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Foundations of Cardiometabolic Health Certification Course

Certified Cardiometabolic Health Professional (CCHP)

Obesity as a Disease

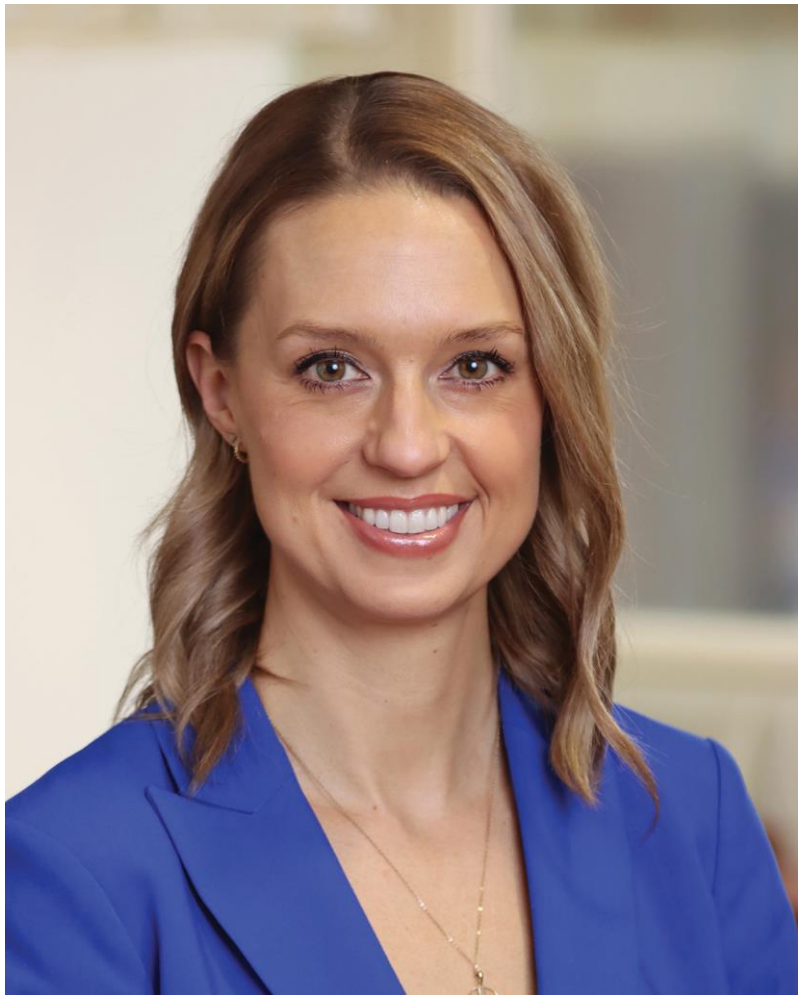
Karli Burridge, PA-C, MMS, FOMA

Owner: Gaining Health

Obesity Specialist: Ascension Weight Loss Solutions

Disclosures

- Advisor/ Consultant: Currax, Novo Nordisk
- Speaker: Currax, Vivus
- Owner: Gaining Health



Goals

- Explain the epidemiology of obesity and the impact of obesity on cardiometabolic health
- Discuss the prevalence and impact of weight bias and stigma on healthcare and health behaviors
- Describe the pathophysiology of obesity
- Demonstrate how to initiate the discussion and counsel patients on obesity and health behavior change using evidence-based behavioral strategies

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Obesity: Definitions & Epidemiology

Karli Burridge, PA-C, MMS, FOMA

Gaining Health

Ascension

Defining Obesity

- WHO:
  - “Overweight and obesity are defined by excessive fat accumulation that presents a risk to health”.
- Obesity Medicine Association:
  - “Obesity is defined as a chronic, progressive, relapsing, and treatable multi-factorial, neurobehavioral disease, wherein an increase in body fat promotes adipose tissue dysfunction and abnormal fat mass physical forces, resulting in adverse metabolic, biomechanical, and psychosocial health consequences.”

World Health Organization: https://www.who.int/health-topics/obesity#tab=tab_1 (Accessed 12/07/2021)
Bays HE, McCarthy W, Burridge K et al. Obesity Algorithm eBook, presented by the Obesity Medicine Association. www.obesityalgorithm.org. 2021. https://obesitymedicine.org/obesity-algorithm/ (Accessed = 12/07/2021)

Classifying Obesity

NIH and the World Health Organization (WHO) for White, Hispanic, and Black individuals:

- Overweight (pre-obesity) – BMI ≥25 to 29.9 kg/m²
- Obesity – BMI ≥ 30 kg/m²
- Waist circumference ≥35 inches women
- Waist Circumference ≥ 40 inches men

Asian and South Asian population

- Overweight (pre-obesity) - BMI ≥23 - 24.9 kg/m²
- Obesity - BMI ≥ 25 kg/m²
- Waist Circumference ≥31.5 inches women
- Waist Circumference ≥35 inches men

Weir CB, Jan A. BMI Classification Percentile And Cut Off Points. [Updated 2021 Jun 29]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-. Available from:https://www.ncbi.nlm.nih.gov/books/NBK541070/

Classifying Obesity in Children

BMI (kg/m^2)	Classification
≥ 95th percentile	Obesity
85th to < 95th percentile	Pre-Obesity (Overweight)
5th to < 85th percentile	Healthy Weight
< 5th percentile	Underweight

https://www.cdc.gov/nccdphp/dnpao/growthcharts/training/bmiage/page4.html. Accessed 12/07/2021

Worldwide Statistics

- Obesity rates have tripled worldwide since 1976
- 2016:
  - 39% adults with pre-obesity (overweight)- 1.9 billion adults
  - 13% obesity- 650 million adults
- 2020:
  - 39 million children under the age of 5 with pre-obesity or obesity
  - Over 340 million children and adolescents (5-19) with pre-obesity or obesity
  - Obesity rates children have increased from 4% in 1975 to over 18% in 2016,
- 2035 Global Projections:
  - Pre-obesity: >50%
  - Obesity: >25% with obesity (>4 billion people)
  - Obesity in boys: 20%, Obesity in girls: 18% (>400 million children with obesity)
- Obesity kills more people than underweight
- Undernutrition and obesity often co-exist in many low-and middle-income countries (“double burden” of malnutrition).

World Obesity Federation, World Obesity Atlas 2023. https://data.worldobesity.org/publications/?cat=19

World Health Organization: https://www.who.int/health-topics/obesity#tab=tab_1 (Accessed 12/07/2021)

United States of America

ADULTS WITH OBESITY 2035
58%
VERY HIGH

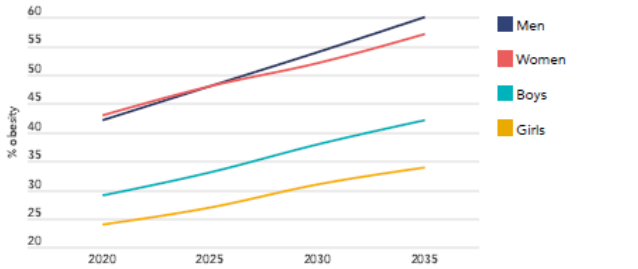
ANNUAL INCREASE IN ADULT OBESITY 2020–2035
2.1%
HIGH

ANNUAL INCREASE IN CHILD OBESITY 2020–2035
2.4%
HIGH

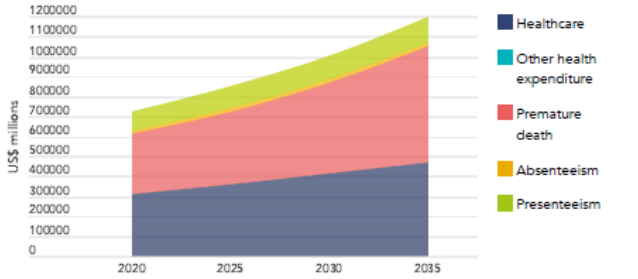
OVERWEIGHT IMPACT ON NATIONAL GDP 2035
4.0%
VERY HIGH

GLOBAL PREPAREDNESS RANKING
41/183
FAIRLY GOOD

PROJECTED TRENDS IN THE PREVALENCE OF OBESITY (BMI ≥30kg/m²)



PROJECTED ECONOMIC IMPACT OF OVERWEIGHT (BMI ≥25kg/m²)



