GLP-1 AGONISTS AND THE PSYCHOLOGY OF EATING: IMPLICATIONS FOR EATING DISORDER RECOVERY

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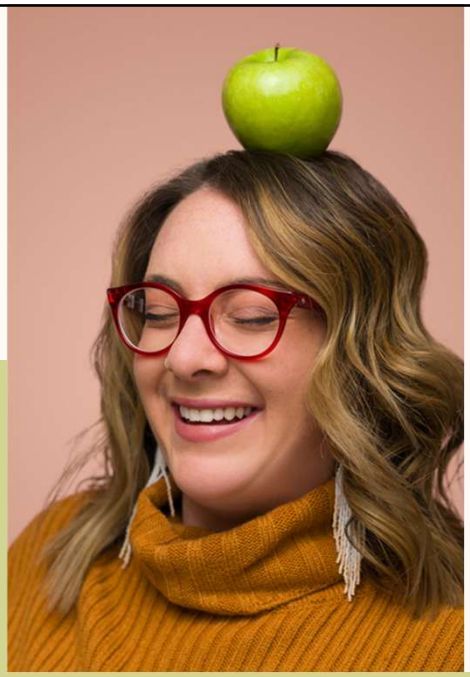

NICE TO MEET YOU

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OBJECTIVES

- Understand how GLP-1 medication functions in appetite regulation, insulin secretion, and gastric emptying
- Define key psychological drivers of eating behavior
- Differentiate between homeostatic and hedonic eating
- Potential impacts that GLP-1s have in recovery spaces

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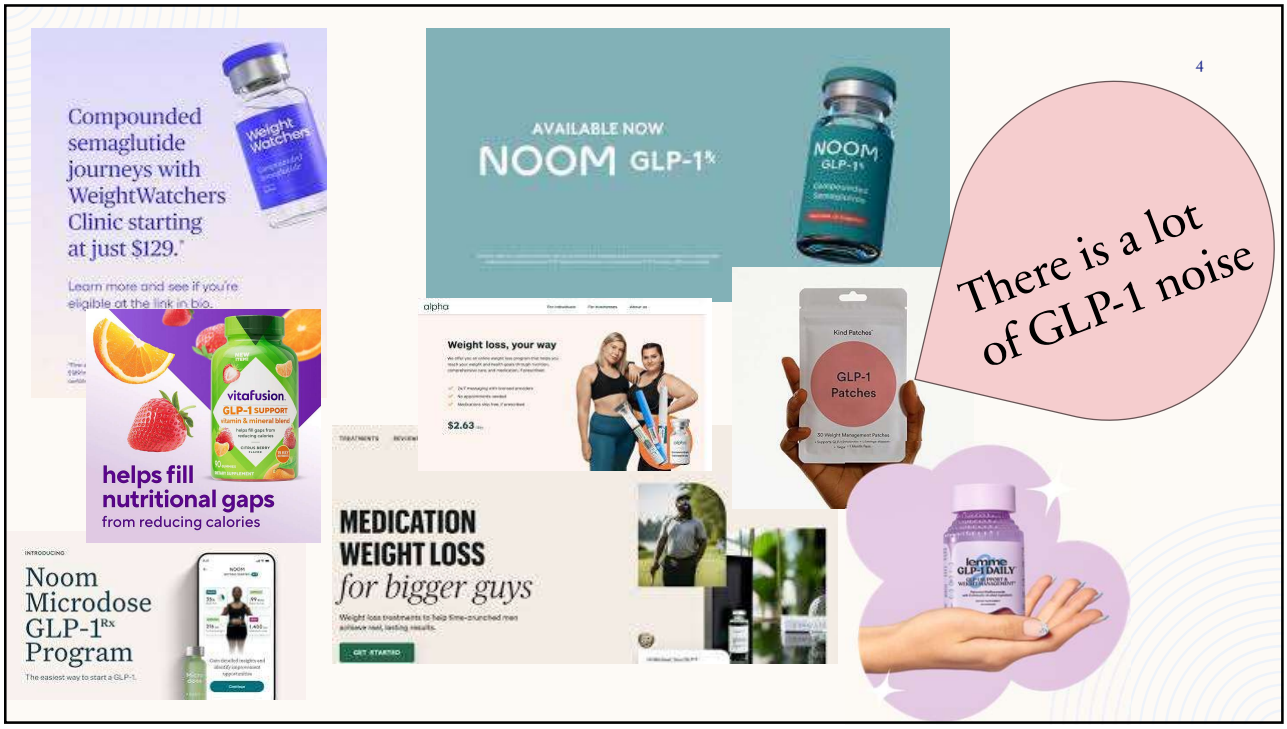

