AMGA Foundation

Chronic Care Roundtable

Tackling the Obesity Epidemic

Strategies for addressing Diabetes, Cardiovascular and Kidney Disease Comorbidities

November 8, 2023

Thank You Chronic Care Roundtable Partners

CCR Theme

Today's meeting will be focused on preparing medical groups and health systems to ensure equitable access to the next generation of obesity therapies and address obesity comorbidities of diabetes, cardiovascular disease, and kidney disease.


OBESITY HEADLINES IN THE NEWS
CDC Centers for Disease Control and Prevention
CDC 24/7: Saving Lives, Protecting People™
CDC Newsroom
How Does Stigma Affect Patients with Overweight, Obesity, or Diabetes?
Newsroom Home
CDC Newsroom Releases
Historical News Releases
Adult Obesity Prevalence Remains High; Support for Prevention and
Adult Obesity Prevalence Remains High; Support for Prevention and Treatment Needed
Print
Press Release
HOME
HEALTH NEWS
Weight-loss drug can reverse heart failure symptoms, study finds
Turning the Tide on Obesity: Are We on the Brink of a Medical Miracle?
NEWS / HEALTH
ONLY IN NEWSDAY
Diabetes patients face supply problems from weight-loss drugs' popularity
CNN health
Life, But Better
Fitness
Food
Sleep
Mindfulness
Relationships
Obesity is becoming more common in a growing number of states, CDC data show
New heart syndrome identifies link among obesity, diabetes and kidney disease
For the first time, the American Heart Association has defined cardiovascular-kidney-metabolic syndrome, or CKM.

Start Time	End Time	Agenda Item
9:30 am	10:00 am	**Welcome & Introductions** John W. Kennedy, MD & Christopher M. Steer , Esq.
10:00 am	10:45 am	**Obesity Keynote** Christopher Still, DO, FACN, FACP, FTOS
10:45 am	11:00 am	**Keynote Q&A**
11:00 am	11:15 am	**Roundtable Discussions**
11:15 am	11:30 pm	**Morning Break**
11:30 am	12:00 pm	**Speaker: Patient Advocate** Patricia Nece, JD
12:00 pm	1:00 pm	**Networking lunch**

Start Time	End Time	Agenda Item
1:00 pm	2:15 pm	**Breakout Sessions** Kidney Disease: Sandra J. Taler, MD Diabetes: Brian C. Jameson, DO Cardiovascular Disease: John Clark, MD, PhD
2:15 pm	2:30 pm	**Afternoon Break**
2:30 pm	3:30 pm	**Panel Discussion & Q&A**
3:30 pm	4:00 pm	**Roundtable Discussions**
4:00 pm	4:15 pm	**AC24 Preview & Upcoming Materials**
4:15 pm	4:30 pm	**Closing Remarks**
4:30 pm	6:00 pm	**Break**
6:00 pm	6:30 pm	**Cocktail Reception**
6:30 pm	8:30 pm	**Dinner**

Keynote Speaker

Christopher Still, DO, FACN, FACP, FTOS

Director, Geisinger Obesity Institute
Medical Director, Center for Nutrition & Weight Management

OBESITY:

Blame it on the brain.

Supporting the need for Pharmacologic or Surgical Treatment

Christopher D. Still, DO, FACP, FTOS

Professor of Medicine
Department of Clinical Sciences
Geisinger Commonwealth School of Medicine

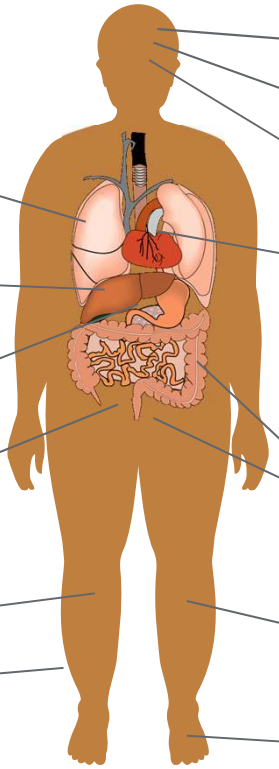
Medical Director, Center for Nutrition & Weight Management
Director, Center for Obesity and Metabolic Research
Geisinger Clinic

November 8, 2023

We Love to Lay Blame for OBESITY

- On the people with obesity (whom internalize that blame)
- On food companies
- On sugar (or fat, or artificial sweeteners, etc.)
- On urban planners
- **On the BRAIN**

Cardiometabolic Diseases & Obesity



Idiopathic intracranial hypertension

Stroke

Cataracts

Coronary heart disease

Diabetes
Dyslipidemia
Hypertension

Pulmonary disease
Abnormal function
Obstructive sleep apnea
Hypoventilation syndrome

Nonalcoholic fatty liver disease
Steatosis
Steatohepatitis
Cirrhosis

Gall bladder disease

Gynecologic abnormalities
Abnormal menses
Infertility
Polycystic ovarian syndrome

Cancer
Breast, uterus, cervix, colon, esophagus, pancreas, kidney, prostate

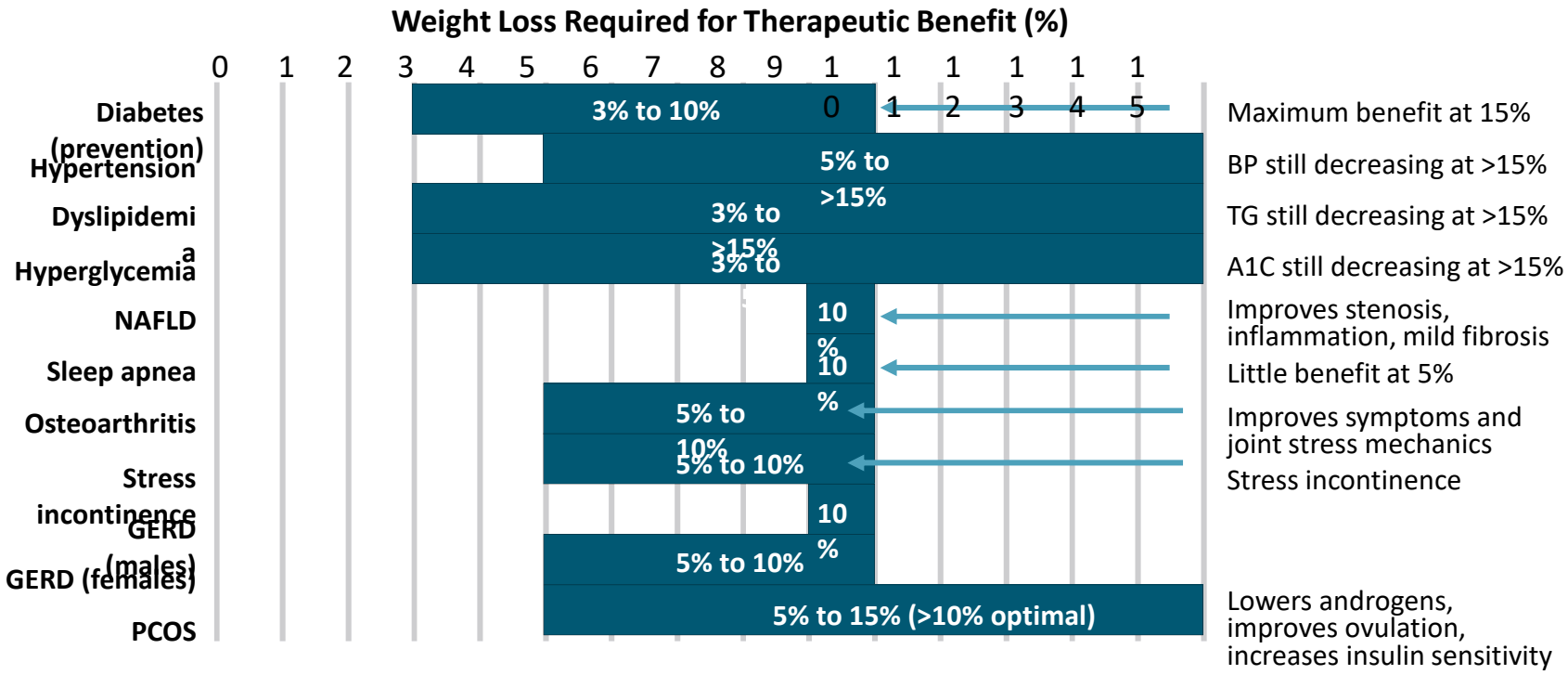
Osteoarthritis

Phlebitis
Venous stasis

Skin

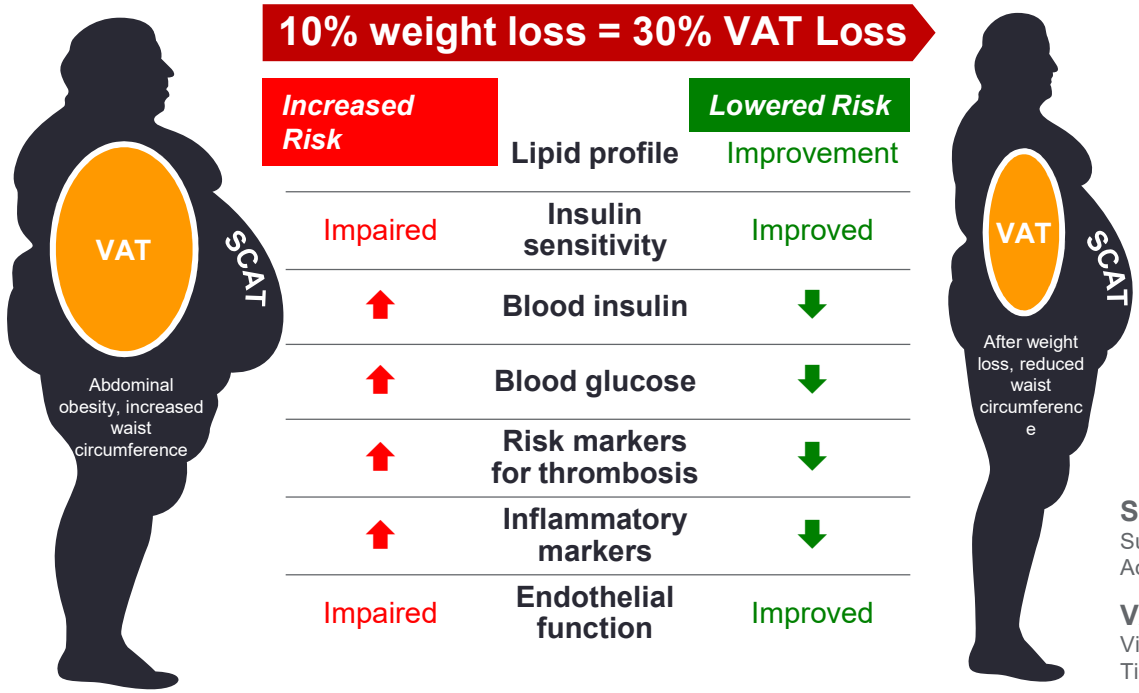
Gout

Therapeutic Weight Loss Reduces Complications



Cefalu. Diabetes Care. 2015;38:1567.

Why is modest weight loss beneficial in Cardiometabolic Diseases?



10% weight loss = 30% VAT Loss

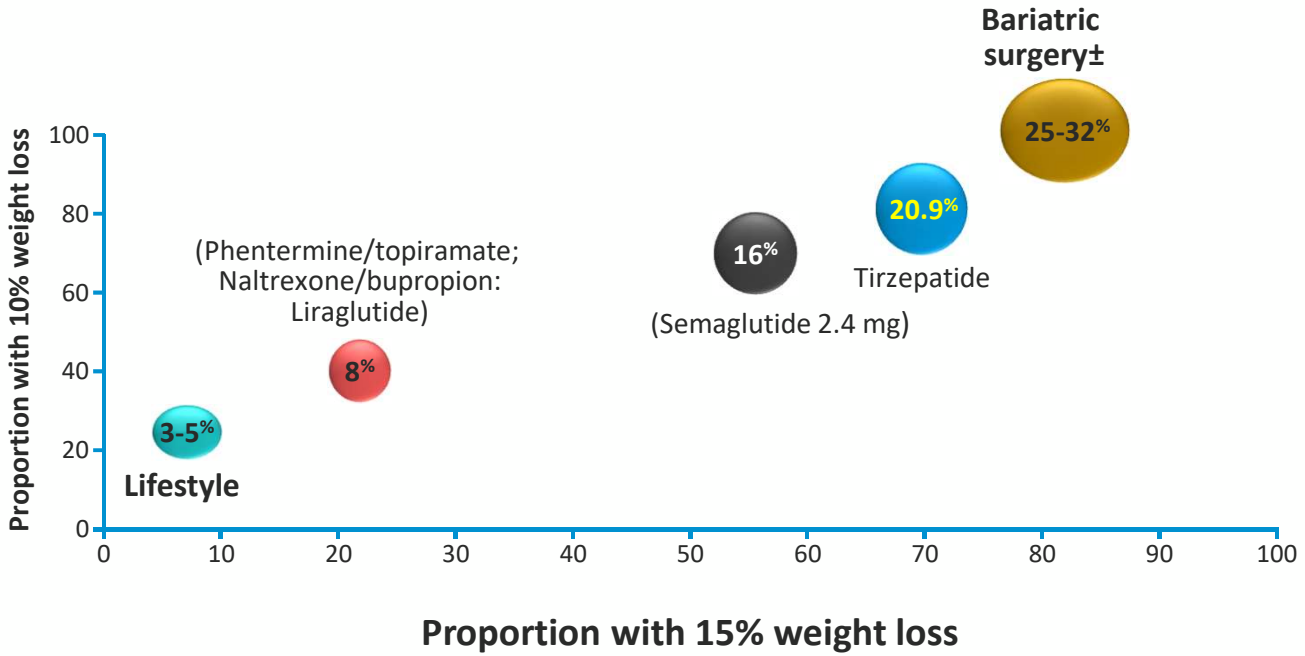
Increased Risk		*Lowered Risk*
	Lipid profile	Improvement
Impaired	**Insulin sensitivity**	Improved
⬆	**Blood insulin**	⬇
⬆	**Blood glucose**	⬇
⬆	**Risk markers for thrombosis**	⬇
⬆	**Inflammatory markers**	⬇
Impaired	**Endothelial function**	Improved

SCAT = Subcutaneous Adipose Tissue

VAT = Visceral Adipose Tissue

Adapted from: Després J, et al. BMJ. 2001;322:716-720.

