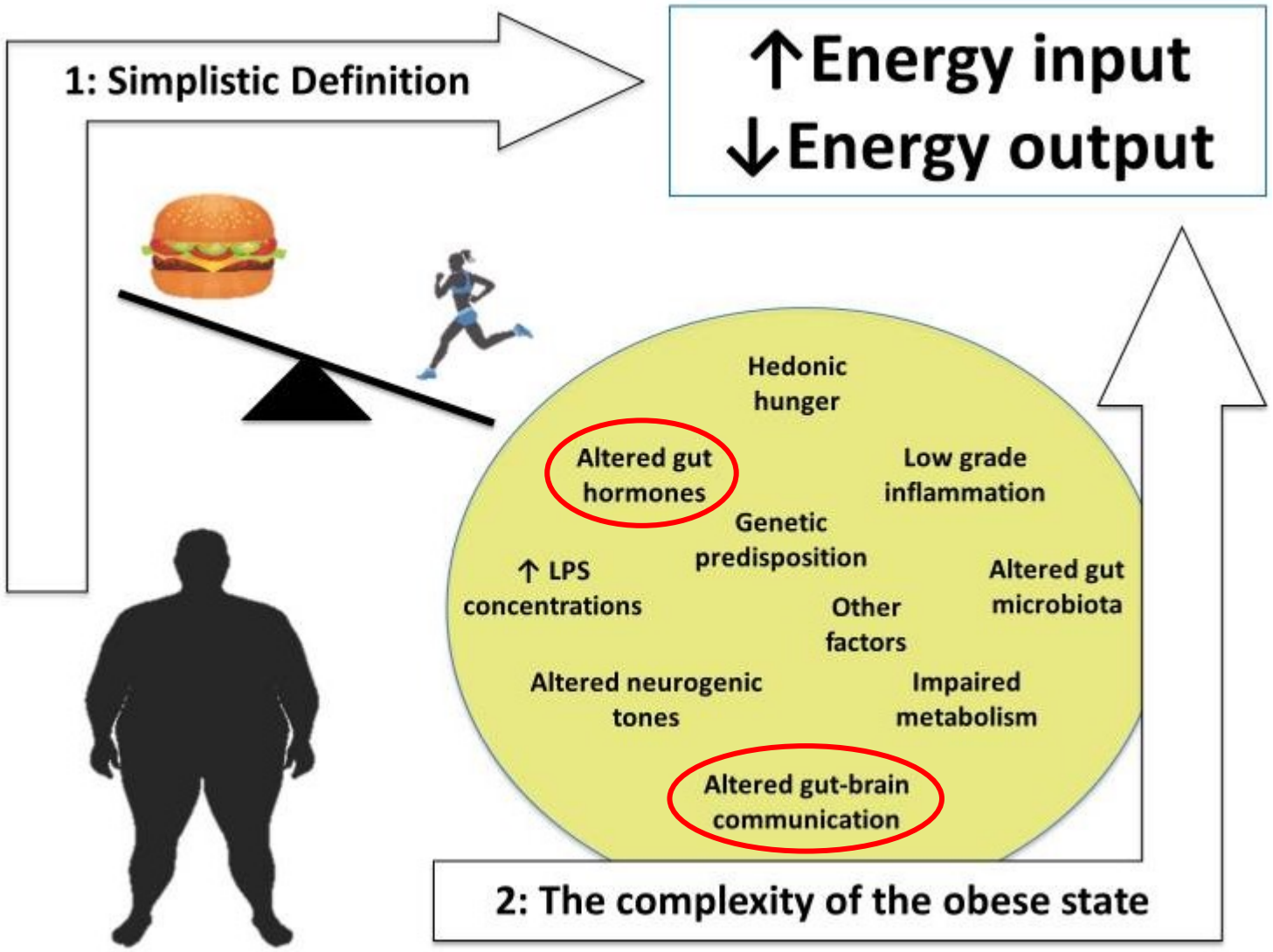
Session I. Obesity and Gut

Gut Hormone Overview

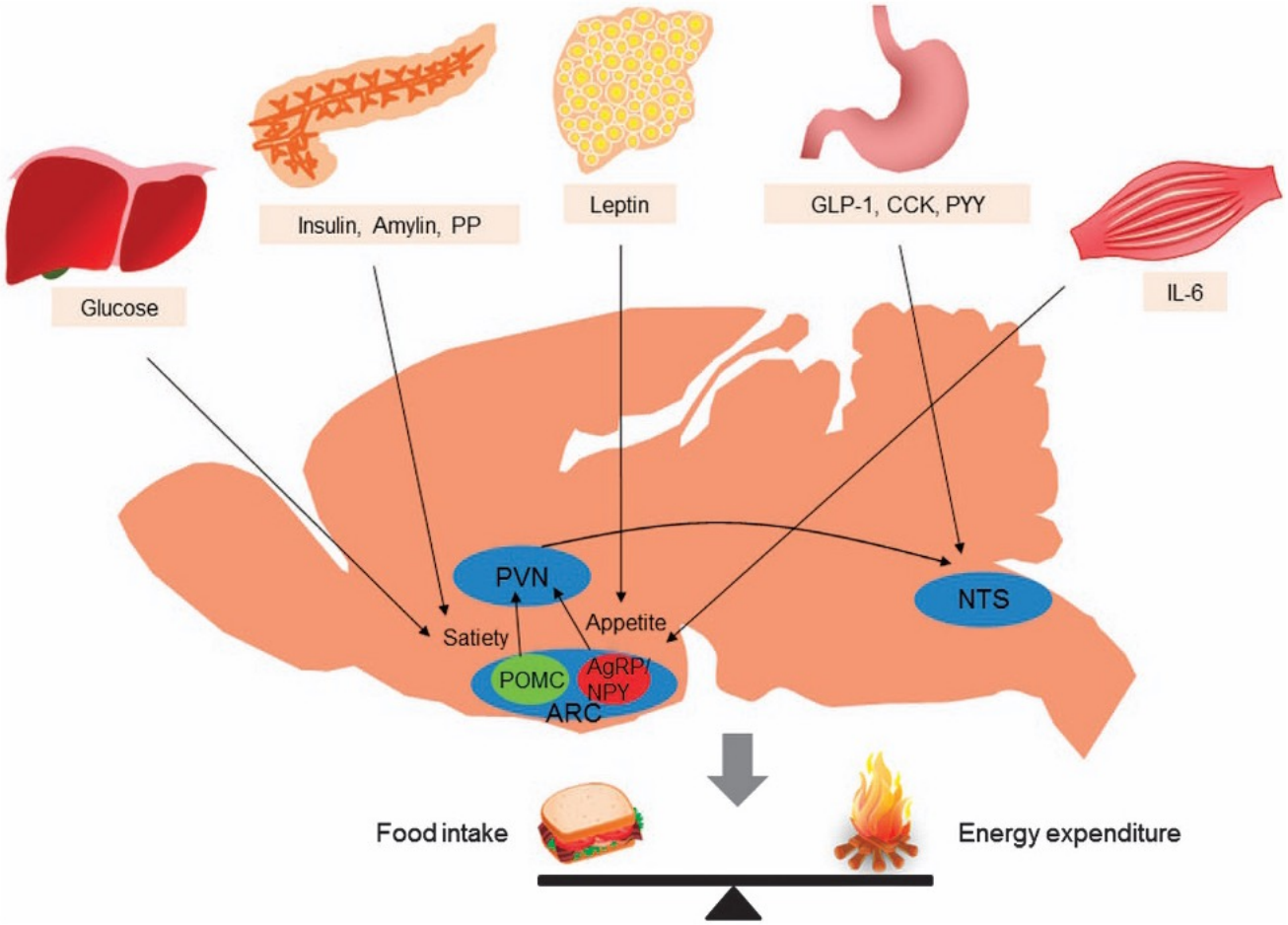
원광대산본병원

서 유 빈

Pathophysiology of the obese state



Central regulation of energy metabolism

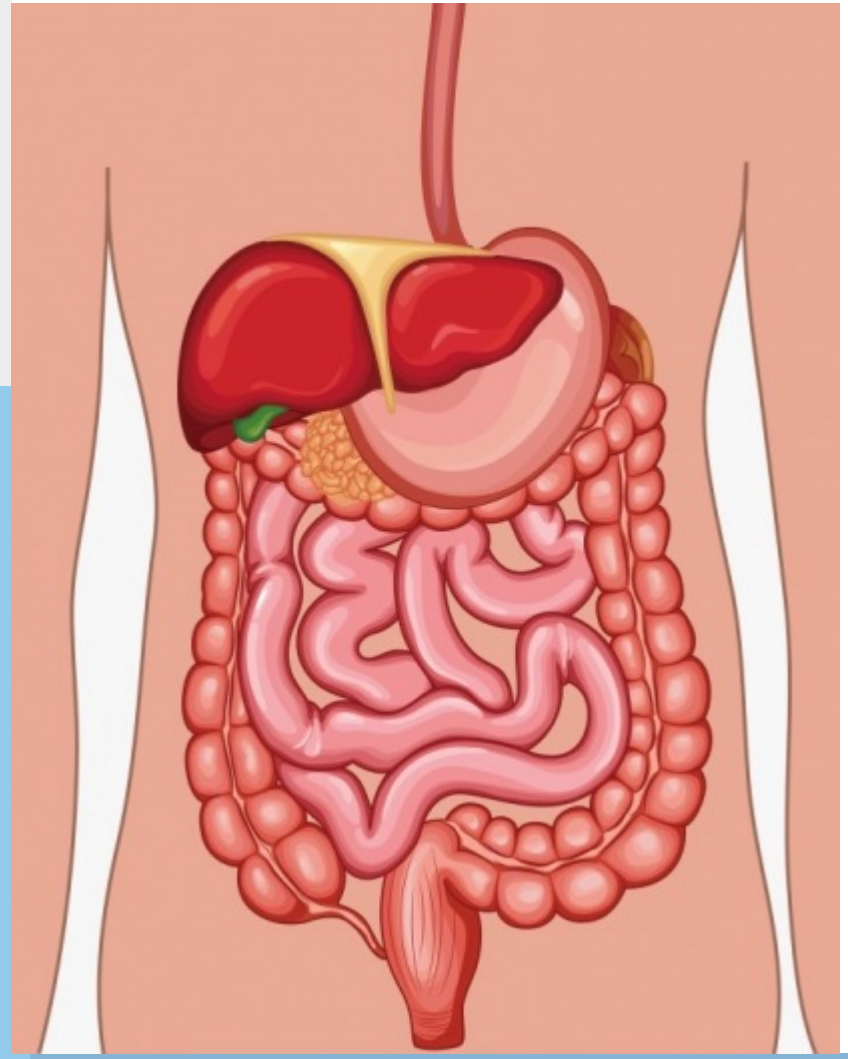


Peripheral signals of energy availability

Short-term : nutritional state
Long-term : adiposity

Roh, E., et al. (2016). Exp Mol Med, 48(3), e216-e216.