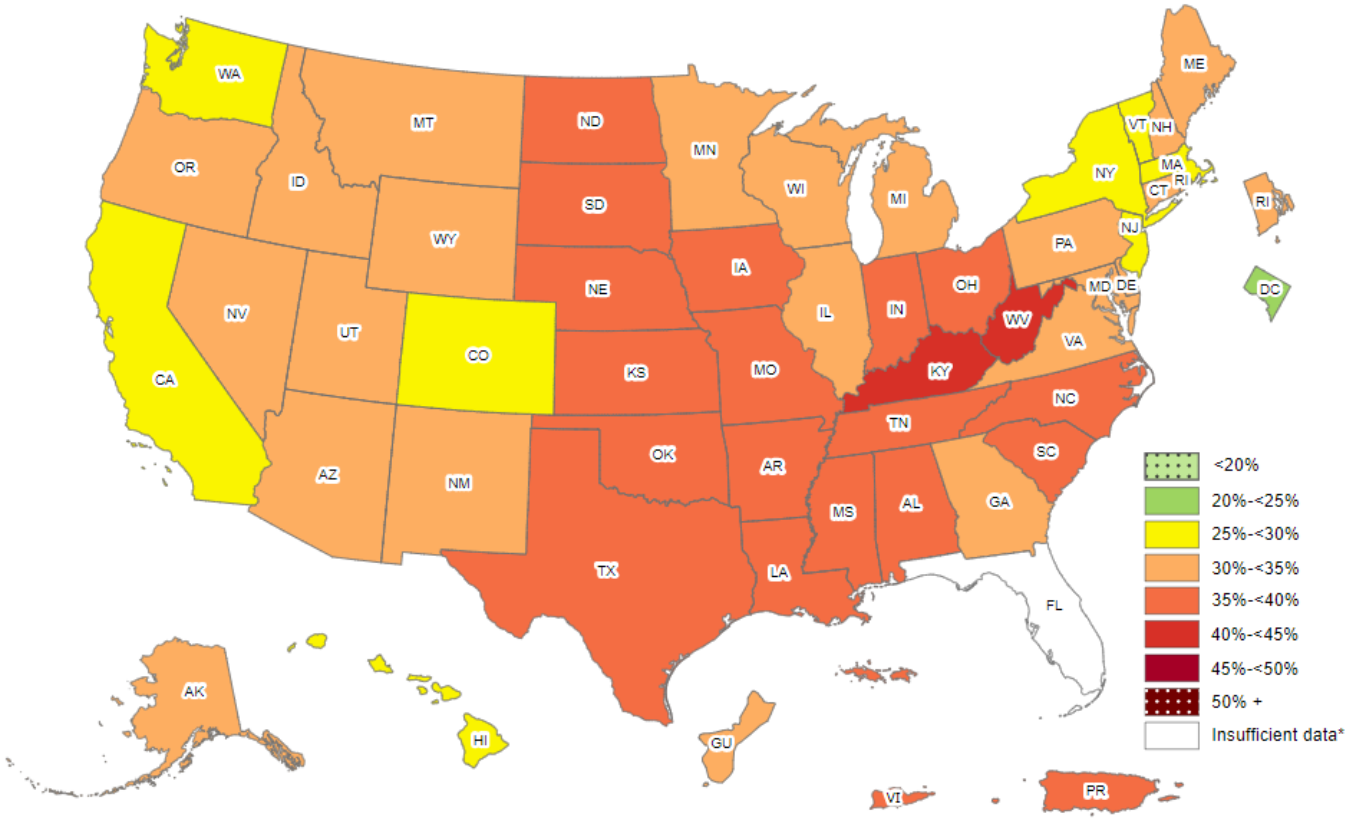
IMPACT OF OVERWEIGHT (BMI ≥25kg/m²) 2020–2035

	Healthcare impact of BMI ≥25kg/m², US$ million	Total economic impact of BMI ≥25kg/m², US$ million	Estimated GDP US$ billion	Impact of BMI ≥25kg/m² on GDP
2020	312,964	726,152	20,645	3.5%
2025	360,101	850,469	23,862	3.6%
2030	411,595	1,005,286	26,623	3.8%
2035	467,470	1,198,463	30,205	4.0%

World Obesity Federation, World Obesity Atlas 2023. https://data.worldobesity.org/publications/?cat=19

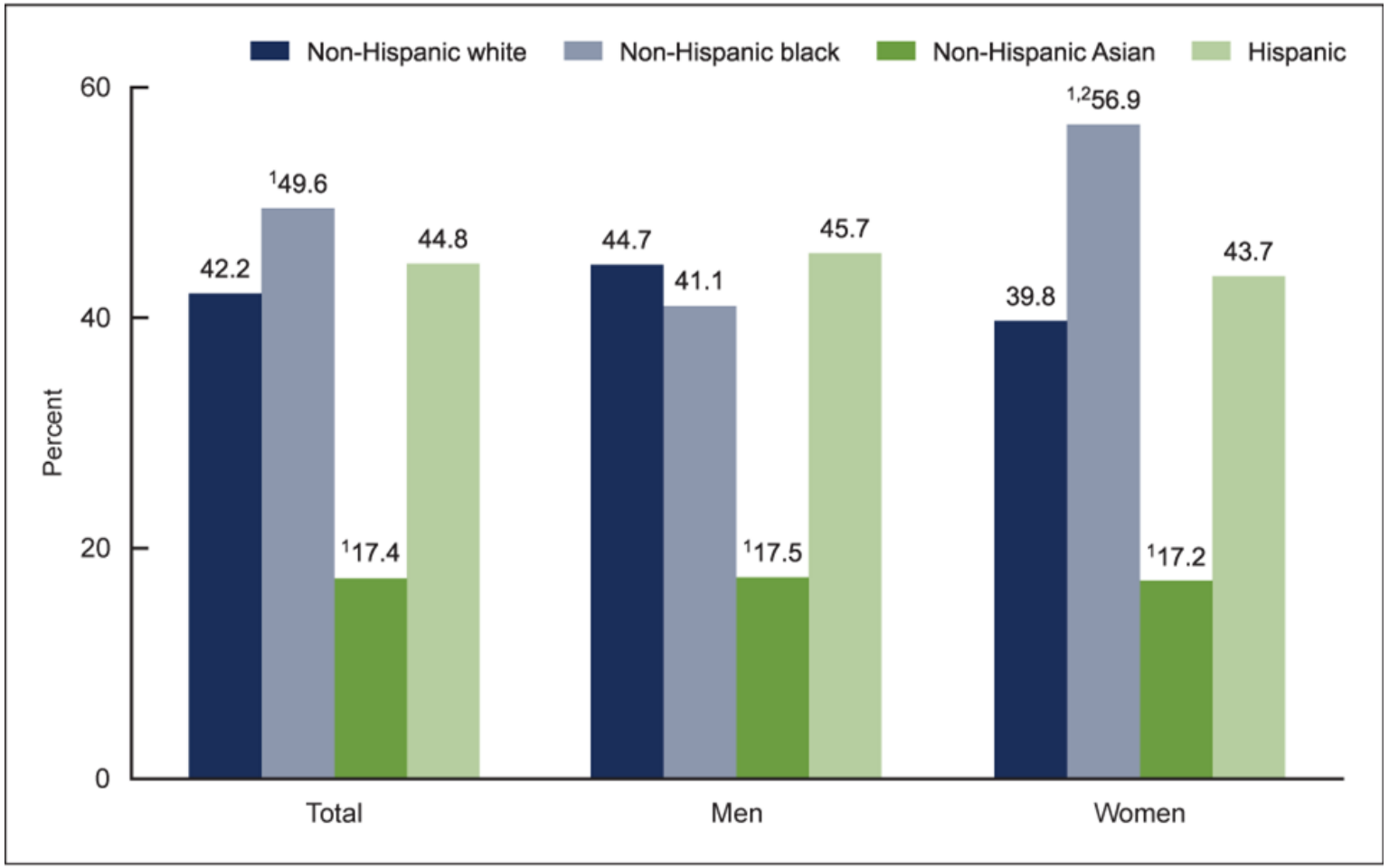
Prevalence† of Self-Reported Obesity Among U.S. Adults by State and Territory, BRFSS, 2021

†Prevalence estimates reflect BRFSS methodological changes started in 2011. These estimates should not be compared to prevalence estimates before 2011.



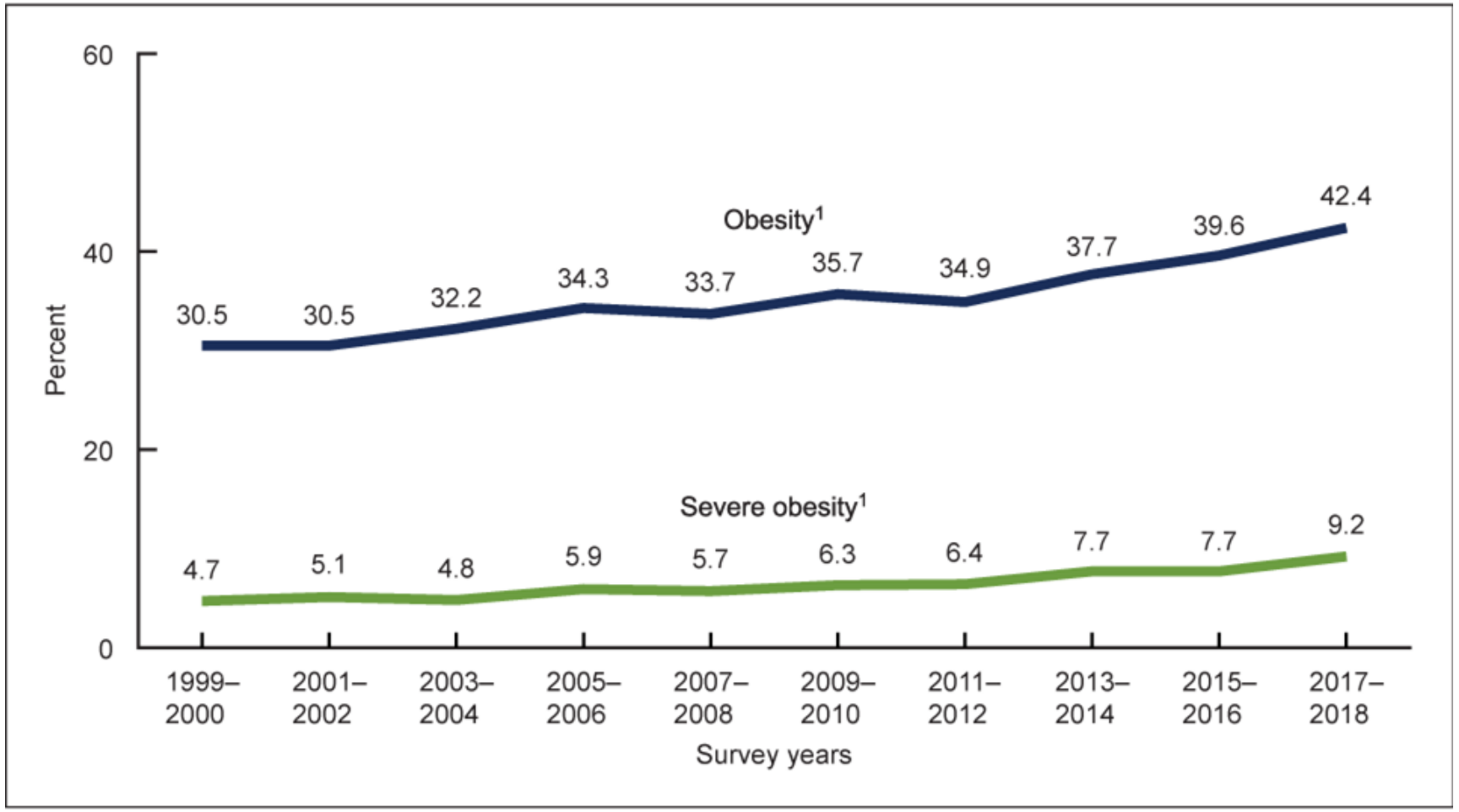
CDC Adult Obesity Prevalence Maps. https://www.cdc.gov/obesity/data/prevalence-maps.html (accessed 3/21/23)