Efficacy of Existing Obesity Interventions



AoM, anti-obesity medications.
Bubble size represents mean % weight loss

Allison DB, et al. *Obesity*. 2012;20:330-342. [EQUIP]; Gadde KM, et al. *Lancet*. 2011;37:1341-1352. [CONQER]; Greenway FL, et al. *Lancet*. 2010;376:595-605. [COR-I]; Apovian CM, et al. *Obesity*. 2013;21:935-943 [COR-II]; Wadden TA, et al. *Obesity*. 2011;19(1):110-120. [COR-BMOD]; Pi-Sunyer X, et al. *N Engl J Med*. 2015;373(1):11-22. [SCALE]; Wadden TA, et al. *In J Obes*. 2013;37:1443-1451. [SCALE MAIN]; Enebo LB, et al. *Lancet*. 2021;397(10286):1736-1748. [Cag + Sema]; Wilding JPH, et al. *N Engl J Med*. 2021;384(11):989. [STEP 1]; Wadden TA, et al. *JAMA*. 2021;325(14):1403-1413. [STEP 3]; Rubino D, et al. *JAMA*. 2021;325(14):1414-1425. [STEP 4]; Ryan D. *Lancet Diabetes Endocrinol*. 2021;9(5):252-254. [STEP]; Sjöström L, et al.
N Engl J Med. 2007;357:741-52; Jastreboff AM, et al. *N Engl J Med*. 2022;387(3):205-216.

Obesity Is a Chronic Disease With a Complex Etiology[1-6]

Possible interrelated factors contributing to obesity:

Physiological[1-3]
- Altered levels of hormones and gastrointestinal peptides
- Altered homeostatic and reward system pathways
- Weight-positive medications
- Health conditions (IR, PCOS, DM, etc.)
- Sleep hygiene/quality

Behavioral[3]
- Physiologic "diet"
- Inactivity/sedentariness
- Emotional factors/ depression
- Lack of sleep
- Smoking cessation

Genetic[4]
- Epigenetics
- Mutations
- Single nucleotide polymorphisms

Environmental[5,6]
- Socioeconomic status
- Access to/affordability of food
- Built/physical environment
- Cultures
- Sociocultural attitudes
- Endocrine-disrupting chemicals

1. Lean MEJ et al. *Int J Obes (Lond)*. 2016;40:622-632. 2. Yu YH et al. *Obes Rev*. 2015;16:234-247.
3. National Heart, Lung, and Blood Institute. 2012.www.nhlbi.nih.gov/health/health-topics/topics/obe/causes#. Accessed July 14, 2016.
4. Moleres A et al. *Curr Obes Rep*. 2013;2:23-31. 5. Sharma AM et al. *Obes Rev*. 2010;11:362-370. 6. Chaput JP et al. *Obes Rev*. 2012;13:681-691.

Obesity is a Complex Disease: Possible Causes

Genetic mutations leading to obesity remain to be elucidated[1,2]

High heritability of body weight as indicated by twin and adoption studies[2]

Multiple gene variations in key metabolic and homeostatic pathways may also contribute to obesity[1]

~5% of obesity cases may be due to single gene variations in[1]:

- *LEP, LEPR, POMC, MC4R*, and *PCSK1*

LEP=leptin; LEPR=leptin receptor; MC4R=melanocortin receptor 4; PCSK1=proprotein convertase subtilisin/kexin type 1; POMC=proopiomelanocortin.

1. Moleres A et al. *Curr Obes Rep*. 2013;2:23-31.
2. Chesi A et al. *Trends Endocrinol Metab*. 2015;26:711-721.

Possible Causes of Poor Weight Loss Maintenance

Adherence

One explanation for the poor long-term outcome of weight-loss diets relates to behavior:

Motivation to adhere to restrictive regimens typically diminishes with time

Hypothalamic Injury

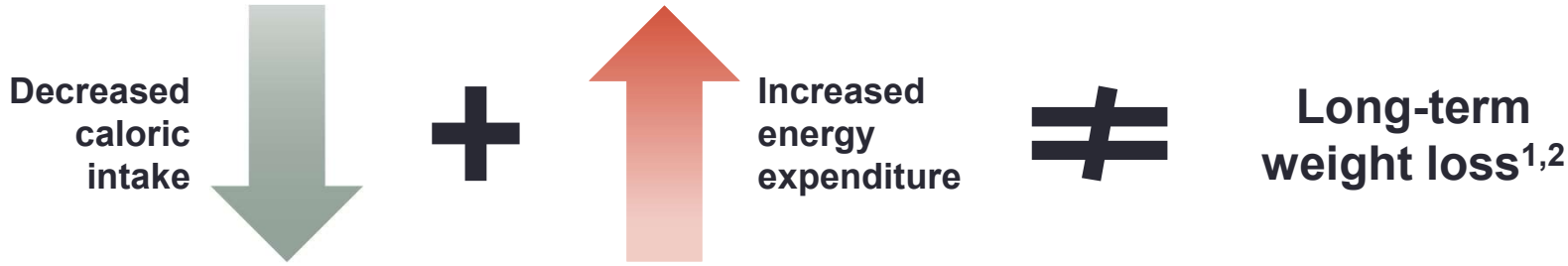
Weight loss elicits biological adaptations that promote weight regain:

Specifically, a decline in energy expenditure (adaptive thermogenesis) and an increase in hunger

Ebbling CB, et al. JAMA. 2012 Jun 27;307(24):2627-34.

Regulation of Food Intake and Body Weight Regulation

Counting Calories Is Not Enough to Achieve Long-term Weight Loss



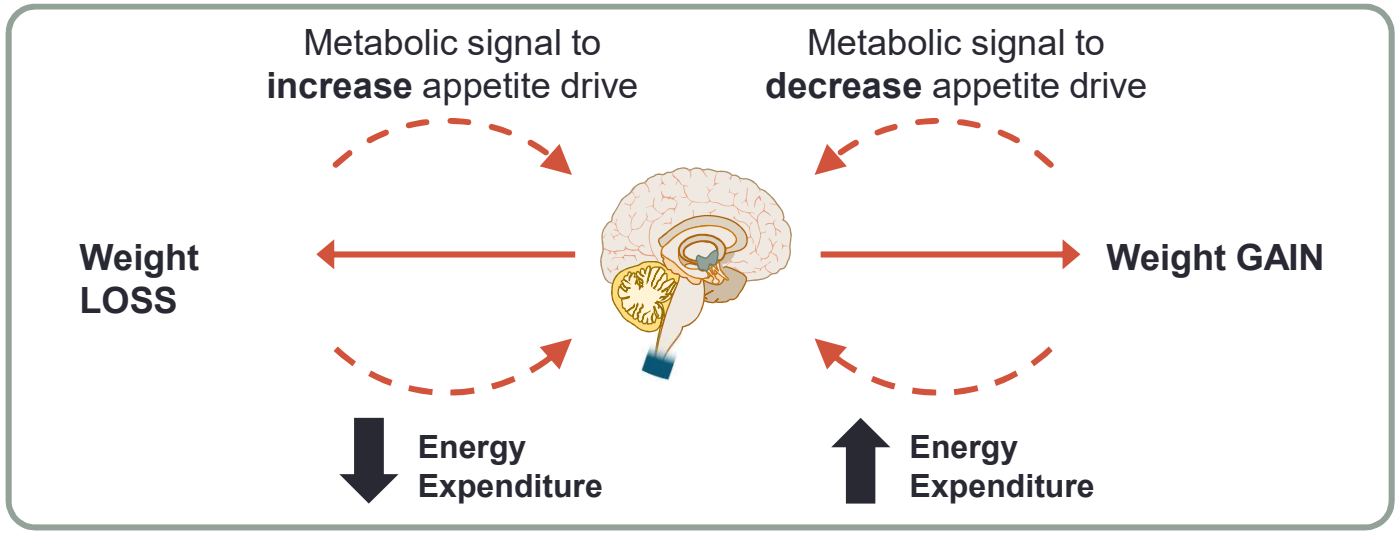
CNS pathways sense changes in weight and body energy stores and **exert opposing effects on energy balance** to promote homeostasis[3]

CNS=central nervous system.

1. Chaput JP et al. *Obes Rev*. 2012;13:681-691.
2. National Heart, Lung, and Blood Institute. 2012. www.nhlbi.nih.gov/health/health-topics/topics/obe/causes#. Accessed July 14, 2016.
3. Schwartz MW et al. *Diabetes*. 2003;52:232-238.

Homeostatic Regulation of Set Point Body Weight[1]

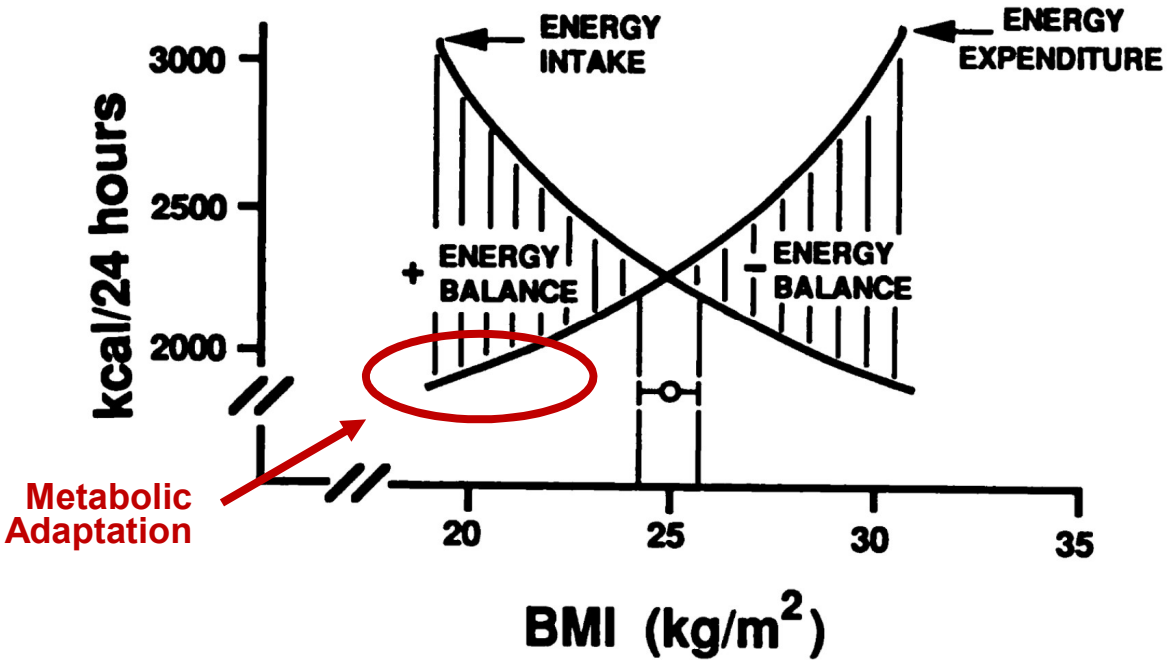
A homeostatic weight regulatory system prevents deviation from a body weight set point



Deviation from this set point elicits a **physiological** compensatory mechanism controlling **food intake** and **energy expenditure**

1. Yu YH et al. *Obes Rev*. 2015;16:234-247.

Defense of a Body Fat Storage "Set Point"



