

Satiety: Targeting Food Intake Regulation Pathways

Focus: GLP-1, GIP, GDF15, Asprosin, Insulin, Isthmin-1 and Famsin

Satiety is a central regulator of feeding behavior, energy balance and metabolic health. It arises from the integration of hormonal and neuronal signals that link nutrient intake to appetite control, glucose metabolism and energy storage. These signals originate primarily from the gastrointestinal tract, pancreas, adipose tissue and liver, and converge on the central nervous system to coordinate short- and long-term regulation of food intake.

Disruption of satiety signaling contributes to obesity, diabetes and cardiometabolic disease, making this pathway a major focus of basic and translational research. Recent advances have revealed an increasingly complex endocrine network, extending beyond classical appetite hormones to include stress- and disease-associated regulators that influence feeding behavior and metabolic adaptation.

Key Hormonal Pathways in Satiety Regulation

Satiety control is mediated by multiple interacting hormone classes with distinct temporal and physiological roles. **Gut-derived incretins**, including **glucagon-like peptide-1 (GLP-1)**, **glucose-dependent insulinotropic polypeptide (GIP)** and **glucagon**, couple nutrient ingestion to insulin secretion, gastric emptying and appetite suppression. Other gut-hormones, such as oxyntomodulin (OXM), cholecystokinin (CCK) and peptide YY are also important anorexigenic hormones secreted upon food intake. **Pancreatic hormones**, most notably insulin, act as both metabolic regulators and central satiety signals. **Adipose-derived factors** such as **leptin** and **acyl-CoA-binding protein (ACBP)** provide long-term feedback on energy stores, while liver-, adipose- and gut-secreted peptides and proteins including **asprosin** and **famsin** influence glucose production, feeding behavior and systemic metabolism. Orexigenic hormones such as **ghrelin** promote hunger during fasting states.

In addition, **growth differentiation factor 15 (GDF15)** has emerged as a key regulator of appetite suppression in response to metabolic stress, inflammation and disease, acting through central nervous system pathways distinct from classical satiety hormones. Together with emerging regulators such as **isthmin**, a new insulin-like hormone regulating glucose uptake while suppressing lipid accumulation, these molecules form an integrated endocrine network that dynamically controls appetite, metabolism and energy homeostasis.

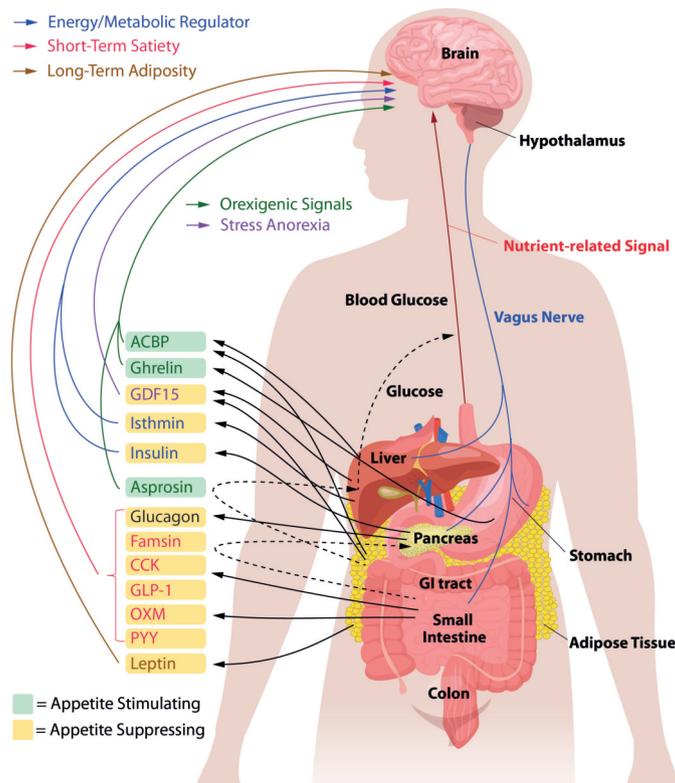


FIGURE: Schematic Overview of Satiety-related Proteins.

