Amylin

is a nutrient-stimulated hormone (NuSH)

What is a NuSH?

The term "NuSH" refers to a hormone secreted by cells in the gastrointestinal (GI) tract and pancreas in response to nutrients via oral meal intake, which regulate nutrient and energy fluxes[1,2]

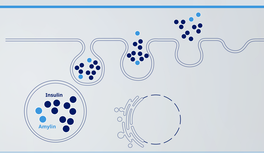
What are the characteristics of a NuSH?[2–8]

NuSH name	Nutrient stimuli	Origin	Sites of action	Physiological function
Amylin	Glucose; protein/ amino acids[3]	Pancreatic β cells[3]	Central nervous system (CNS)	Increase satiety; reduce gastric emptying; reduce food intake[2]
Glucagon-like peptide-1 (GLP-1)	Glucose; fatty acids (protein/amino acids)[4,6]	Ileum/colon L cells[2,4–6]	CNS, β cells, heart, lung, GI tract, kidney[2]	Increase satiety; reduce gastric emptying and motility; increase postprandial insulin secretion[2,5]
Glucose-dependent insulinotropic peptide (GIP)	Glucose; fatty acids (protein/amino acids)[3]	Duodenum/ jejunum K cells[2–4]	β cells, CNS, bone, heart, adipocytes[2]	Reduce food intake; increase LPL activity; increase postprandial insulin secretion[2]
Glucagon (GCG)	Decrease in blood glucose level[2]	Pancreatic α cells[2,7]	CNS, pancreas, adipocytes, kidney, liver[2]	Increase satiety; increase glycogenolysis; increase gluconeogenesis[2]
Cholecystokinin (CCK)	Fatty acids[4]	Duodenum/ jejunum I cells[2,4]	Pancreas, CNS (GI tract)[2]	Increase satiety;[2,4] reduce gastric emptying and motility;[2] mediate reduced glucose production via lipids[4]
Ghrelin	Fasting via noradrenaline; glucose and insulin decrease ghrelin levels[8]	Stomach P/D1 cells[2,4,8]	CNS (pancreas, adrenal glands, pituitary)[2]	Increase food intake[2]
Oxyntomodulin (OXM)	Fatty acids[5]	Ileum/colon L cells[2,4,5]	CNS, pancreas[2]	Increase satiety; reduce gastric emptying; reduce food intake[2,5]
Pancreatic polypeptide (PP)	Stomach distension; circadian rhythm[3]	Pancreatic F cells (GI tract)[2,3]	CNS[2]	Increase satiety; reduce gastric emptying[2]
Peptide tyrosine tyrosine (PYY)	Fatty acids (protein/amino acids, glucose)[3]	Ileum/colon L cells[2,4,5]	CNS[2]	Increase satiety; reduce gastric emptying and motility[2]

Text within parentheses indicates weak/secondary stimuli or weak receptor expression in potential target organs

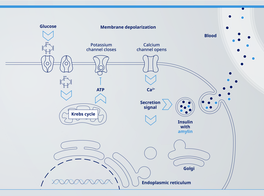
The role of amylin as a NuSH

Amylin is co-located with insulin in secretory granules in pancreatic β cells, which are co-secreted into the bloodstream following nutrient stimulation[7]

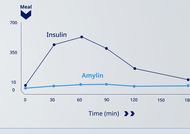


Glucose stimulates biphasic amylin and insulin secretion, via "triggering" and "amplifying" pathways[7,9]

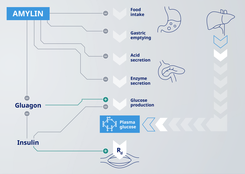
Both hormones are also co-secreted in response to **amino acids**, either via direct effects on metabolic processes or via indirect effects of α/β-cell communication[7]



Plasma amylin and insulin levels are **correlated** after glucose ingestion or mixed-meal tolerance test, although insulin concentrations are up to 70 times higher than those of amylin[10,11]

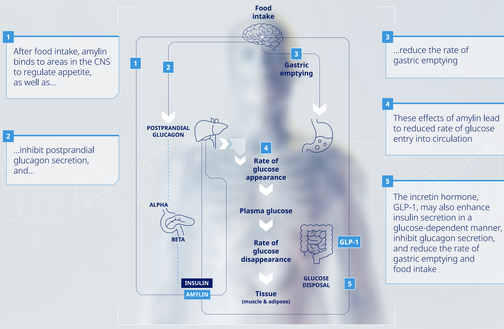


Amylin and insulin have complementary functions to **maintain glucose homeostasis** by decreasing the rate of nutrient entry into circulation and increasing storage in peripheral tissues, respectively[12]



Interaction with other hormones

Amylin can mediate the effects of another NuSH or have a synergistic effect on eating[1]

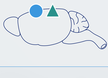


Amylin may interact with GLP-1 to reduce food intake[1] and maintain glucose homeostasis[13]

Preclinical data in rodents suggest that amylin may interact with CCK to reduce food intake[1]

Amylin and CCK may stimulate similar intracellular signaling pathways, albeit in different areas of the brain, in the CNS of animal models. The interaction mechanism needs to be elucidated, but the effect of CCK is thought to be mediated by amylin signaling

Amylin may interact with PYY to reduce food intake[1]



Acute PYY administration in animal models enhances amylin-induced c-Fos expression in the area postrema, leading to increased signaling

Amylin may interact with insulin to reduce food intake[1]

Insulin may potentiate amylin signaling in the CNS of animal models, interacting centrally and potentially mediated by projections from amylin-activated areas in the area postrema to the hypothalamus

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ATP, adenosine triphosphate; CCK, cholecystokinin; c-Fos, Fos proto-oncogene (activator protein-1 transcription factor subunit); CNS, central nervous system; GCG, glucagon; GI, gastrointestinal; GIP, glucose-dependent insulinotropic peptide; GLP-1, glucagon-like peptide-1; LPL, adipocyte lipoprotein lipase; NuSH, nutrient-stimulated hormone; OXM, oxyntomodulin; PP, pancreatic polypeptide; PYY, peptide tyrosine tyrosine.

1. Lutz TA. *Diabetes Obes Metab* 2013;15:99–111; **2.** Jastreboff AM, Kushner RF. *Annu Rev Med* 2023;74:125–39; **3.** Moore CX, Cooper GJ. *Biochem Biophys Res Commun* 1991;179:1–9; **4.** Côté CD et al. *J Biol Chem* 2014;289:11642–9; **5.** Stanley S et al. *Am J Physiol Gastrointest Liver Physiol* 2004;286:G693–7; **6.** Parker HE et al. *Expert Rev Mol Med* 2010;12:e1; **7.** White PJ et al. *Nat Rev Endocrinol* 2025;21:375–412; **8.** Akalu Y et al. *Int J Endocrinol* 2020;2020:1385138; **9.** Rohli KE et al. *Biomolecules* 2022;12:335; **10.** Pullman J et al. *Visc Health Risk Manag* 2006;2:203–12; **11.** Butler PC et al. *Diabetes* 1990;39:752–6; **12.** Young A. *Adv Pharmacol* 2005;52:67–77; **13.** Grandy S et al. *J Diabetes Metab* 2016;7:1000643.