Age-adjusted prevalence of obesity among adults aged 20 and over, by sex and race and Hispanic origin: The United States, 2017-2018



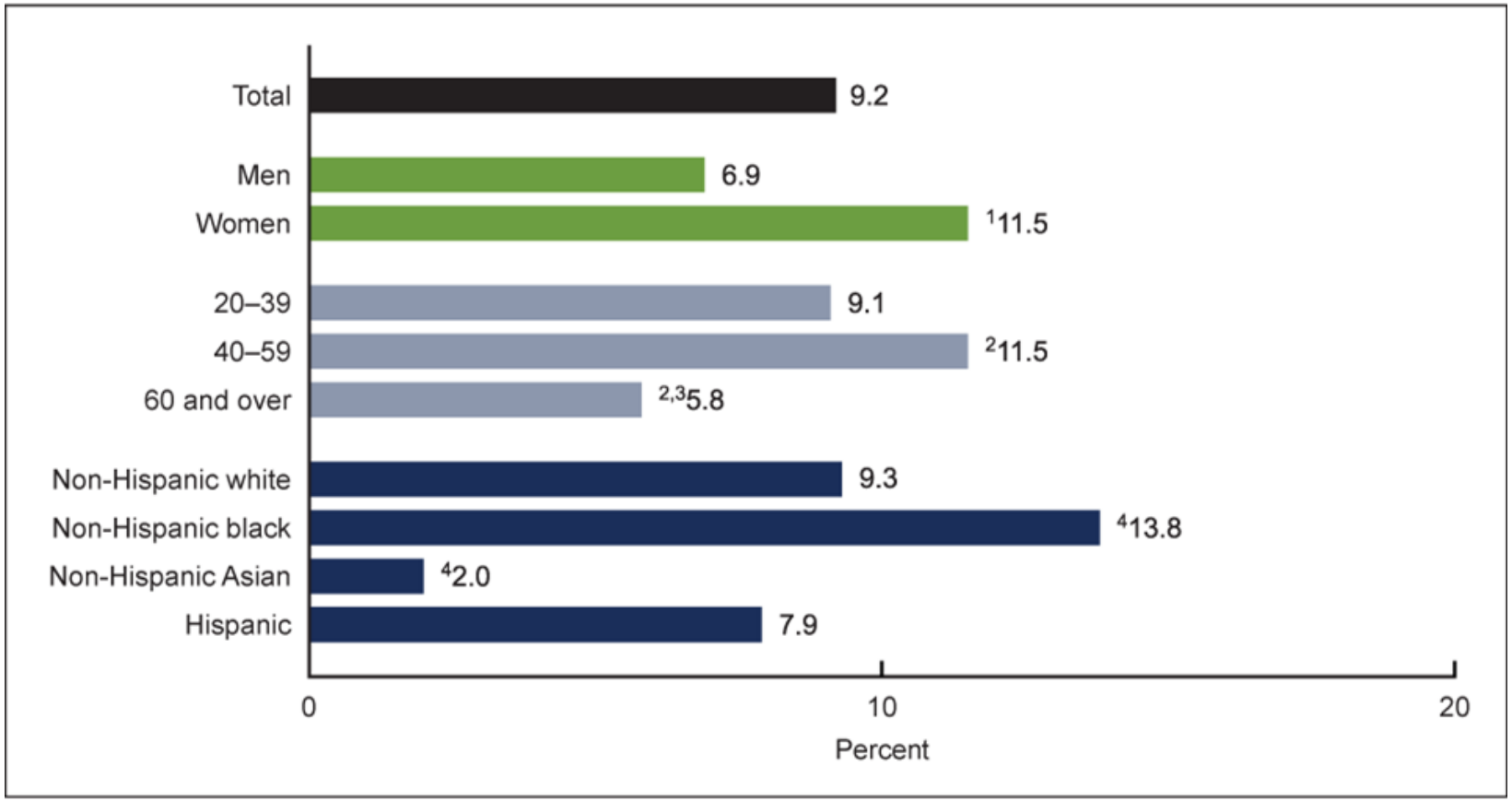
[1]Significantly different from all other race and Hispanic-origin groups.
[2]Significantly different from men for same race and Hispanic-origin group.
NOTES: Estimates were age adjusted by the direct method to the 2000 U.S. Census population using the age groups 20–39, 40–59, and 60 and over. Access data table for Figure 2 at: https://www.cdc.gov/nchs/data/databriefs/db360_tables-508.pdf#2.
SOURCE: NCHS, National Health and Nutrition Examination Survey, 2017–2018.

Trends in age-adjusted obesity and severe obesity prevalence among adults aged 20 and over: the United States, 1999-2000 through 2017-18



[1]Significant linear trend.
NOTES: Estimates were age adjusted by the direct method to the 2000 U.S. Census population using the age groups 20–39, 40–59, and 60 and over. Access data table for Figure 4 at: https://www.cdc.gov/nchs/data/databriefs/db360_tables-508.pdf#4.
SOURCE: NCHS, National Health and Nutrition Examination Survey, 1999–2018.

Age-adjusted prevalence of severe obesity among adults aged 20 and over, by sex, age, and race and Hispanic origin: United States, 2017-18



[1]Significantly different from men.
[2]Significantly different from adults aged 20–39.
[3]Significantly different from adults aged 40–59.
[4]Significantly different from all other race and Hispanic-origin groups.
NOTES: Estimates for adults aged 20 and over were age adjusted by the direct method to the 2000 U.S. Census population using the age groups 20–39, 40–59, and 60 and over. Crude estimates are 9.0% for total, 6.8% for men, and 11.1% for women. Access data table for Figure 3 at: https://www.cdc.gov/nchs/data/databriefs/db360_tables-508.pdf#3.
SOURCE: NCHS, National Health and Nutrition Examination Survey, 2017–2018.

Childhood Obesity in the US: 2017-2018

Age	% obesity rates
2-19 years	19.3%
2-5 years	13.4%
6-11 years	20.3%
12-19 years	21.2%

Ethnicity	% Obesity Rates
Hispanic	25.6%
Non-Hispanic Black	24.2%
Non-Hispanic White	16.1%
Non-Hispanic Asian	8.7%

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Obesity Bias and Stigma

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Gaining Health, Enara Health

ACTION Study: People with Obesity

- PwO (people with obesity)
  - 65% recognize obesity as a disease
  - Only 50% saw themselves as having obesity
  - 54% worried that their weight might affect their future health
  - 82% felt “completely responsible” for their own weight loss
- Gap exists between recognizing obesity as a disease and treating it as such

Lee Kaplan et al. Perceptions of Barriers to Effective Obesity Care: Results from the National ACTION Study. Obesity Volume 6, Issue 1 January 2018. https://doi.org/10.1002/oby.22054

ACTION Study: Health Care Providers

Only 55% of patients with obesity were given the diagnosis of obesity.

Only 24% of those people formally diagnosed with obesity had a follow-up appointment scheduled.

72% of health care providers feel poorly prepared by their medical training and report insufficient knowledge about nutrition.

Lee Kaplan et al. Perceptions of Barriers to Effective Obesity Care: Results from the National ACTION Study. Obesity Volume 6, Issue 1 January 2018. https://doi.org/10.1002/oby.22054

Weight Bias and Stigma in Healthcare

- Study 2006: 2,449
  - 67% women and 64% of men with obesity experienced weight bias from their doctors
    - Most frequent source of stigma for women, 2nd most frequent source of stigma for men
- Far-reaching consequences: health, social, psychological, economic
  - 52% of women reported avoidance of preventative screening or delaying medical care due to their BMI
  - 79% identified eating as a common coping strategy to deal with obesity stigma

Puhl & Brownell. (2006). Obesity.
Puhl, Luedicke, & Heuer. (2011). Journal of School Health
Hayward, Vartanian, Pinkus. (2018) Obesity
Fruh et al. (2016) J Nurse Pract.

