotion and reward, facilitating the eventual transmission of this information to the cortex through direct and indirect projections.

Ultimately, the cortex receives the information and issues instructions for conscious, action-oriented activity.

The diagram “Mapping the hunger-satiety circuit” describes how subconscious signals are passed to the conscious parts of the brain. Subconscious signals are generated in the hypothalamus, passed on to the brain stem, and ends in the cortex, where the signals are expressed by body actions.

In hypothalamus, leptin released by fat cells and ghrelin released by empty gut act on the arcuate nucleus, known as the ARC, which contains neurons that sense energy levels. Leptin excites the satiety-producing neurons and inhibits the hunger-producing neurons, while ghrelin excites hunger-producing neurons. After that the satiety-producing neurons excites the receptor in the paraventricular hypothalamic nucleus, known as the PVH, whereas the hunger-producing neurons inhibits both the satiety-producing neurons and the receptor in the PVH. The PVH consists of melanocortin neurons, whose activity controls satiety and hunger.

After the hypothalamus has done its work, the signals pass on to the brain stem. The melanocortin neurons excites the “Holy Grail” neurons in the parabrachial nucleus, the key way station to higher-order brain areas, known as the PBN. The PBN also receives input about digestion from the gut.

In the PBN, subcortical structure involved in emotion and reward help the information from the neurons reach the cortex via direct and indirect projections. The information provokes conscious and action-oriented activity in the cortex.

The image clearly illustrates the brain’s hunger-satiety regulation mechanism, explaining how subconscious signals are transmitted to conscious brain regions.

Firstly, fat cells secrete leptin in proportion to fat stores, which activates neurons responsible for generating satiety while inhibiting those linked to hunger. In contrast, ghrelin, known as the “hunger hormone,” stimulates hunger. Additionally, glucose, insulin, and other absorbed nutrients also contribute to this complex regulatory process.

Next, neurons located in the arcuate nucleus (ARC) of the hypothalamus, which generate satiety and hunger signals, collect and process these inputs. They relay information about the body’s energy levels to melanocortin receptors in the paraventricular nucleus (PVH) of the hypothalamus. This process, often called the “satiety switch,” signals either fullness or hunger. These signals are then transmitted to the parabrachial nucleus (PBN), a critical relay point for information on its way to higher brain centers.

Finally, with the help of various subcortical structures and neural pathways, the cortex integrates these signals, translating the body’s internal state into conscious actions, such as initiating or stopping eating.

In conclusion, this pathway demonstrates how the brain converts physiological conditions into conscious behaviors, forming a complete feedback loop between the body and mind.

Class: C2

This diagram outlines how subconscious hunger signals reach the conscious parts of the brain.

Firstly, in the hypothalamus, hormones are generated due to our hunger/satiety. In detail, leptin will be released by fat cells in proportion to the storage of fat. Ghrelin is released when one’s gut is empty. The hormones affect the arcuate nucleus (ARC), which contains neurons that sense energy levels. Leptin excites satiety-producing neurons and inhibits hunger-producing neurons. Ghrelin excites the hunger-producing neurons exclusively. And the hunger-producing neurons will inhibit satiety-producing neurons. The next scene occurs in the paraventricular hypothalamic nucleus (PVH), which plays a big role in metabolism and other autonomic functions. Melanocortin neurons in the PVH serves as the satiety switch, causing satiety at high activity while causing hunger at low activity. It is supposed to be inhibited by hunger-producing neurons and excited by satiety-producing neurons.

Then the signals will travel to the brain and reach the parabrachial nucleus (PBN). PBN is the key way station to higher-order brain areas. It also receives input about digestion from the gut. Particularly, the melanocortin neurons excite “Holy Grail” neurons, consisting of tens of thousands of neurons. Exactly which of them are connected to appetite still remains unclear.

Finally the “Holy Grail” activates subcortical structures involved in emotion and reward, helping this information eventually reach the cortex via direct and indirect projections.

This diagram illustrates the neural mechanisms of the hunger-satiety circuit, showing how subconscious signals travel through the brain to drive conscious, goal-oriented behavior. The process begins in the hypothalamus, specifically the arcuate nucleus (ARC), which senses energy levels through hormones like leptin and ghrelin. Leptin, released by fat cells, activates satiety-producing neurons and inhibits hunger-producing neurons, signaling the body has enough energy. Conversely, ghrelin, released by an empty stomach, activates hunger-producing neurons, promoting food-seeking behavior.

Signals from the ARC pass through the paraventricular hypothalamic nucleus (PVH), a key hub regulating hunger and metabolism. PVH neurons, particularly melanocortin neurons, act as “satiety switches” that control appetite. When active, they suppress hunger, but when inactive, they trigger the need to eat.

Next, these signals reach the parabrachial nucleus (PBN) in the brainstem, which integrates hunger signals with other bodily information. Researchers believe the PBN contains the elusive "Holy Grail" neurons that connect hunger signals to higher-order brain functions, such as motivation and decision-making.

Finally, the signals reach the cortex, where they transform into conscious actions. The interaction between hormonal signals and neural pathways demonstrates how fundamental biological needs influence complex behaviors, providing valuable insights into appetite control and potential treatments for obesity.