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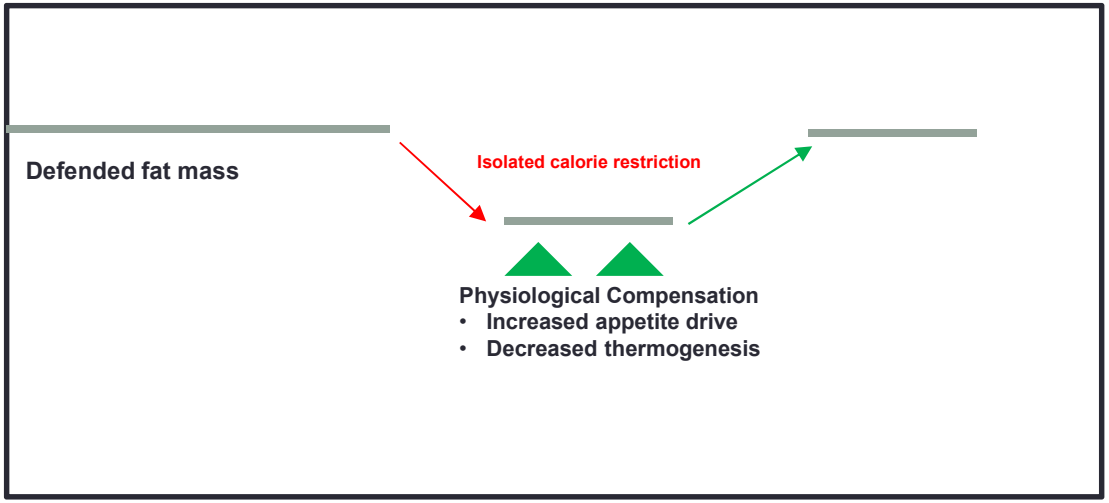
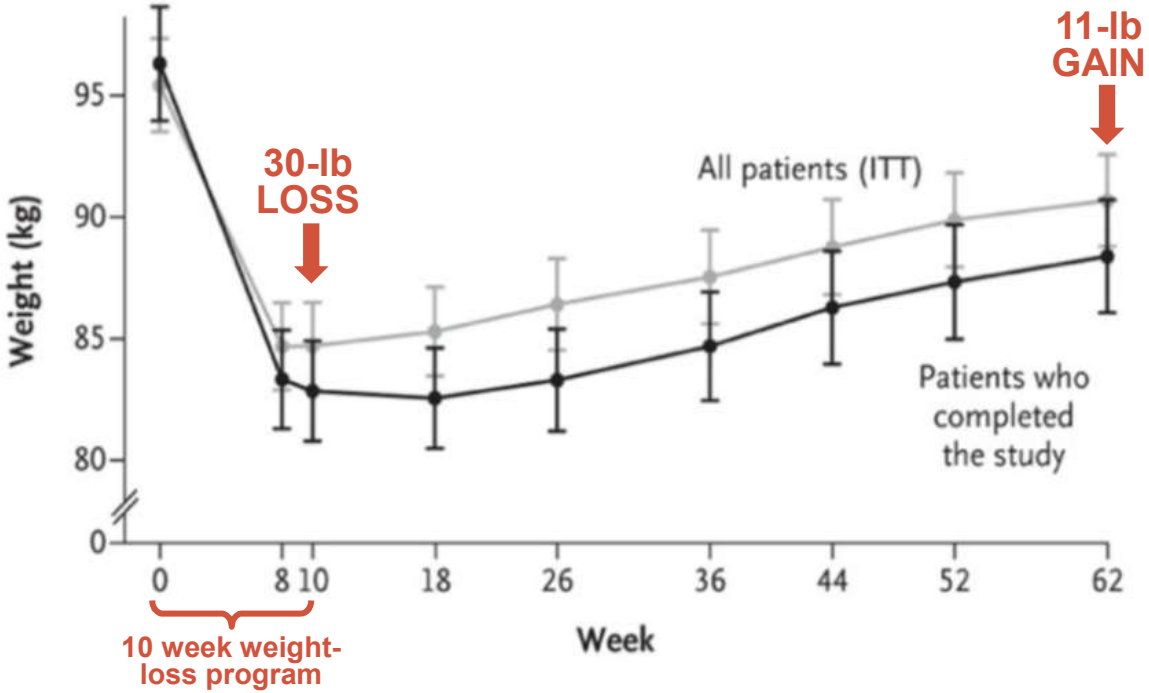
Counter-Physiologic Weight Loss: Caloric Restriction



14% Weight Loss Led to Major **Hormonal** Adaptations Which Lasted for 1 Year

Changes in Weight from Baseline to Week 62



Sumithran P, et al. *N Engl J Med.* 2011;365:1597-1604.

14% Weight Loss Produced Changes in 8 Hormones That Encourage Weight Regain

Mean fasting and postprandial levels of some peripheral signals at baseline and 62 weeks

14% Weight Loss ***Reduced:***	***Increased:***
• **Leptin - 65%** ↓ - Adipose hormone - Regulates appetite - Control of metabolism & energy homeostasis	• **Ghrelin** - Gastric hormone - Promotes hunger - Fat deposition
	• **Measures of appetite**

10-week, lifestyle-based weight loss intervention in healthy overweight and obese adults (n=34)

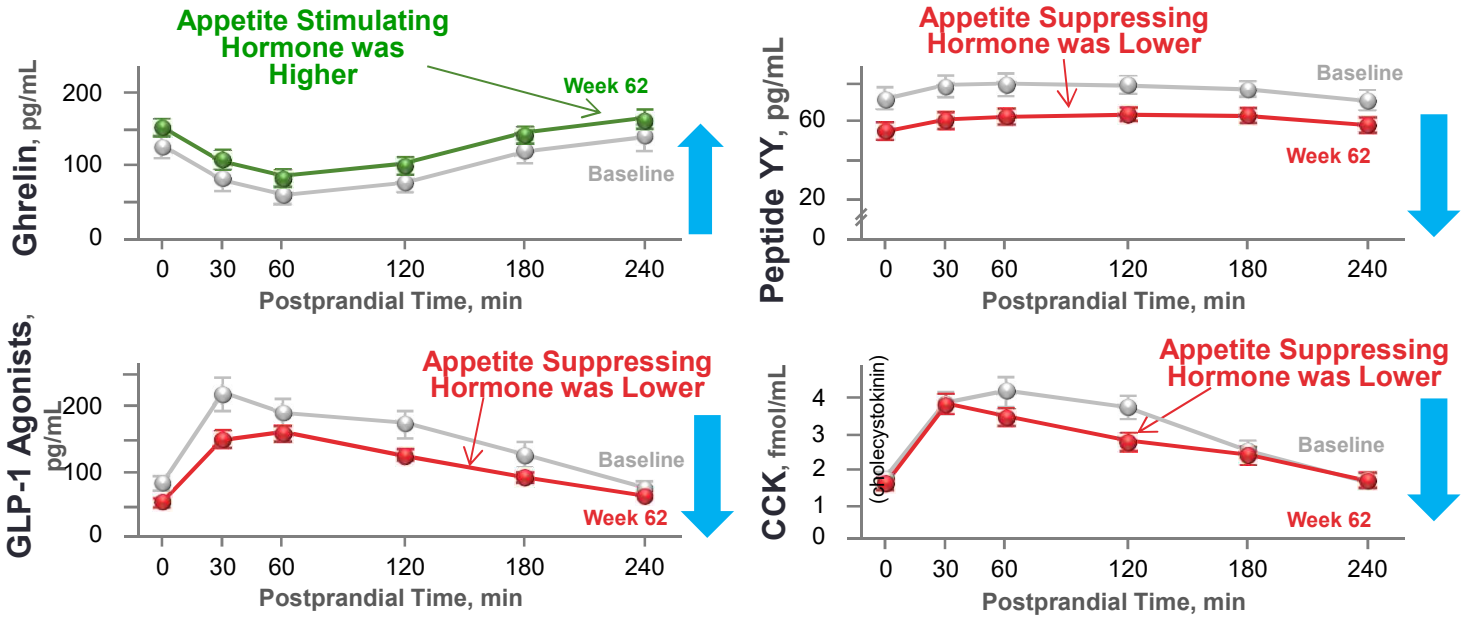
Led to sustained elevations in appetite stimulating hormone(s) and decreases in appetite suppressing hormones

Net result of these hormonal changes is **WEIGHT GAIN**

Sumithran P, et al. *N Engl J Med.* 2011;365:1597-1604.

Sustained Changes in Peripheral Signals for Up to One Year Following Weight Loss

Mean fasting and postprandial levels of some peripheral signals at baseline and 62 weeks



10-week, lifestyle-based weight loss intervention in healthy overweight and obese adults (n=34) led to sustained elevations in appetite stimulating hormone(s) and decreases in appetite suppressing hormones

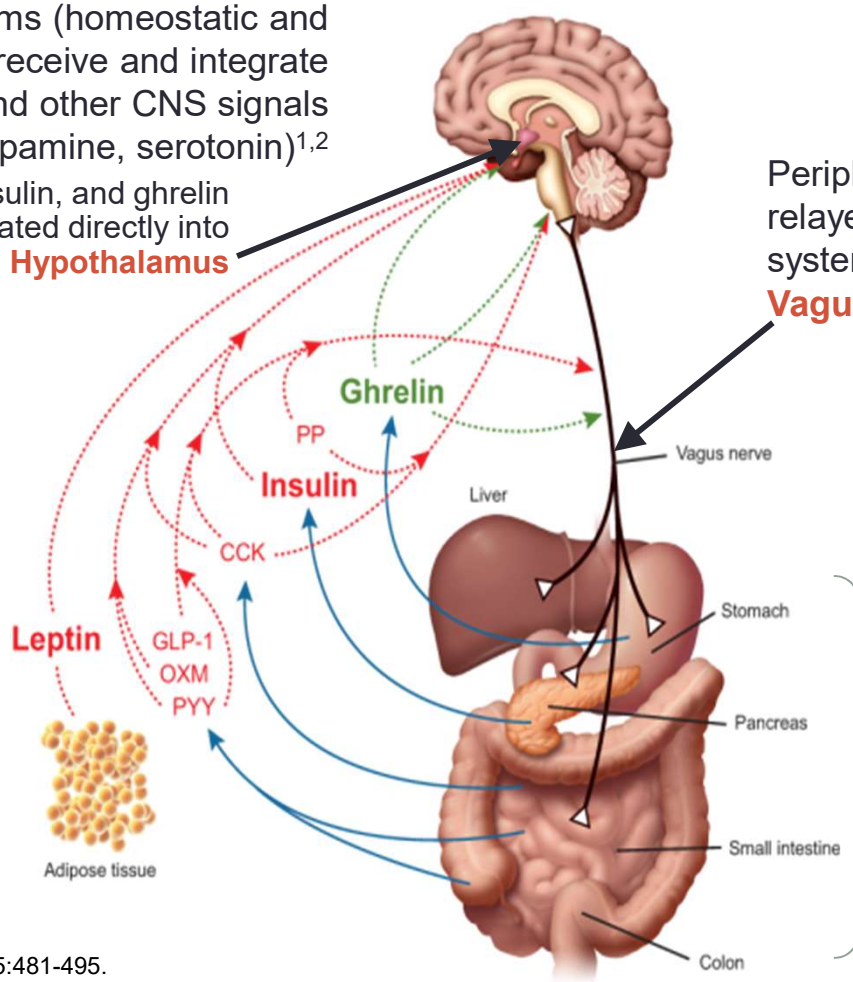
Sumithran P, et al. *N Engl J Med.* 2011;365:1597-1604.

Complex Peripheral Signals are Integrated Into CNS Systems to Regulate Body Weight

Brain systems (homeostatic and reward) receive and integrate peripheral and other CNS signals (eg, dopamine, serotonin)[1,2]

Leptin, insulin, and ghrelin are integrated directly into **Hypothalamus**

Peripheral signals are relayed to brain systems via blood and **Vagus Nerve** [1,2]



Peripheral signals are released by pancreas, gastrointestinal system, and adipose tissue[1,2]

CNS, central nervous system
PfC, prefrontal cortex
NAc, nucleus accumbens
VTA, ventral tegmental area
PP, pancreatic polypeptide
CCK, cholecystokinin;
GLP-1, glucagon-like peptide 1
OXM, oxyntomodulin
PYY, peptide YY.
Primarily based on data from animal studies.

Appetite Stimulating
Appetite Suppressing

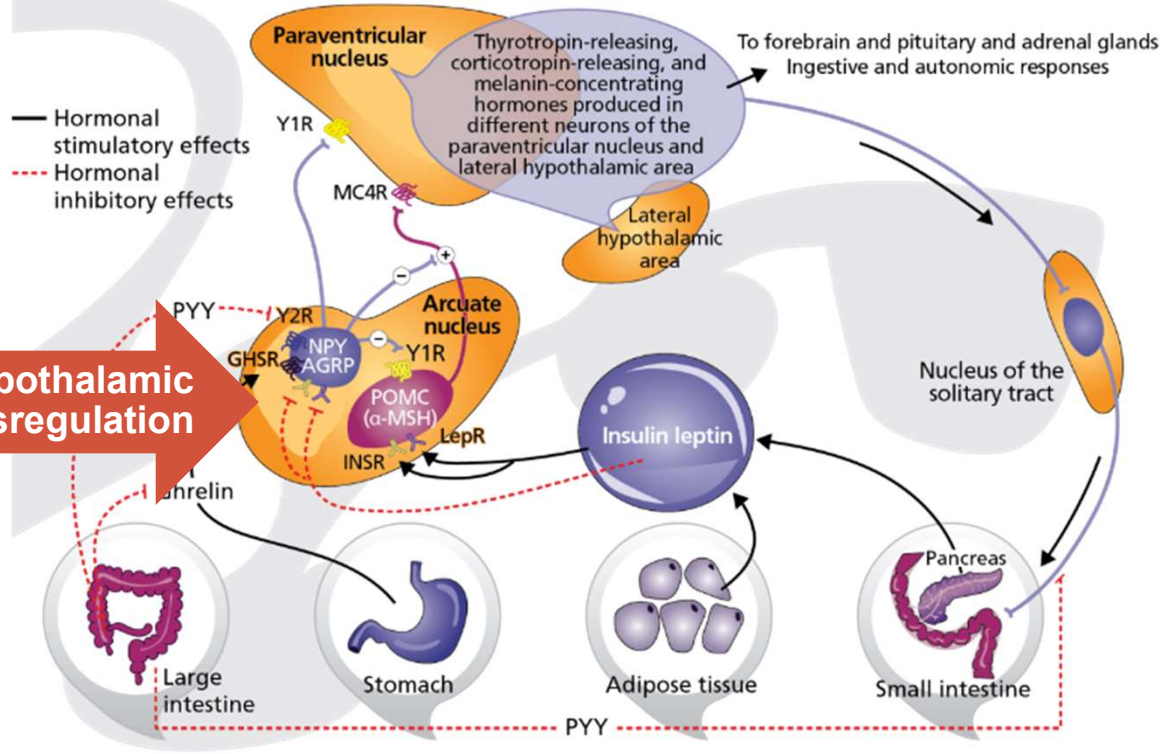
1. Yu JH et al. *Diabetes Metab J*. 2012;36(6):391-398.
2. Mendieta-Zerón H et al. *Gen Comp Endocrinol*. 2008;155:481-495.