LIT: Exercise-induced appetite suppression: An update on potential mechanisms: S.F. McCarthy, et al; *Physiol. Rep.* **12**, e70022 (2024) • The gut-brain axis in appetite, satiety, food intake, and eating behavior: Insights from animal models and human studies: G.S. Clarke, et al; *Pharmacol. Res. Perspect.* **12**, e70027 (2024) • Proteins and Peptides from Food Sources with Effect on Satiety and Their Role as Anti-Obesity Agents: A Narrative Review: A. Ignat-Gutierrez, et al; *Nutrients* **16**, 3560 (2024)

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Incretins and Satiety: Hormonal Signals That Shape Appetite

Incretins (gastric inhibitory polypeptide (GIP), glucagon-like peptide-1 (GLP-1), and glucagon) are gut-derived hormones released after food intake that play a central role in regulating blood glucose and appetite. GIP and GLP-1 are the two primary incretins secreted from the intestine on ingestion of nutrients or glucose to stimulate insulin secretion from pancreatic β -cells. GIP enhances and GLP-1 inhibits postprandial glucagon response. In adipose tissues, only GIP, not GLP-1 facilitates fat deposition. In bone, GIP promotes bone formation while GLP-1 inhibits bone absorption. In the brain, both GIP and GLP-1 are thought to be involved in memory formation and in control of appetite. Glucagon is secreted from pancreatic α -cells and acts in opposition to insulin by promoting gluconeogenesis and glycogenolysis and plays an essential role as regulator of glucose and lipid metabolism. Recently, synthetic long-acting GLP-1 and/or GIP analogs have been shown to improve insulin sensitivity, insulin secretory response and to reduce appetite.

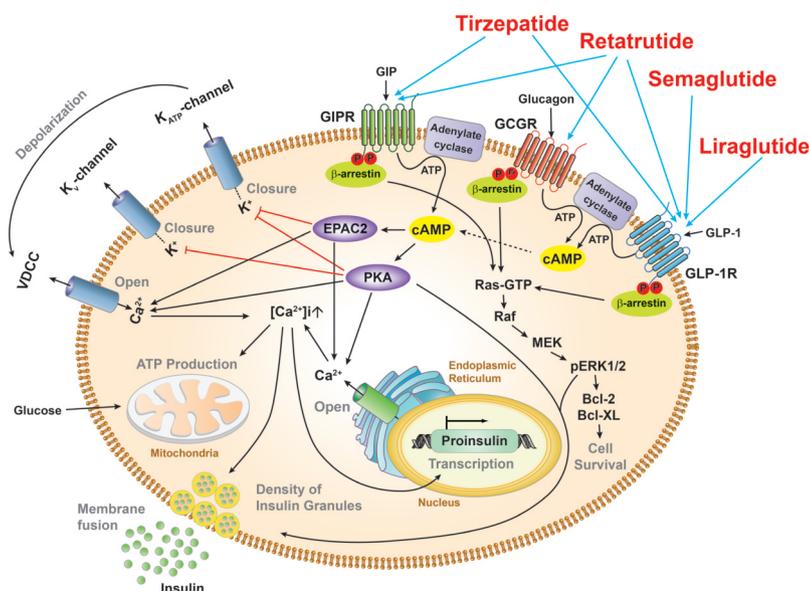


FIGURE: GLP-1R, GIPR and GCGR Signaling.

LIT: GLP-1 Receptor Agonists: Beyond Their Pancreatic Effects: X. Zhao, et al.; Front. Endocrinol. 12, 721135 (2021) • GLP-1 and GIP receptor signaling in beta cells – A review of receptor interactions and co-stimulation: A. Mayendraray, et al.; Peptides 151, 170749 (2022)