Central regulation of energy metabolism



Gut

- The first point of contact with ingested nutrients
- Metabolically active
- Key sensory organ
- Largest endocrine system

  - secretes more than 20 peptide hormones

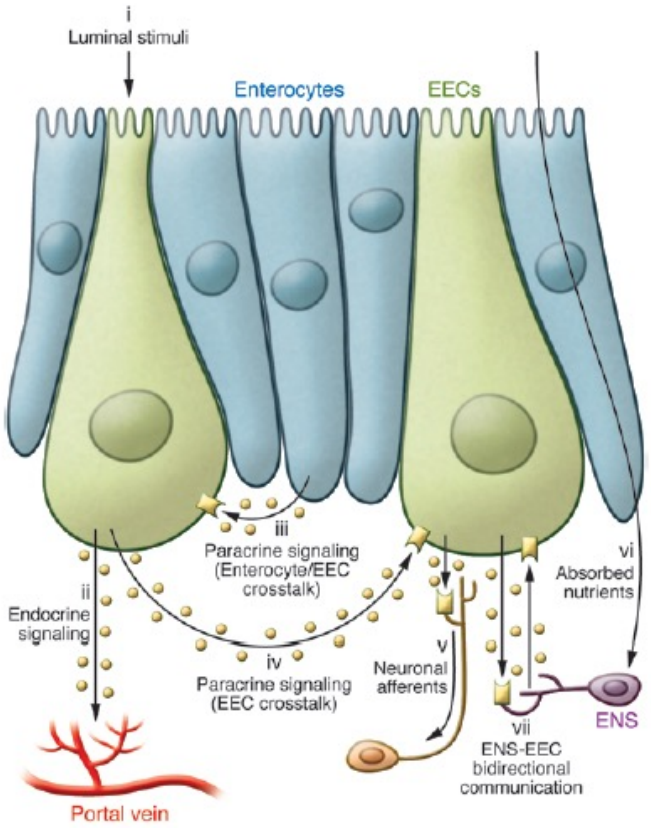
Gut hormones

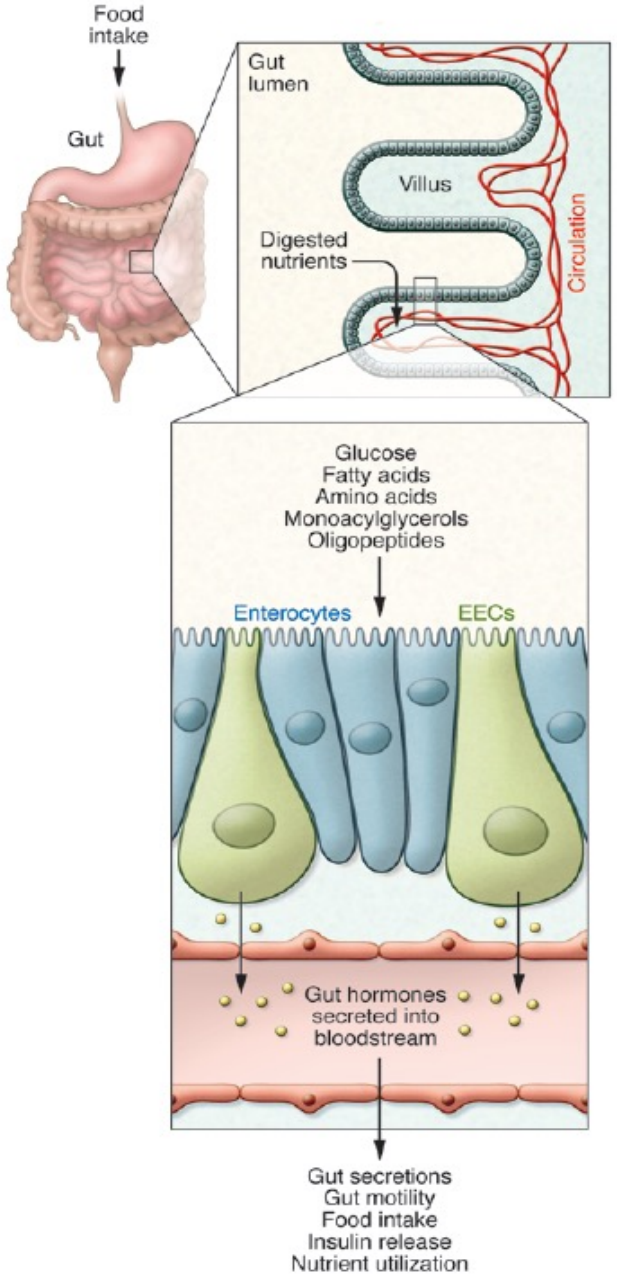
- GI motility and secretion
- Regulate eating behavior
- Energy and glucose homeostasis

Roh, E., et al. (2016). Exp Mol Med, 48(3), e216-e216.

Enteroendocrine cells (EECs)

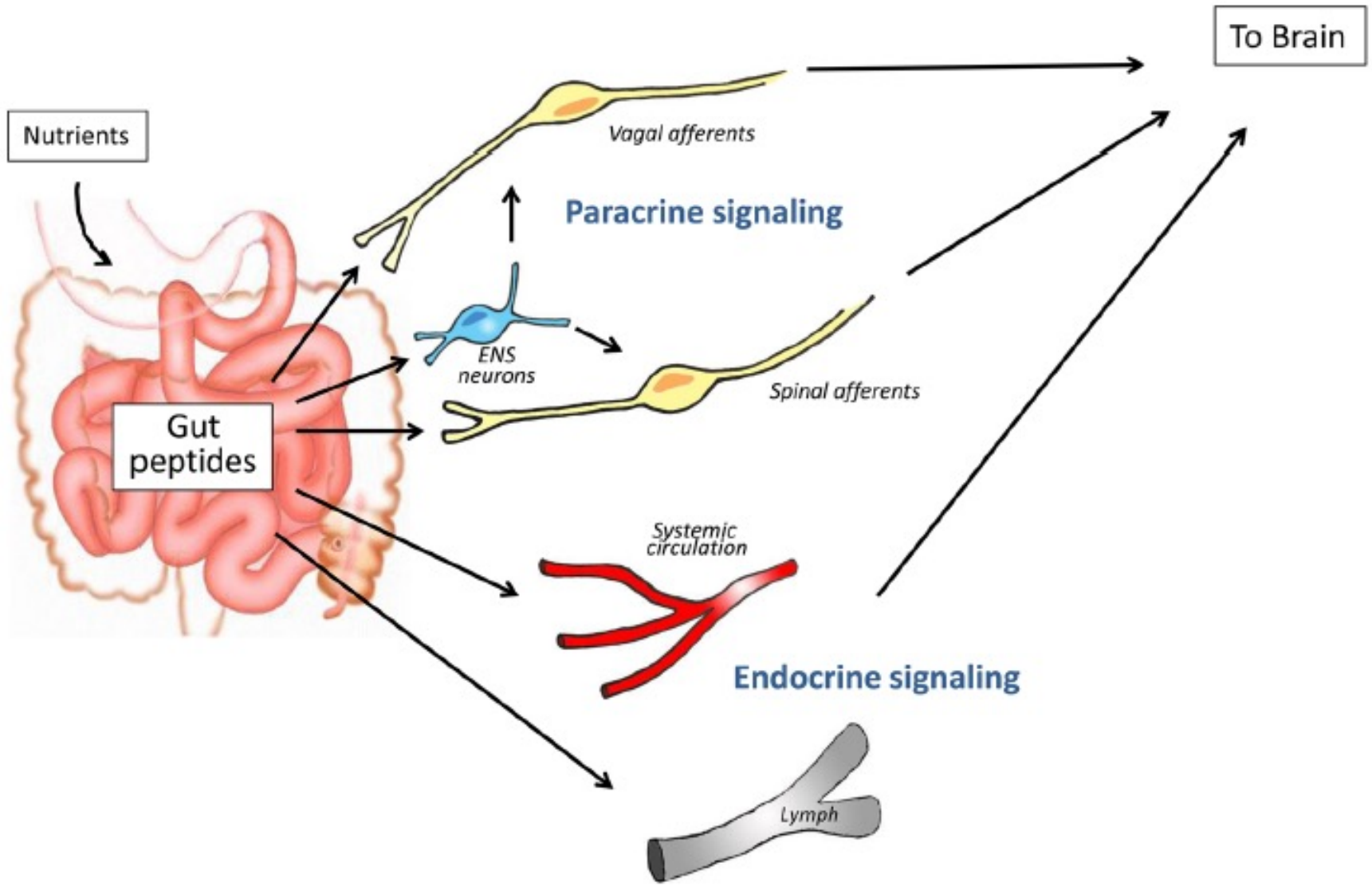
- Specialized trans-epithelial cells
- Constitutes < 1% of the epithelial cells on GI tract
- Stimulated by preabsorptive nutrients and secretes gut hormones





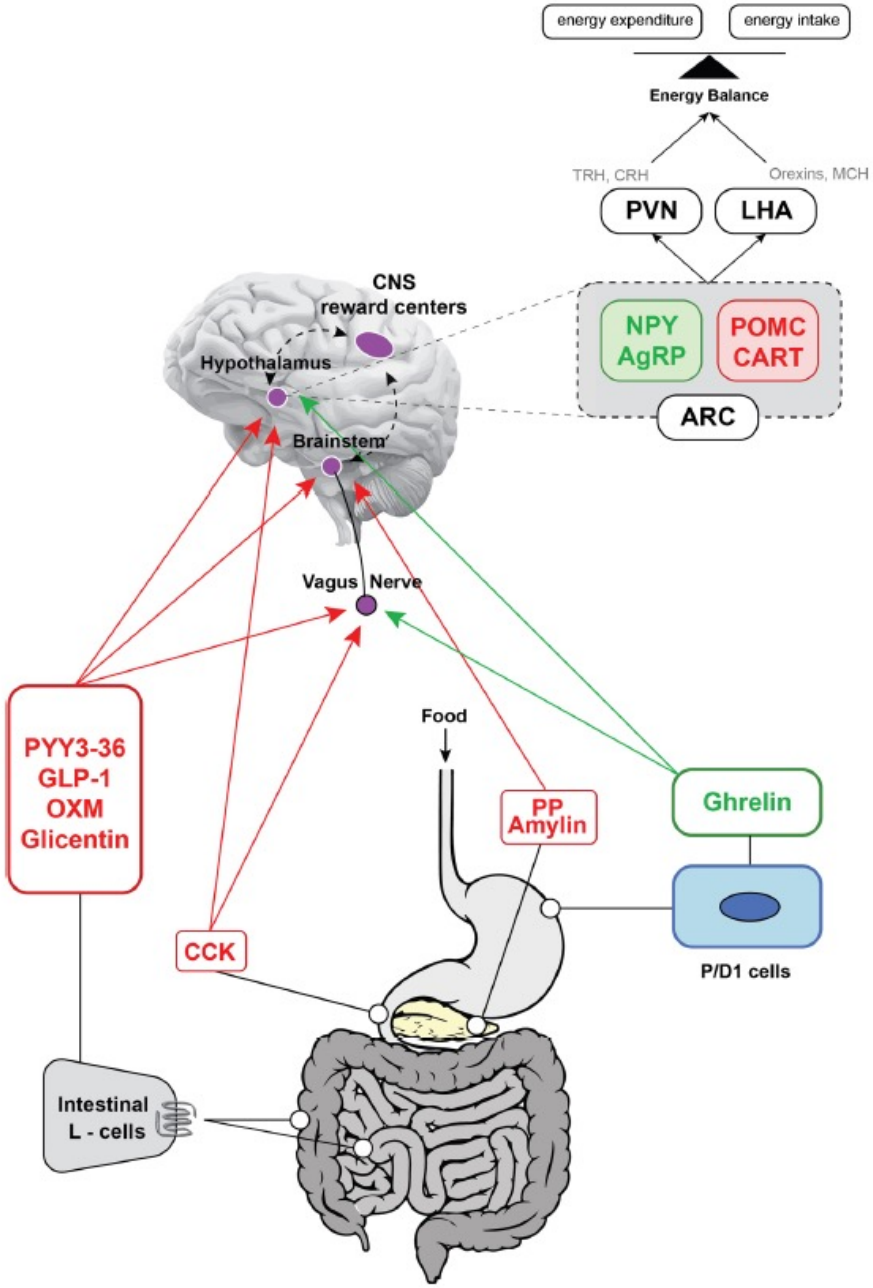
Psichas, A., et al. (2015). J Clin Investig, 125(3), 908-917.

Gut–brain communication through gut peptides



Bauer, P. V. et al. (2016) CMLS, 73(4), 737-755.

Gut brain axis



Koliaki, C., et al. (2020). Curr Obes Rep, 9(3), 255-271.