Bias and Stigma: Internalization

- Women who internalized experiences of weight stigma and blamed themselves engaged in more frequent binge eating, emotional eating, loss-of-control eating
- Impairs weight loss efforts
  - Higher calorie intake
  - Higher program attrition
  - Less weight loss
  - Avoidance of physical activity

Image: © Obesity Action Coalition

Puhl, Moss-Racusin, & Schwartz, 2007 ;Schvey, Puhl, & Brownell, 2011;Carels et al., 2009 Wott & Carels, 2010;O'Brien at al , 2016

Two Crucial Steps to Overcome Weight Bias and Stigma

1. Understanding the Pathophysiology of Obesity and explaining it to our patients

"People often reserve their harshest judgments for those conditions about which the least is known"

~Dr. Friedman

2. Overcoming internalized weight bias (shame) through empathy

"Empathy is the antidote to shame"

~ Brene Brown

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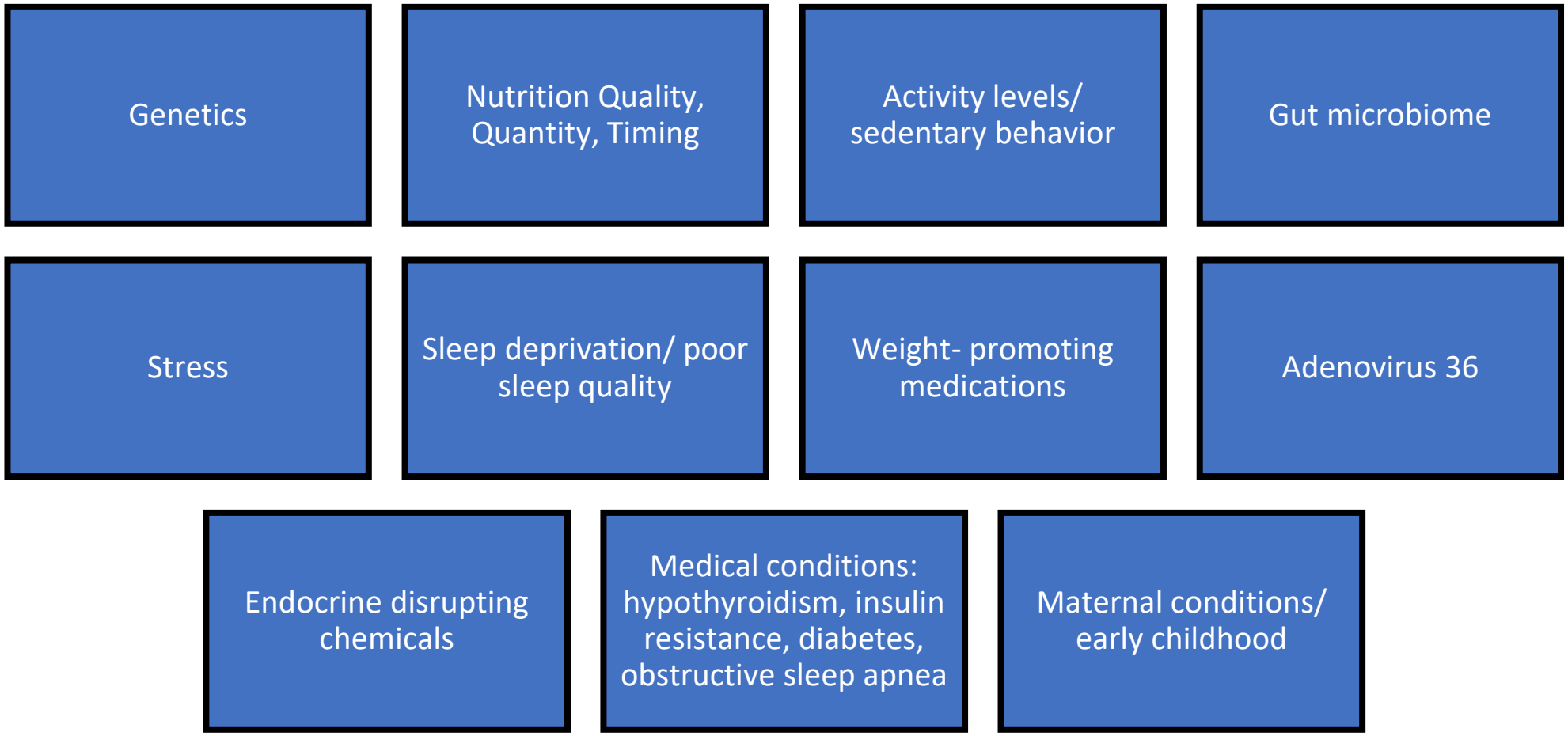
Pathophysiology of Obesity: An Overview

Karli Burridge, PA-C, MMS, FOMA

Gaining Health, Enara Health

Obesity is a Dysregulation of the Energy Regulatory System

Factors That Can Contribute to the Development of Obesity

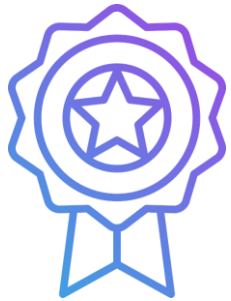


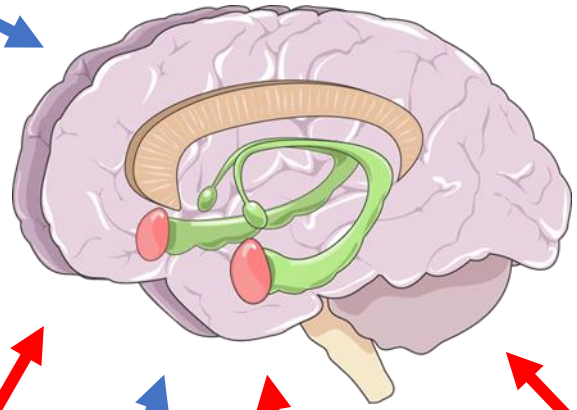
NPY and AgRP Neurons in hypothalamus Increase hunger and cravings

Dopamine
Serotonin
Opioids

Eating Behaviors
Metabolic Rate
Energy Expenditure

POMC and CART neurons in hypothalamus Reduce hunger and cravings (increase satiety)





Ghrelin

GLP-1
CCK

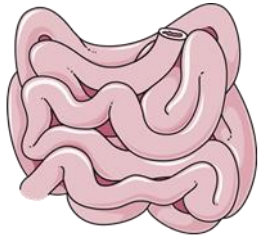
Myokines

OXM
PYY

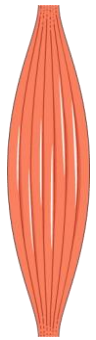
Insulin
Amylin

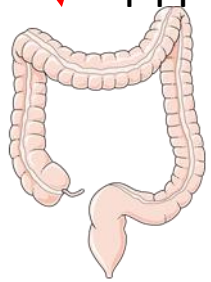
Leptin

Stomach

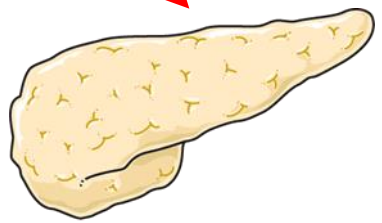


Small intestine

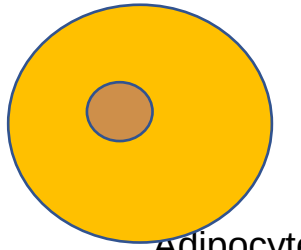




Large intestine



Pancreas



Adipocytes

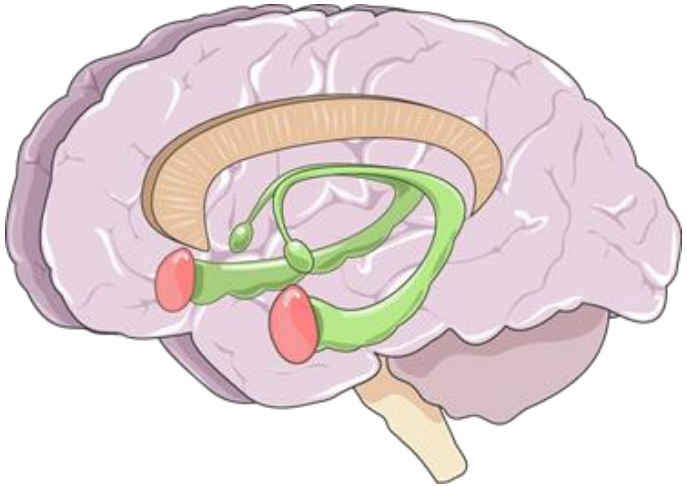
Ghrelin

- The “hunger hormone”
- Produced by gastric fundus and proximal small intestines
- Secreted when stomach is empty
- Distention of stomach inhibits ghrelin secretion
- Levels drop after meal, **but less so in some patients with obesity**

Kim G. Expert Rev Clin pharmacol. 2011

Ghrelin

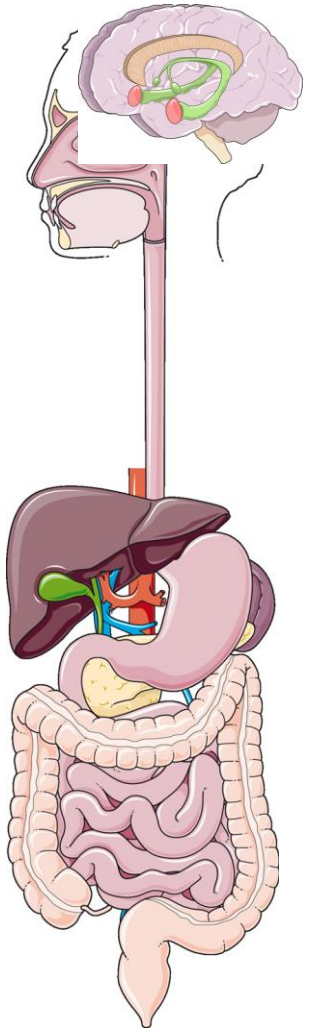
- Stimulates NPY/AgRP neurons in the arcuate nucleus of the hypothalamus
- Reaches brain via vagus nerve and nucleus tractus solitarius
- Stimulates food intake
- Stress and sleep deprivation increase ghrelin
- Sleeve gastrectomy decreases ghrelin
- Prader-Willi syndrome: hypersecretion of ghrelin, causing uncontrolled appetite, hyperphagia, and severe obesity