GLP-1 Receptor Agonists

Available from Stock in **BULK**

PRODUCT NAME	PID	SIZE	DESCRIPTION
Liraglutide	AG-CP3-0034	1 mg 5 mg 25 mg 100 mg	Long-acting acylated GLP-1 receptor agonist.
Retatrutide . Na	AG-CP3-0044	1 mg 5 mg 25 mg 100 mg	Novel triple agonist peptide of the GCG receptor, GIP receptor and GLP-1 receptor.
Semaglutide	AG-CP3-0040	1 mg 5 mg 25 mg 100 mg	Longer-acting alternative GLP-1 receptor agonist to Liraglutide.
Semaglutide . acetate	AG-CP3-0032	1 mg 5 mg 25 mg 100 mg	Semaglutide salt form.
Tirzepatide	AG-CP3-0043	1 mg 5 mg 25 mg 100 mg	Novel dual GIP and GLP-1 receptor agonist.
Cagrilintide . acetate	AG-CP3-0047	1 mg 5 mg 25 mg 100 mg	Novel long-acting nonselective AMYRs agonist and CTR agonist. Developed in combination with Semaglutide as " CagriSema ".
Mazdutide [LY-3305677]	AG-CP3-0045	1 mg 5 mg 25 mg 100 mg	Novel long-acting dual GLP-1 and GCG receptor agonist.
Survodutide [BI 456906]	AG-CP3-0046	1 mg 5 mg 25 mg 100 mg	Novel long-acting dual GLP-1 and GCG receptor agonist.
DMB [GLP-1R Agonist]	AG-CR1-3759	1 mg 5 mg 25 mg	Small molecule GLP-1 receptor agonist.

For Research Use Only (RUO). Our products are not intended or approved for human, diagnostics, therapeutic or veterinary use.

GLP-1 Receptor Proteins for Screening of GLP-1R Modulators

PRODUCT NAME	PID	SIZE	DESCRIPTION
GLP-1R (human) (rec.) (His)	AG-40B-0279	50 μ g	Binds to GLP-1, Semaglutide and Tirzepatide. For high-throughput screening (HTS) assays (binding of GLP-1 analogs).
GLP-1R (human) (rec.) (His) (Biotin)	AG-40B-0279B	50 μ g	
GLP-1R (human) (monomer):Fc (silent) InVivoKine™	AG-40B-0272	50 μ g	Binds to GLP-1, Semaglutide and Tirzepatide. For high-throughput screening (HTS) assays (binding of GLP-1 analogs). Can be used to block GLP-1 <i>in vivo</i> .

GDF15 – Exercise-induced Factor Regulating Appetite

Growth and differentiation factor 15 (GDF15; Macrophage Inhibitory Cytokine 1; MIC-1) is a member of transforming growth factor- β (TGF- β) superfamily. GDF15 is a cytokine, secreted from cells undergoing mechanical or chemical stress and it increases during exercise. GDF15 acts through a recently identified receptor expressed in the brain called glial-derived neurotrophic factor (GDNF) receptor α -like (GFRAL) which signals through the "Rearranged during Transfection" (RET) tyrosine kinase receptor. Functions of GDF15 are pleiotropic and include appetite regulation/suppression, actions on metabolism, pregnancy, cell survival, aging, immune response and inflammation. Recombinant GDF15 administered to mice drives weight loss. Inhibition of the GDF15-GFRAL axis with a blocking antibody leads to inhibition of cachexia, preventing weight loss and reversing anorectic behavior.

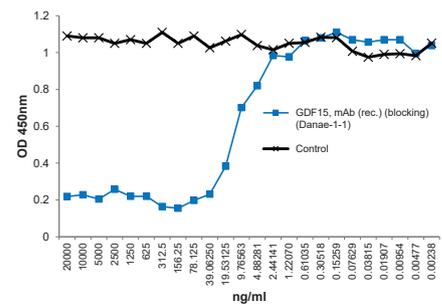
LIT: GDF15 in Appetite and Exercise: Essential Player or Coincidental Bystander? A.B. Klein, et al.; *Endocrinol.* **163**, bqab242 (2022) • GDF15 is still a mystery hormone: C.M. Sigvardsen, et al.; *Trends Endocrinol. Metab.* **36**, 591 (2025)

Unique GDF15 Recombinant Blocking Antibody

PRODUCT NAME	PID	ISOTYPE	APPLICATIONS	SPECIES
anti-GDF15, mAb (rec.) (blocking) (Danae-1-1)	AG-27B-0024PF	Mouse IgG2b	ELISA, FUNC	Hu, Ms

BINDING ASSAY: Binding of GDF15 (mouse) to GFRAL (mouse) is inhibited by GDF15, mAb (rec.) (blocking) (Danae-1-1) (preservative free) (Prod. No. AG-27B-0024PF).