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GLP-1 AGONIST STATS

- US spending on GLP-1s increased 500% between 2018-2023
  - Ie. Ozempic spending went from $0.4billion to $26.4 billion
- Discontinuation of GLP-1s higher in those with "obesity" than those with DM2
- Older patients less likely to discontinue
- 1 in 8 adults (12%) have taken a GLP-1
- 1 in 5 who have been told they are overweight (22%)
- 4 in 10 took for weight loss

Table 1. Prevalence of GLP-1 Receptor Agonist Discontinuation and PDC Among New GLP-1 Receptor Agonist Users by Baseline Status[a]

Outcome	No. (%)			
		T2D and obesity status at baseline		
	Overall (N = 195 915)	T2D only (n = 87 611)	Obesity only (n = 20 217)	T2D and obesity (n = 88 087)
At 3-mo assessment				
Discontinued GLP-1 agonist[b]				
No	144 598 (73.8)	64 613 (73.8)	12 978 (64.2)	67 007 (76.1)
Yes	51 317 (26.2)	22 998 (26.3)	7239 (35.8)	21 080 (23.9)
PDC, mean (SD), %[c]	81.1 (24.5)	81.2 (24.6)	76.5 (25.9)	82.0 (23.9)
At 6-mo assessment				
Discontinued GLP-1 agonist[b]				
No	135 552 (69.2)	61 015 (69.6)	11 167 (55.2)	63 370 (71.9)
Yes	60 363 (30.8)	26 596 (30.4)	9050 (44.8)	24 717 (28.1)
PDC, mean (SD), %[c]	72.3 (29.0)	72.5 (29.2)	65.6 (30.2)	73.7 (28.3)
At 12-mo assessment				
Discontinued GLP-1 agonist[b]				
No	124 331 (63.5)	56 272 (64.2)	10 055 (49.7)	58 004 (65.9)
Yes	71 584 (36.5)	31 339 (35.8)	10 162 (50.3)	30 083 (34.2)
PDC, mean (SD), %[c]	64.3 (31.9)	64.9 (32.1)	54.4 (32.3)	66.1 (31.1)

Abbreviations: GLP-1; glucagon-like peptide 1; PDC, proportion of days covered; T2D, type 2 diabetes.

[a] At 3, 6, and 12 months, the prevalence of GLP-1 agonist discontinuation and the PDC were significantly different across groups (for both, $P < .001$, Pearson χ^2 test).

[b] Discontinuation was defined as no GLP-1 agonist fill in the 135 days following the 3-, 6-, and 12-month post-index date assessment.

[c] Allowing at most 10 days between prescriptions.

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HELPFUL DEFINITIONS

- **Insulin:** Hormone made by the cells of the pancreas to regulate blood sugar levels, takes glucose into cells
- **Glucagon:** Hormone made by the cells of the pancreas that breakdown stored CHO (glycogen) in the liver to raise blood glucose
- **Receptor:** Region of tissues that responds to a specific hormone or other substances
- **Agonist:** Substance which initiates a physiological response when combined with a receptor
- **Palatable food**: Hedonic value of food, observation that some food stimulate more intake than others, ie. Food high in CHO or fat

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TERMS

Hunger: Painful sensation caused by a lack of food that initiates food seeking behavior

Appetite: The psychological need to eat that can occur in the response to sight, smell, thought, or taste of food that initiates eating

Craving: Intense and irresistible desire to consume specific foods. Desire, specificity, and reward-seeking