Hypothalamic **Dysregulation** Diminishes Signaling to Cortex and NTS, Leading to Greater Weight Gain

When damaged, the brain can't tell how much fat is stored or how much has been eaten

Brain becomes resistant to key hormone, leptin

Hypothalamic dysregulation



AGRP: agouti-related peptide
α-MSH: α-melanocyte-stimulating hormone
GHSR: growth hormone secretagogue receptor
INSR: insulin receptor;
LepR: leptin receptor
MC4R: melanocortin-4 receptor
NPY: neuropeptide Y
POMC: proopiomelanocortin
PYY: peptide YY
Y1R; neuropeptide Y1 receptor
Y2R: neuropeptide Y2 receptor

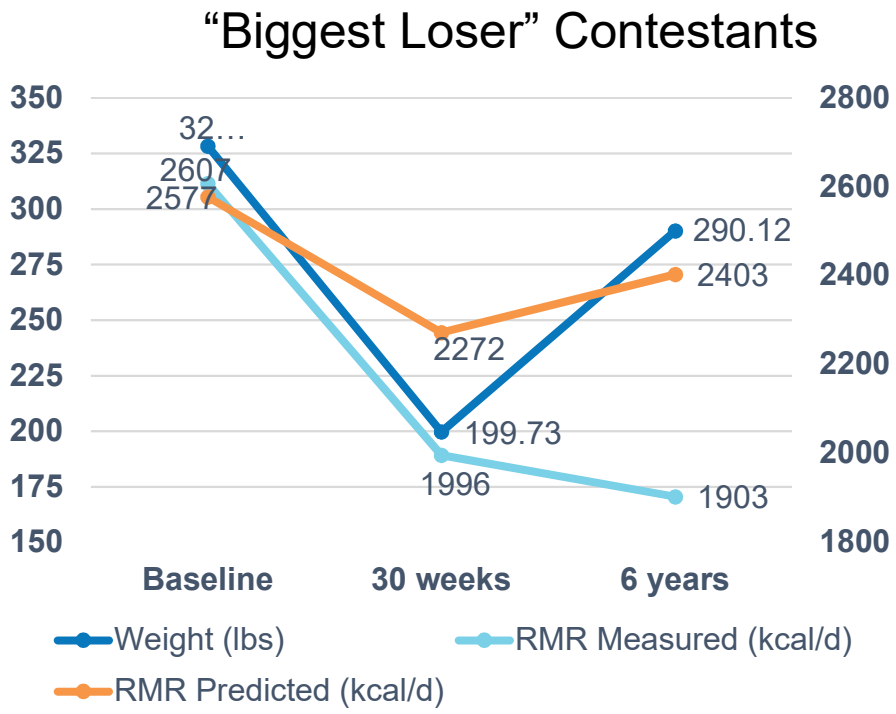
Apovian CM, Aronne LJ, Bessesen D et al. *J Clin Endocrinol Metab*. 2015;100:342-362.

Metabolic Adaptation to Weight Reduction

- Reduction in resting metabolic rate greater than that predicted with weight loss alone
- Associated with degree of reduction in leptin levels greater than the percentage of weight loss alone
- Greater weight loss = greater metabolic adaptation
- Subject to individual variability
- Metabolic adaptation after weight loss has been demonstrated for up to 6 years.

Ravussin, E. and Ryan, D. H. Obesity, 2016. 24: 1607-1608.

Resting Metabolic Weight Decreases During Weight Loss and Weight Regain



N = 14
Competition = 30 weeks

Weight regain was not significantly correlated with metabolic adaptation at the competition's end (r = -0.1, P = 0.75)

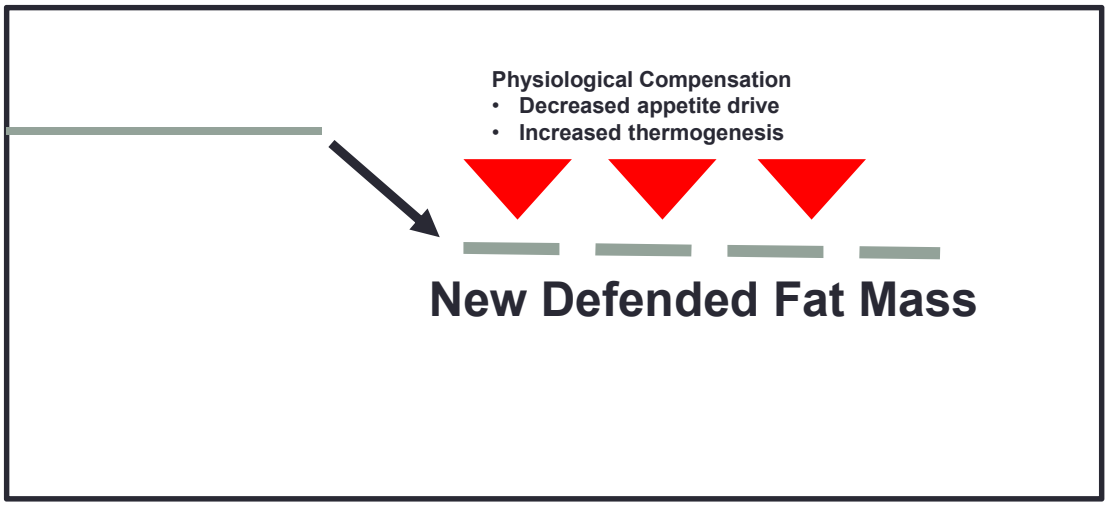
Those maintaining greater weight loss at 6 years also experienced greater concurrent metabolic slowing (r = 0.59, P = 0.025)

Fothergill E, et al. Obesity (Silver Spring). 2016 Aug;24(8):1612-9.

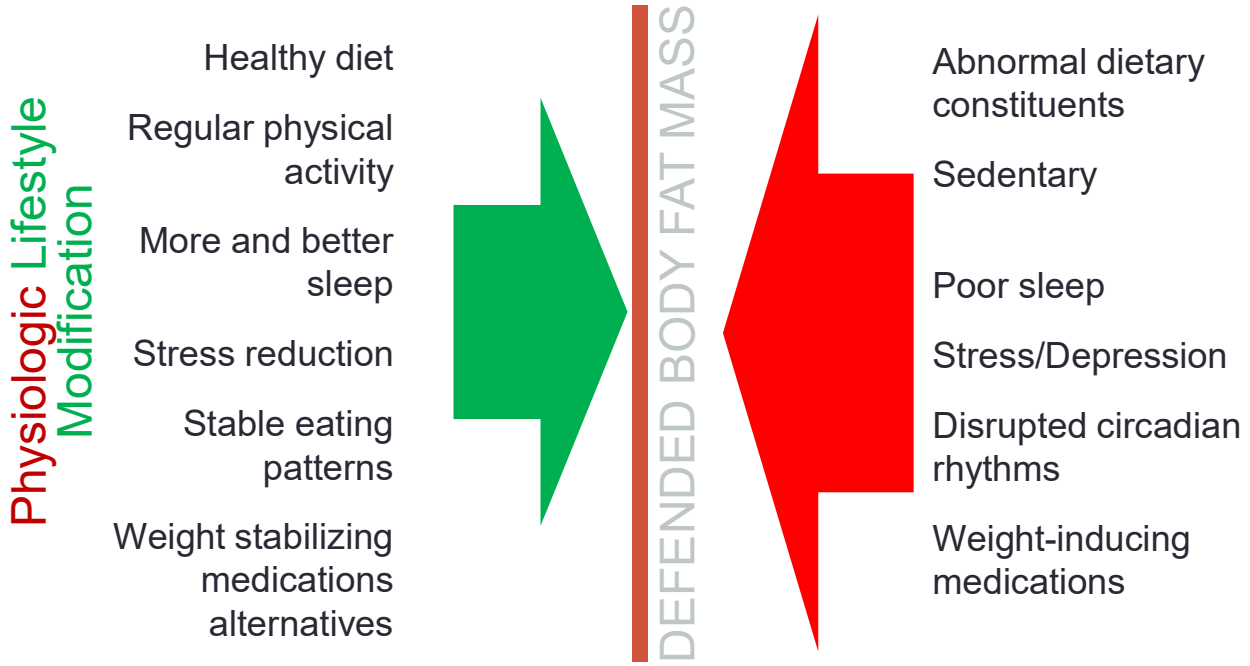
The major problem – nearly all obesity treatment is
not physiologically driven

IMPLICATIONS FOR OBESITY TREATMENT

Physiologic Weight Loss:
Targeted Lifestyle Modification, Effective Medications, Surgery



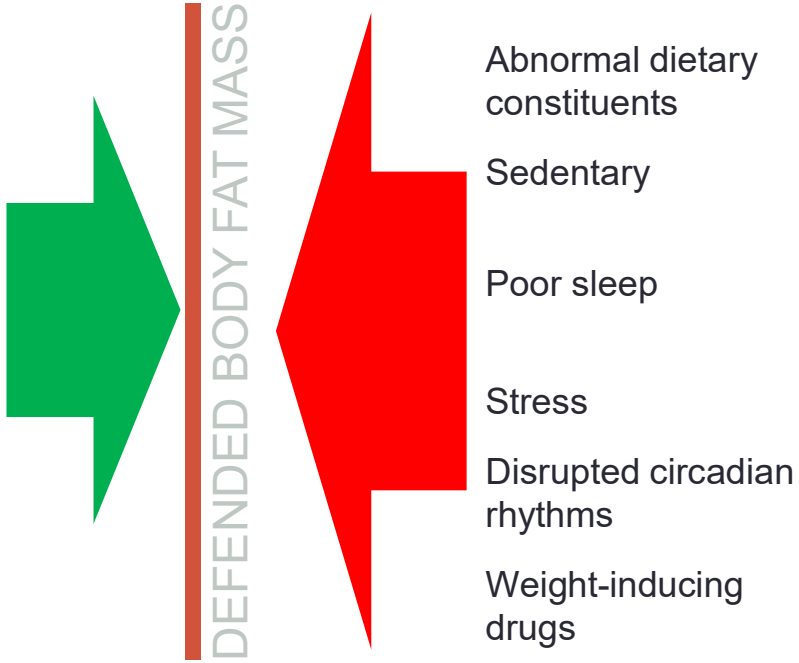
Battle of **Physiologic** Forces that Influence Fat Mass



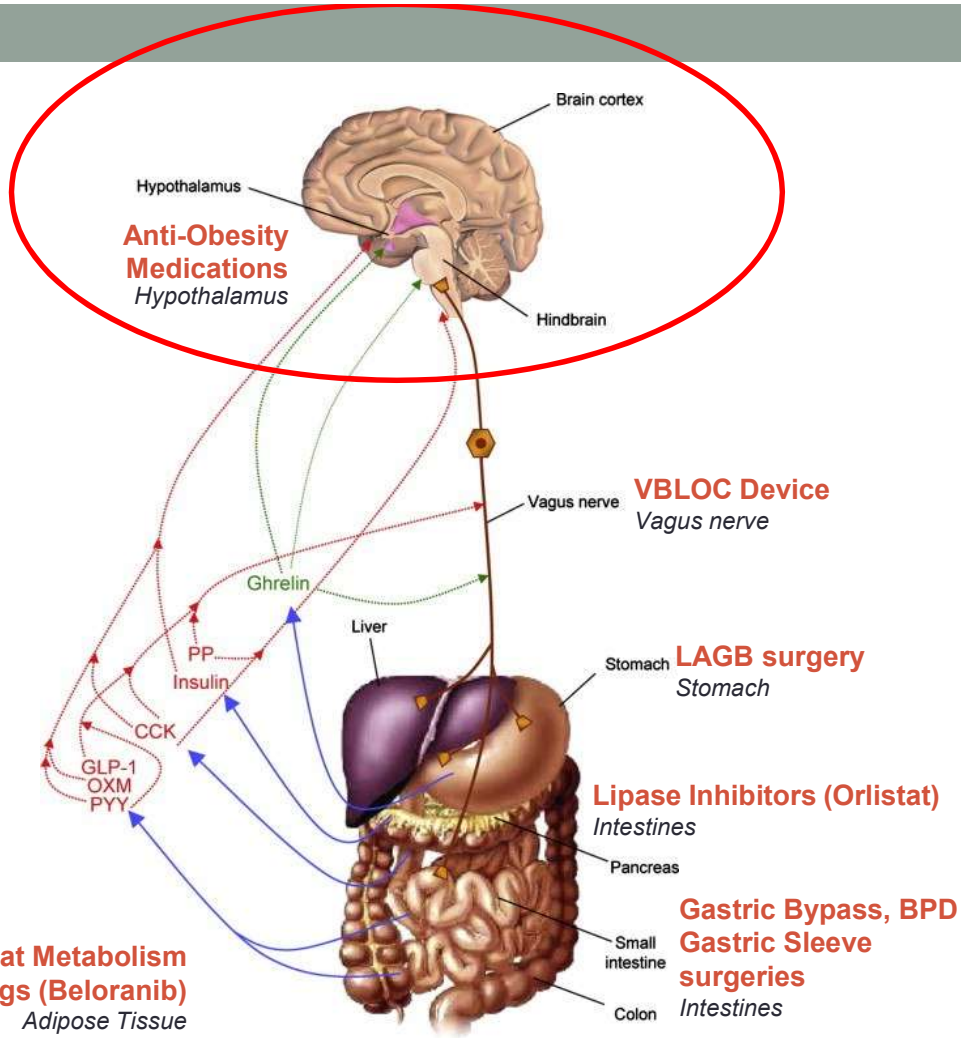
Battle of **Physiologic** Forces that Influence Fat Mass