Gut hormone secretion along the GI tract

Table 2 | Types of enteroendocrine cells and their secreted products

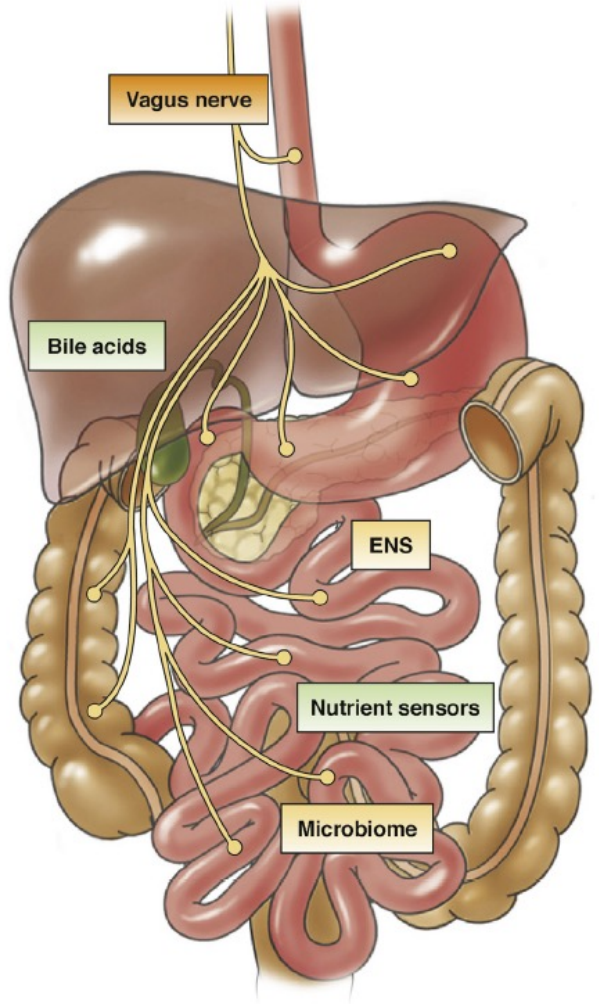
Cell type	Secreted hormone	Location
α cells	Glucagon	Pancreas (islets of Langerhans)
β cells	Insulin, islet amyloid polypeptide	Pancreas (islets of Langerhans)
PP cells	Pancreatic polypeptide	Pancreas (islets of Langerhans)
δ cells (D cells)	Somatostatin	Pancreas (islets of Langerhans)
G cells	Gastrin	Stomach Occasionally in the pancreas
X/A-like cells	Ghrelin, nesfatin-1	Stomach Occasionally in the small intestine
GIP cells (K cells)	GIP, xenin	Small intestine
S cells	Secretin	Small intestine
I cells (CCK cells)	Cholecystokinin	Small intestine
N cells	Neurotensin	Small intestine
L cells	PYY, GLP-1, GLP-2, oxyntomodulin	Small and large intestine

Abbreviations: CCK, cholecystokinin; GIP, gastric inhibitory polypeptide; GLP, glucagon-like peptide; PYY, peptide YY.

Duodenum
- CCK
- GIP
- Ghrelin

Jejunum
- GIP
- GLP1
- Apo A-IV
- Guanylin
- Uroguanylin

Ileum
- GLP1
- Oxyntomodulin
- PYY
- Neurotensin
- Apo A-IV
- Guanylin
- Uroguanylin



Stomach
- Ghrelin
- Nesfatin-1
- Leptin

Lipid derived molecules
- Endocannabinoid agonists
- Anorexic lipid OEA

Colon
- GLP1
- GLP2
- Oxyntomodulin
- PYY

Monteiro, M. P., et al. (2017). Gastroenterology, 152(7), 1707-1717.

Gut hormones

Orexigenic hormones

Ghrelin

Insulin- like peptide 5 (INSL 5)

Anorexigenic hormones

Glucagon-like Peptide-1 (GLP-1)

Oxyntomodulin (OXM)

Glicentin

Proglucagon-derived peptides

Peptide YY (PYY)

Pancreatic polypeptide (PP)

Pancreatic polypeptide (PP-fold) peptides

Glucose-dependent insulinotropic polypeptide (GIP)

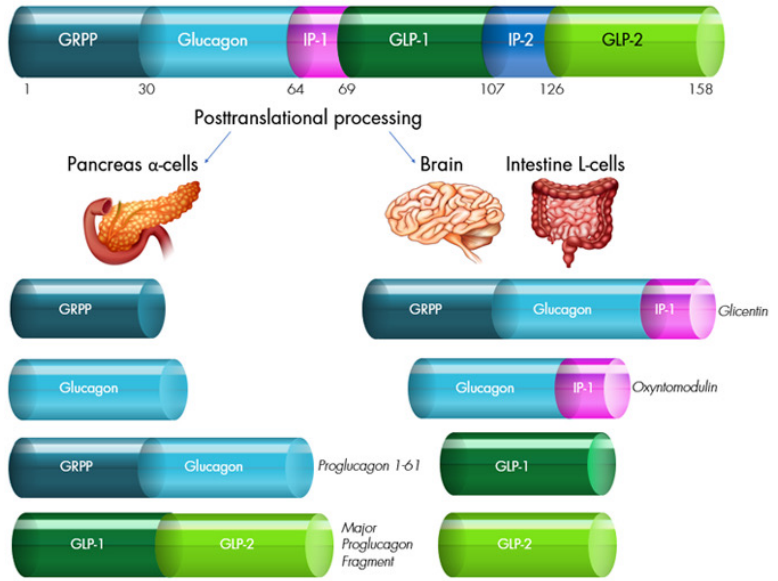
Amylin

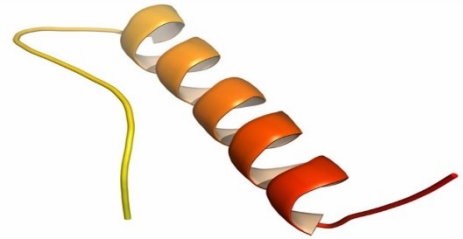
Cholecystokinin (CCK)

Uroguanylin

Neurotensin (NT)

Fibroblast Growth Factor 19 (FGF 19)





Ghrelin

Production and activation

P/D1-type cells in the gastric fundus and duodenum

activated by acylation (ghrelin O-acyl transferase (GOAT))

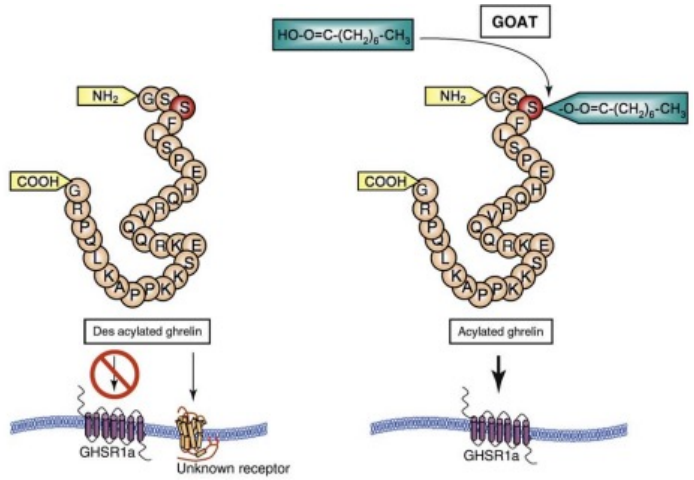
Stimulus for release

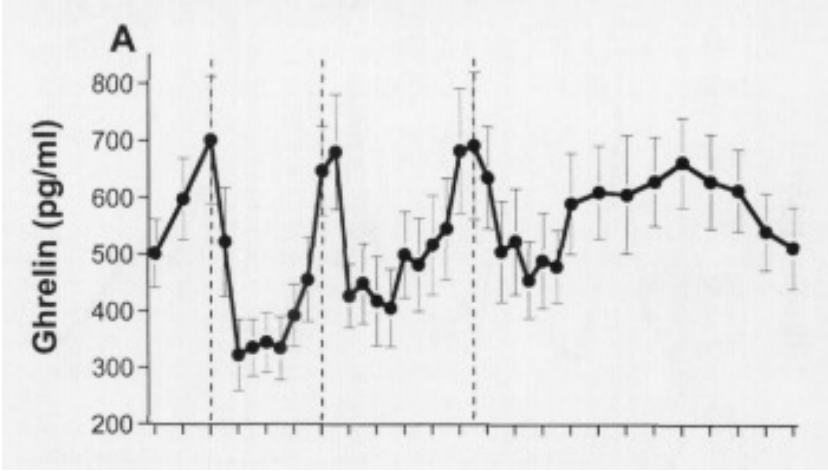
Fasting, Food cues

Circulating level

Peak prior to intake, fall rapidly in the postprandial state

(carbohydrates > fats)





Koliaki, C., et al. (2020). Curr Obes Rep, 9(3), 255-271.

Ghrelin

Targets

growth hormone secretagogue-receptor (GHS-R) in hypothalamus, brainstem, CNS reward centers, vagus nerve)

Effects

1) Brain (hypothalamus, brainstem)

Increases appetite, hedonic response to food cues

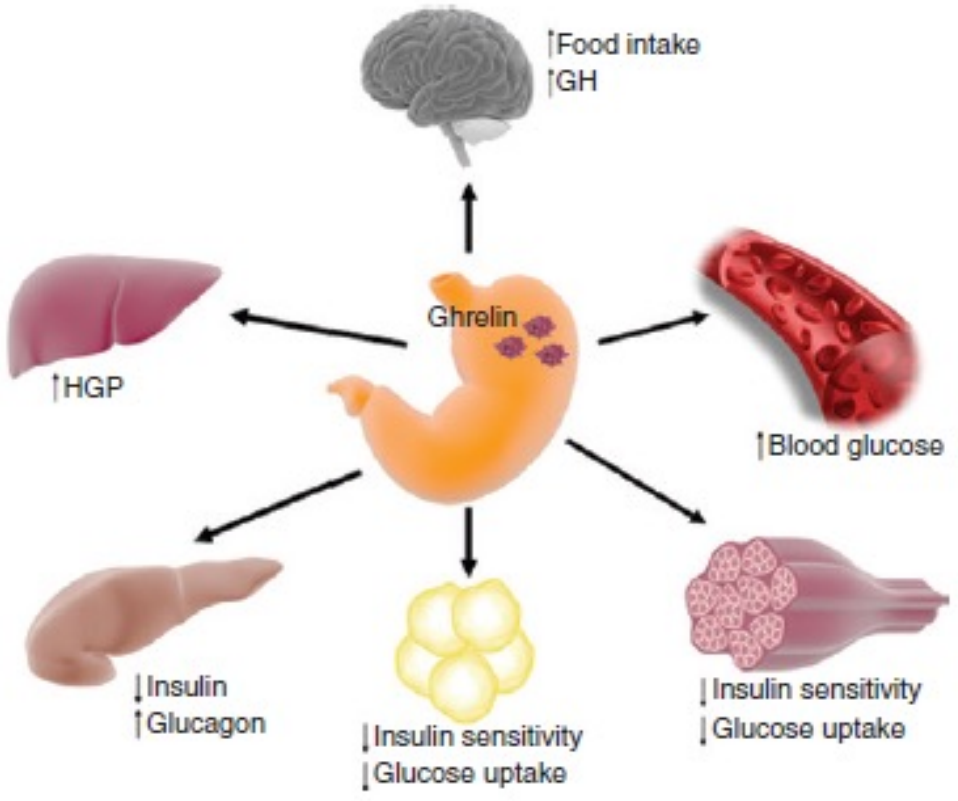
2) Pancreas

Reduces insulin secretion

Stimulates hepatic glucose production

3) GI tract

Prokinetic effects - gastric emptying, gastric acid secretion



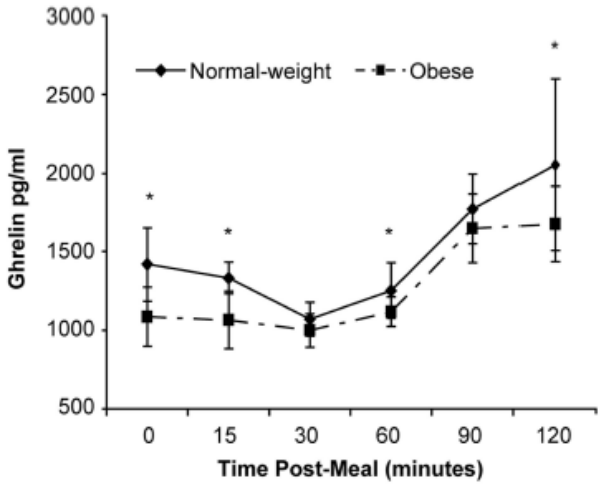
Alamri et al. (2016) Horm Mol Biol Clin Invest; 26(1): 3–11