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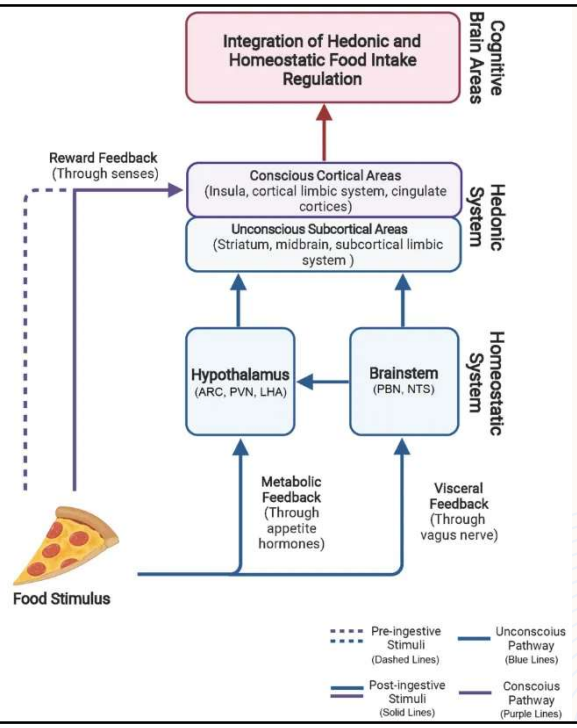
HOMEOSTATIC VS HEDONIC EATING

Homeostatic: Driven by physiological need to consume energy for survival.

Hedonic: Driven by the desire for pleasure, consuming foods for sensory qualities (taste, smell, etc) can occur outside of physiological hunger, develop reward-based regulation



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Supplemental Table 2. Exploratory factor analysis of Power of Food Scale (2-factor solution, oblique rotation)

	Item	Factor 1	Factor 2
1	I find myself thinking about food even when I am not physically hungry	.66	
2	I get more pleasure from eating than I do from almost anything else	.67	
3	If I see or smell a food I like, I get a powerful urge to have some	.61	
4	When I'm around fattening food I love, it's hard to stop myself from at least tasting it	.72	
5	It's scary to think of the power that food has over me	.76	
6	When I know a delicious food is available, I can't help myself from thinking about having some	.63	
7	I love the taste of certain foods so much that I can't avoid eating them even if they're bad for me	.60	
8	Just before I taste a favorite food, I feel intense anticipation	.36	.44
9	When I eat delicious food I focus a lot on how good it tastes		.70
10	Sometimes, when I'm doing everyday activities, I get an urge to eat 'out of the blue' (for no apparent reason)	.56	
11	I think I enjoy eating, a lot more than most other people	.47	.34
12	Hearing someone describe a great meal makes me really want to have something to eat		.54
13	It seems like I have food on my mind a lot	.58	
14	It's very important to me that the foods I eat are as delicious as possible		.70
15	Before I eat a favorite food my mouth tends to flood with saliva		.62

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WHAT IMPACTS HOW WE EAT

- Environment
- Emotions
- Physical activity
- Traditions/culture
- Disease/illness
- Body image
- Social norms/learned behaviors
- Energy needs
- Availability
- Socioeconomic status
- Medications
- Habits

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REGULATION OF EATING AND GLP-1S

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INTRODUCING THE HYPOTHALAMUS

- Regulates hormones
- Controls autonomic nervous system (CNS), wants things stable!
- Balances bodily functions
  - Hunger/satiety, heart rate, mood, sleep, etc.

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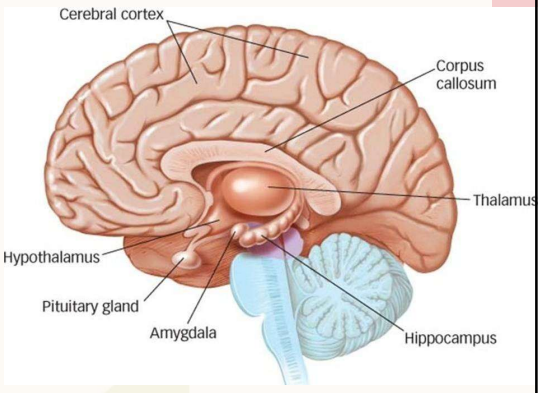
HYPOTHALAMUS REGULATION

- Neural and nutrient inputs:
  - Vagus nerve: Detects distension
  - Sense nutrient availability
  - Regulates hunger/satiety through hormones
- Controls reward pathways to eating
  - Ie. Dopamine