Kim G. Expert Rev Clin pharmacol. 2011

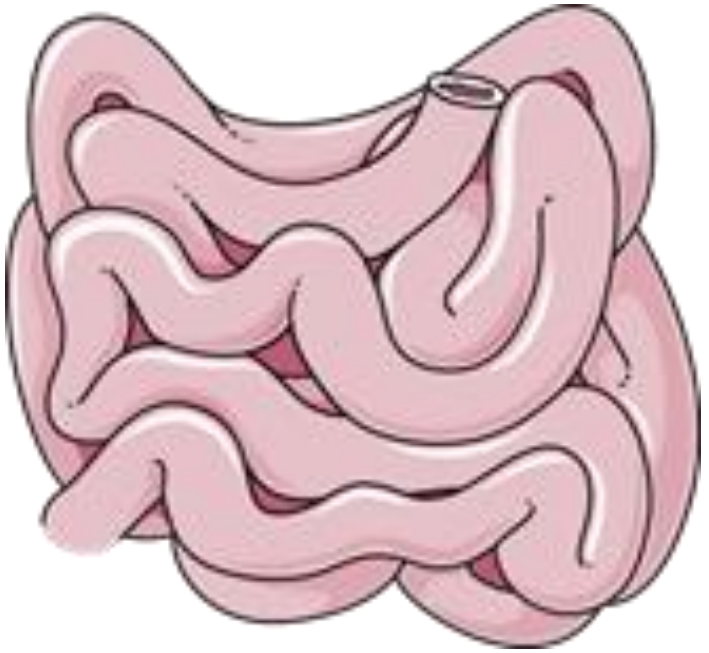
Satiety Hormones Secreted by the Gut

- Short acting
- Signal satiety from gut to brain via vagus nerve
- Several hormones
    1. CCK (cholecystokinin)
    2. GLP-1 (glucagon-like peptide-1)
    3. OXM (oxyntomodulin)
    4. PYY (peptide YY)



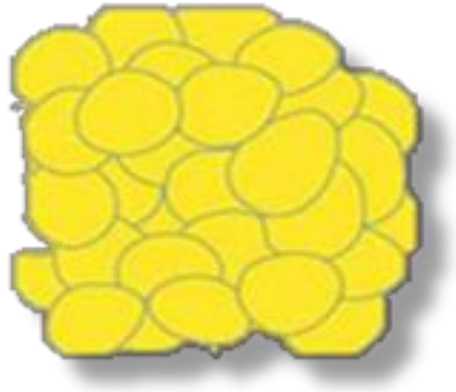
GLP-1 (Glucagon-Like Peptide-1)

- Short acting (half-life: 5 minutes)
- Secreted by L cells of distal small intestines and colon in response to carbohydrate ingestion
- Delays gastric emptying
- Reduces hepatic gluconeogenesis
- Glucose-dependent insulin secretion
- Induces satiety
- Levels are decreased in obesity, pre-diabetes, and type 2 diabetes
- Levels are increased after metabolic and bariatric surgery (MBS)



Leptin

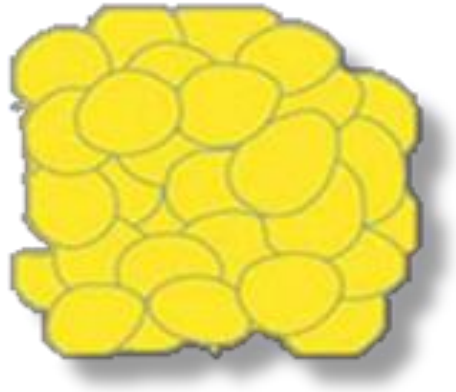
- Secreted by white adipose tissue adipocytes
- Primary hormone for long-term adiposity control and energy balance
- Metabolic signal of energy sufficiency
- Proportional to the level of adiposity
- Stimulates POMC neurons in hypothalamus; inhibits NPY/AgRP pathway (promoting weight loss and inhibiting weight gain)
- Levels decrease with weight loss



Perry & Yang. Appetite Regulation and Weight Control. Nutrition and Diabetes 2012

Leptin (cont.)

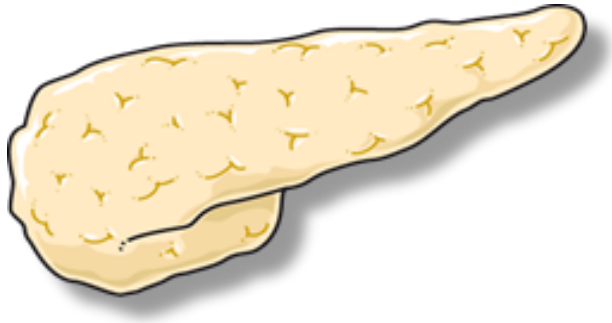
- Genetic leptin deficiency increases obesity
- Leptin receptor dysfunction increases obesity
- Most patients with obesity have **high** leptin…
- Leptin resistance!



Perry & Yang. Appetite Regulation and Weight Control. Nutrition and Diabetes 2012

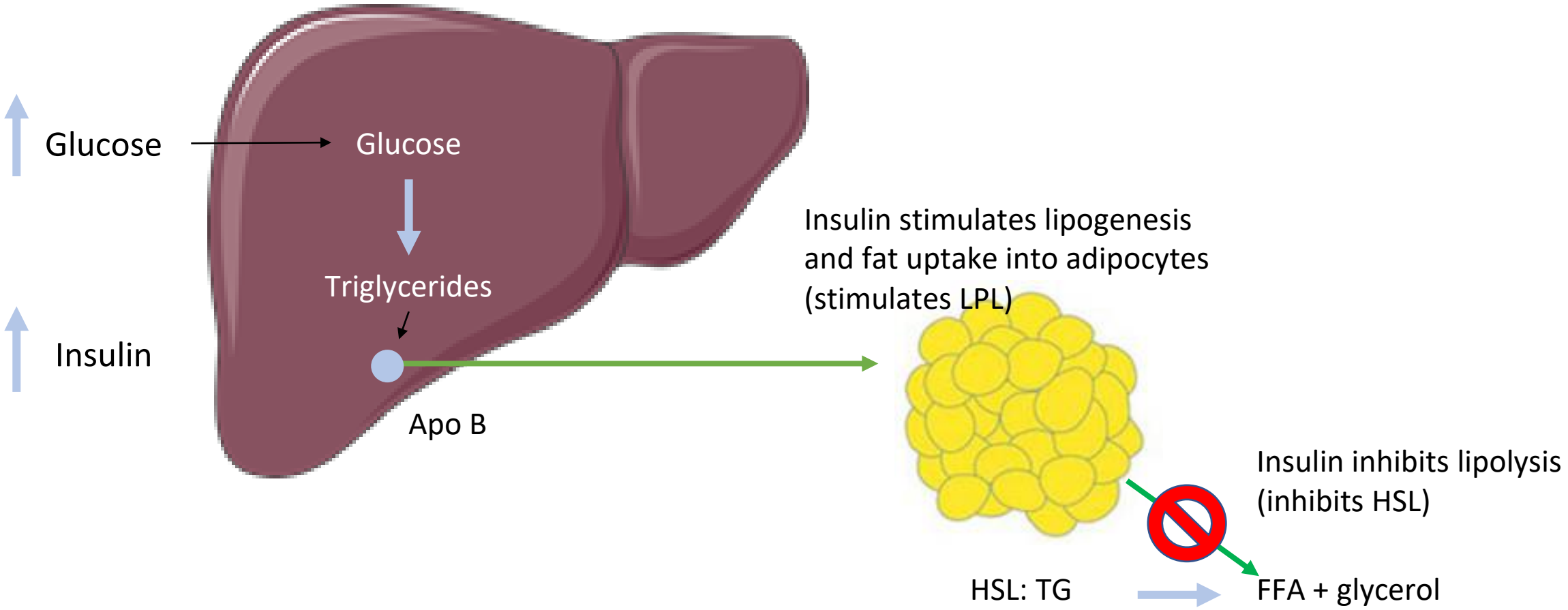
Insulin

- Secreted by beta-cells of pancreas
- Less potent than leptin, but also inhibits AgRP neurons, thus inducing satiety
- Many patients with obesity have insulin resistance, attenuating the appetite suppressing response of insulin



Perry & Yang. Appetite Regulation and Weight Control. Nutrition and Diabetes 2012

Insulin Controls Lipogenesis and Lipolysis

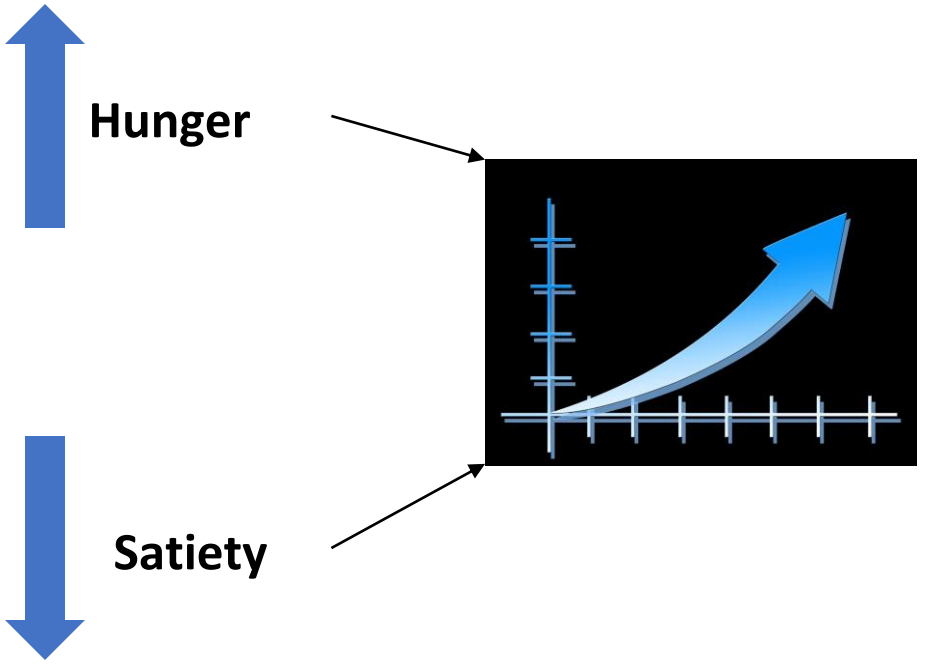


There are Many Forms of Obesity

- Some individuals with obesity have abnormal appetite hormone signaling which can result in hunger that is out of proportion to actual energy needs
- Some individuals with obesity have an overactive reward center
- Some individuals with obesity have inflammation or damage to the energy regulatory center in the brain (hypothalamus)
- Some people with obesity have a lower-than-expected metabolism
- There are MANY subtypes of obesity and MANY factors that can contribute to the development of obesity

Biologic Adaptations to Weight Reduction

- With nonsurgical weight reduction:
  - Disproportional ghrelin increases: **increased hunger**
  - Disproportional leptin, insulin, CCK, PYY, or amylin decreases: **decreased satiety**
  - These effects persists for over a year, even if weight is regained!
- Leads to ***increased appetite***, and the frequent ***weight regain*** commonly seen in weight loss with lifestyle changes alone



Sumithran, P. (2011). The New England Journal of Medicine, 365(17), 1597–1604.
Wadden, T. A. et al. (2011). Obesity, 19(10), 1987–1998.

