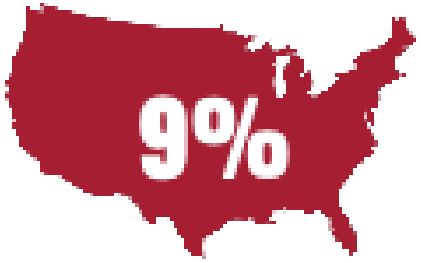
Session 1A: Ingestive Eating Disorders

Moderator: Dr. Kimberlei Richardson

Howard University College of Medicine

No disclosures or conflicts of interest

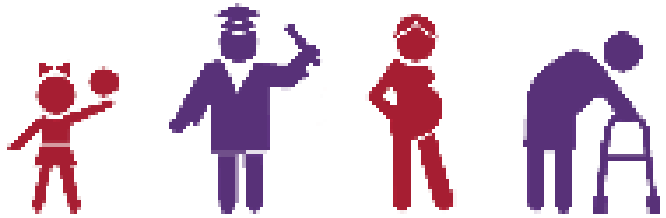
Eating Disorders: Prevalence and Mortality



Percent of the U.S. population, or **28.8 million Americans**, that will **have an eating disorder in their lifetime**

10,200 deaths per year as a direct result of an eating disorder, equating to **1 death every 52 minutes**

EATING DISORDERS AFFECT EVERYONE:



- **All ages**, starting as young as 5 years old to over 80 years old
- **All races**, however, people of color with eating disorders are **half as likely to be diagnosed or to receive treatment**[1]
- **All genders**, with females being **2x more likely to have an eating disorder**
- **All sexual orientations**

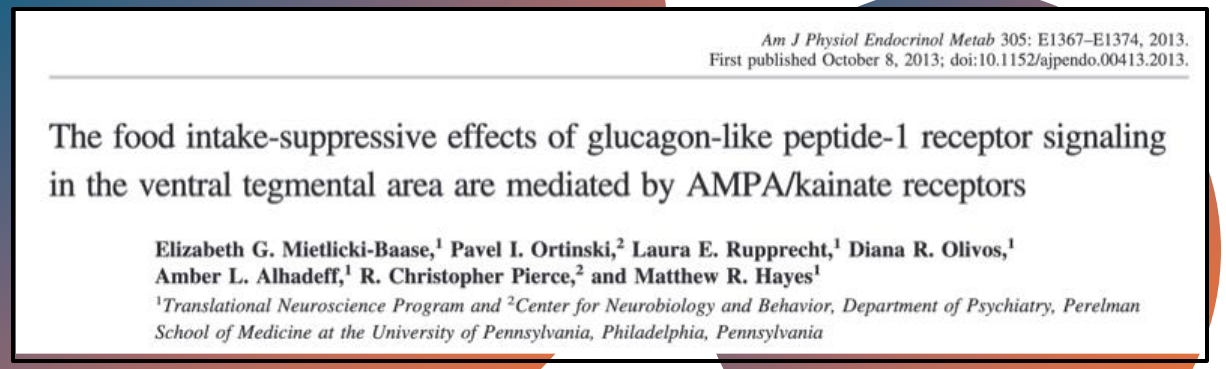
Source: Strategic Training Initiative for the Prevention of Eating Disorders, Academy for Eating Disorders, and Deloitte Access Economics, 2020

Characterization of Eating Disorders: Diagnostic and Statistical Manual (DSM) of Mental Disorders-5th edition

- Anorexia Nervosa
- Bulimia Nervosa (BN)*
- Binge Eating Disorder (BED)*

- Binge eating is a core symptom of BN and BED.

- BED has the highest prevalence of comorbid obesity, followed by BN (Villarejo et al 2012; Udo et al 2018; Aguera et al 2021).

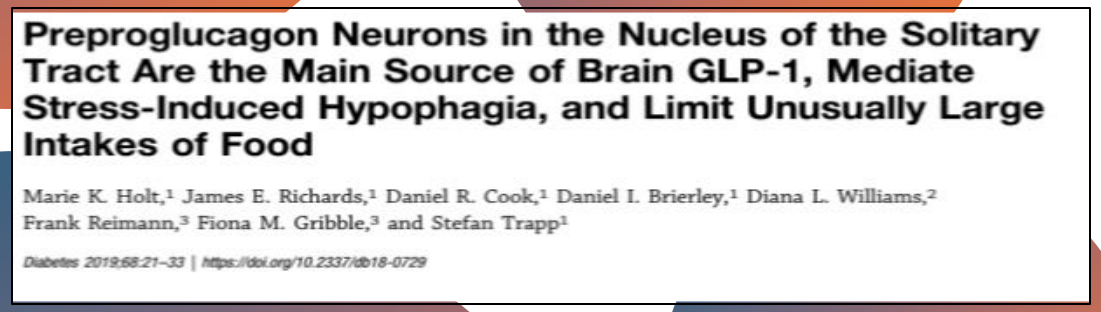


Am J Physiol Endocrinol Metab 305: E1367–E1374, 2013.
First published October 8, 2013; doi:10.1152/ajpendo.00413.2013.