Anti-Obesity Medications

Phentermine plus topiramate-ER *(3.75 mg/23 mg for 2 weeks, increased to 7.5 mg/46 mg, escalating to a max of 15 mg/92 mg; 1×/d*	Noradrenergic + GABA-receptor activator, kainite /AMPA glutamate receptor inhibitor causing appetite suppression
Bupropion/ naltrexone	Inhibitor of dopamine and noradrenaline reuptake + μ opiate antagonist
Liraglutide 3.0 mg **Sema 2.4 mg**	Glucagon-like peptide 1 (GLP-1) agonist

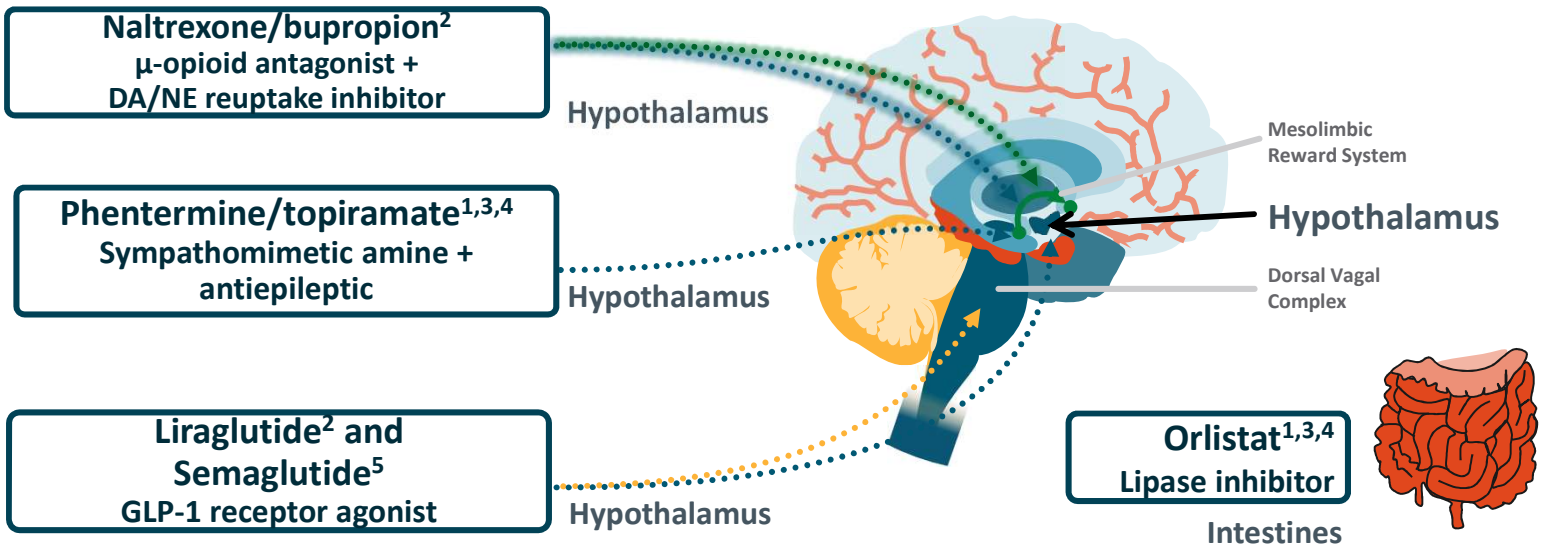


Where Obesity Treatments Work



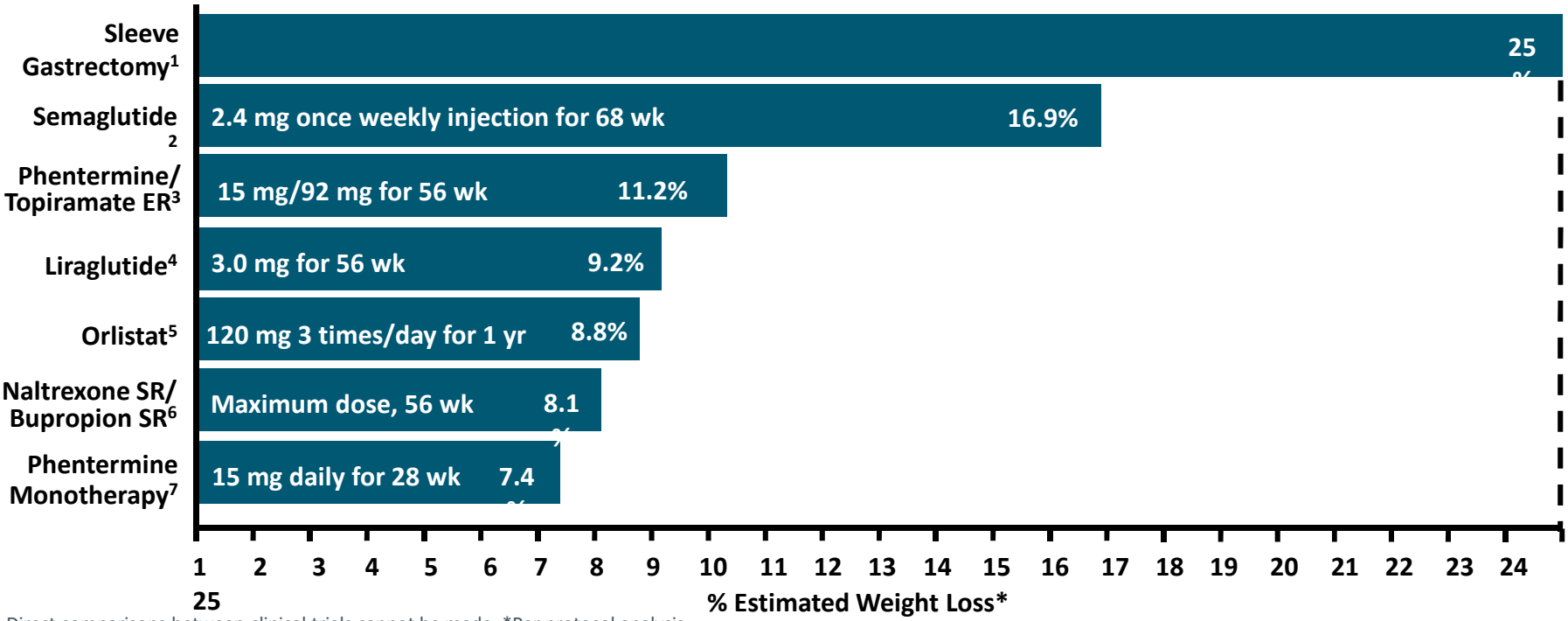
Current Obesity Pharmacotherapy for Long-term Use

- Multiple pharmacotherapies with varying MoA currently approved in US for long-term treatment of obesity[1-4]



1. Yanovski. JAMA. 2014;311:74. 2. Apovian. J Clin Endocrinol Metab. 2015;100:342. 3. Kim. Clin Pharmacol Ther. 2014;95:53. 4. Dietrich. Nat Rev Drug Discov. 2012;11:675. 5. Christou. Obes Rev. 2019;20:805.

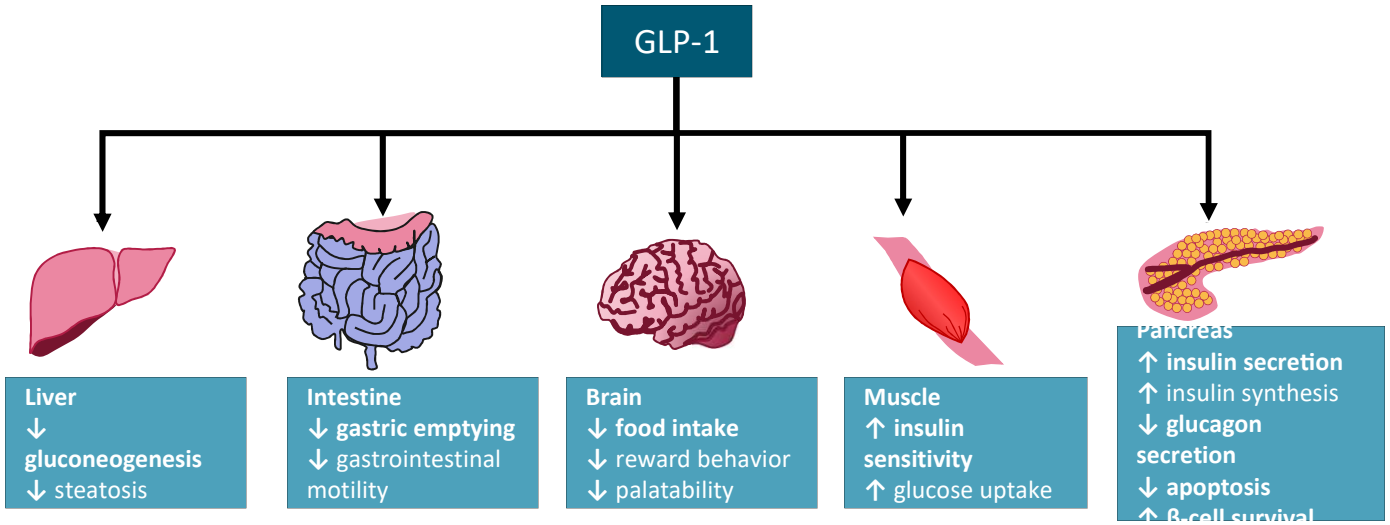
Efficacy of Current FDA-Approved Obesity Therapy



Direct comparisons between clinical trials cannot be made. *Per protocol analysis.

1. Mechanick. Endocr Pract. 2019;25(12):1346. 2. Wilding. NEJM. 2021;384:989. 3. Allison. Obesity (Silver Spring). 2012;20:330. 4. Pi-Sunyer. NEJM. 2015;373:11. 7. Finer. Int J Obes Relat Metab Disord. 2000;24:306. 6. Greenway. Lancet. 2010;376:595. 7. Aronne. Obesity (Silver Spring). 2013;21:2163.

Regulation of Body Weight and Glucose Metabolism by GLP-1 Receptor Agonism



- The specific mechanism of action is multifactorial, with gut, brain, and systemic improvements in insulin sensitivity each contributing a finite fraction to the total efficacy

Müller. Nat Rev Drug Discov. 2022;21:201.

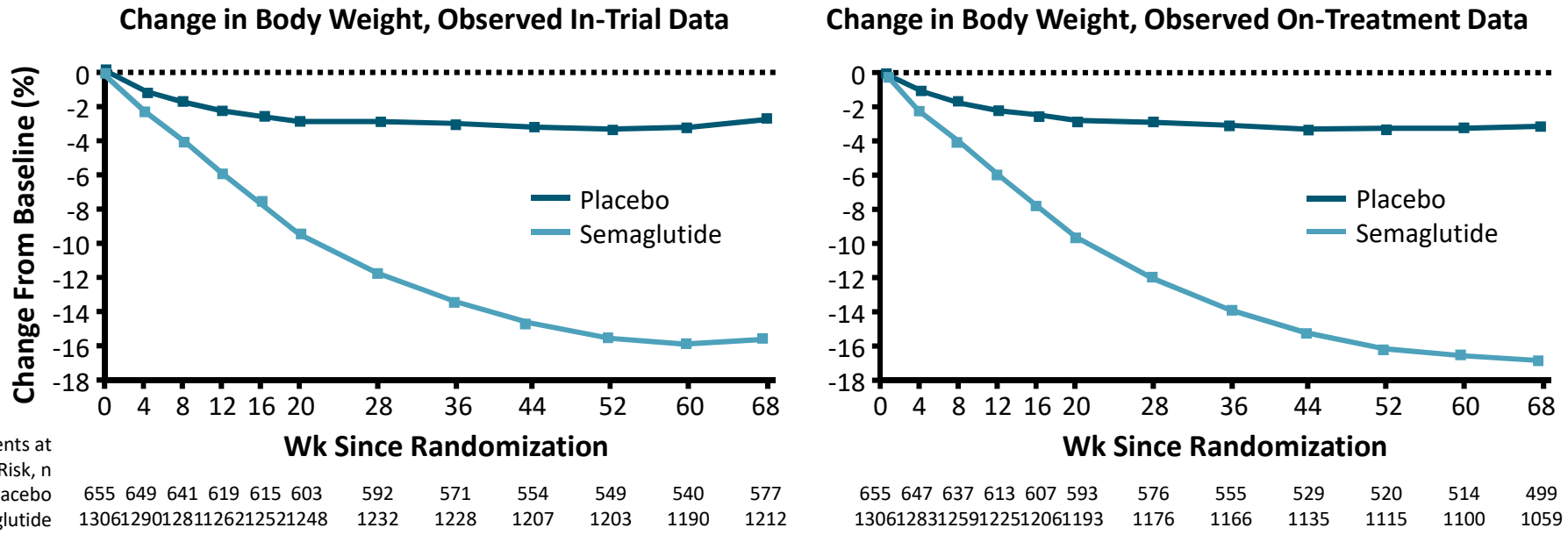
Semaglutide (2.4 mg): Efficacy vs Placebo

5% Weight Loss	10% Weight Loss	15% Weight Loss	Average Weight Loss:
86.4% vs 31.5%	69.1% vs 12.0%	50.5% vs 4.9%	15.3 kg vs 2.6 kg

Wilding. NEJM. 2021;384:989.