Effects of Dietary Weight Loss on Gut Hormones

Hormone	Overall Effect of Energy Restriction on Circulating Levels	Specific diets/ circumstances
Ghrelin	Increase	No change or decrease during ketosis
GLP-1	Decrease	No change in low carbohydrate diet
PYY	Decrease/ No change	
CCK	Decease	
Amylin	Decrease	

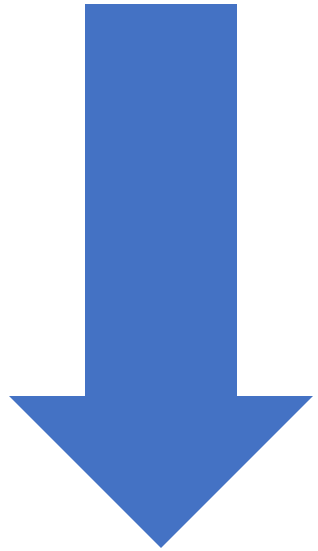
Table adapted from Busetto et al. Mechanisms of Weight Regain. European Journal of Internal Medicine Jan 2021

Metabolic Adaptation Drives Weight Regain

- The metabolic handicap: reduction in metabolism which is disproportionate to weight reduction
- Metabolism decreases 15% *beyond* expected (avg. 300 kcals)

- ↓ SNS activity
- ↓ Body mass
- ↑ Muscle efficiency
- ↑ Energy efficiency

Decreased metabolism



- This “metabolic handicap” persists for many years and drives weight regain

Melby, C. L., Paris, H.L., Foright, R. M., & Peth, J. (2017). Attenuating the biologic drive for weight regain following weight loss: Must what goes down always go back up? Nutrients, 9, 468.
Busetto et al. Mechanisms of Weight Regain. European Journal of Internal Medicine Jan 2021

Overcoming Biology

- Evidence- Based tools available to overcome biologic adaptations that drive weight re-gain:
  - Specific Nutrition Plans
  - Specific Physical Activity Plans
  - Stress Management and Sleep optimization
  - Treatment of underlying medical conditions/ management of weight promoting medications
  - Prescription Weight Loss (anti-obesity) Medications
  - Bariatric Devices/ endoscopic procedures
  - Metabolic and Bariatric Surgery

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Impact of Obesity on Cardiometabolic Health

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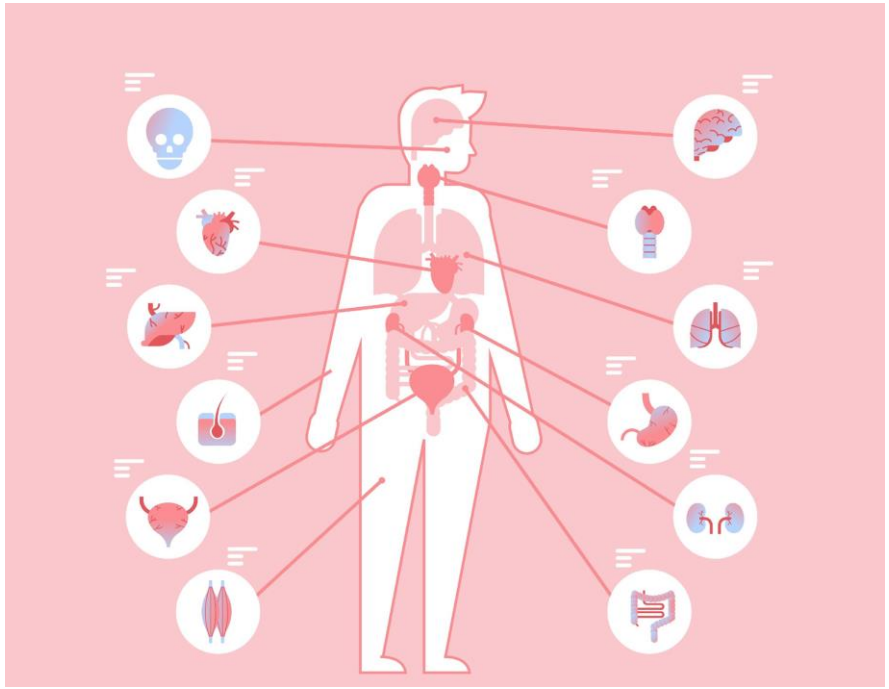
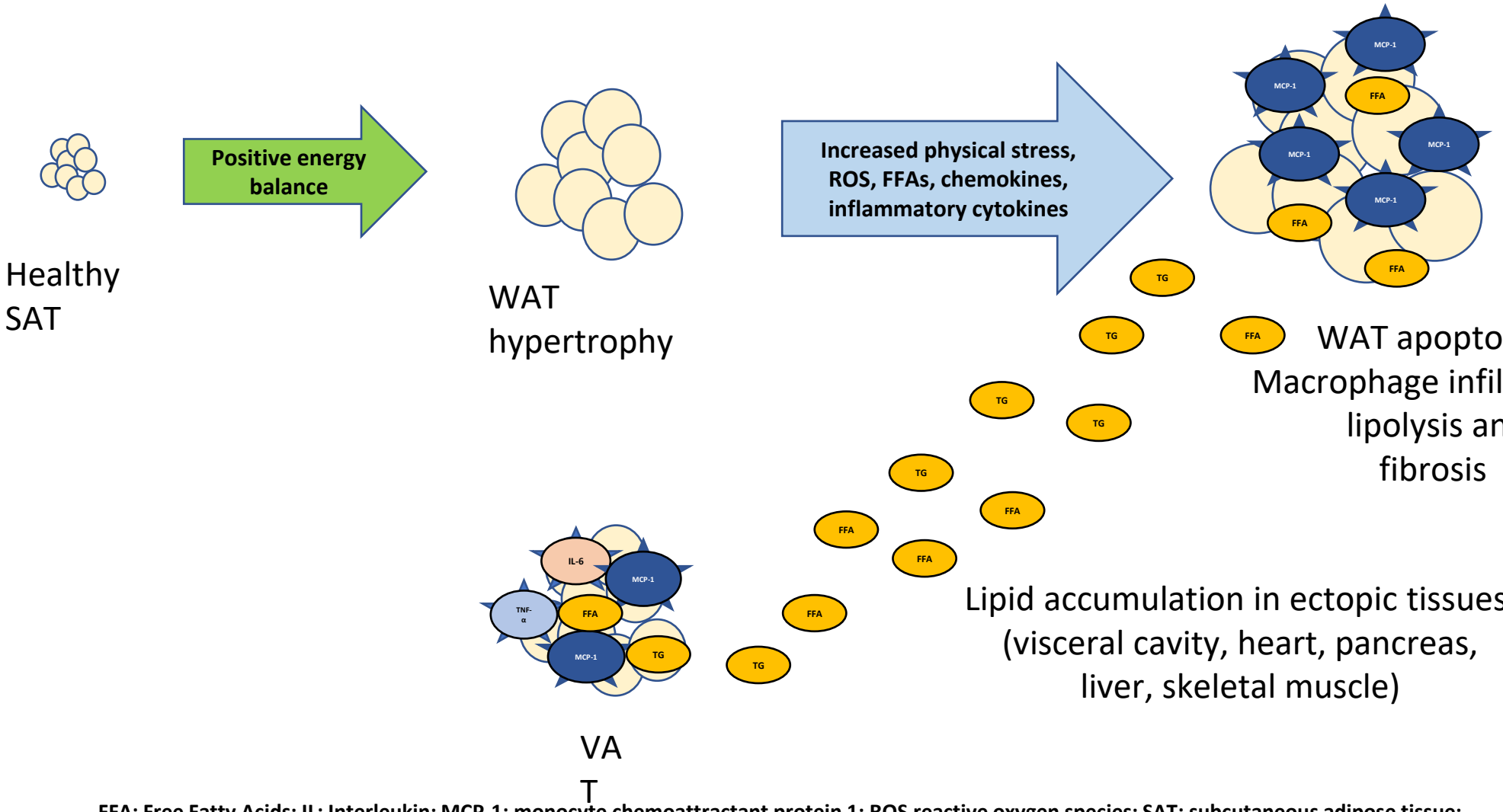
There are 236+ Potential Medical Complications of Obesity



- Metabolic
- Biomechanical
- Immunologic
- Psychologic
- Reproductive
- 13 types of cancers

- WHY????