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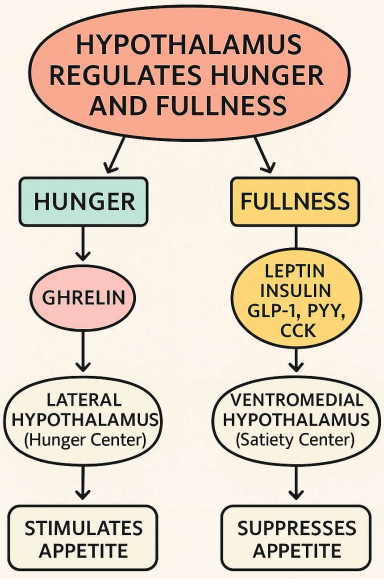

Lateral Hypothalamus (LH) – The Hunger Center
Function: Stimulates appetite
Activation: Triggered by low blood sugar, ghrelin, and other hunger signals
Effect: Promotes eating behavior

Ventromedial Hypothalamus (VMH) – The Satiety Center
Function: Suppresses appetite
Activation: Triggered by rising blood sugar, leptin, insulin, and gut hormones
Effect: Signals fullness and stops eating

Arcuate Nucleus (ARC) – Signal Integrator
Function: Receives hormonal signals and regulates LH and VMH Neurons:
- Orexigenic neurons (NPY/AgRP): Stimulate hunger
- Anorexigenic neurons (POMC/CART): Promote satiety

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HORMONE	SOURCE	ROLE
Ghrelin	Stomach	Stimulates hunger via LH and NPY/AgRP neurons
Leptin	Fat cells	Promotes satiety via VMH and POMC/CART neurons
Insulin	Pancreas	Reduces appetite and regulates energy balance
GLP-1	Intestines	Slows digestion and promotes satiety
PYY	Small intestine	Inhibits hunger neurons and enhances fullness
CCK	Small intestine	Stimulates satiety centers and reduces meal size

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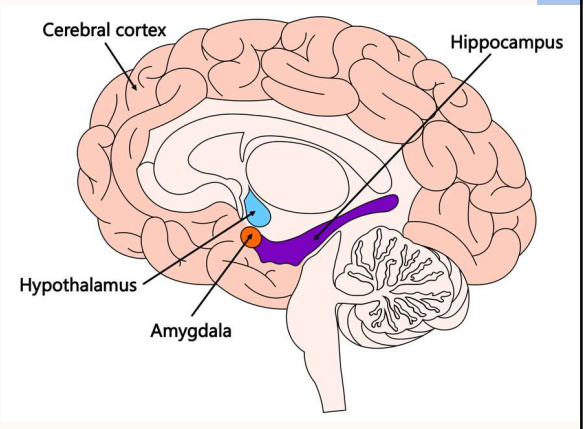
CONTINUED…

- **Amygdala:**
  - Can override hunger cues:
    - Learned environmental cues which can trigger eating behavior, amplify cravings
    - Suppress normal satiety cues
    - During stress: adrenaline and cortisol, override signals from the hypothalamus
  - Disrupt our ability to listen to physical fullness
  - Contributes to reward systems and emotional processing of eating
  - GLP-1 decreases Amygdala activity
    - Decreases dopamine response



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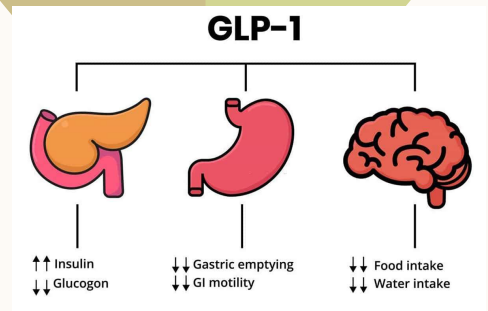
ENDOGENOUS GLP-1

- Naturally occurring hormone in the body
- Produced in the small intestine
- Activated in response to ingestion of nutrients
  - Rapid response with CHO ingestion
  - Main job to maintain glucose homeostasis
- Inhibits glucagon secretion, promotes insulin secretion



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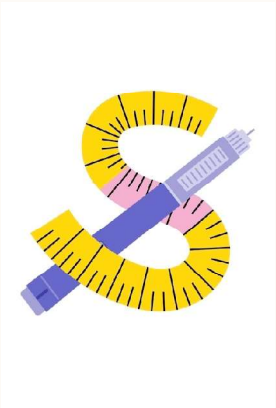
GLP-1 AGONISTS



