Nutrition Physiology

Fasting and Feeding

Simge Aykan, PhD
Department of Physiology
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Regulation of Absorptive and Postabsorptive States

- Absorptive state: ingested nutrients enter the blood from the gastrointestinal tract

- Postabsorptive state: the gastrointestinal tract is empty of nutrients and the body's own stores must supply energy

- Fasting: 24 hour without eating

Regulation of Absorptive and Postabsorptive States

- Nightly starved-fed cycle
  1. the postabsorptive state after a meal,
  2. the early fasting during the night, and
  3. the refed state after breakfast.

- A major goal of the many biochemical alterations in this period is to maintain *glucose homeostasis*—that is, a constant blood-glucose level.

Postabsortive States

- Glucose and amino acids are transported from the intestine to the blood
- The dietary lipids are packaged into chylomicrons and transported to the blood by the lymphatic system
- Insulin is secreted

Insulin signals the fed state—it stimulates the storage of fuels and the synthesis of proteins in a variety of ways

Postabsorptive States

- Insulin → Beta cells
  - Increased during absorptive state
  - Decreased during postabsorptive state
  - Insertion of GLUT-4
  - High glucose transport into the cells

Postabsorptive States

- The liver helps to limit the amount of glucose in the blood during times of plenty by storing it as glycogen so as to be able to release glucose in times of scarcity.
- Insulin accelerates the uptake of blood glucose into the liver by GLUT2.
- The level of glucose 6-phosphate in the liver rises
  - the catalytic sites of glucokinase become filled with glucose
- The increase in glucose 6-phosphate coupled with insulin action leads to a buildup of glycogen stores

Postabsorptive States

- High insulin level
  - Promotes *the entry of glucose into muscle and adipose tissue*. Insulin stimulates the synthesis of glycogen by muscle as well as by the liver.
  - Entry of glucose into adipose tissue provides glycerol 3-phosphate for the synthesis of triacylglycerols.
  - Promotes the uptake of branched-chain amino acids (valine, leucine, and isoleucine) by muscle.
    - general stimulating effect on protein synthesis, which favors a building up of muscle protein.
    - inhibits the intracellular degradation of proteins.

The Early Fasting State

- The blood-glucose level begins to drop several hours after a meal, leading to a decrease in insulin secretion and a rise in *glucagon* secretion

- Glucagon is secreted by the α cells of the pancreas in response to a *low blood-sugar level in the fasting state.*

Glucagon signals the starved state

The Early Fasting State

- Liver is the main target organ of glucagon
  - Stimulates glycogen breakdown and inhibits glycogen synthesis by triggering the cyclic AMP cascade leading to the phosphorylation and activation of phosphorylase and the inhibition of glycogen synthase
  - Inhibits fatty acid synthesis by diminishing the production of pyruvate and by lowering the activity of acetyl CoA carboxylase by maintaining in an unphosphorylated state.
  - Stimulates gluconeogenesis in the liver and blocks glycolysis by lowering the level of F-2,6-BP.

The net result is to increase the release of glucose by the liver.

The Early Fasting State

- Both muscle and liver use fatty acids as fuel when the blood-glucose level drops. Thus, *the blood-glucose level is kept at or above 80 mg/dl*
  1. *the mobilization of glycogen and the release of glucose by the liver*
  2. *the release of fatty acids by adipose tissue*
  3. *the shift in the fuel used from glucose to fatty acids by muscle and the liver*

The Refed State

- Fat: processed as in the normal fed state
- Glucose:
  - Liver does not initially absorb glucose from the blood, but rather leaves it for the peripheral tissues
  - Liver remains in a gluconeogenic mode
    - the newly synthesized glucose is used to replenish the liver's glycogen stores
  - As the blood-glucose levels continue to rise, the liver completes the replenishment of its glycogen stores and begins to process the remaining excess glucose for fatty acid synthesis

Regulation of Apetite

- Digestion is controlled by the nervous system and hormones
  - Food triggers nervous system responses
    - The thought of food stimulates the hypothalamus, which controls many involuntary responses of the nervous system
    - The hypothalamus stimulates the nervous pathways that prepare the digestive system to process food
      - Salivation, for example, increases and the stomach produces more acid and protective mucus

Regulation of Apetite

- Hormones regulate hunger
  - Two appetite-regulating hormones discovered in the 1990s are leptin and ghrelin
    - **Leptin** is a peptide secreted by fat cells
      - When calorie intake is restricted, leptin in the bloodstream is reduced, stimulating the hypothalamus to trigger hunger
    - **Ghrelin** is a peptide secreted by gastric gland cells in the stomach lining
      - An increase in circulating ghrelin occurs prior to mealtime, stimulating hunger via the hypothalamus

Regulation of Appetite

- Ghrelin → hunger
- Insulin → supression of appetite
- Leptin → supresses appetite
- Peptide YY → supresses the appetite