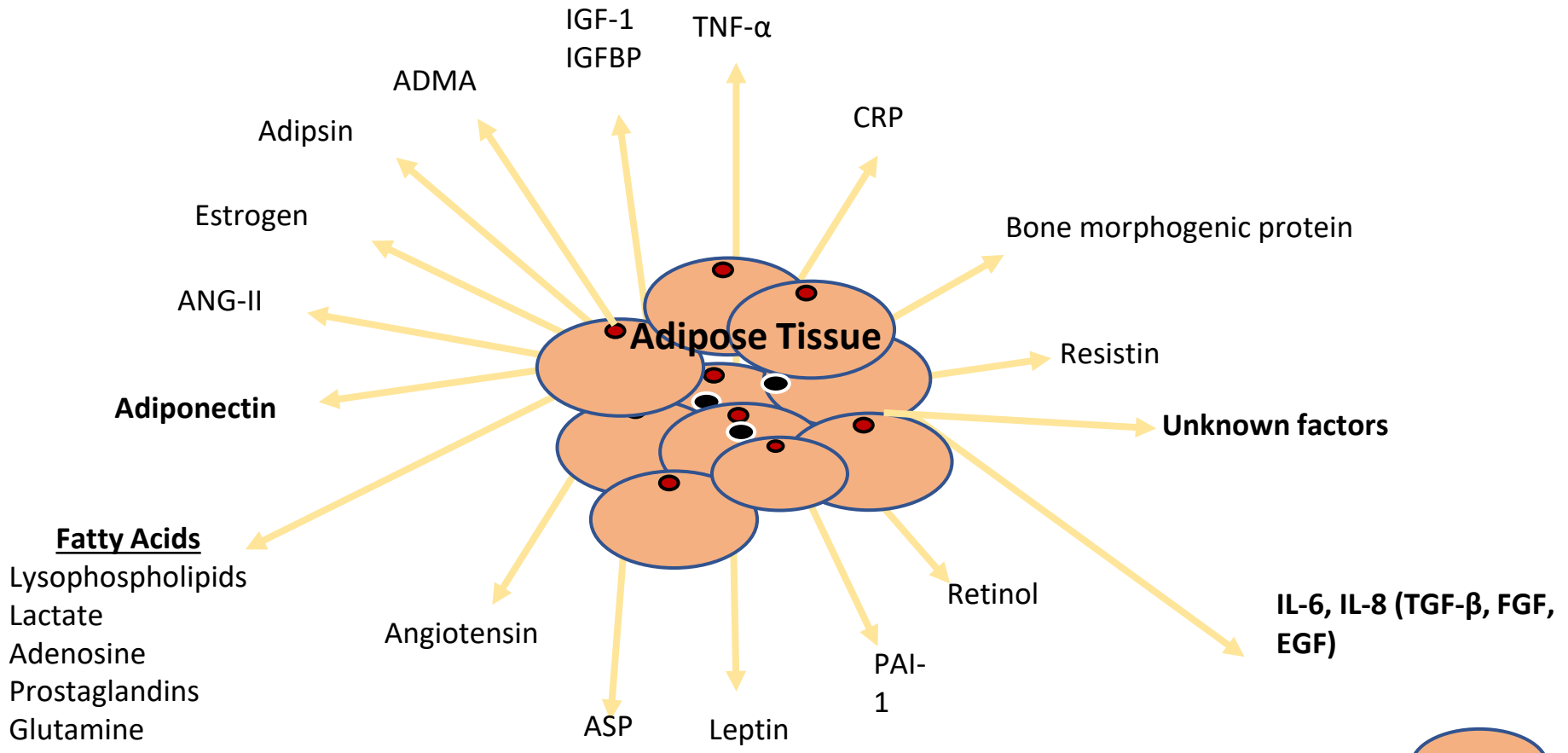
Consequences of WAT Expansion



FFA: Free Fatty Acids; IL: Interleukin; MCP-1: monocyte chemoattractant protein 1; ROS reactive oxygen species; SAT: subcutaneous adipose tissue; TG: triglyceride; TNF-alpha: Tumor necrosis factor-alpha; VAT: Visceral adipose tissue; EAT: white adipose tissues

Gustafson B, Smith U. Atherosclerosis. 2015;241:271-35

Factors Secreted by Adipose Tissue Under inflammatory Conditions



ADMA= asymmetric dimethyl-arginine; ANG-II= angiotensin II; ASP= acylation-stimulating protein; CRP= C-reactive protein; EGF: epidermal growth factor; FGF: Fibroblast growth factor; IGF-1= insulin-like growth factor 1; IGFBP: insulin-like growth factor binding protein; PAI-1= plasminogen activator inhibitor 1; TGF-beta= transforming growth factor beta; TNG-alpha: Tumor necrosis factor alpha

Itoh M. et al. Int J Inflam. 2011;2011:720926. doi: 10.4061/2011/720926. Epub 2011 Jul 7. Dixit VD. J Leuko Biol 2008;84:882-892

Adiposopathy

ADIPOCYTE EXPANSION BEYOND CAPACITY ⍰ ADIPOCYTE INSULIN RESISTANCE

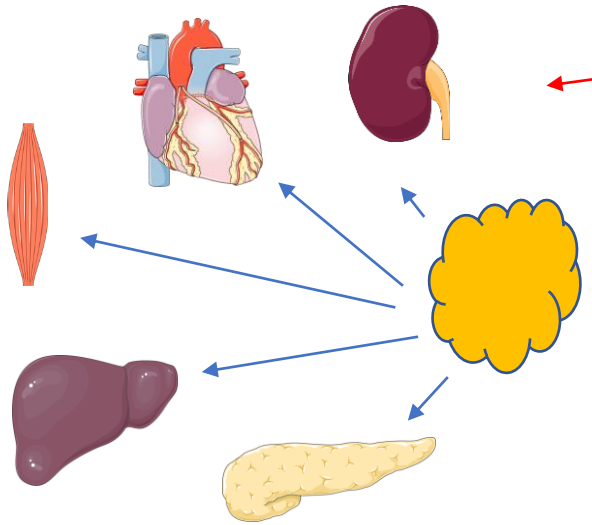
Estrogen

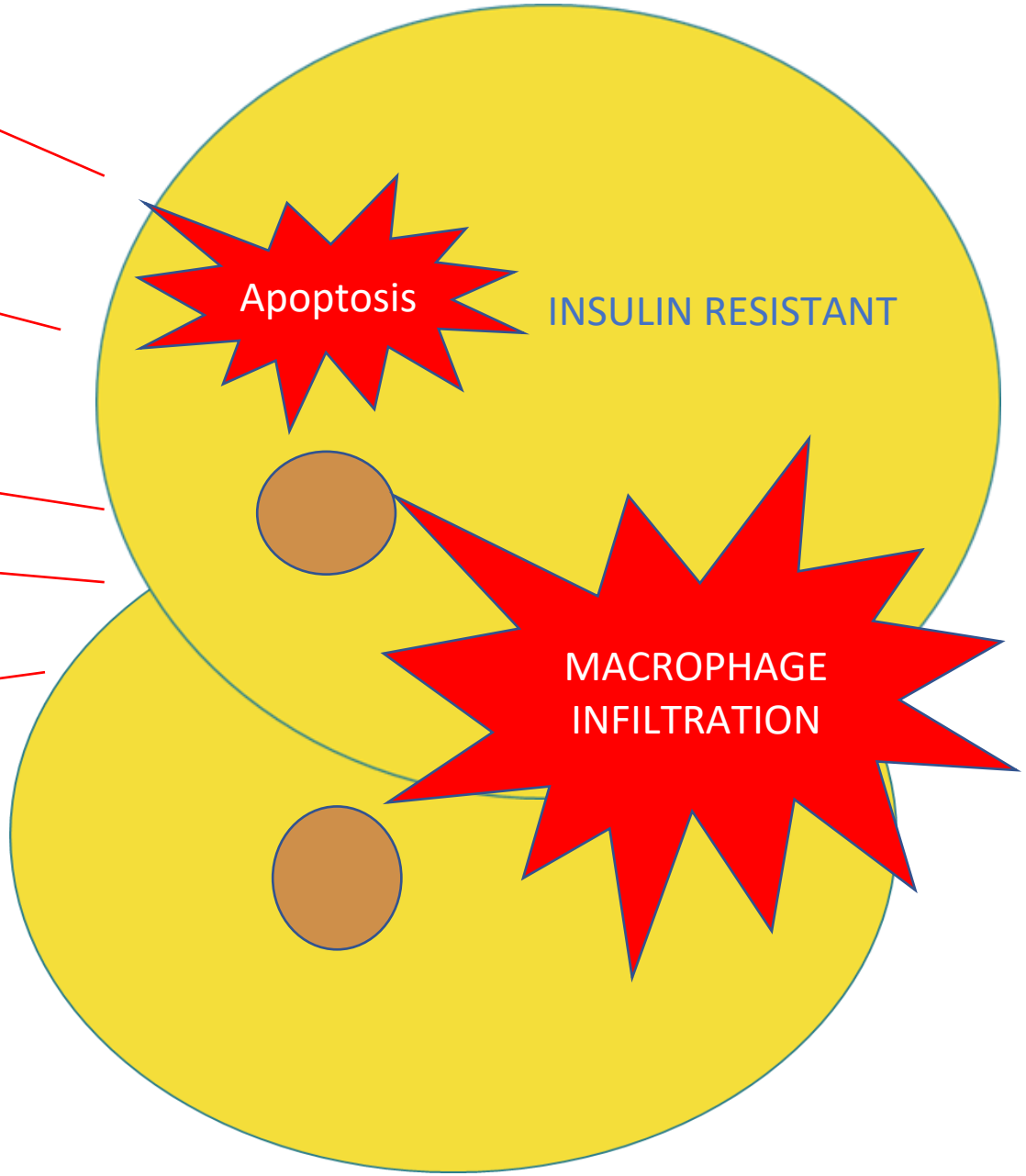
Adiponectin

Leptin

INFLAMMATORY CYTOKINES (TNF-a, IL-6, IL-8, CRP)