The food intake-suppressive effects of glucagon-like peptide-1 receptor signaling in the ventral tegmental area are mediated by AMPA/kainate receptors

Elizabeth G. Mietlicki-Baase,[1] Pavel I. Ortinski,[2] Laura E. Rupprecht,[1] Diana R. Olivos,[1] Amber L. Alhadeff,[1] R. Christopher Pierce,[2] and Matthew R. Hayes[1]

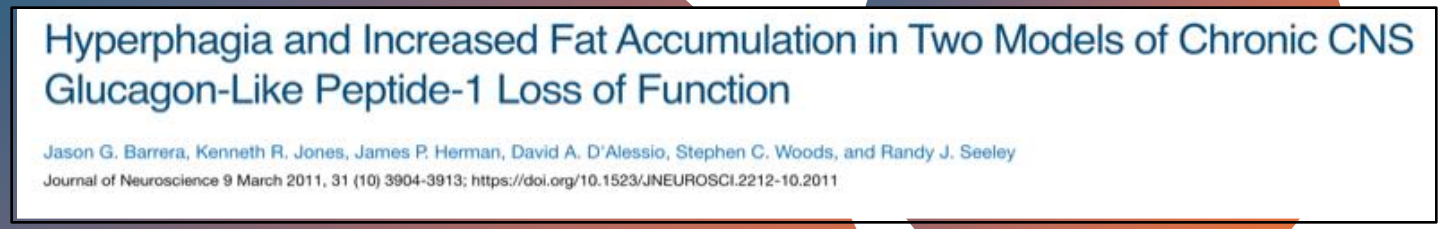
[1]*Translational Neuroscience Program and* [2]*Center for Neurobiology and Behavior, Department of Psychiatry, Perelman School of Medicine at the University of Pennsylvania, Philadelphia, Pennsylvania*



Preproglucagon Neurons in the Nucleus of the Solitary Tract Are the Main Source of Brain GLP-1, Mediate Stress-Induced Hypophagia, and Limit Unusually Large Intakes of Food

Marie K. Holt,[1] James E. Richards,[1] Daniel R. Cook,[1] Daniel I. Brierley,[1] Diana L. Williams,[2] Frank Reimann,[3] Fiona M. Gribble,[3] and Stefan Trapp[1]

Diabetes 2019;68:21–33 | https://doi.org/10.2337/db18-0729



Hyperphagia and Increased Fat Accumulation in Two Models of Chronic CNS Glucagon-Like Peptide-1 Loss of Function

Jason G. Barrera, Kenneth R. Jones, James P. Herman, David A. D'Alessio, Stephen C. Woods, and Randy J. Seeley

Journal of Neuroscience 9 March 2011, 31 (10) 3904-3913; https://doi.org/10.1523/JNEUROSCI.2212-10.2011

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Physiology & Behavior
Volume 171, 15 March 2017, Pages 158-164