- Mesolimbic system effects:
  - Modulate the reward we receive from food, decreasing hedonic eating
- Decrease anticipation from "palatable" food
- Decreased dopamine receptor gene expression
- Increased energy expenditure: brown adipose tissue thermos regulation
- **Insula:** increased activity when fasting decreased activity when in response to palatable food
  - Ie: palatable food more palatable when hungry
  - Makes body more aware of lack of energy
  - GLP1s decrease this response

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- Decreased gastric and intestinal motility
- Decreases production of ghrelin, both circulating and pre-meal rise
- To sum up: Less food craving: dopamine, less hedonic eating: reduced drive/pleasure, increased cognitive control: improved impulse control via prefrontal cortex, decreased emotional eating: Less amygdala driven reactivity to food

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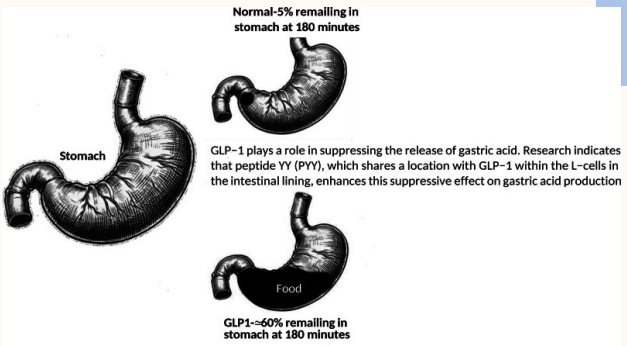
GLP-1 AGONISTS AND THE GUT

- Delayed gastric emptying
  - Stomach volume impacts on satiety
  - Slows down gastric emptying in hypoglycemia
- Can impact absorption of some medications
- Increases nausea/potential vomiting/constipation
- Going to increase postprandial fullness



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PATIENT EXPERIENCES

"I felt normal for the first time in my life"

"I didn't have to think about food anymore, there was no more food noise"

"I just lost appetite. I don't even feel hungry in the whole day. Sometimes I forget about eating." (P3, female, obesity). "

My dinner size has gone down by half." (Pierret et al., 2025

"I've lived with binge-eating disorder for like 25 years…Literally overnight, when I started taking it, it was like a switch had been turned off in my brain and all my thoughts about food vanished…So the binge eating has gone completely. I've not had a single binge episode in all that time." (Pierret et al., 2025).

"It felt nice not to be hungry"

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TAKING CARE OF OUR PATIENTS

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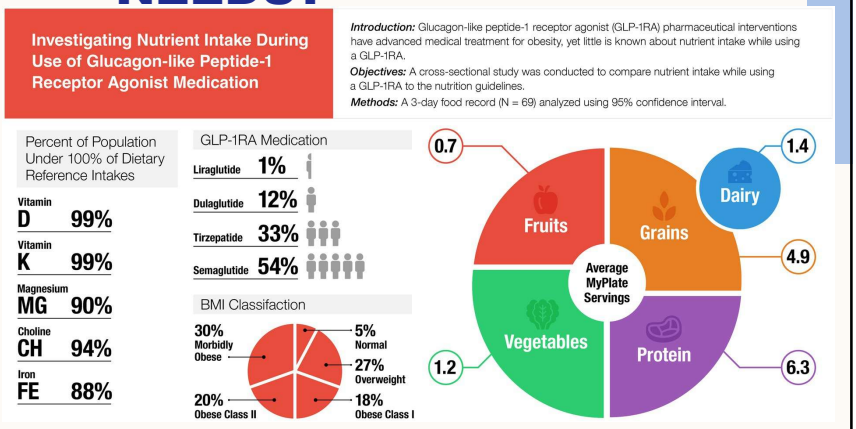
WHAT ARE WE USING TO DETERMINE NEEDS?

- American Diabetes Association recommendations a diet of 800-1,000kcal/d
- 20-50% of more lean muscle mass loss, more than traditional efforts to lose weigh



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CONCERNS

- Overemphasis on weight loss
- Unsupervised use
  - No medical oversight, no labs, no nutritional counseling, no face to face
- Decreased appetite, trading one E.D. for another?
- Symptom decrease only with medication use
- Limited E.D. screening
- Reaffirming weight stigma
- Increased risk of weight cycling
- Social implications from weight cycling

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CONTRADICTIONS TO ED TREATMENT

- Delayed gastric emptying: Impact consistently of nutritional needs required in E.D. treatment
- Reduced interest in eating
  - Nausea
  - Less Hedonic eating
- Potential significant weight loss
- Impacts on body image
- Reinforce negative beauty ideals
- Role of interoceptive awareness in recovery

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WHAT HAPPENS AFTER?