Class: C3

The diagram titled “Mapping the hunger-satiety circuit” illustrates the pathways through

which subconscious signals related to hunger and satiety are processed by the brain,

eventually reaching the conscious parts of the brain. The diagram is divided into three key

regions based on their location and function: the hypothalamus, the brainstem, and the

cortex, which correspond to the stages of hunger regulation as studied by Lowell and his

team.

Two primary hormones, leptin and ghrelin, control hunger and satiety. Leptin, released by

fat cells in proportion to fat stores, promotes satiety by stimulating satiety-producing neurons,

while inhibiting hunger-producing neurons in the arcuate nucleus (ARC). Ghrelin, released by

an empty stomach, has the opposite effect: it stimulates hunger-producing neurons and

inhibits satiety-producing ones. The ARC is critical for sensing the body's energy levels,

acting as the first hub in the hunger-satiety circuit.

Signals from the ARC are then transmitted to the paraventricular hypothalamic nucleus

(PVH), the second hub in this circuit. The PVH, which plays a crucial role in metabolism and

other autonomic functions, receives input from both satiety and hunger-producing neurons,

with each type of signal having an opposite effect on the melanocortin neurons within the

PVH.

From the PVH, signals are sent to the parabrachial nucleus (PBN) in the brainstem, which

acts as a key relay station to higher-order brain areas. The PBN is also responsible for

integrating signals from the digestive system. Researchers are particularly focused on

identifying specific neurons within the PBN, sometimes referred to as the “Holy Grail

neurons,” due to their critical role in appetite regulation. However, the large number of

neurons in this region makes it challenging to map them precisely.

Finally, subcortical structures involved in emotion and reward help transmit this information

to the cortex, the final destination where conscious, action-oriented decisions related to

hunger and satiety are made.

Overall, the hunger-satiety circuit is a complex process, with many aspects still being

explored by scientists. Continued research into this system promises to yield deeper insights

into how the brain regulates hunger, potentially leading to more effective treatments for

conditions like obesity.

Class: C4

The diagram illustrates how subconscious signals reach the conscious parts of the brain through the hunger-satiety circuit, which involves multiple brain regions working in concert to regulate our feelings of hunger and fullness.

The regulation of hunger begins in the arcuate nucleus (ARC) located in the hypothalamus. This area responds to signals such as leptin, a satiety hormone released by fat cells in proportion to fat stores, and ghrelin, a hunger hormone released when the gut is empty. When leptin levels rise, the ARC activates satiety-producing neurons that release melanocortin hormones, which promote feelings of fullness and suppress appetite. Conversely, an increase in ghrelin levels stimulates hunger-producing neurons, which act in opposition to the satiety-producing neurons to inhabit the release of melanocortin hormones. Additionally, the ARC integrates information about insulin, glucose, and other absorbed nutrients, providing the brain with a comprehensive understanding of the body's energy levels.

The melanocortin hormones can bind to their receptors in the paraventricular hypothalamic nucleus (PVH), another crucial region in the hunger-satiety circuit that plays a vital role in metabolism and other autonomic functions. These receptors act as a satiety switch; high receptor activity promotes feelings of fullness, while low activity triggers hunger.

The parabrachial nucleus (PBN) in the brain stem serves as another essential hub within the hunger-satiety circuit. It receives signals from the PVH and interacts with higher-order brain structures, while also processing input about digestion from the gut. However, identifying the elusive “Holy Grail” neuron connected to appetite among the tens of thousands of unmapped neurons in the PBN remains a significant challenge for scientists studying the hunger-satiety circuit.

Ultimately, signals originating from the hunger-satiety circuit propagate to higher brain areas (the cortex) through both direct and indirect projections, aided by subcortical structures involved in emotion and reward. This connection enables basic impulses, such as the need for food, to evolve into complex behaviors aimed at obtaining nourishment.

wk6 diagram description

This diagram illustrates the hunger-satiety circuit, demonstrating how subconscious signals make it to the conscious parts of the brain. The circuit starts from hypothalamus, where leptin and ghrelin, hormones that control hunger and satiety, are generated to act on the ARC, the arcuate nucleus contains neurons that sense energy levels. Leptin, released by fat cells, excites the satiety-producing neurons in the ARC and inhibits the hunger-producing neurons at the same time (Ghrelin, released by empty gut, has the opposite effect compared to leptin, while it cannot inhibit the satiety-producing neurons).

Then signals are transmitted to the receptor “melanocortin neurons” located in the PVB, the paraventricular hypothalamic nucleus that plays an important role in the circuit as well as other autonomic functions. This neuron can act as a satiety switch: its high activity causes satiety, while low activity causes hunger.

After that, melanocortin neurons pass the impulse to an area in the brain stem called the parabrachial nucleus (PBN)—the third key hub involved in the hunger-satiety circuit. This is where the “Holy Grail” neurons (those make the final command) lie, and it also receives input about digestion from the gut. However, as there are tens of thousands of unmapped neurons, it’s hard to determine which ones are connected to appetite, and the research is still ongoing.

Finally subcortical structures involved in emotion and reward help this information eventually reach the cortex via direct and indirect projections. Soon in cortex, the conscious, action-oriented activity is formed and that’s when we put things into action.