STEP 1 Trial: Body Weight Changes With Semaglutide

- Double-blind, placebo-controlled phase III trial in adults with BMI >30 kg/m^2 without diabetes (N = 1961)

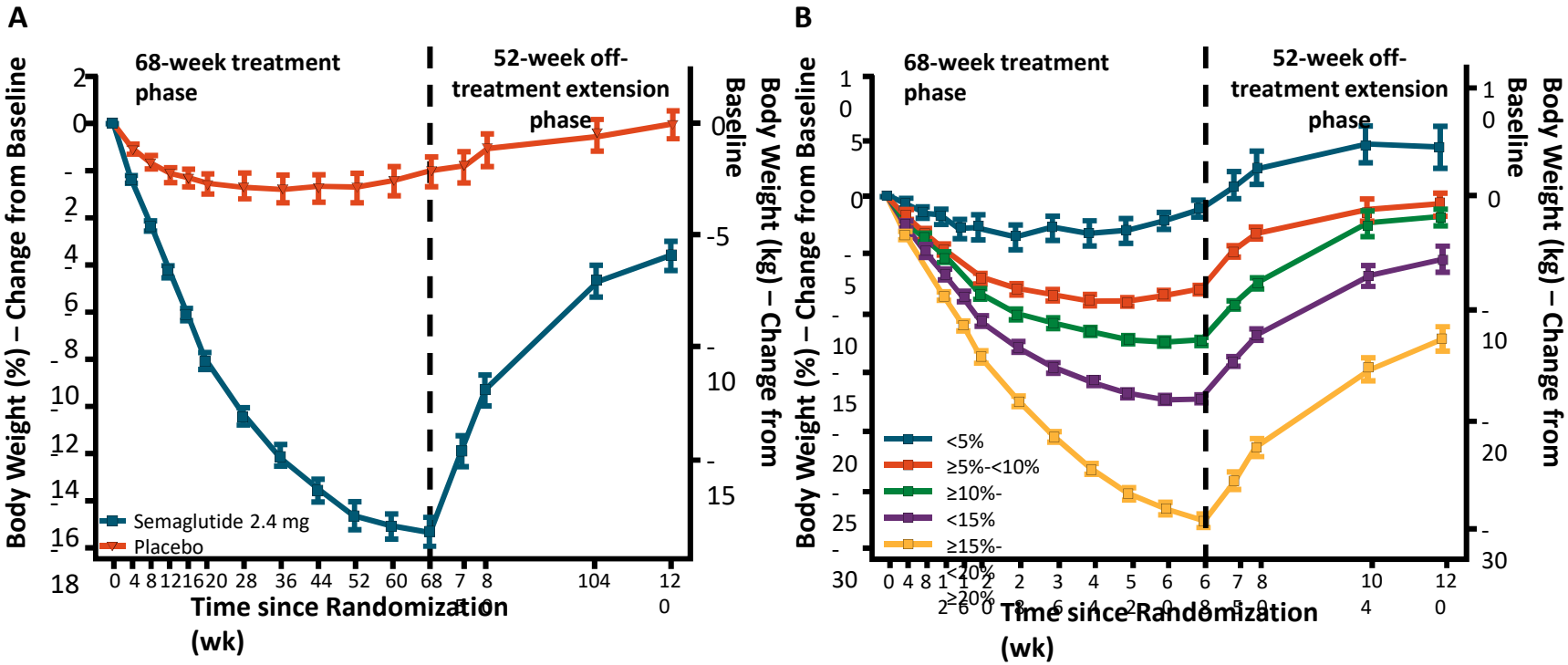


Patients at Risk, n												
Placebo	655	649	641	619	615	603	592	571	554	549	540	577
Semaglutide	1306	1290	1281	1262	1252	1248	1232	1228	1207	1203	1190	1212

Placebo	655	647	637	613	607	593	576	555	529	520	514	499
Semaglutide	1306	1283	1259	1225	1206	1193	1176	1166	1135	1115	1100	1059

Wilding. NEJM. 2021;384:989.

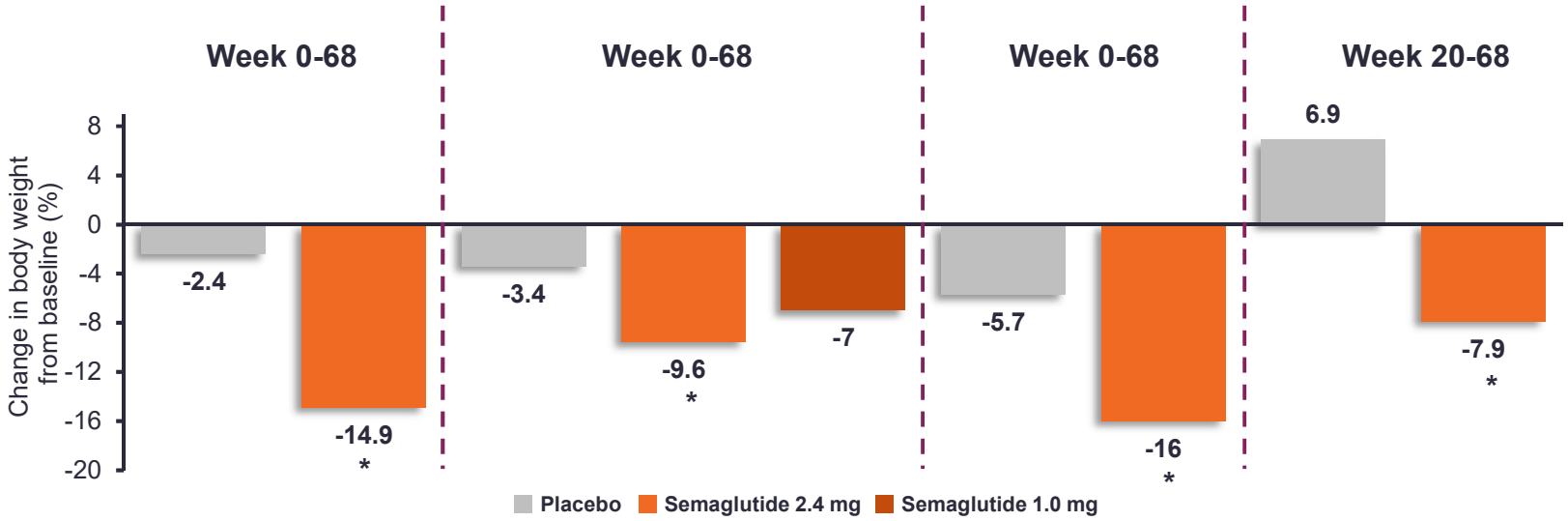
STEP 1 Trial Extension of Semaglutide 2.4 mg



Wilding. Diab Obes Metab. 2022:1.

Semaglutide 2.4 mg: Mean Weight Loss (STEP Trials)

	STEP 1[1] N=1961	STEP 2[2] N=1210	STEP 3[3] N=611	STEP 4[4] N=902
	Weight management	Weight management in type 2 diabetes	Weight management with IBT	Sustained weight management*
Baseline BW	105.3 kg	99.8 kg	105.8 kg	96.1 kg



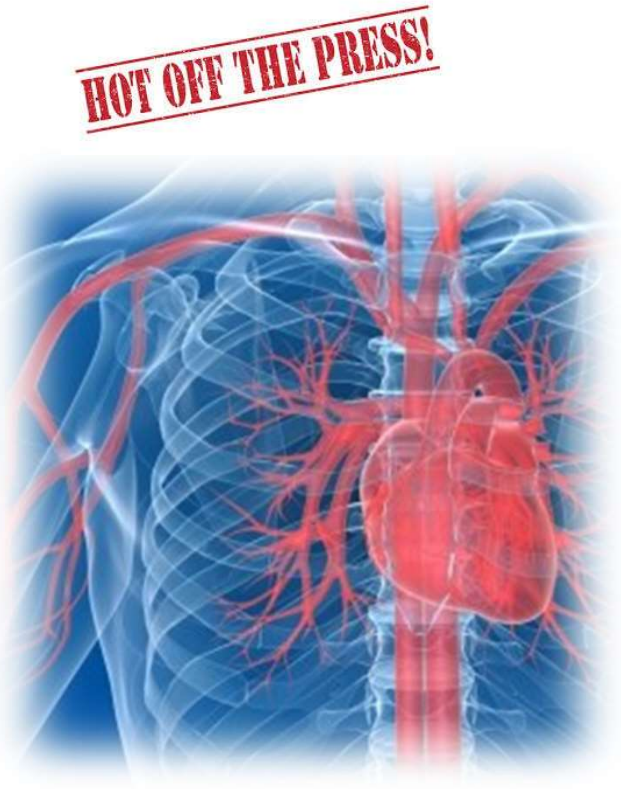
Primary endpoint. *Statistically significant vs placebo.
BW = body weight; IBT = intensive behavioral therapy

1. Wilding JPH et al. *N Engl J Med.* 2021;384(11):989-1002. 2. Davies M et al. *Lancet*. 2021;397(10278):971-84. 3. Wadden TA et al. *JAMA*. 2021;325(14):1403-13.
4. Rubino D et al. *JAMA*. 2021;325(14):1414-25.

SELECT Trial: Cardiovascular Outcomes, August 2023

Randomised, double-blind, parallel-group, placebo-controlled trial

Semaglutide 2.4 mg **reduced risk of major adverse cardiovascular events (MACE) by 22%** in adults with overweight or obesity

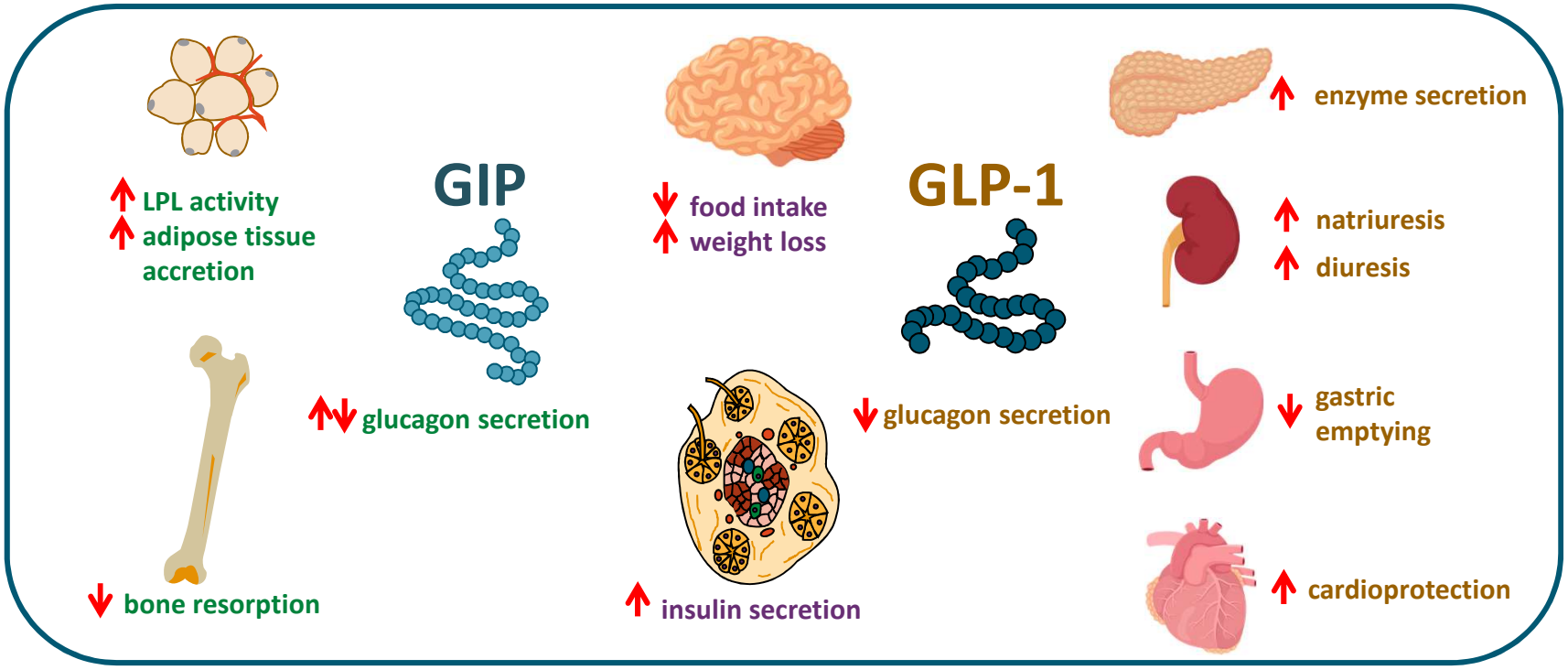


- n = 17,604 adults
- ≥ 45 years
- BMI ≥27 kg/m^2
- with established CVD and no prior history of diabetes

Evaluated subcutaneous once-weekly semaglutide 2.4 mg vs placebo as an adjunct to standard of care for prevention of MACE, over a period of up to five years

https://www.novonordisk.com/news-and-media/news-and-ir-materials/news-details.html?id=166301