- two-thirds of the weight lost on treatment was regained after discontinuation of semaglutide, with worsening of cardiometabolic parameters such as glucose levels, blood pressure, and cholesterol levels.
- Weight regain common despite lifestyle interventions
- Helping our clients have an "exit" strategy
- Discussing outcomes from weight changes
  - Positive affirmations w/ weight loss, shame around weight gain

Where does this leave our clients?

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CHALLENGES WITH THE RESEARCH

- Looking mainly at anthropometric measurements to measure "success"
- Short term
- Looking at cessation of bingeing behaviors and weight loss, not identifying increased restrictive thoughts
- Adequate nutrition, are we looking at restriction in binge eating studies?

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Nov. 2022 | Version 1.4

I. Introduction

This resource was created by Medical Students for Size Inclusivity (MSSI), an international community of medical students dedicated to addressing weight bias in medicine. We have seen the huge increase in GLP-1 agonist prescriptions specifically aimed at promoting weight loss, and are concerned that patients may receive misleading or insufficient information from their healthcare providers before being started on them.

While MSSI does not believe weight & BMI are accurate measures of health, or that weight loss improves health outcomes, we also champion patient autonomy. Weight discrimination permeates so many aspects of society, and the physical and mental harm it directly causes fat people is immeasurable and far-reaching. Our goal is to give patients desiring to start medications to lose weight a more comprehensive understanding of the risks and benefits associated. Knowing what alternatives are available is part of full informed consent, so we also include evidence-based options for improving health that do not require weight loss.

II. An Overview of GLP-1 Agonists

Glucagon-like peptide 1 (GLP-1) receptor agonists are medications that cause a release of insulin from the pancreas, delaying stomach emptying and causing early satiety ("feeling full"), which reduces blood sugar and appetite. The medications are originally marketed for glycemic (blood sugar) control in adults with type 2 diabetes mellitus, in tandem with diet and exercise.

GLP-1 agonists have a side effect of weight loss, and recently made headlines as "miracle, game-changing, weight-loss drugs," increasing their desirability and overall use. As an unintended consequence, there has been decreased medication availability for those living with diabetes.

List of GLP-1 Agonists:

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1	Do I have health condition that should be addressed more directly rather than with a trial of weight loss?
2	Do I feel short term weight loss is worth the cost and potential side effects?
3	Do I have a history of disordered eating that might be triggered by beginning this medication, or by a loss of appetite?
4	If I were to set weight aside, what would make the biggest difference in my health? Am I pursuing that as well?
5	Do I need help caring for myself in the body I have now? What professionals or community could I access for help?

Medical Students for Size Inclusivity. (2023, Nov.). GLP-1 Agonist Medications: Informed Consent Resource. https://sizeinclusivemedicine.org/glp1/

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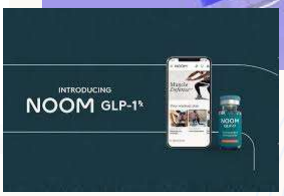
WE ARE UP AGAINST IT



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Thank You!