METHOD: GFRAL (mouse) is coated on an ELISA plate at 1 μ g/ml. anti-GDF15, mAb (rec.) (blocking) (Danae-1-1) (PF) (Prod. No. AG-27B-0024PF) or an unrelated recombinant mAb Control were added (starting at 20 μ g/ml with a twofold serial dilution) together with 30 ng/ml of GDF15 (mouse):Fc (silent) InVivoKine™ (Prod. No. AG-40B-0245). After incubation for 1 hour at RT, the binding was detected using an anti-Fc human antibody (HRP).



Biologically Active GDF15 and GFRAL Proteins

PRODUCT NAME	PID	SOURCE	ENDOTOXIN	SPECIES
GDF15 (human):Fc (silent) InVivoKine™	AG-40B-0253	HEK 293 cells	<0.01EU/ μ g	Hu, Ms
GDF15 (mouse):Fc (silent) InVivoKine™	AG-40B-0245	HEK 293 cells	<0.01EU/ μ g	Ms
Fc (silent) InVivoKine™ Human IgG1 Control	AG-35B-0018	HEK 293 cells	<0.01EU/ μ g	Hu, Ms
GFRAL (human):Fc (human) (rec.)	CHI-HF-210GFRAL	HEK 293 cells	<1EU/mg	Hu
GFRAL (mouse):Fc (mouse) (rec.)	CHI-MF-110GFRAL	HEK 293 cells	<0.06EU/ μ g	Ms

Insulin & Isthmin-1: Linking Metabolic Control to Appetite Regulation

Insulin is a central metabolic hormone that not only regulates glucose homeostasis but also plays a critical role in the control of satiety and energy balance. In response to an elevation in plasma glucose and amino acids (after consumption of a meal), insulin is released from the β -cells in the pancreas. When plasma glucose falls (during fasting or exercise), glucagon is secreted by α -cells, which surround the β -cells in the pancreas. In the brain, insulin modulates key appetite-regulating neuronal circuits. By activating anorexigenic pathways and suppressing orexigenic signals, insulin contributes to reduced food intake and the promotion of satiety following meals. Disruption of central insulin signaling has been linked to Types I/II diabetes, obesity, insulin resistance and impaired appetite control. Isthmin-1 (ISM1) is a liver-derived metabolic regulator that improves glucose uptake and insulin sensitivity in peripheral tissues and supports energy homeostasis indirectly by enhancing metabolic efficiency and promoting a balanced energy state.

Sensitive Insulin & Isthmin-1 ELISA Assays

PRODUCT NAME	PID	SENSITIVITY	RANGE	SAMPLE TYPE
Insulin (human) ELISA Kit	AG-45B-0031	10 pg/ml	7.81 to 500 pg/ml	Plasma, Serum, CCS
Isthmin-1 (human) ELISA Kit	AG-45B-0032	0.4 ng/ml	0.625 to 40 ng/ml	Plasma, Serum, CCS
PRODUCT NAME	PID	SOURCE	ENDOTOXIN	SPECIES
Isthmin-1 (human) (rec.) (His)	AG-40B-0214	HEK 293 cells	<0.01EU/ μ g	Hu
Isthmin-1 (mouse) (rec.) (His)	AG-40B-0215	HEK 293 cells	<0.01EU/ μ g	Ms
PRODUCT NAME	PID	ISOTYPE	APPLICATIONS	SPECIES
anti-Isthmin-1, mAb (rec.) (Giusepi-1-4)	AG-27B-0022	Mouse IgG2by	ELISA, WB	Hu, Ms

Asprosin

Asprosin is the C-terminal cleavage product of the protein Fibrillin-1. Asprosin is a fasting-induced glucogenic and orexigenic hormone that plays a critical role in the regulation of energy homeostasis. Within the central nervous system, asprosin works by stimulating the orexigenic AgRP+ (Agouti related neuropeptide) neurons via a cAMP-dependent pathway and by inhibiting the anorexigenic POMC+ (proopiomelanocortin) neurons in a GABA- dependent manner, promoting food intake and reducing satiety signals. Elevated circulating asprosin levels have been associated with obesity, insulin resistance and metabolic syndrome, highlighting its relevance in metabolic disease research.