Ghrelin

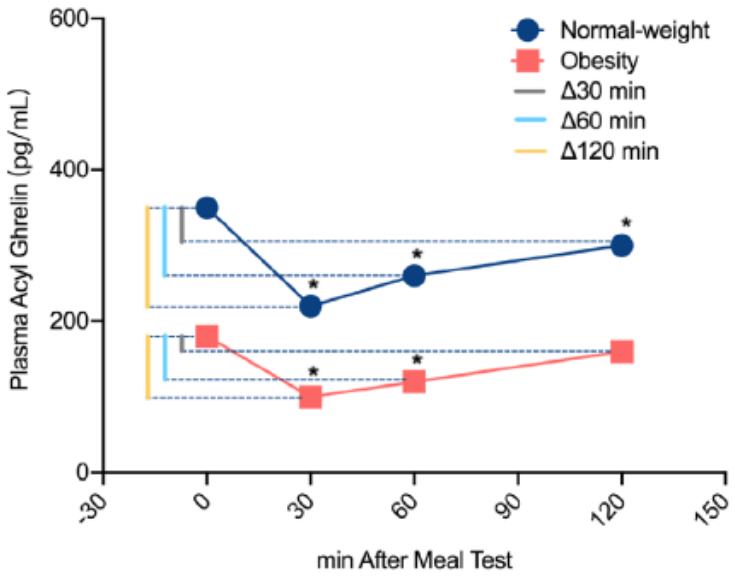
Change in obesity

- negatively correlated with BMI and insulin resistance
- attenuated postprandial suppression

: related with compensatory adaptation of (+) energy balance



Carlson JJ, et al. (2009) Nutr Metab 6: 32.



scientific reports

Circulating acyl and des-acyl ghrelin levels in obese adults: a systematic review and meta-analysis

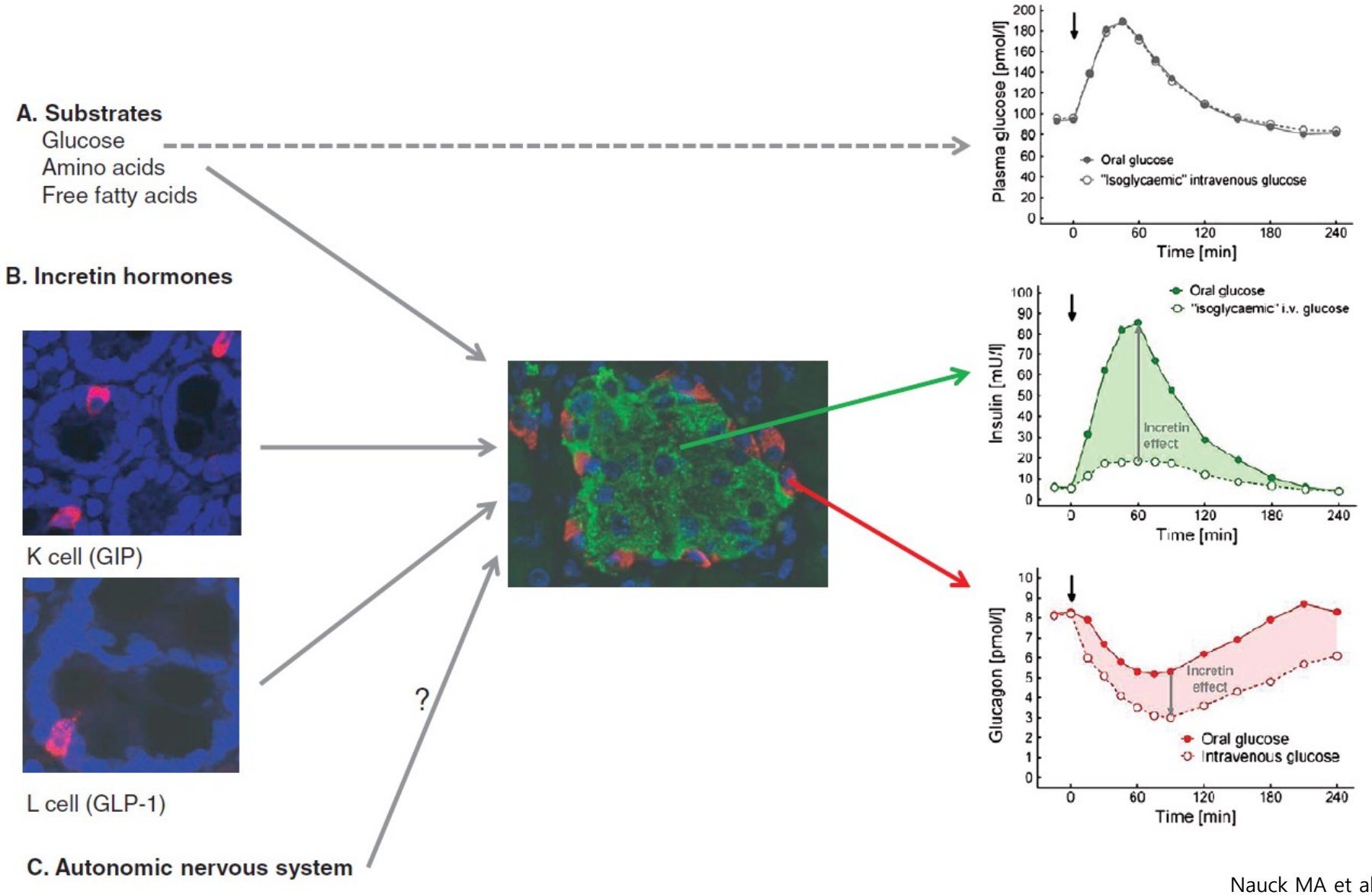
Yanmei Wang[1,2,4], Qianxian Wu[2,4], Qian Zhou[1], Yuyu Chen[3], Xingxing Lei[1], Yiding Chen[1] & Qiu Chen[1]✉

* P<0.05 for difference between postprandial (30 min, 60 min,120 min) vs. fasting (0 min) → A shorter duration of AG suppression in obese groups

P≥0.05 for changes in postprandial AG (Δ30 min, Δ60 min, Δ120 min) Obesity vs. Normal-weight → A similar extent of AG decline in obese and normal-weight groups

Wang, Y., et al. (2022). Sci Rep 12(1), 1-17.

Incretins



Insulinotropic incretin effect

Glucgonostatic incretin effect

Nauck MA et al. Diabetes Obes Metab. 2018;20(Suppl. 1):5–21.

GLP-1 (glucagon-like peptide 1)

Source of production

L cells of the distal jejunum, ileum, and colon

Stimulus for release

Ingested nutrients (mainly carbohydrates and fats)

Circulating level

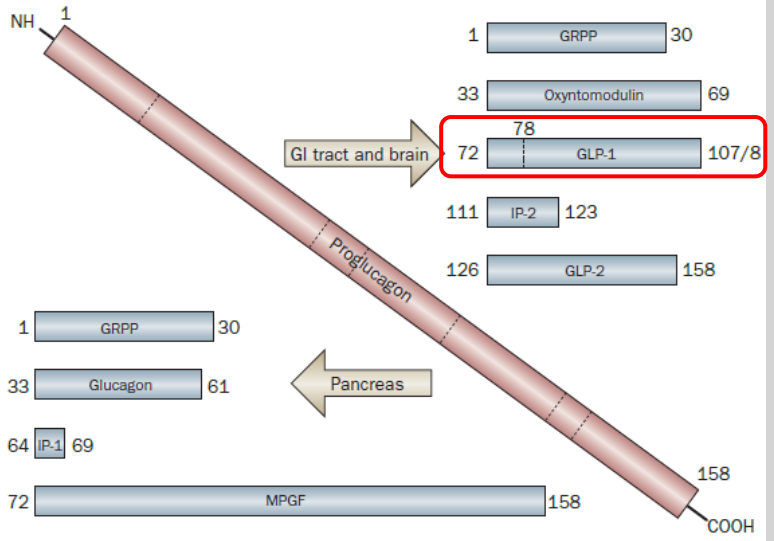
Biphasic secretion

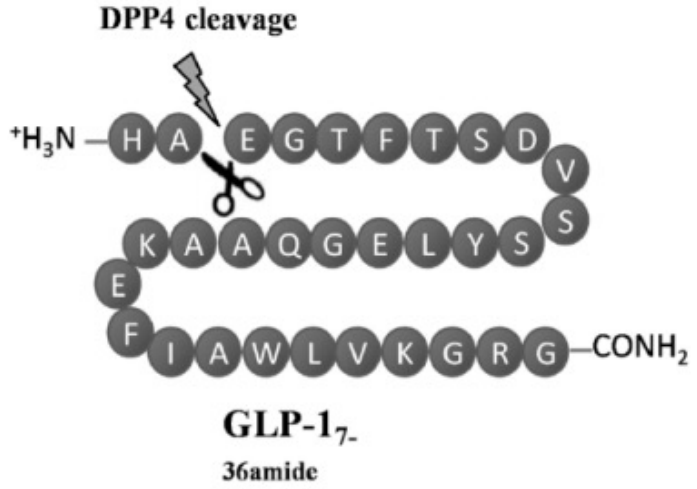
: early phase within 15 min after intake → second, longer peak at 30-60min

Reach basal concentrations again after several hours

* Inactivated by DPP-IV, having plasma half-life of less than 2 min

→ only about 10-15% reaches the peripheral tissues





Field, B. C., et al. (2010). Nat. Rev. Endocrinol., 6(8), 444-453.

GLP-1 (glucagon-like peptide 1)

Targets

GLP-1 receptors : widely distributed including hypothalamus, liver, pancreas and skeletal muscle

Effects

1) Brain (hypothalamus, brainstem)