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THANK YOU

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REFERENCES

- Chiappini, S., Vickers-Smith, R., Harris, D., Papanti Pelletier, G. D., Corkery, J. M., Guirguis, A., Martinotti, G., Sensi, S. L., & Schifano, F. (2023). Is There a Risk for Semaglutide Misuse? Focus on the Food and Drug Administration's FDA Adverse Events Reporting System (FAERS) Pharmacovigilance Dataset. Pharmaceuticals (Basel, Switzerland), 16(7), 994. https://doi.org/10.3390/ph16070994
- Dailey, M. J., & Moran, T. H. (2013). Glucagon-like peptide 1 and appetite. *Trends in endocrinology and metabolism: TEM*, *24*(2), 85–91. https://doi.org/10.1016/j.tem.2012.11.008
- Do D, Lee T, Peasah SK, Good CB, Inneh A, Patel U. GLP-1 Receptor Agonist Discontinuation Among Patients With Obesity and/or Type 2 Diabetes. *JAMA Netw Open*. 2024;7(5):e2413172. doi:10.1001/jamanetworkopen.2024.13172
- Eren-Yazicioglu CY et al (2020) Can GLP-1 Be a target for reward system related disorders? A qualitative synthesis and systematic review analysis of studies on palatable food, drugs of abuse, and alcohol. Front Behav Neurosci 14:614884
- Frewer, L., & Van Trijp, H. (2007). Liking wanting and eating: Drivers of food intake in obesity.*Understanding consumers of food products*. (pp.10-17) Woodhead Publishing.
- Holst JJ. The physiology of glucagon-like peptide 1. ***Physiol Rev*** **87**: 1409–1439, 2007.
- Ida S, Kaneko R, Imataka K, Okubo K, Shirakura Y, Azuma K, Fujiwara R, Murata K. Effects of Antidiabetic Drugs on Muscle Mass in Type 2 Diabetes Mellitus. Curr Diabetes Rev. 2021;17(3):293-303. doi: 10.2174/1573399816666200705210006. PMID: 32628589.
- Kałas, M., Stępniewska, E., Gniedziejko, M., Leszczyński-Czeczatka, J., & Siemiński, M. (2025). Glucagon-like Peptide-1 Receptor Agonists in the Context of Eating Disorders: A Promising Therapeutic Option or a Double-Edged Sword?. Journal of clinical medicine, 14(9), 3122. https://doi.org/10.3390/jcm14093122
- Khan SS, Ndumele CE, Kazi DS. Discontinuation of Glucagon-Like Peptide-1 Receptor Agonists. JAMA. 2025 Jan 14;333(2):113-114. doi: 10.1001/jama.2024.22284. PMID: 39535741.
- Kffmarleyp. (2025, August 12). *KFF Health Tracking Poll May 2024: The public's use and views of GLP-1 Drugs*. KFF. https://www.kff.org/health-costs/kff-health-tracking-poll-may-2024-the-publics-use-and-views-of-glp-1-drugs/
- Medical Students for Size Inclusivity. (2023, Nov.). *GLP-1 Agonist Medications: Informed Consent Resource*. https://sizeinclusivemedicine.org/glp1/
- Radkhah, H., Rahimipour Anaraki, S., Parhizkar Roudsari, P. *et al.* The impact of glucagon-like peptide-1 (GLP-1) agonists in the treatment of eating disorders: a systematic review and meta-analysis. *Eat Weight Disord* **30**, 10 (2025). https://doi.org/10.1007/s40519-025-01720-9

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- Richards, J. R., & Khalsa, S. S. (2024). Highway to the danger zone? A cautionary account that GLP-1 receptor agonists may be too effective for unmonitored weight loss. Obesity reviews : an official journal of the International Association for the Study of Obesity, 25(5), e13709. https://doi.org/10.1111/obr.13709
- Pierret, A.C.S., Benton, M., Sen Gupta, P., & Ismail, K. (2025). A qualitative study of the mental health outcomes in people being treated for obesity and type 2 diabetes with glucagon-like peptide-1 receptor agonists. Acta Diabetologica, 62, 731–742. https://doi.org/10.1007/s00592-024-02392-0
- Shankar, A., Sharma, A., Vinas, A., & Chilton, R. J. (2024). GLP-1 receptor agonists and delayed gastric emptying: implications for invasive cardiac interventions and surgery. *Cardiovascular endocrinology & metabolism*, *14*(1), e00321. https://doi.org/10.1097/XCE.0000000000000321
- Tsipas S, Khan T, Loustalot F, Myftari K, Wozniak G. Spending on Glucagon-Like Peptide-1 Receptor Agonists Among US Adults. *JAMA Netw Open.* 2025;8(4):e252964. doi:10.1001/jamanetworkopen.2025.2964
- Wilding JPH, Batterham RL, Davies M, Van Gaal LF, Kandler K, Konakli K, Lingvay I, McGowan BM, Oral TK, Rosenstock J, Wadden TA, Wharton S, Yokote K, Kushner RF; STEP 1 Study Group. Weight regain and cardiometabolic effects after withdrawal of semaglutide: The STEP 1 trial extension. Diabetes Obes Metab. 2022 Aug;24(8):1553-1564. doi: 10.1111/dom.14725. Epub 2022 May 19. PMID: 35441470; PMCID: PMC9542252.