LIT: Asprosin in health and disease, a new glucose sensor with central and peripheral metabolic effects: M. Farrag, et al.; Front. Endocrinol. 13, 110191 (2023) • Asprosin: its function as a novel endocrine factor in metabolic-related diseases: Y. Zhang, et al.; J. Endocrinol. Invest. 47, 1839 (2024)

Asprosin ELISA Assays, Antibodies & Recombinant Proteins

PRODUCT NAME	PID	SENSITIVITY	RANGE	SAMPLE TYPE
Asprosin (human) ELISA Kit	AG-45B-0010	230 pg/ml	0.3125 to 20 ng/ml	Plasma, Serum, CCS
Asprosin (human) Matched Pair Detection Set	AG-46B-0011	150 pg/ml	0.156 to 10 ng/ml	CCS
PRODUCT NAME	PID	ISOTYPE	APPLICATIONS	SPECIES
anti-Asprosin, mAb (Birdy-1)	AG-20B-0073	Mouse IgG1	WB, ELISA	Hu, Ms
anti-Asprosin (human), mAb (Birdy-2)	AG-20B-0074	Mouse IgG2a	WB, ELISA	Hu
anti-Asprosin, Rabbit Monoclonal (RM463)	REV-31-1355-00	Rabbit IgG	WB, IHC	Hu
PRODUCT NAME	PID	SOURCE	ENDOTOXIN	SPECIES
Asprosin (human) (rec.) (His)	AG-40B-0174	E. coli	<0.01EU/μg	Hu

Famsin – Linking Metabolic Control to Appetite Regulation

Famsin is an emerging gut-derived hormone that plays an important role in the body's adaptation to fasting. During periods of food deprivation, circulating famsin levels rise, helping maintain blood glucose by stimulating glucagon release and supporting hepatic glucose production. By acting as part of a gut-pancreas-liver signaling axis, famsin helps to balance energy needs during fasting while interacting with pathways that influence hunger and metabolic regulation.

LIT: Famsin and fasting adaptation: A glucagon connection: S. Li, et al.; Cell Metab. 37, 561 (2025)

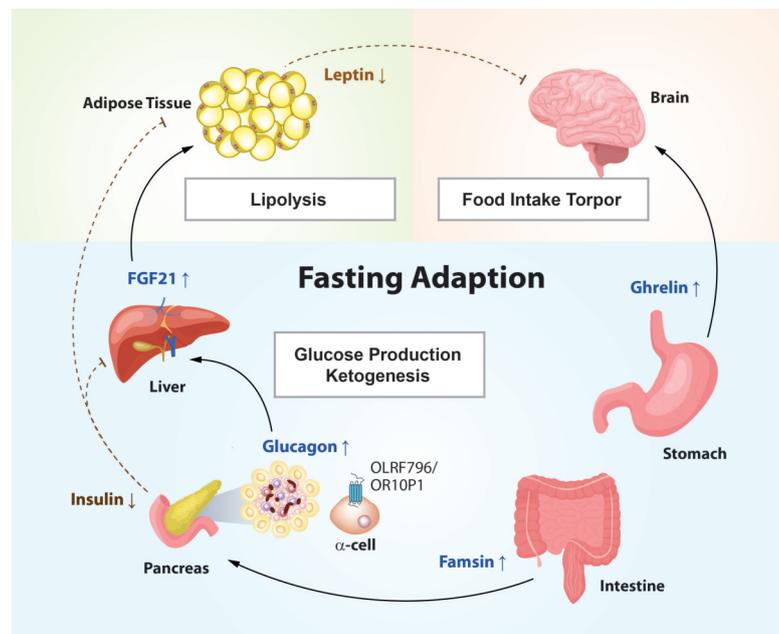


FIGURE: Schematic Overview of Fasting Adaption Pathway, including the Factor Famsin.

PRODUCT NAME	PID	SOURCE	ENDOTOXIN	SPECIES
Famsin (mouse) (rec.) (His)	AG-40B-0278	CHO cells	<0.01EU/μg	Ms