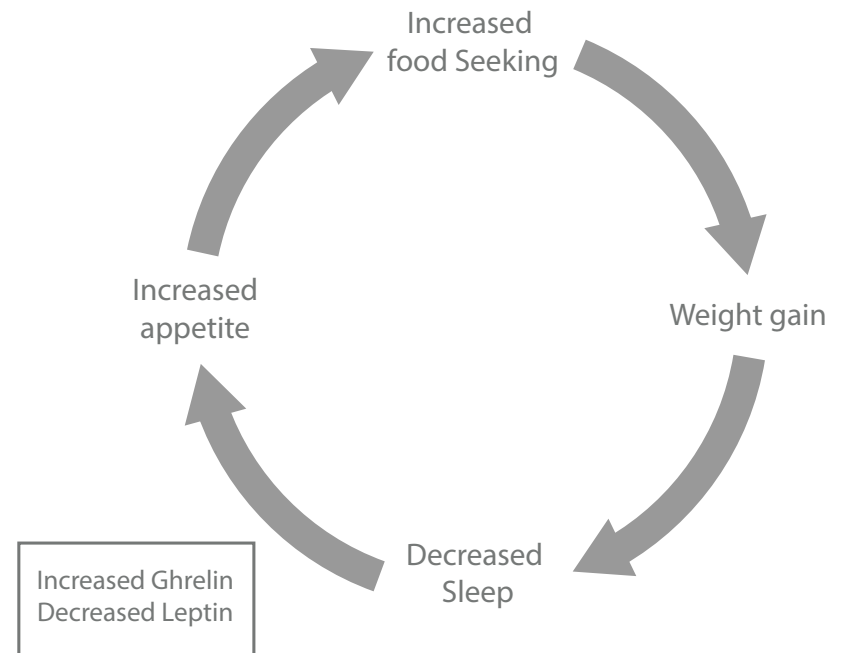
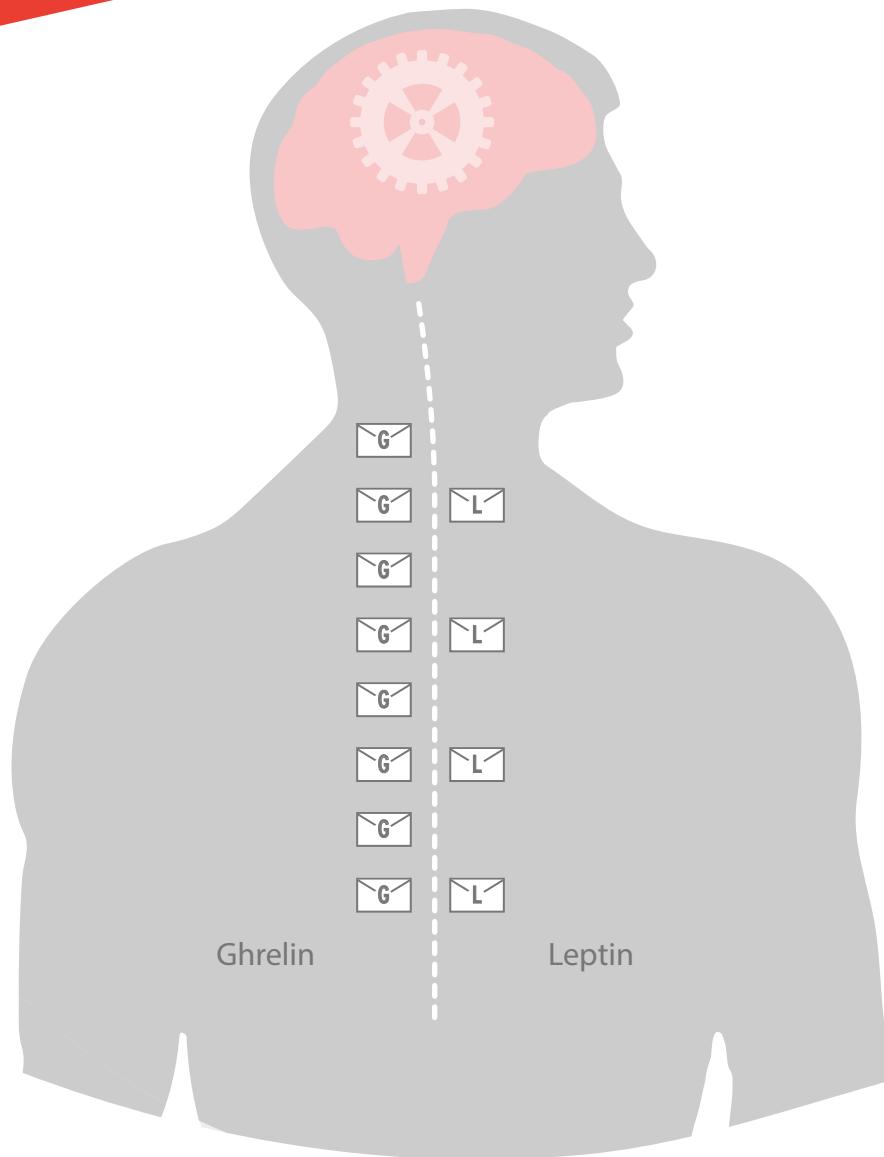