Reduces appetite and food intake

2) Pancreas

Incretin effect : glucose-dependent insulin secretion

Suppresses post prandial glucagon release

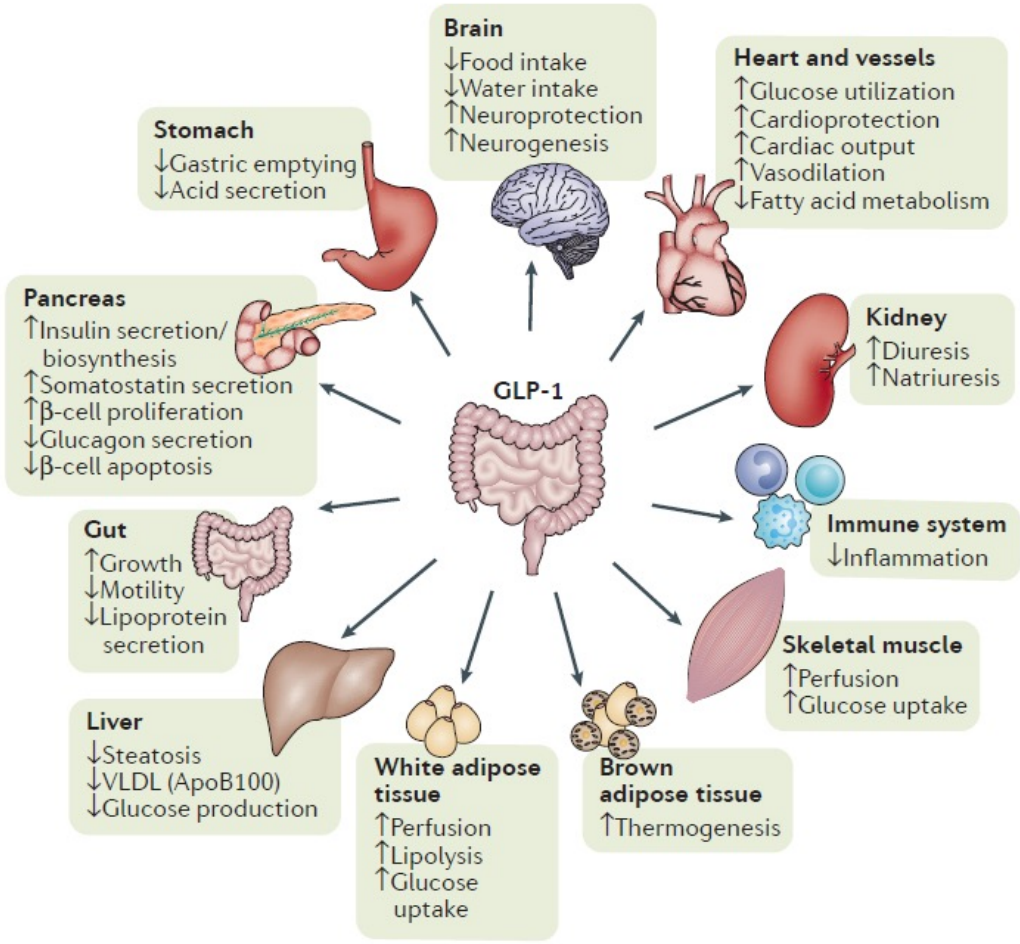
Enhances β-cell proliferation

3) GI tract

Delays gastric emptying

4) Brown adipose tissue

Increases thermogenesis

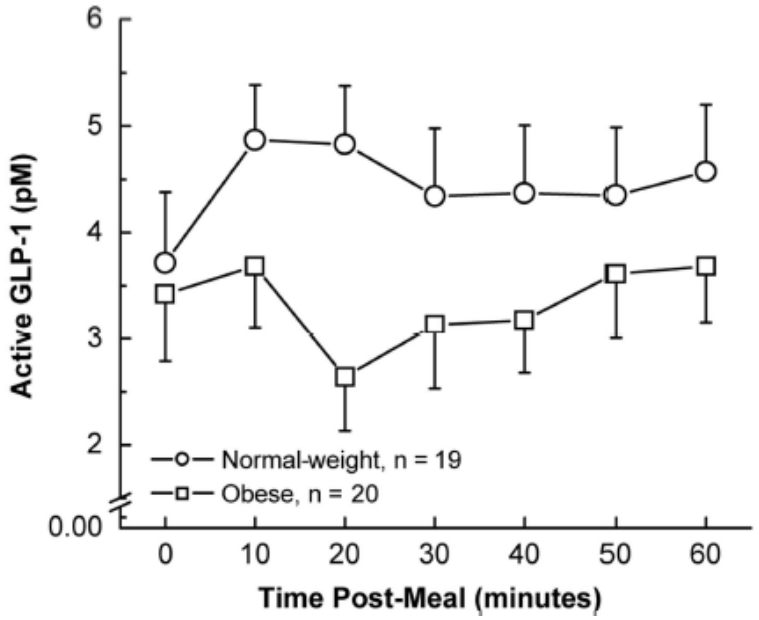


Muskiet, M. H., et al. (2017). Nat. Rev. Nephrol., 13(10), 605-628.

GLP-1 (glucagon-like peptide 1)

Change in obesity – attenuated postprandial response

GLP-1 responses to a liquid test meal
19 normal-weight and 20 obese subjects



Obesity 2007; 15: 2974–2983.

GLP-1 Response to Oral Glucose Is Reduced in Prediabetes, Screen-Detected Type 2 Diabetes, and Obesity and Influenced by Sex: The ADDITION-PRO Study

Diabetes 2015;64:2513–2525 | DOI: 10.2337/db14-1751

Obese and overweight individuals had up to 20% reduced GLP-1 response to oral glucose compared with normal weight individuals independent of glucose tolerance status. Higher GLP-1 responses were associated with better insulin sensitivity and β-cell function, older age, and lesser degree of obesity. Our findings indicate that a reduction in GLP-1 response to oral glucose occurs prior to the development of type 2 diabetes and obesity, which can have consequences for early prevention strategies for diabetes.

GIP (glucose-dependent insulinotropic polypeptide)

Source of production

K cells of the proximal small intestine

Stimulus for release

Ingested nutrients (mainly carbohydrates and lipids)

Circulating level

Rise a few minutes after nutrient intake, peak after approximately 1 h

Reach basal concentrations again after several hours

Targets

GIP receptor in pancreatic islet cells, hypothalamus, adipose tissue

Effects

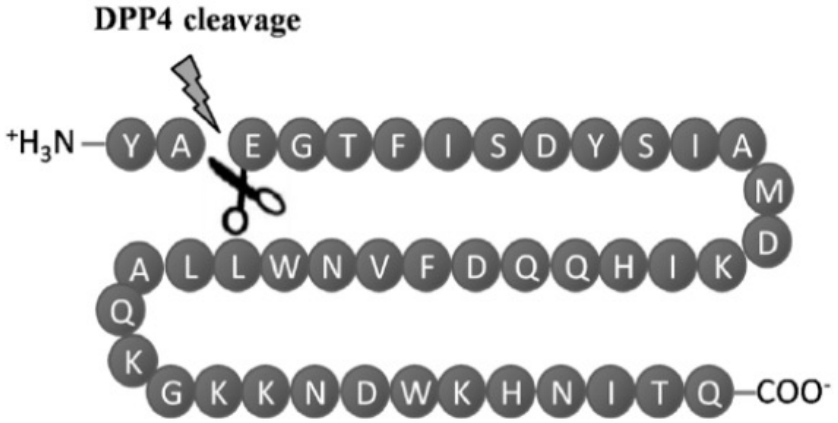
Incretin effect

Strong anabolic effects on adipose tissue, promoting fat accumulation

Weak effect in reducing energy intake

Changes in obesity

Increased



Zhu, Q., et al. Nature Reviews Nephrology, 14(2), 105-120. 2018

OXM (Oxytomodulin)

Source of production

cosecreted with GLP-1 by L cells in distal gut

Stimulus for release

Ingested nutrients, in proportion to caloric load

Targets

GLP-1 receptor, glucagon receptor, unknown receptor in hypothalamus

(50-fold lower affinity, 100-fold lower affinity than GLP-1, glucagon respectively)

Effects

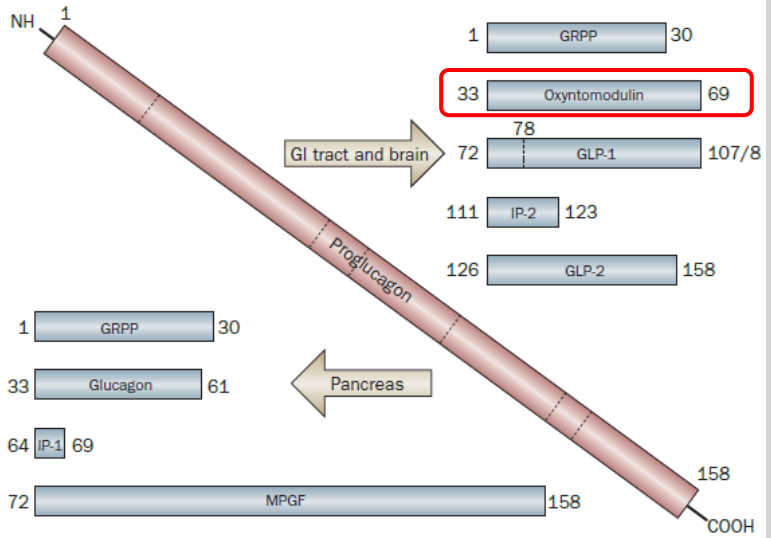
Reduces appetite and food intake (also by ghrelin suppression)

Neutral action in glucose homeostasis

Delays gastric emptying

Change in obesity

Unknown



Koliaki, C., et al. (2020). Curr Obes Rep., 9(3), 255-271.

PYY (peptide YY)

Source of production and activation

cosecreted with GLP-1 by L cells in distal gut

activated by DPP4 (secreted form : PYY(1-36) / active form : PYY (3-36))

Stimulus for release

Ingested nutrients (fat>protein>carbohydrate)

Circulating level

begin to increase 15–30 min after meals, peak 60–90 min after meals, and remain elevated for several hours

Targets

Y2 receptor in hypothalamus, hedonic circuits

Effects

Reduces appetite and food intake

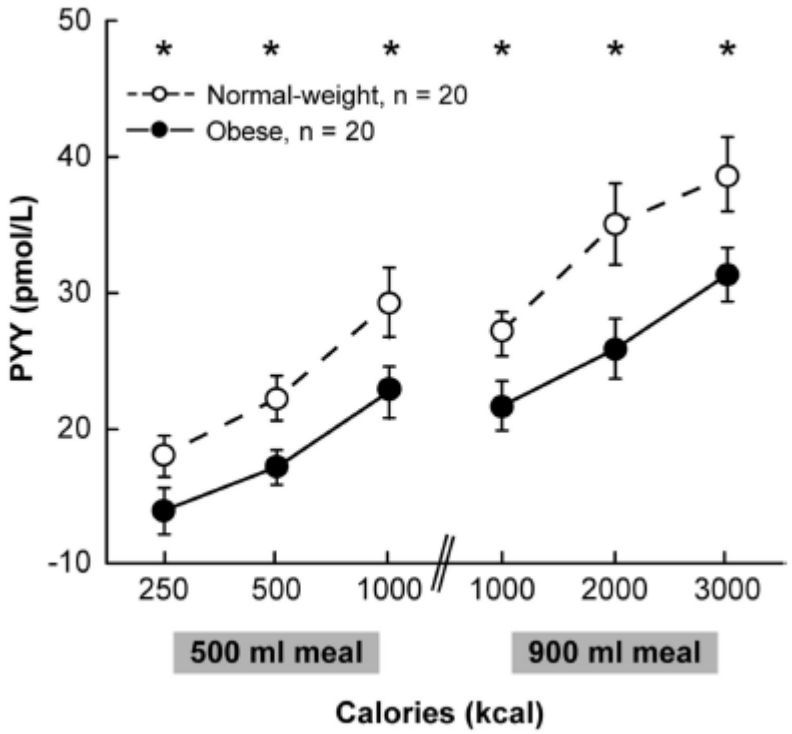
Promotes insulin secretion

Delays gastric emptying

Neary, M. T., et al. (2009). Physiolo Behav, (2009)97(5), 616-619.

PYY (peptide YY)

Change in obesity – lower fasting level, attenuated postprandial response



90 min after six test meals of increasing caloric content
20 obese and 20 normal-weight subjects.
***P<0.05 (unpaired t-test).**

le Roux CW et al. (2006) Endocrinology 147: 3–8.

PP (pancreatic polypeptide)

Source of production

F-cells (PP cells) located in the islets of Langerhans

Stimulus for release

Ingested nutrients, adrenergic stimulation

Circulating level

Increase after meal, remain for up to 6 hours post-prandially

Targets

Y4 receptor within the brainstem and hypothalamus

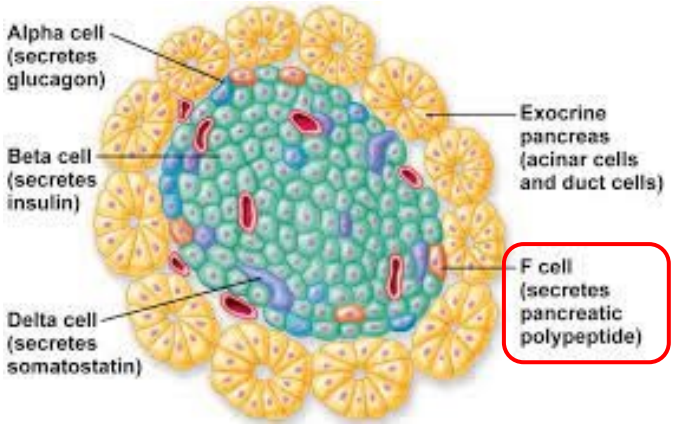
Effects

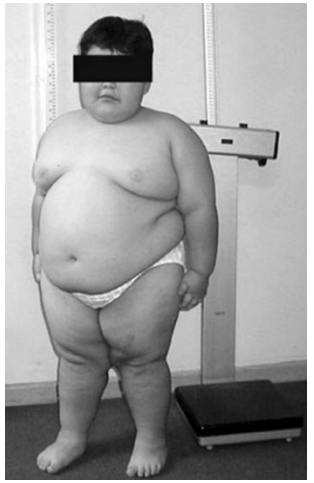
Suppresses appetite

Gallbladder relaxation, inhibits pancreatic secretion and delays gastric emptying

Changes in obesity

lower fasting, post-prandial level in Prader-Willi syndrome





Koliaki, C., et al. (2020). Curr Obes Rep., 9(3), 255-271.

