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Effects of Endogenous GLP-1 on Hunger, Weight, and Blood Sugar Regulation

1. GLP-1 Basics: Origin and Physiology

• GLP-1 is produced in enteroendocrine L-cells and certain brainstem neurons.

• Secreted rapidly after meals, especially with carbohydrate and fat intake.

• Rapidly degraded by DPP-4, giving it a very short half-life.

• Acts locally via vagal pathways and paracrine mechanisms.

2. Effects on Hunger and Eating Behavior

• Acts as a meal-driven satiation hormone.

• Reduces meal size and suppresses reward-driven eating.

• Activates GLP-1 receptors in vagal afferents, brainstem, hypothalamus, and mesolimbic reward systems.

• Helps diminish cravings for highly palatable food.

3. Effects on Body Weight

• Supports long-term weight regulation by modestly reducing overall intake.

• Impairment of GLP-1 signaling often increases weight in animal models.

• Bariatric surgery greatly enhances endogenous GLP-1 response, contributing to weight loss and altered food preference.

• Drugs amplify this physiology but at much higher receptor activation levels.

4. Effects on Blood Sugar Regulation

• Key player in the incretin effect.

• Enhances glucose-dependent insulin secretion.

• Suppresses glucagon when glucose is elevated.

• Slows gastric emptying to smooth post-meal glucose spikes.

5. Diet, Obesity, and Disease Modulation

• Carbs, fats, protein, and fermentable fiber stimulate GLP-1 release.

• Obesity and type 2 diabetes impair GLP-1 secretion and/or islet responsiveness.

• GLP-1 resistance may occur in metabolic disease.

Summary

Endogenous GLP-1 reduces hunger, supports healthy body weight, and is a cornerstone hormone in blood glucose regulation through its incretin role, glucagon suppression, and gastric emptying delay.