The Evolving GIP–GLP-1 Partnership in Metabolism



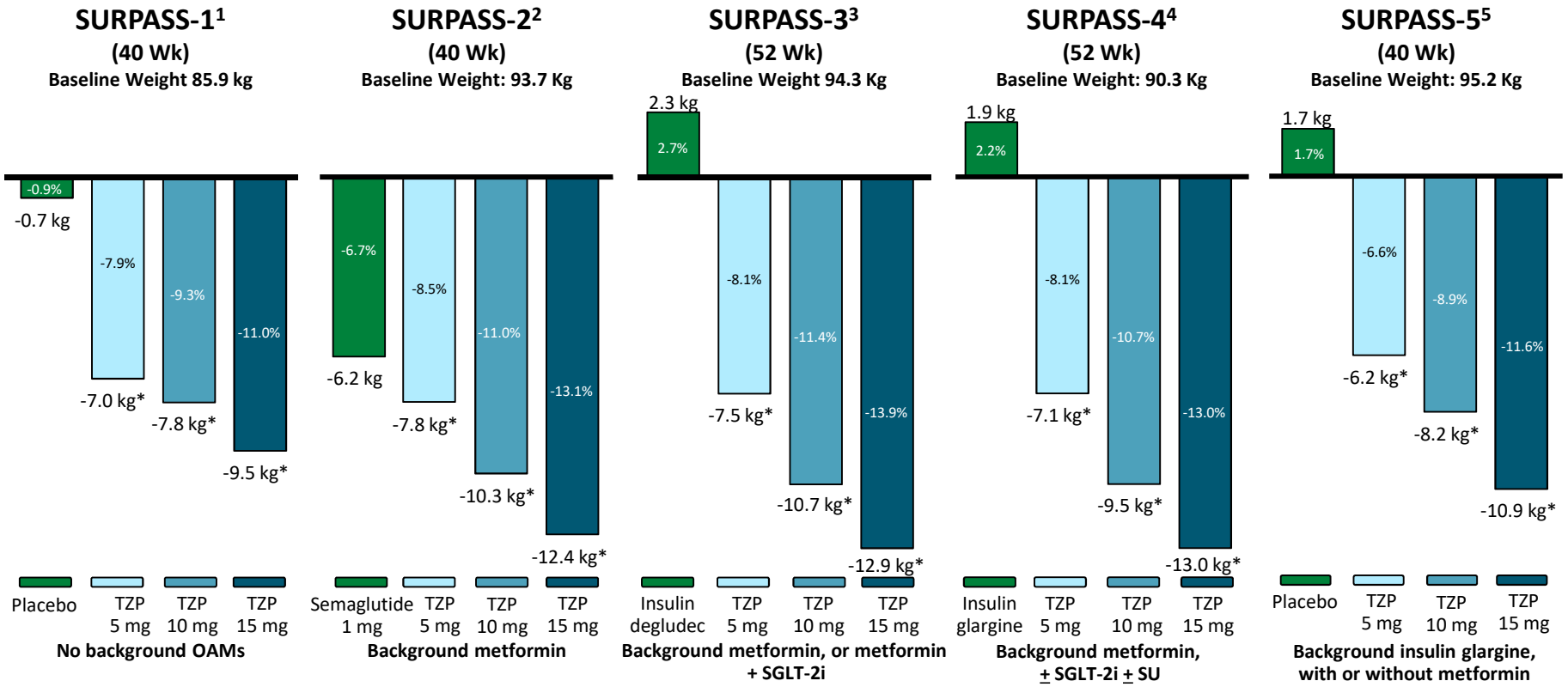
Baggio. J Mol Metabolism. 2020;46:101090.

Tirzepatide: Novel Dual GIP and GLP-1 Receptor Agonist

- Tirzepatide is multifunctional 39 amino acid peptide based on native GIP peptide sequence and modified to bind to GIP or GLP-1 receptors
- Administered as once-weekly injection as half-life of 5 days
  - Starting dose 2.5 mg weekly, titrated at 2.5-mg increments monthly to max dose of 15 mg
- Demonstrated dose-dependent reduction in HbA1c (up to 2.4%) and body weight (up to 11.3 kg) in patients with T2D in phase I and II trials
- Contraindications and AEs similar to GLP-1 RAs
- Contraindications: personal or family history of MTC or MEN2
  - Precautions: pancreatitis, AKI, diabetic retinopathy, gallbladder disease
  - Adverse events: GI including nausea, vomiting, diarrhea, constipation, abdominal pain

Min. Diabetes Ther. 2021;12:143. Coskun. Mol Metab. 2018;18:3. Tirzepatide PI.

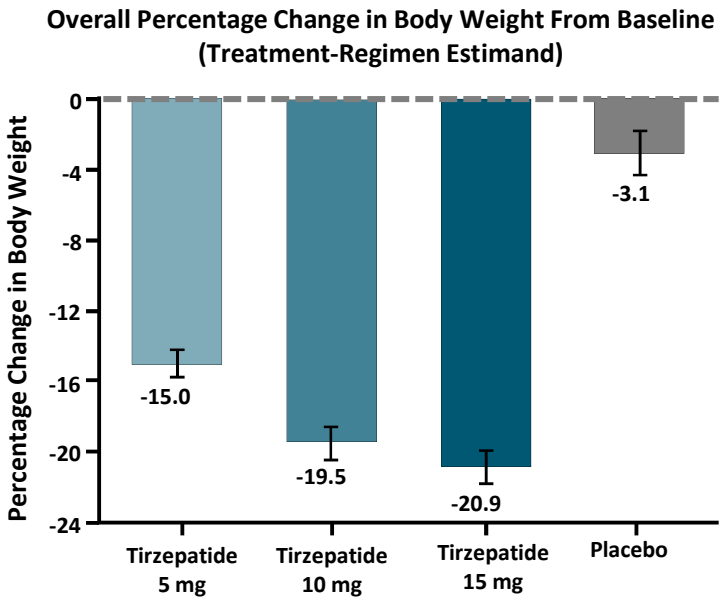
SURPASS: Weight Loss With Tirzepatide in T2D

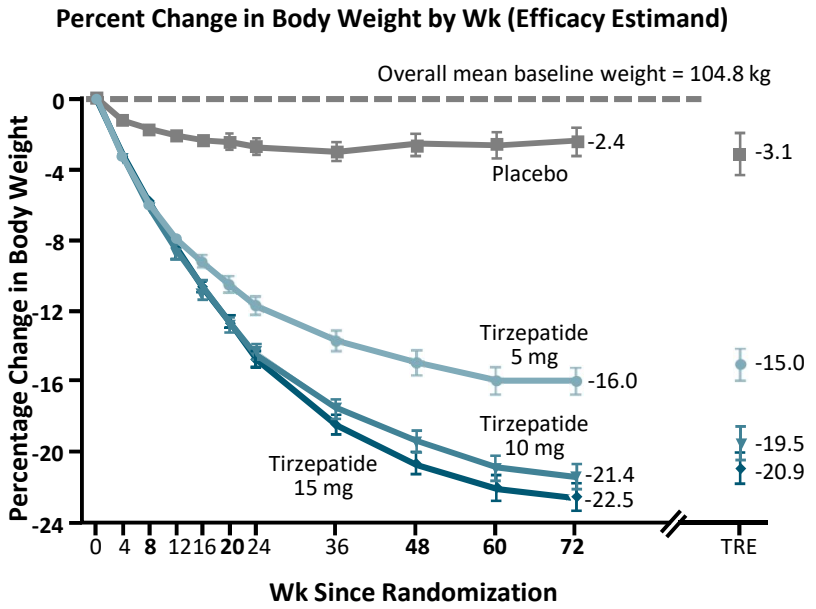


*Denotes statistical significance to comparator.

1. Rosenstock. Lancet. 2021;398:143. 2. Frias. NEJM. 2021;385:503. 3. Giorgino. ADA 2021. Abstr 78-LB. 4. Del Prato. Lancet. 2021;398:1811. 5. Dahl. ADA 2021. Abstr 80-LB.

SURMOUNT 1: Weight Loss With Tirzepatide

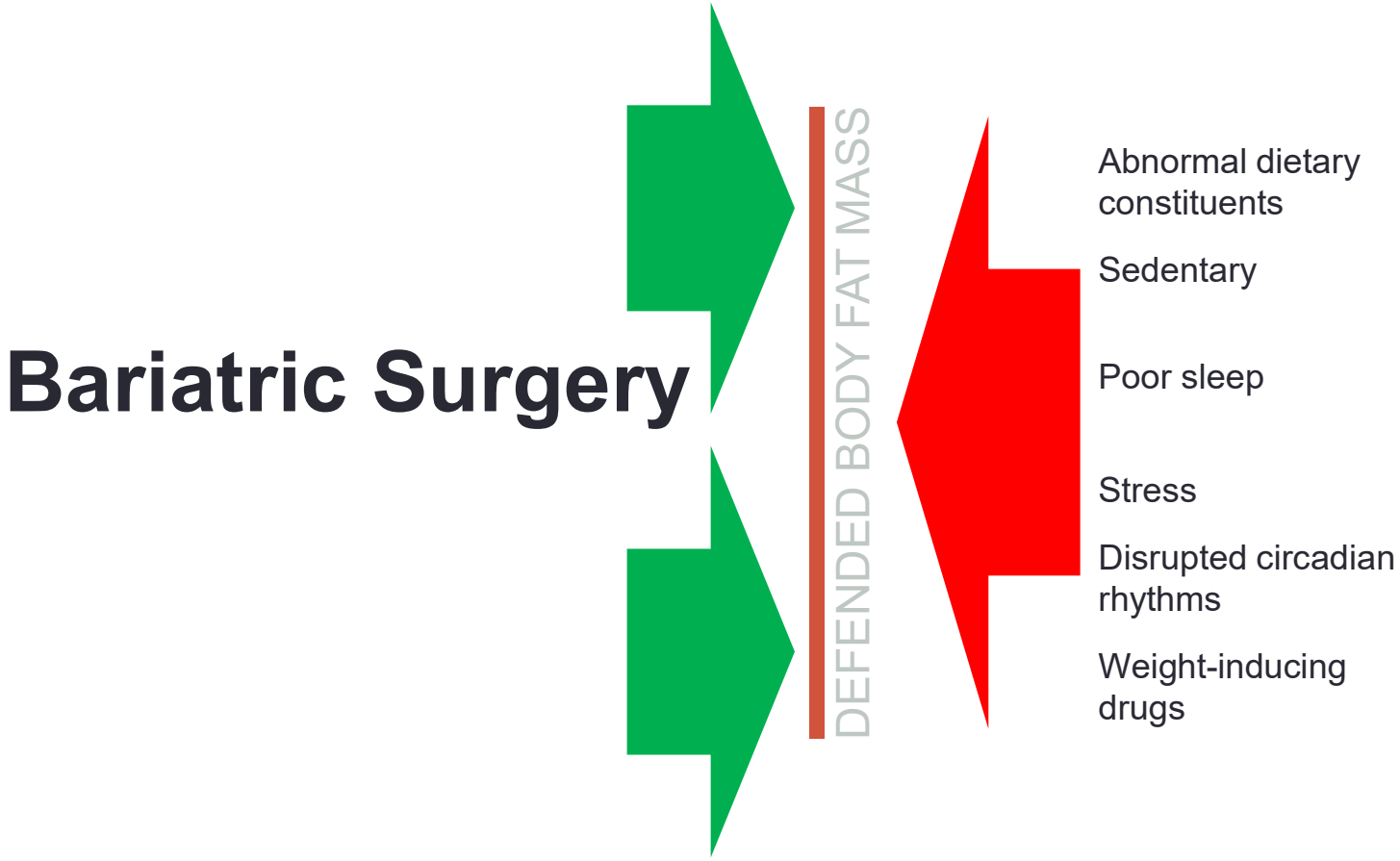




Not Currently FDA Approved for Weight Loss

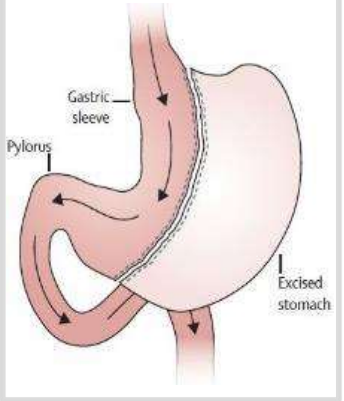
Jastreboff. NEJM. 2022;387(3):205.

Battle of **Physiologic** Forces that Influence Fat Mass

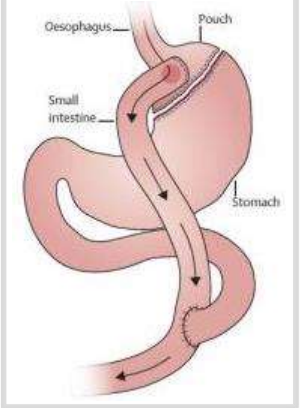


Most Common Bariatric Procedures

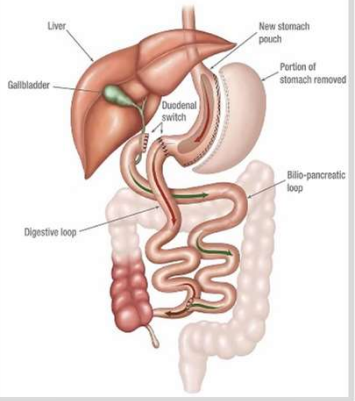
Sleeve Gastrectomy



Roux-en-Y Gastric Bypass



Duodenal Switch



98% performed laparoscopically
Average length of stay – 1.2 days

Madsbad S, et al. *Lancet Diabetes Endocrinol*. 2014;2(2):152-64.
ASMBS. Estimate of Bariatric Surgery Numbers, 2011-2017. http://asmbs.org/resources/estimate-of-bariatric-surgery-numbers. Accessed Sept 17, 2018.

Why does bariatric surgery work so well?

Food Intake

- Changes in hunger and fullness via enhanced satiety leading to decrease in calorie intake
- Mean caloric intake 600-700 one month postop to 1000-1800 after first year
- Average reduction of 1800 kcal per day from pre-op intake sustained for several years

Potential Mediators of Decreased Food Intake

- Increased transit of food into mid-gut through gastric pouch

Mediators for Food Preferences

- Taste function domains
- Sensory-discriminative *(stimulus identification)*
- Hedonic *(ingestive motivation) altered brain responsivity to high calorie food cues*
- Physiological *(digestive preparation)*

Hormonal

- GLP-1 and PYY increase
- Ghrelin decreases

Change in Gut Microbiota

- Short chain fatty acids – calorie extraction/signals

Energy Expenditure

- Increase/Decreased basal metabolic rate after bariatric surgery – in gut?