CCK (cholecystocknin)

Source of production

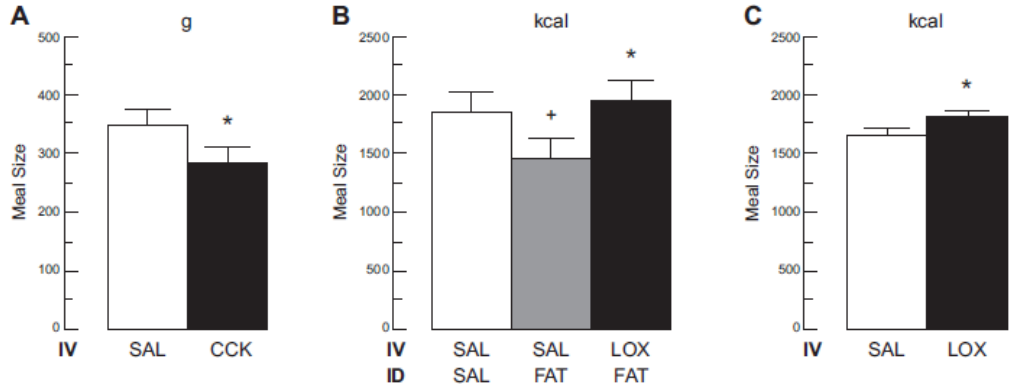
I cells of the duodenum

Stimulus for release

Ingested nutrients (mainly by fat)

Circulating level

increase in 10-15 min, peak in 60 min



Am J Physiol Regul Integr Comp Physiol 2001

Targets

CCK-1 receptors located in peripheral tissues and CCK-2 receptors in the brain

Effects

Reduces intake (Satiation signal)

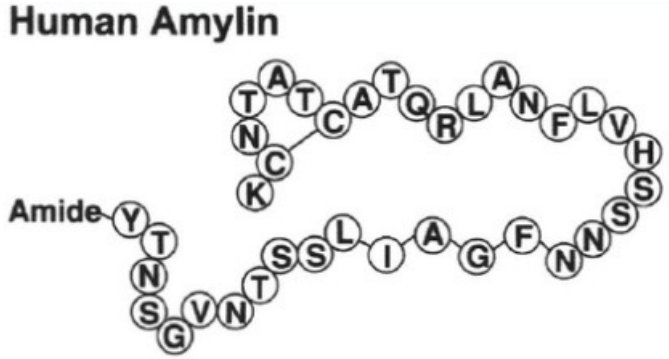
Promotes insulin secretion

Stimulates gallbladder contraction, enhances pancreatic enzyme secretion

Slows gastric emptying, inhibits gastric acid secretion

Steinert, R. E., et al.(2017). Physiol Rev, 97(1), 411-463.

Amylin



Source of production

co-secreted with insulin by pancreatic β-cells

Stimulus for release

Ingested nutrients (glucose, lipids), incretins and neural signals

Circulating level

Rise rapidly following meal, peak within an hour and remain elevated up to 4 h postprandially

Targets

Amylin specific receptors in CNS reward centers, stomach, pancreas

Effects

Reduces intake

Suppresses postprandial glucagon secretion

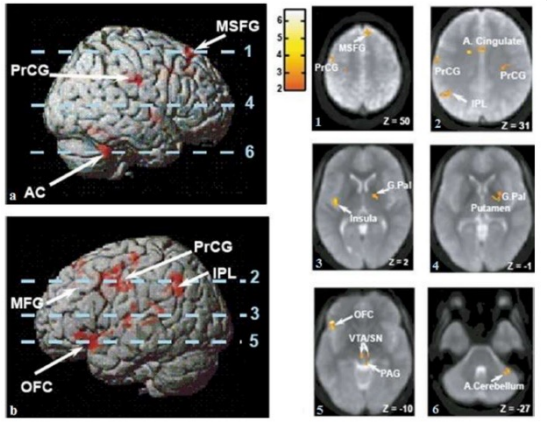
Slows gastric emptying

Change in obesity

increased amylin levels, downregulation of amylin receptor

Koliaki, C., et al. (2020). Curr Obes Rep., 9(3), 255-271.

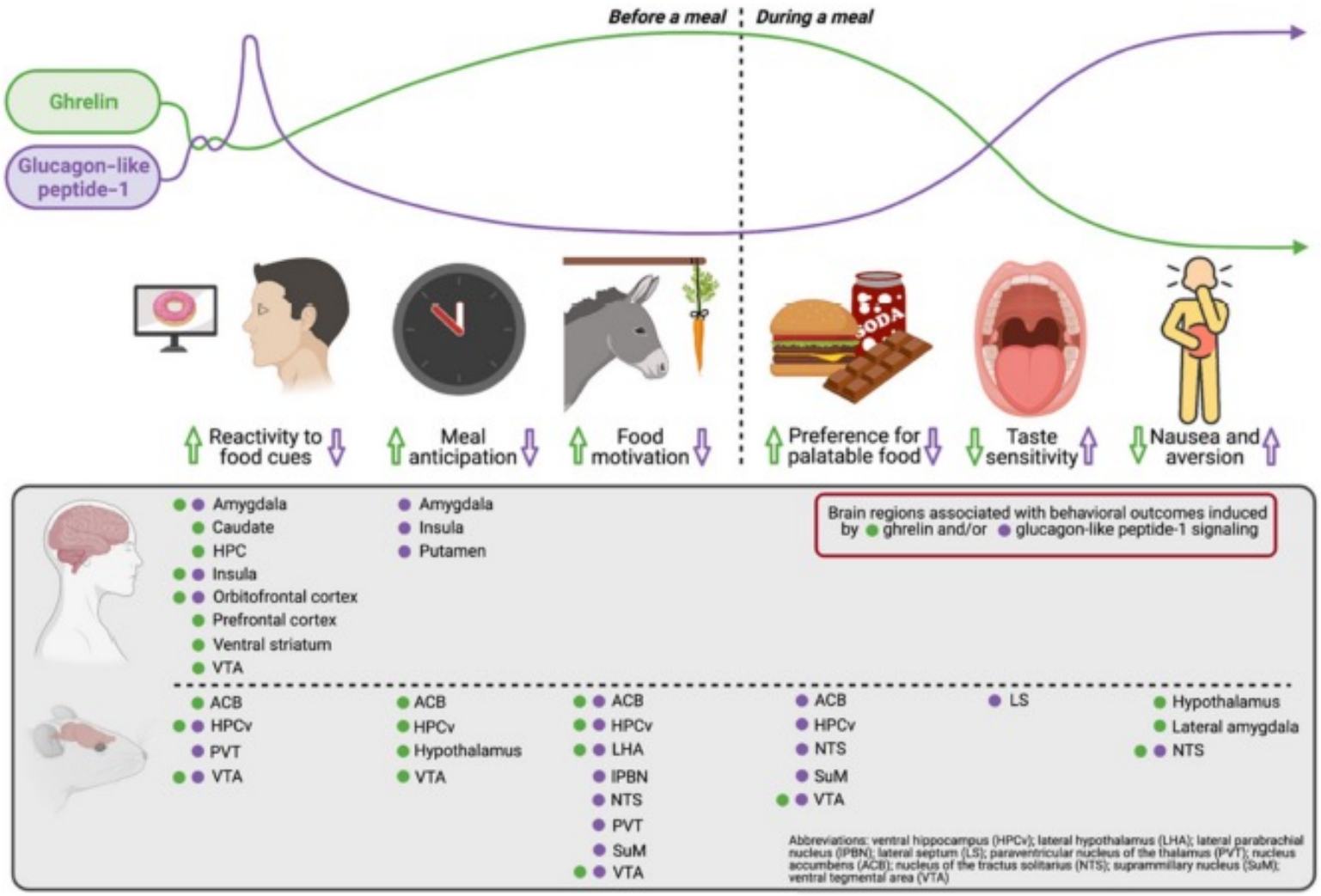
Gut hormones and hedonic eating



- **Ghrelin, GLP-1 and PYY** modulate neural activity in **reward center** (fMRI) Br J Radiol 2018
- Exposure to **food-related stimuli** → change in gut hormone (Ghrelin, PYY) → alteration in reward value (food cue, memory, social factor) Nature 2007
- Gut hormones receptors in **olfactory and gustatory cortex** → **palatability** of food Am J Clin Nutr 2015
- **Amylin** reduce reward value of high-fat, high-carb diet (animal) Neuropharmacology 2017

→ Gut hormones influence both the **intention and desire to eat**, as well as perceived **hedonic value** of food

Makaronidis, J. M., et al. (2019). Curr Opin in Physiol, 12, 1-11.

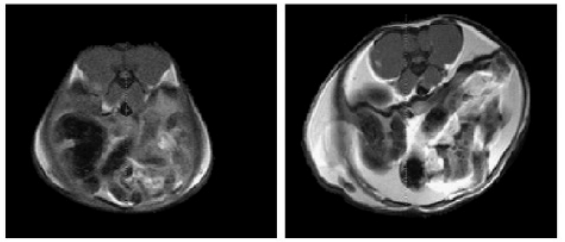


Decarie-Spain, L. et al. (2021). Nutrients, 13(3), 977.

Altered gut hormones profiles in obesity

Gut hormone	Change
Ghrelin	↓ fasting levels ↓ postprandial suppression
GLP-1	↓ fasting levels ↓ postprandial secretion
OXM	Unknown
GIP	↑ levels
PYY	↓ fasting levels ↓ postprandial secretion
PP	↓ fasting levels in Prader–Willi syndrome Conflicting data in non-syndromic obesity
CCK	↓ satiety effect ↓ response to oleic infusion
Amylin	↑ levels ↓ satiety effect due to down-regulation of amylin receptors

Gut hormones in obesity pathogenesis



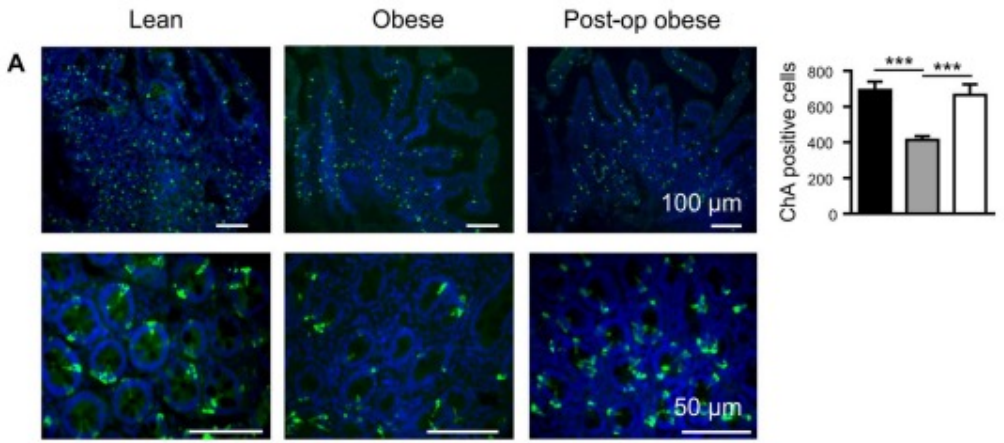
- Gut hormones may induce obesity

  ex) *Pyy* knock-out mouse developed adiposity Physiolo Behav, 2009
- High energy intake per se may chronically impair gut hormone responsiveness to nutrients

  ex) Diet-induced obesity → reduced circulating PYY and GLP-1 Exp Biol Med (Maywood) 2017

- Reduced population numbers and responsivity of gastrointestinal EECs in obesity (human) Sci Rep 2017