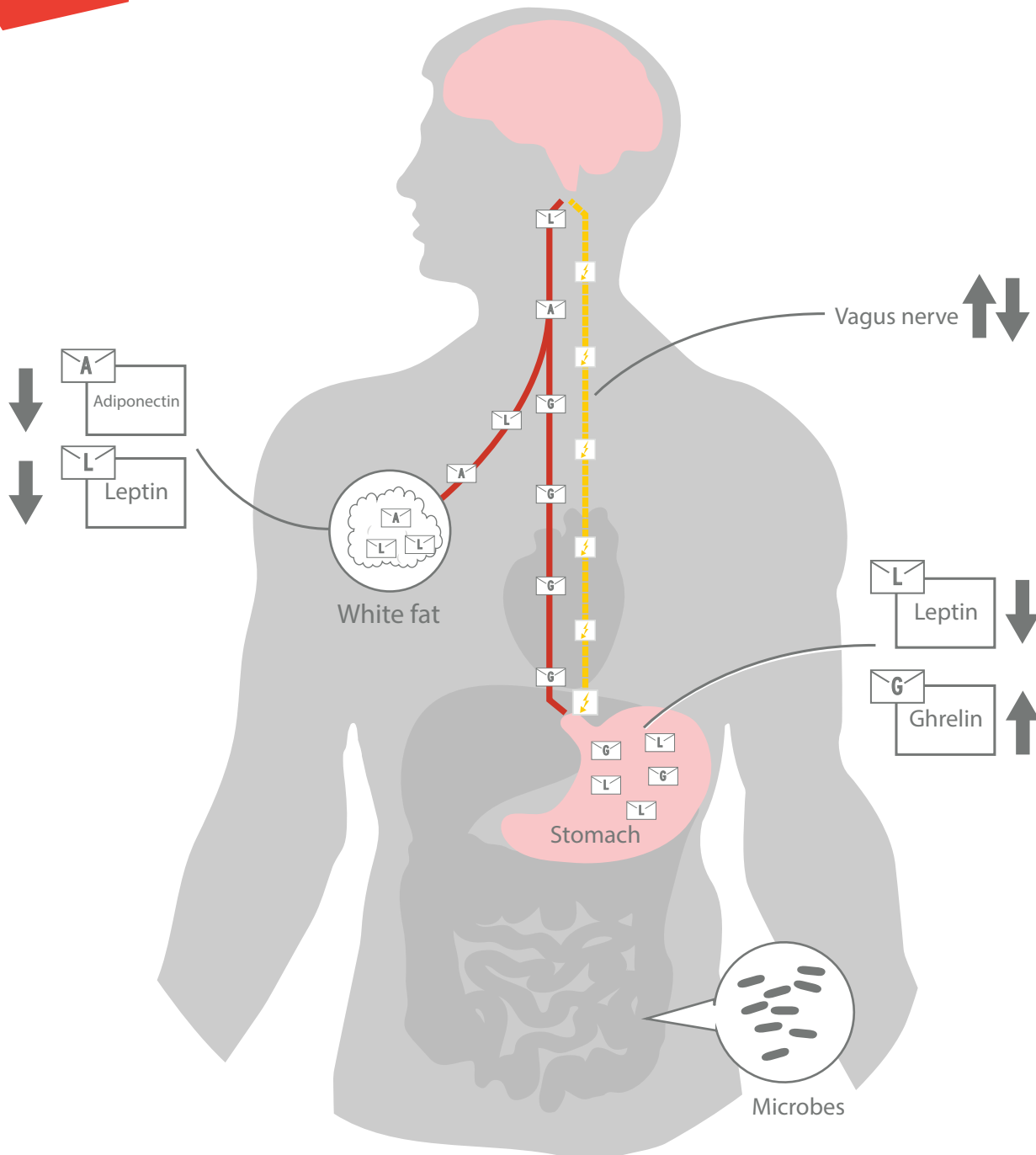