Food Preferences Change

- Dumping syndrome?
- Conditioned food avoidance?

Calorie Malabsorption

- Exclusion of 10% of the bowel after RYGB unlikely to result in malabsorption

Neural

- Vagal and partial vagal transection

Change in Bile Acids

- Partly responsible for intestinal hypertrophy, anorexigenic hormone secretion and alterations in gut microbiota; activation of FXR signaling

PATIENT CASE

Mrs. Jones

38-year-old woman with a history of depression, migraine headaches, premenstrual dysphoric disorder, seasonal allergies, who presents for routine medical follow-up. No significant change in health since last visit 6 months ago but is concerned about a 10-lb weight gain. She has been following a low-fat meal plan but not much physical activity.

Examination:

- Weight, 288 lbs
- Height, 67”
  BMI, 45 kg/m^2
- Waist circumference, 46 inches

Medications

- Propranolol 160 mg QD
- Paroxetine 37.5 mg QD
- Diphenhydramine 25 mg at bedtime

(+) ROS

- Occasional AM headaches and daytime fatigue

Labs

- Unremarkable except A1c is 6.4% and fasting insulin 79 mIU/L

Weight Loss Journey

Initial "**Physiologic"** Recommendations:

- Started Mediterranean-type meal plan instead of low-fat diet
- Screened and treated her OSA with CPAP
- Stopped paroxetine and diphenhydramine
- Started metformin XR
- Started bupropion ER/Naltrexone ER
- Increased physical activity
- Enrolled in bariatric surgery process

3 months:

- 5% weight loss
- Weight 274#

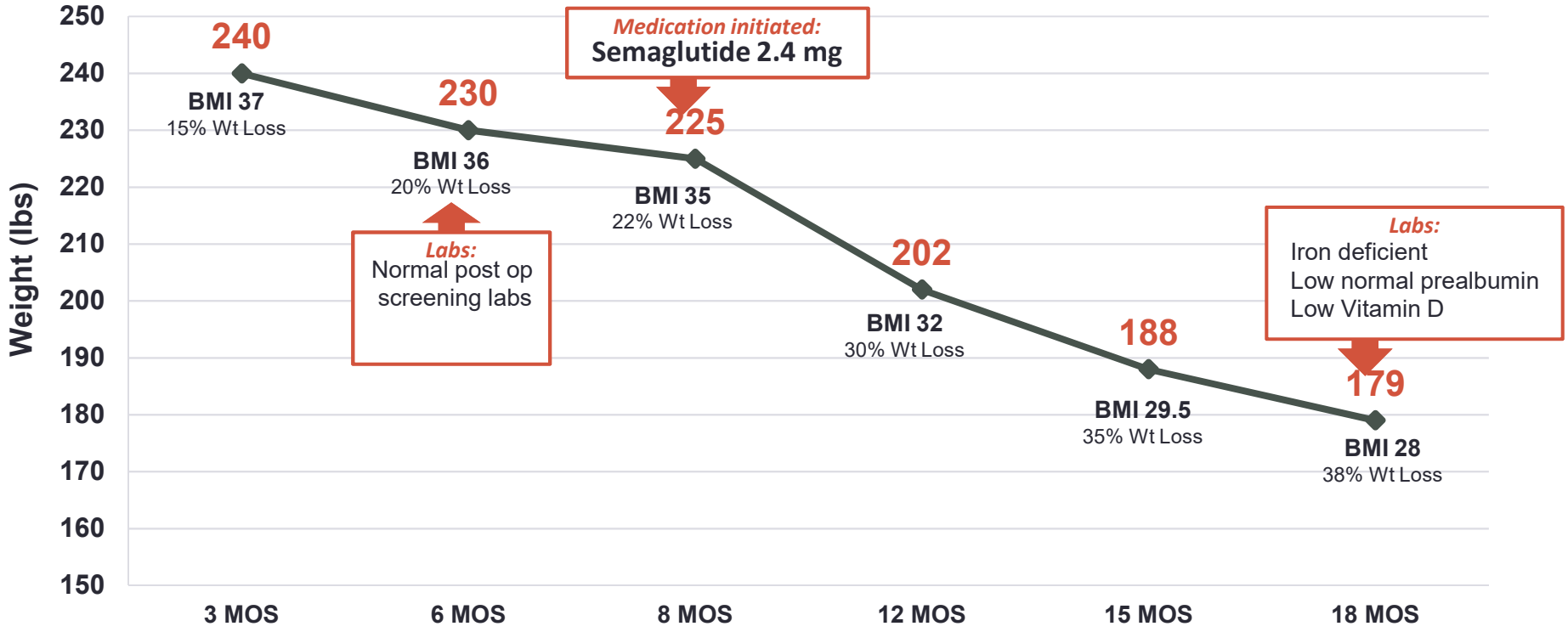
6 months:

- 8% weight loss / 265#
- **Underwent uncomplicated laparoscopic sleeve gastrectomy**

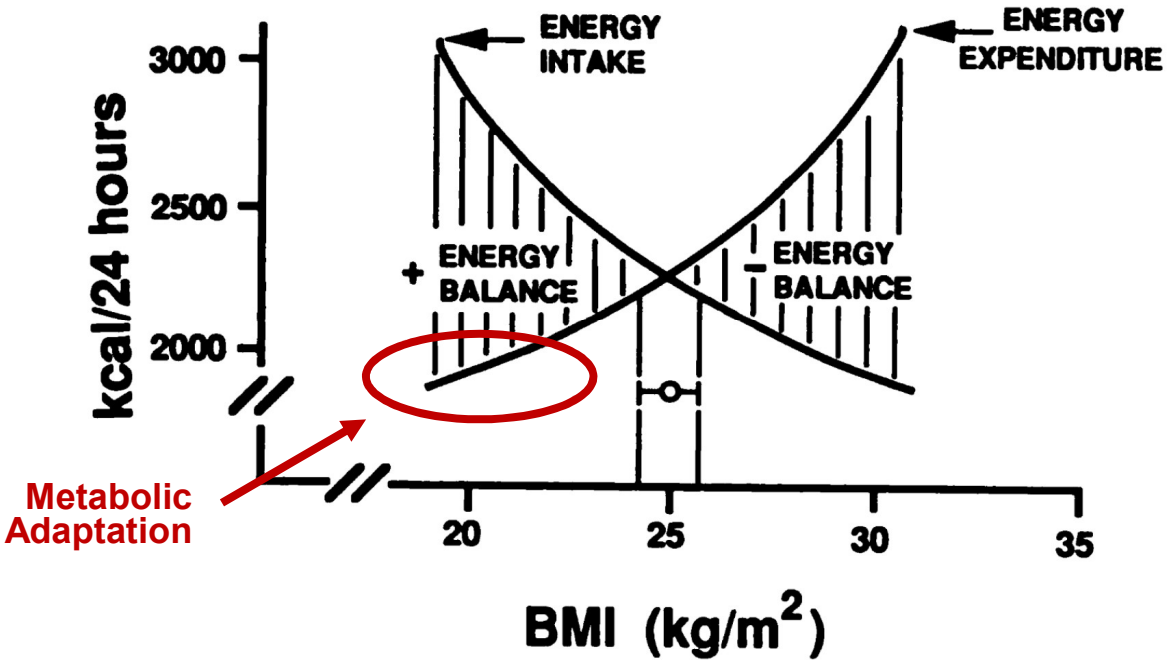
Post Surgery Weight Loss Journey (Con't)

- 3 months post op
  - 15% weight loss
  - 240#/BMI 37
- 6 months post op
  - 20% weight loss
  - 230# / BMI 36
  - **Labs obtained**
- 8 months post op
  - 22% weight loss
  - 225# / BMI 35
  - Initiated Semaglutide 2.4 mg
- 12 months post op
  - 30% weight loss
  - 202# / BMI 32
- 15 months post op
  - 35% weight loss
  - 188# / BMI 29.5
- 18month post op
  - 38% weight loss
  - 179# / BMI 28
  - **Labs obtained**

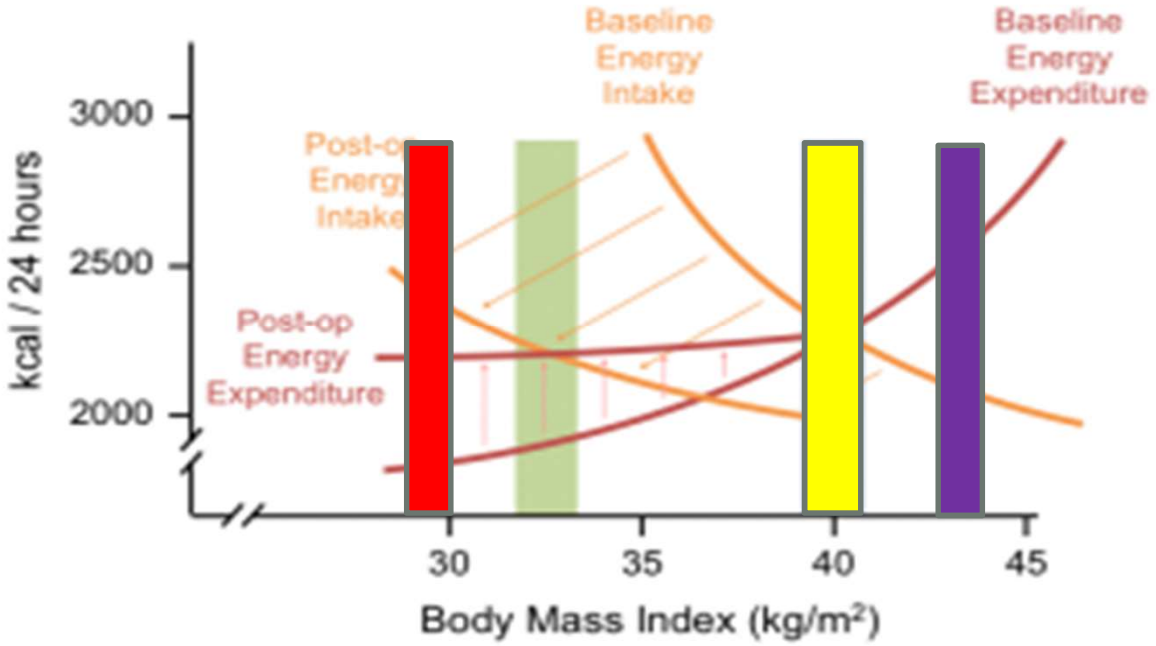
Mrs. Jones: **Post-op** Weight Journey



Defense of a Body Fat Storage "Set Point"



Physiologic Weight Loss:
Physiologic Lifestyle Modification, Effective Medications, Surgery



Conclusion:

- The disease of obesity is a major driver of cardiometabolic diseases
- A modest weight loss of at least 5-10% does have significant metabolic benefits but greater weight loss → greater benefit.
- Obesity is a dysregulation of energy balance which is a function of the brain
- Physiologic lifestyle Modification, effective AO medications, and bariatric surgery are often required for physiologic compensation to a **NEW DEFENDED FAT MASS**

Q & A

Roundtable Discussion on Keynote

1. What resonated with you most in Dr. Still's presentation?
2. What are you now rethinking given the information Dr. Still shared?
3. How are you incorporating therapeutic weight loss into your treatment plans? How do you get patient buy-in for the efficacy of existing obesity interventions?

Speaker: Patient Perspective

Patricia Nece

Breakout Session Overview

- These breakouts will run concurrently.
- The breakout room assignments are only suggestions.
- In fairness to the facilitators, once you've selected a breakout, we politely ask you to remain in that room.
- We encourage conversation from all!

Breakout Facilitators

Diabetes

Brian C. Jameson, DO
Endocrinologist
Geisinger Health System

Kidney Disease

Sandra J. Taler, MD
Consultant, Division of Nephrology/Hypertension,
Professor of Medicine,
College of Medicine,
Mayo Clinic

Cardiovascular

John Clark, MD, PhD
Associate Professor
UC San Diego Health

Suggested Breakout Assignments

Diabetes Breakout

Salon I

- Nkem O Akinsoto
- Fred Bloom
- Ken Bogenschutz
- Suelyn Boucree
- James Gaither
- Thomas Grace
- Insha Haque
- Leslie High
- Leon Jerrels
- Sonya Kokil Raikar
- Chalak Muhammad
- Richard Mulcahey
- Courtney Peters
- Kay Sadik
- Bruce Taylor

Kidney Breakout

Plaza C

- Robert Charles
- Dave Dolton
- Claire Grawburg
- Angie Griffith
- Victoria Harris
- Meredith Milligan
- Tesha Montgomery
- Sara Mukherjee
- Philip Oravetz
- Stacie Smith
- Laura Wilson
- Stephen Winn

Cardiovascular Breakout

Plaza D

- Alka Atal-Barrio
- Alexander Baer
- David Boyd
- Frank Colangelo
- Stephanie Copeland
- Rebecca Fitch
- Joel Ortiz
- Barbara Pritchard
- Crystal Redfern
- Christi Taylor
- Rachel Thomas
- Martine Thurin

Panel Discussion with Facilitators and Audience

John W. Kennedy, MD
President, AMGA Foundation
Chief Medical Officer, AMGA

Christopher M. Steer, Esq.
Founder & CEO
Steer, LLC

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Cardiovascular

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Associate Professor
UC San Diego Health

AC24 Preview & Action

Christopher M. Steer, Esq.

Founder & CEO

Steer, LLC

AC24 Preview & Action

Wednesday, April 10, 2024
10:30 am – 12:30 pm
Rosen Shingle Creek | Orlando, Florida

Tackling the Obesity Epidemic 2.0:
Implementing the Takeaways