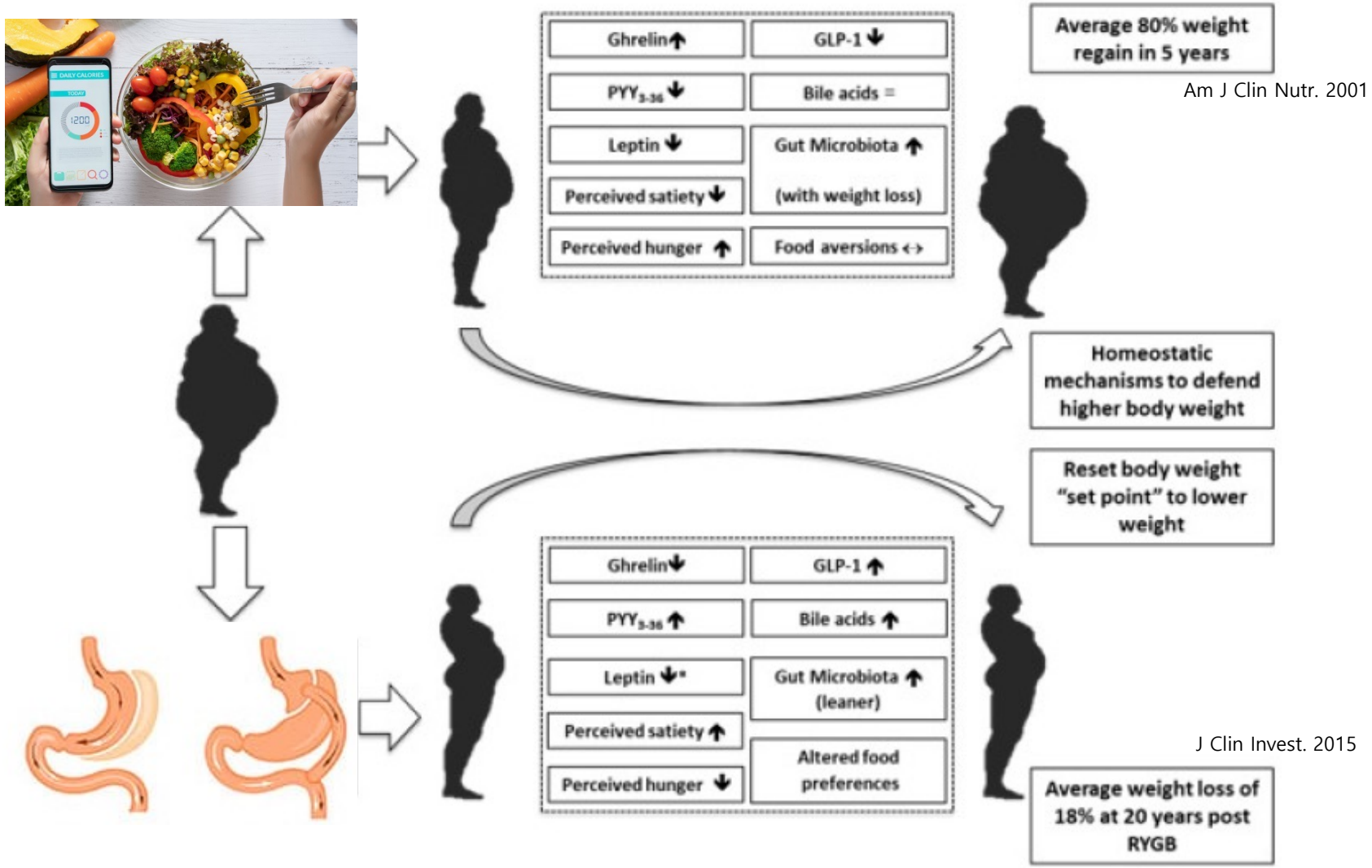
Figure 1. Expression of chromogranin A, CCK and ghrelin proteins in human duodenal biopsies. Profile of the enteroendocrine cell marker, chromogranin A (**A**, ChA) and the gut hormones CCK (**B**) and ghrelin (**C**), was determined in the duodenal biopsies of lean (■), obese (▨) and post-operative obese, (□) by immunohistochemistry. Bar charts (on the right) show number of cells counted expressing ChA or gut hormones. Statistical significance was determined by a One-way ANOVA with differences between means



- Gut hormone receptor expression on the vagus nerve and its responsivity to gut hormones are diminished in obesity (animal) Brain Res 2018

Gut hormones alteration after weight loss

- After energy deficit diet
- After bariatric surgery



Pucci, A., et al. (2020). Endotext [Internet].

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Long-Term Persistence of Hormonal Adaptations to Weight Loss

Participants: **50 obese** patients without diabetes

Intervention: 10-week 'very-low-energy diet'

Outcome: **Gut hormones and appetite**

Time point: at baseline, at 10 weeks, and at 62 weeks

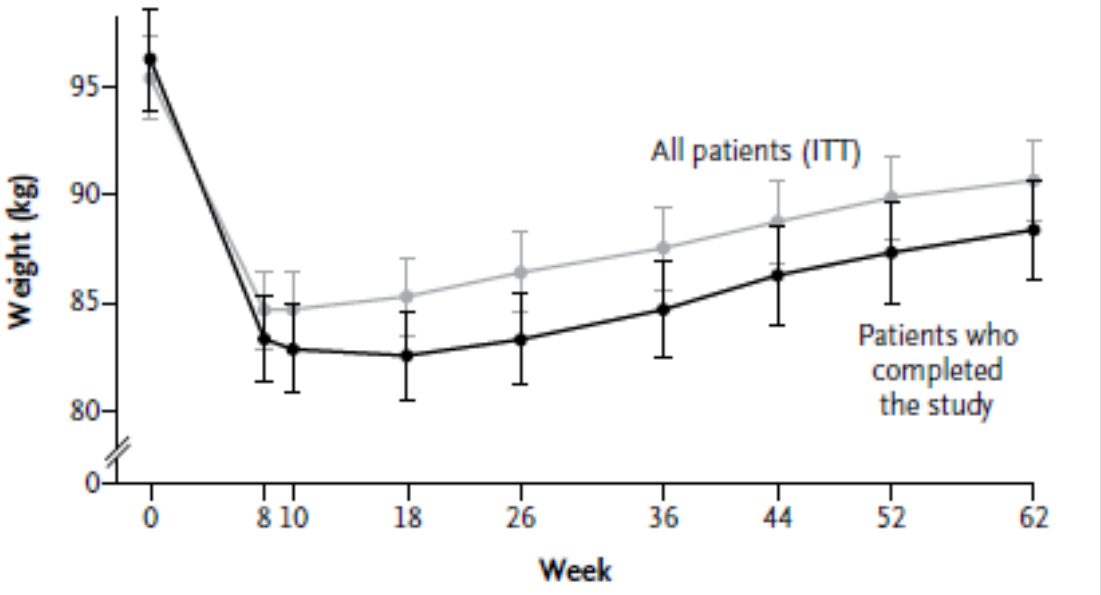


Figure 1. Mean (±SE) Changes in Weight from Baseline to Week 62.
The weight-loss program was started at week 0 and completed at week 10. ITT denotes intention to treat.

Wk 10: - 13.5±0.5 kg (14.0% of initial wt.)

Wk 10-62: + 5.5±1.0 kg

Sumithran et al. NEJM 2011;365:1597-604.

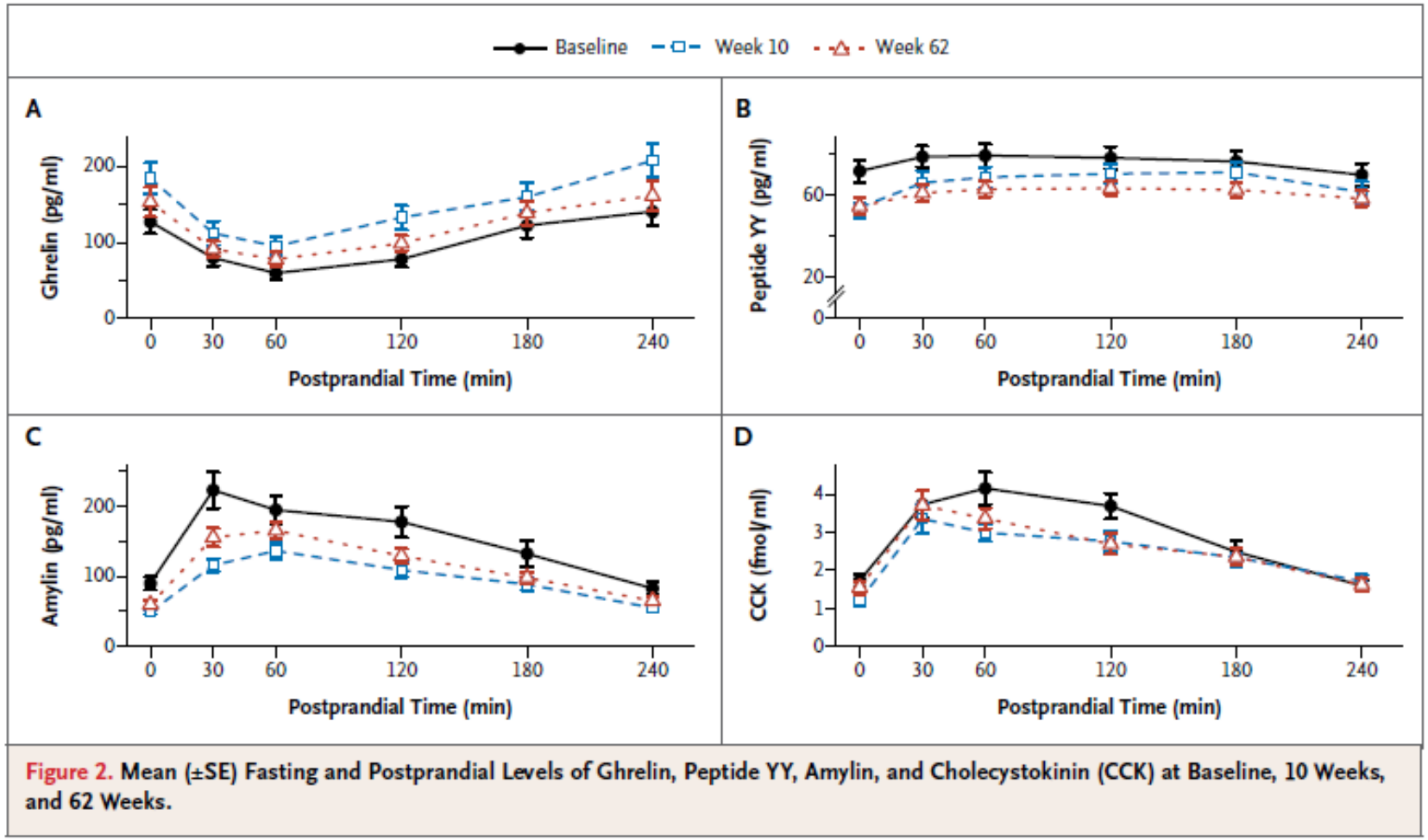


Figure 2. Mean (±SE) Fasting and Postprandial Levels of Ghrelin, Peptide YY, Amylin, and Cholecystokinin (CCK) at Baseline, 10 Weeks, and 62 Weeks.

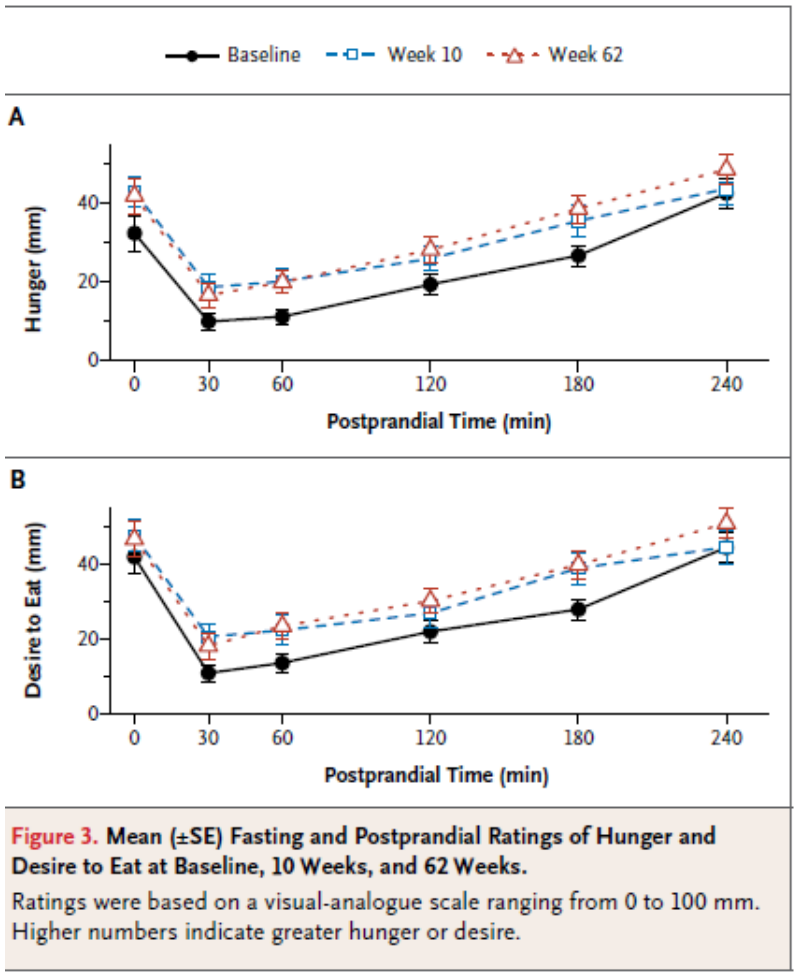


Figure 3. Mean (±SE) Fasting and Postprandial Ratings of Hunger and Desire to Eat at Baseline, 10 Weeks, and 62 Weeks.