The effect of lack of sleep on appetite

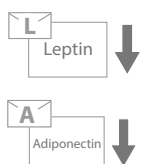


Weight Regulation



Effect on Appetite

Decreases appetite



Increases appetite



Legend

Nerve (represented by a yellow dashed line with a lightning bolt)

Blood vessel (represented by a solid red line)



Hormone



Key messaging for the Weight Regulation Diagram

Vagus nerve

The vagus nerve innervates most of the regions of the gut (gastrointestinal tract) involved in energy intake, satiation and digestion and serves as a crucial link between the brain, brainstem and gut.

The vagus nerve plays dual role, interacting with appetite stimulating and appetite inhibiting pathways.

A part of the vagus nerve in the stomach is involved in regulating appetite and satiation. This part is stimulated directly by mechanical stretch or change in tension when food, or chyme, passes through the gut.

The other part of the vagus nerve controls the motor and secretory apparatus of the gut involved in food digestion and absorption.

Stomach

In the stomach, ghrelin secretion inhibits afferent vagal fibers to induce appetite.

Leptin secreted in the stomach stimulates vagal fibers and induces satiety

Ghrelin-hunger hormone

Ghrelin increases gastric emptying and stimulates appetite. The ghrelin pathway is responsible for stimulating appetite areas in the brain that are involved in food-seeking behavior and prepare the gut for food intake by a vagal response.

During weight loss ghrelin levels may increase, which causes increased food consumption and weight gain.

Short sleep duration is associated with high levels of ghrelin, increase hunger, increase food intake and obesity.

As the sleep duration increases, the level of ghrelin in the blood lowers, thereby potentially reducing appetite and preventing weight gain.

Leptin

Leptin and some other secretions in the body induce satiation (or the feeling of fullness after meal ingestion) and satiety (absence of appetite).

Leptin is secreted from the adipose tissue, pancreas and gut after food intake to induce satiety.

Linkage with satiation or satiety: Leptin decreases motility, increases energy expenditure by inducing hyperthermia, and stimulates the area of the brain that produces food reward and feelings of well-being after meal, therefore inhibiting appetite.

Leptin levels in the blood are reduced by short sleep duration, therefore may lead to increase appetite and weight gain.

Microbes

Emerging evidence suggests that microbes in the gut can contribute to development of obesity.

The microbe in the gut may interact with our metabolism. For example, they can break down other indigestible carbohydrates and increasing short chain fatty acid absorption in the colon. This provides additional energy and increases fat storage in adipose tissue.

The microbiota composition in obese individuals appears to be structurally and functionally different from that of lean individuals, possibly adapted to more efficient energy extraction and storage, contributing to weight gain and its metabolic consequences. This adaptation is referred to as 'obesity associated dysbiosis'.

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