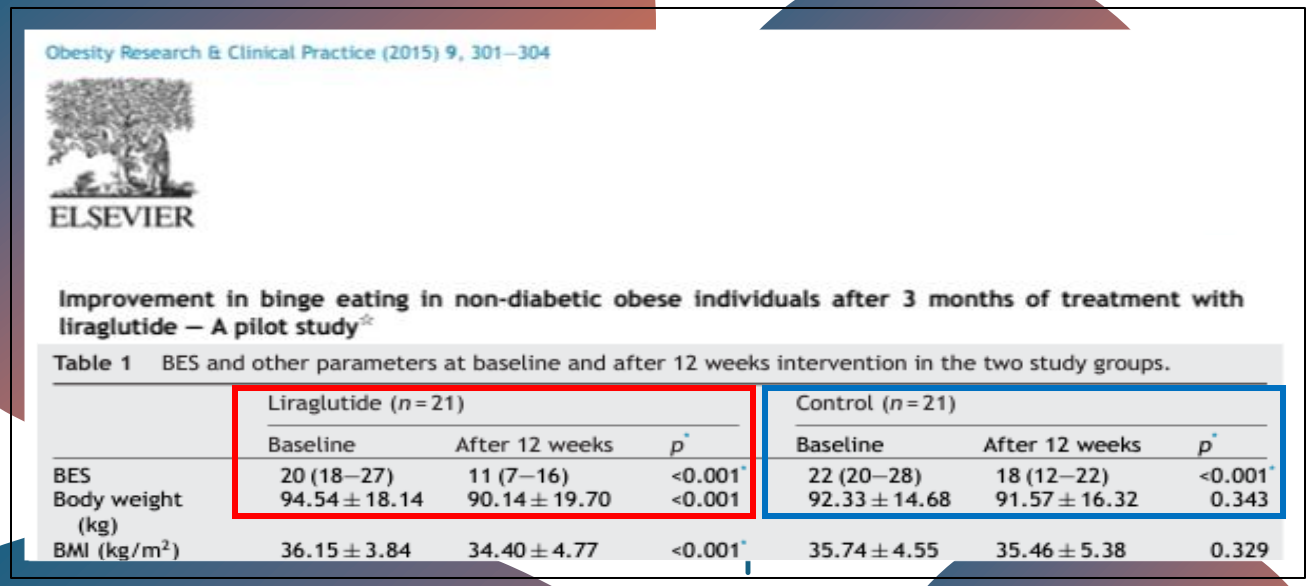
Systemic administration of anorexic gut peptide hormones impairs hedonic-driven sucrose consumption in mice

Erina Yamaguchi, Yasunobu Yasoshima, Tsuyoshi Shimura

Pre-Clinical Studies: Effect of GLP-1 Receptor Agonists on Feeding and Binge Eating Behaviors

- Preclinical evidence showing GLP-1 receptor signaling in the VTA controls food intake via AMPA/kainate receptors on dopamine neurons (Mietlicki-Baase et al 2013).
- Preclinical model demonstrating GLP-1 of central origin is relevant for aspects of feeding behavior (Holt et al, 2019).
- Chronic central GLP-1 loss of function caused hyperphagia along with weight gain and glucose tolerance (Berrera et al 2011).
- Systematic administration of GLP-1 reduced hedonically-mediated sugar consumption in a mouse model of binge -like sucrose overconsumption (Yamaguchi et al 2017).

Clinical Studies: Effect of GLP-1 Receptor Agonists on Binge Eating Behavior



Obesity Research & Clinical Practice (2015) 9, 301–304

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Improvement in binge eating in non-diabetic obese individuals after 3 months of treatment with liraglutide — A pilot study☆

Table 1 BES and other parameters at baseline and after 12 weeks intervention in the two study groups.

	Liraglutide ($n=21$)			Control ($n=21$)		
	Baseline	After 12 weeks	p^*	Baseline	After 12 weeks	p^*
BES	20 (18–27)	11 (7–16)	<0.001*	22 (20–28)	18 (12–22)	<0.001*
Body weight (kg)	94.54 ± 18.14	90.14 ± 19.70	<0.001	92.33 ± 14.68	91.57 ± 16.32	0.343
BMI (kg/m²)	36.15 ± 3.84	34.40 ± 4.77	<0.001*	35.74 ± 4.55	35.46 ± 5.38	0.329

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GLP-1 receptor agonists: A novel pharmacotherapy for binge eating (Binge eating disorder and bulimia nervosa)? A systematic review

Laurence Aoun[a,*], Shaza Almardini[a], Fares Saliba[a], Fadi Haddadin[a], Omar Mourad[a], Jennifer Jdaidani[a], Zeina Morcos[a], Ibrahim Al Saidi[a], Elie Bou Sanayeh[a], Saliba Saliba[b], Michel Almardini[c], Julie Zaidan[d]

- Individuals treated with Liraglutide demonstrated reduced binge eating behavior and lost more weight than those not treated with the drug (Robert et al 2015).

- Promising effects of GLP-1 receptor agonists on binge eating behavior; however more rigorous clinical trials are needed (Aoun et al 2024).

Session Objectives

- To review current knowledge regarding the mechanism of action of GLP-1 receptor agonists and their therapeutic applications in ingestive eating disorders.
- To discuss available scientific evidence on the clinical efficacy of GLP-1 receptor agonists for treating eating disorders.
- To discuss clinical consequences and adverse effects related to the use of GLP-1 receptor agonists.
- To identify unique gaps/challenges in the field and provide suggestions for future research.

Panelists for Session-1a

- Ms. Patricia Nece
- Dr. Jon Davis
- Dr. Elizabeth Mietlicki-Baase
- Dr. Susan McElroy