Ratings were based on a visual-analogue scale ranging from 0 to 100 mm. Higher numbers indicate greater hunger or desire.

→ One year after initial weight reduction, levels of the **circulating mediators of appetite that encourage weight regain** after diet-induced weight loss **do not revert** to the levels recorded before weight loss.

Sumithran et al. NEJM 2011;365:1597-604.

▶ **Table 2** The details of diet-induced weight changes and identified gut hormones among included studies.

Study	Female (%)	Basal BMI (kg/m²)	Weight changes				Gut hormones identified and their changes
			Loss	Maintain	Regain	Change (kg or kg/m²)	
Adam et al. [34]	72	30.3 ± 2.8	#			28.2 ± 2.7	GLP-1 (↓)*
Cahill et al. [35]	69	22.5 ± 2.6			#	2.4 ± 1.3	PYY (↑)*
de Luis et al. [36]	80	Diet I: 35.2 ± 6.6; Diet II: 35.9 ± 7.3	#			Diet I: 33.9 ± 6.6 Diet II: 34.3 ± 6.9	Diet I: GLP-1 (→) Diet II: GLP-1 (↓)*
Essah et al. [25]	83	Diet I: 34.9 ± 0.8 Diet II: 36.0 ± 0.9	#			Diet I: −0.99 ± 0.86 Diet II: −5.8 ± 0.75	Diet I: PYY (↓)* *. PYY_{AUC} (↓)* * * Diet II: PYY (↓)* *. PYY_{AUC} (↓)* * *
Hayes et al. [24]	80	33. 9 ± 1.3	#	#		(BMI) 32.8 ± 1.2	Phase I: ghrelin (→). CCK (↑. postprandial)* Phase II: ghrelin (↑)*. CCK (↑ .postprandial)*
Jensen et al. [16]	55	Diet I: 32.1 ± 4.8 Diet II: 32.6 ± 5.9	#			Diet I: 30.9 ± 4.9 Diet II: 31.6 ± 6.0	Diet I/II: total amylin (↓)*. PP (↓)*. GIP (↓)*. ghrelin (→). PYY (→). GLP-1(→)
Leidy et al. [26]	100	HP: 30.5 ± 0.9 NP: 30.1 ± 0.8	#			HP: -6.9 ± 0.4 NP: −6.9 ± 0.6	HP-AM: ghrelin (↓)*. CCK (↓)* * * NM-AP: ghrelin (→). CCK (→)
Lien et al. [23]	59	32.58 (30.76, 38.19)	#	#	#	−6.31 (−8.46, −3.63)	Phase I: ghrelin (↑)* * *. PYY (↓)* * *. NPY (→) Phase II: ghrelin (↓)* * *. PYY (↓)* * *. NPY(→)
Lobley et al. [31]	0	36.6 ± 5.8	#	#		−4.1	NP Diet: GIP (↓)*. PYY (↓)*. ghrelin (→) HP Diet: GIP (↓)*. PYY (↓)*. ghrelin (↓)* NPAA Diet: GIP (↓)*. PYY (↓)*. ghrelin (↓)*
Moran et al. [29]	100	35.3 ± 1.5	#			−4.2 ± 3.9	CCK (→). PYY (→). fasting ghrelin (↑)*. postprandial ghrelin (↓)*
Neacsu et al. [32]	0	34.8 ± 4.8	#			Diet I: −2.41 ± 0.22 Diet II: −2.27 ± 0.19	Soy-HPWL diet: PYY (↓)* *. ghrelin (↑)* * *. GLP-1 (→) Meat-HPWL diet: PYY (↓)* *. ghrelin (↑)* * *. GLP-1 (→)
O'Connor et al. [28]	29	25 ± 3	#			(BW)EB: 74.2 ± 14 ED: 72.4 ± 13.5	ED diet: fasting PP (↑)*. PYY (↓)*. ghrelin (↓)* *. GLP-1 (→). PP_{AUC} (↑)* * *. $GLP\text{-}1_{AUC}$ (↑)*
Ratliff et al. [27]	0	25–37	#			Diet I: −6.7 Diet II: −5.9	ghrelin (→). PYY (→). PP (→)
Sainsbury et al. [30]	0	34.9 ± 4.9		#		−1.5 ± 0.2	ghrelin (→). PYY (→)
Sloth et al. [33]	52	MUFA: 30.7 ± 0.6; LF: 30.8 ± 0.6; CTR: 32 ± 0.9	#	#		MUFA: 27.5 ± 0.6 LF: 27.3 ± 0.5 CTR: 28.5 ± 0.7	MUFA: GLP-1(↓)*. GLP-2 (↓)*. PP (↓)*. PYY (↓)*. GIP (↓)* LF: GLP-1 (↓)*. GLP-2 (↓)*. PP (↓)*. PYY (↓)*. GIP (↓)*
Sumithran et al. [14]	68	34.7 ± 3.2	#	#		−13.5 ± 0.5	Phase I: PYY (↓)* * *. CCK (↓)* * *. ghrelin (↑)* * * GLP-1 (→). PP (↑)* *. amylin (↓)* *. GIP (↑)* * Phase II: PYY (↓)* *. ghrelin (↓)* * *. CCK (↓)* amylin (↓)*. PP (↓)* *. GIP (↑)* * *. GLP- 1 (↓)* *

HP: High protein diet group; NP: Normal protein group; MUFA: Moderate-fat diet; LF: Low-fat diet; CTR: Control-fat diet; EB: Energy balance; ED: Energy deprivation; HPWL diet: High protein weight-loss diet; PYY: Peptide tyrosine-tyrosine; GLP-1: Glucagon-like peptide-1; GIP: Glucose-dependent insulinotropic polypeptide; PP: Pancreatic polypeptide; CCK: Cholecystokinin; GLP-2: Glucagon-like peptide-2; NPY: Neuropeptide Y: * $p<0.05$; * * $p<0.01$; * * * $p<0.001$. # refers to the existence of weight changes in each study.

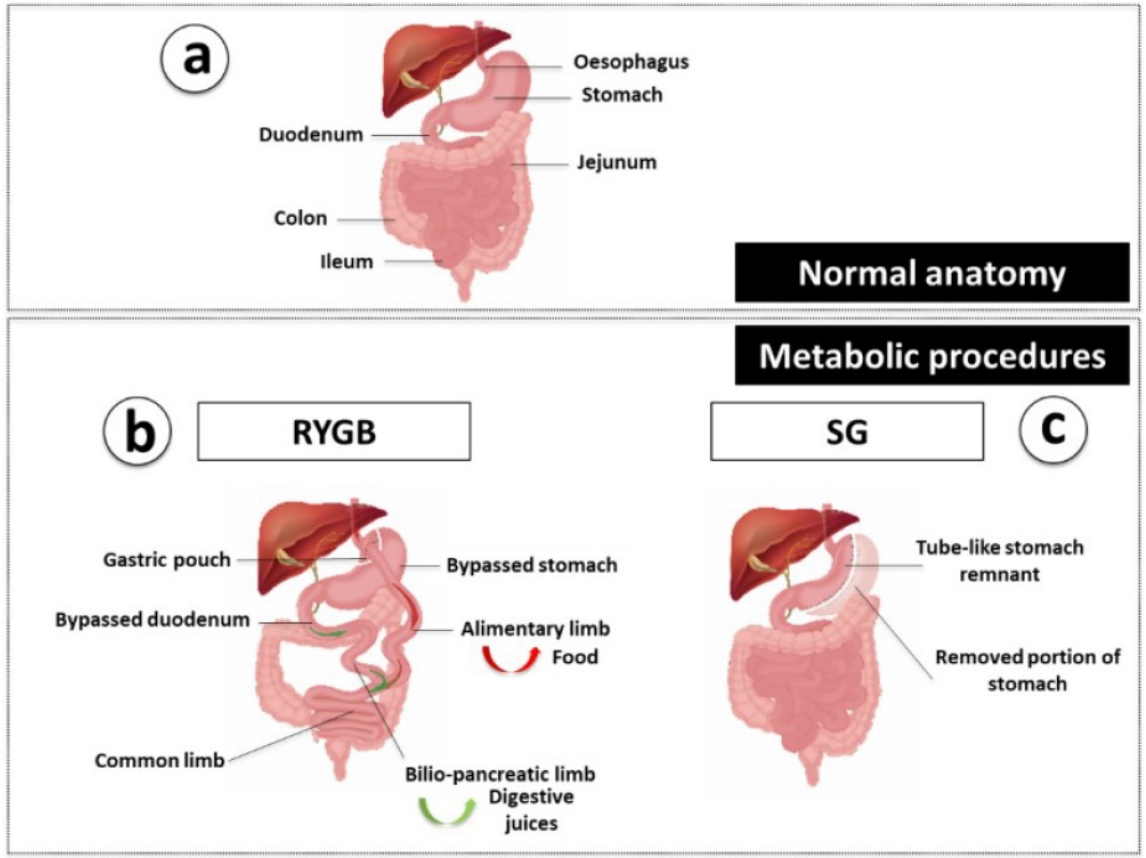
The Role of Gut Hormones in Diet-Induced Weight Change: A Systematic Review

ABSTRACT

Gut hormones are known to play an important role in long-term weight loss maintenance after bariatric surgery. However, the interplay between gut hormones and diet-induced weight changes remains unclear. Our aims were to evaluate the alterations of gut hormones in diet-induced weight loss, weight maintenance, and weight regain periods. Available studies were searched on MEDLINE, EMASE, ClinicalTrials.gov, the Cochrane Library, and Web of science from inception to October 2016. After selection, 16 studies with 656 participants were included. Based on current evidence, we found significant alterations of gut hormones induced by different diets. In weight-loss diets, decreased fasting total PYY, GLP-1, CCK, GIP, PP, and amylin along with increased ghrelin levels were observed in most studies. After weight loss, the persistent decreases of fasting total PYY and GLP-1 levels as well as increased appetite were reported, suggesting the profound impact of altered gut hormones on later weight regain after dietary intervention. The differences between diet-induced changes in gut hormones and other treatments such as bariatric surgery and exercise are also discussed in this review. Although significant alterations of gut hormones were found during weight changes, huge heterogeneity exists in methods and populations. More large-scale studies with elaborate design addressing the gut hormone alterations in dietary weight regulation are required in the future.

Zhao X et al. Horm Metab Res 2017; 49: 816–825

Changes in gut hormones following bariatric surgery



- Long term durability of weight loss
- Metabolic benefits
- Increased satiety
- Reduced appetite
- Changes in taste and food preference

→ Not only from restricted stomach size or malabsorption

Pucci, A., et al. (2020). Endotext [Internet].

Changes in gut hormones following bariatric surgery

Gut hormone	Roux-en-y gastric bypass	Sleeve gastrectomy
Ghrelin	Fasting ↓ or ↔ or ↑ Postprandial ↓ or ↔ or ↑	Fasting ↓ ↓ Postprandial ↓
GLP-1	Fasting ↔ Postprandial ↑ ↑	Fasting ↔ Postprandial ↑
GIP	Fasting ↔ Postprandial ↓ or ↔ or ↑	Fasting ↔ Postprandial ↑
PYY	Fasting ↔ or ↑ Postprandial ↑ ↑	Fasting ↔ Postprandial ↑
OXM	Fasting ↔ Postprandial ↑ ↑	Fasting ↔ Postprandial ↔ or ↑

"Hindgut hypothesis"

Intestinal rearrangement → Large nutrient loads to distal gut → Exposure to EEC (L cell) → GLP-1, PYY ↑

Papamargaritis, D et al (2021). Nutrients, 13(3), 762.

Summary

- 장호르몬은 중추신경계와 말초조직에 작용하여 식이행동과 에너지항상성을 조절하는 중요한 요소이다.

- 비만에서는 장호르몬의 농도와 식이에 대한 반응이 변화되어 있다.

: 혈중 장호르몬의 변화가 비만의 발생에 기여하며,
고열량식이로 유도된 비만은 장호르몬의 분비와 장호르몬에 대한 반응성에 영향을 주기도 한다.

- 체중감소 중재방법에 따른 식이행동 및 체중변화 양상의 차이는 장호르몬과 밀접한 연관이 있다.

Thank you for your attention