FREE FATTY ACIDS





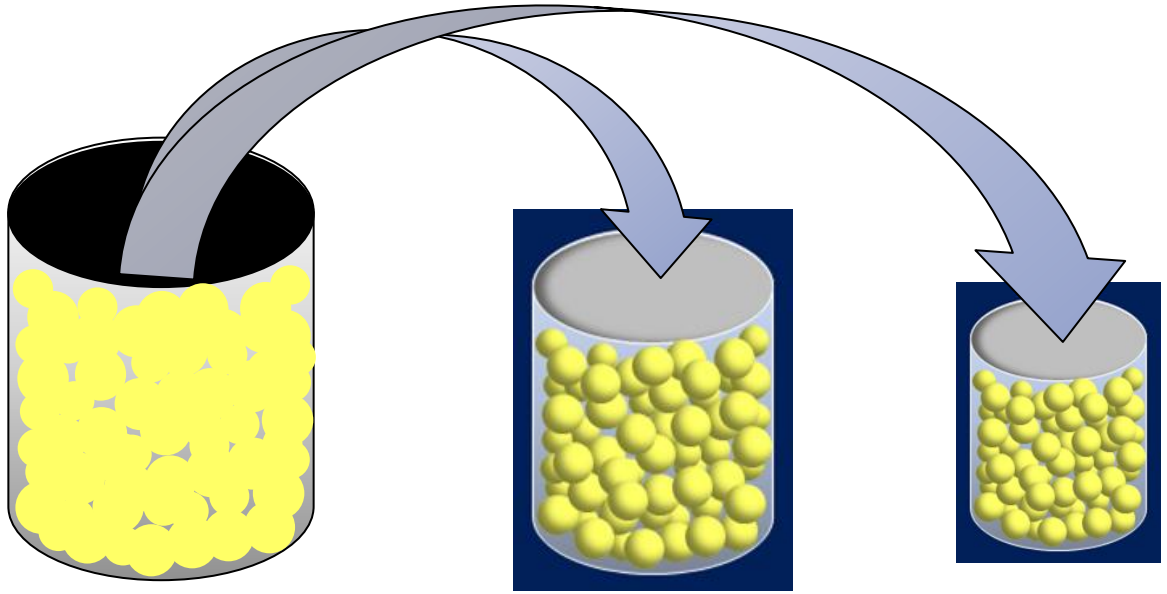
Ectopic Fat Deposits Associated With Cardiometabolic Disorders

Pancreatic fat	B-cell dysfunction, insulin resistance, impaired glucose metabolism, inflammation, lipotoxicity
Intra-muscular fat	Insulin resistance, mitochondrial dysfunction, impaired lipid, and glucose metabolism
Liver fat	Hepatic insulin resistance, hepatic insulin resistance, oxidative stress, inflammation, increased lipogenic transcription factors, increased VLDL-TG
Visceral fat	Inflammation, macrophage infiltration, insulin resistance, altered release of adipokines, altered FFA metabolites, RAS activation, oxidative stress
Perivascular/ cardiovascular fat	Inflammation, increased TNF-α, IL-6, leptin, MCP-1, cell adhesion molecules, calcification, decreased diastolic function, coagulation defects
Kidney fat	Hypertension, vascular resistance, glomerulosclerosis, proteinuria, increased intra-renal pressure

Adapted from Gustafson B. Smith U. Atherosclerosis. 2015.241(1)27-35

Ectopic White Adipose Tissue

Non-adipose Compartment



Subcutaneous Fat (70-75%)

Visceral Fat (10-15%)

Ectopic Fat (10-15%)

Clinical Clues

BMI

Waist Circ.
Triglycerides

ALT

Viscera
Heart
Pancreas
Liver
Skeletal Muscle

Metabolic syndrome

Cardiovascular disease, type 2 diabetes

Gustafson B, Smith U. Atherosclerosis.2015;241:27-35.

Obesity and Cardiovascular Disease: A Scientific Statement From the American Heart Association (2021)

- The GBD (Global Burden of Disease) Obesity Collaborators found high BMI accounting for **4.0 million deaths in 2015**, more than **two-thirds** of which were caused by cardiovascular disease (CVD), even after accounting for smoking and ill health.
- **41 % of BMI-related deaths** and **34% of BMI-related disability-adjusted life-years** were caused by CVD among individuals with obesity.
- High waist circumference (WC) even in individuals with normal weight may unmask higher CVD risk because WC is an indicator of abdominal body fat, which is associated with cardiometabolic disease and CVD and is predictive of mortality.
- WC as a measure of abdominal obesity provides an indicator of body composition and adds critical information along with BMI.

Obesity and Cardiovascular Disease: A Scientific Statement From the American Heart Association. Tiffany M. Powell-Wiley, Paul Poirier, Lora E. Burke, Jean-Pierre Després et al Originally published22 Apr 2021https://doi.org/10.1161/CIR.0000000000000973. Circulation. 2021;143:e984–e1010

Obesity and Atherosclerosis/ CAD

- Visceral adiposity promotes systemic and vascular inflammation, which is fundamental to all aspects of the atherosclerotic process, from fatty streak development to atherothrombosis.
- Inflammation induced by obesity increases the likelihood of low-density lipoprotein oxidation, which in turn promotes atherogenesis.
- Insulin resistance is associated with dyslipidemia and metabolic syndrome, which are linked to atherosclerosis.

Obesity and Cardiovascular Disease: A Scientific Statement From the American Heart Association. Tiffany M. Powell-Wiley, Paul Poirier, Lora E. Burke, Jean-Pierre Després et al Originally published22 Apr 2021https://doi.org/10.1161/CIR.0000000000000973. Circulation. 2021;143:e984–e1010

Obesity and Heart Failure

- Excess adiposity promotes changes in cardiac function both directly through the effects on the myocardium and vasculature and indirectly through obesity-related comorbidities.
- Excess adiposity also leads to higher blood pressure as a result of activation of the renin-angiotensin-aldosterone and sympathetic nervous systems.
- Obesity directly affects the myocardium with myocardial fat accumulation and subsequent fibrosis that can lead to the development of left ventricular diastolic dysfunction (LVDD) and heart failure with preserved ejection fraction (HFpEF).
- Atherosclerotic heart disease related to obesity can lead to systolic dysfunction and, ultimately, HF with reduced ejection fraction (HFrEF).
- Finally, comorbidities associated with obesity such as diabetes, sleep apnea, and hypoventilation syndrome can increase the risk for pulmonary hypertension and right ventricular and LV failure.

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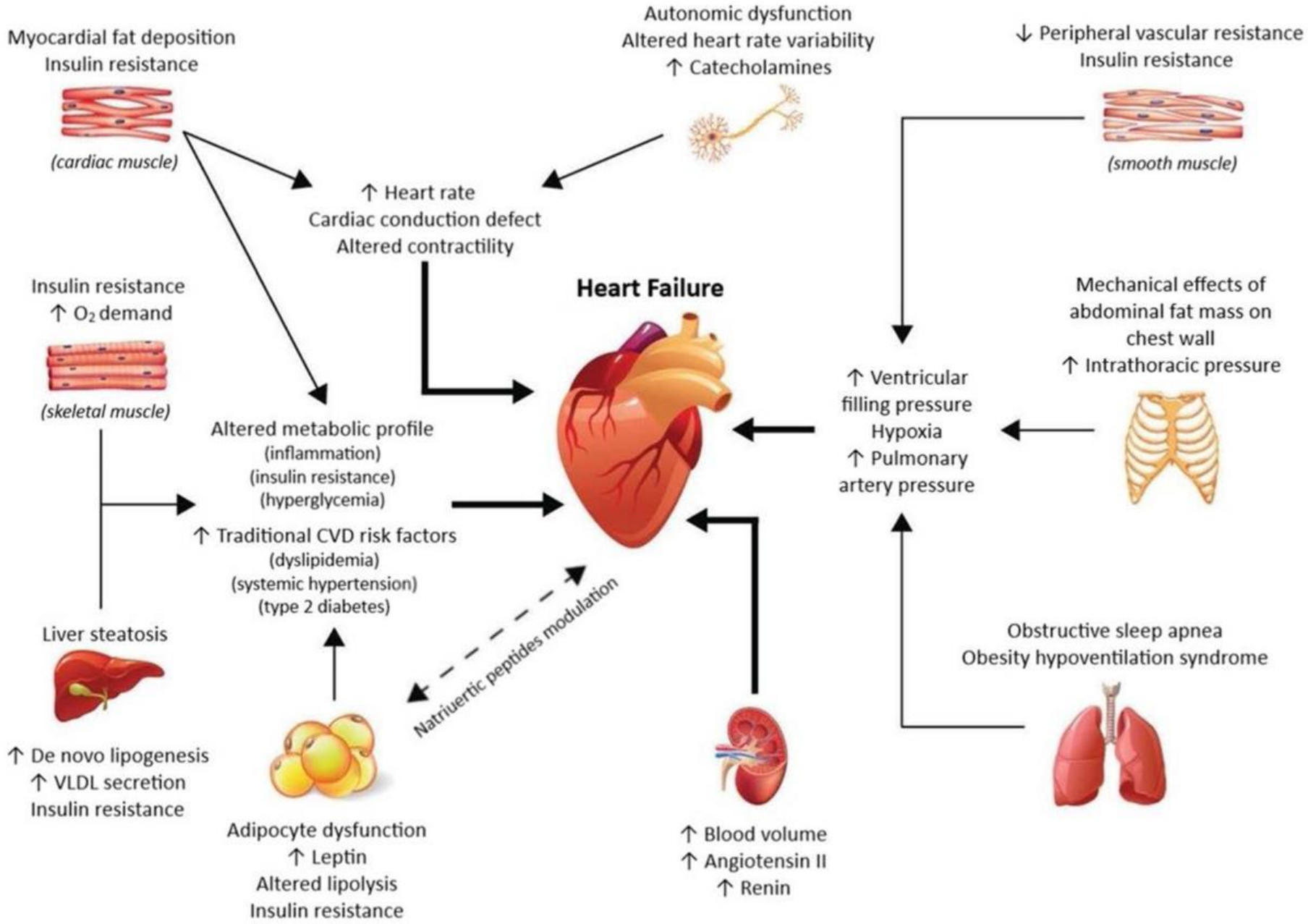

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Obesity and Arrhythmias

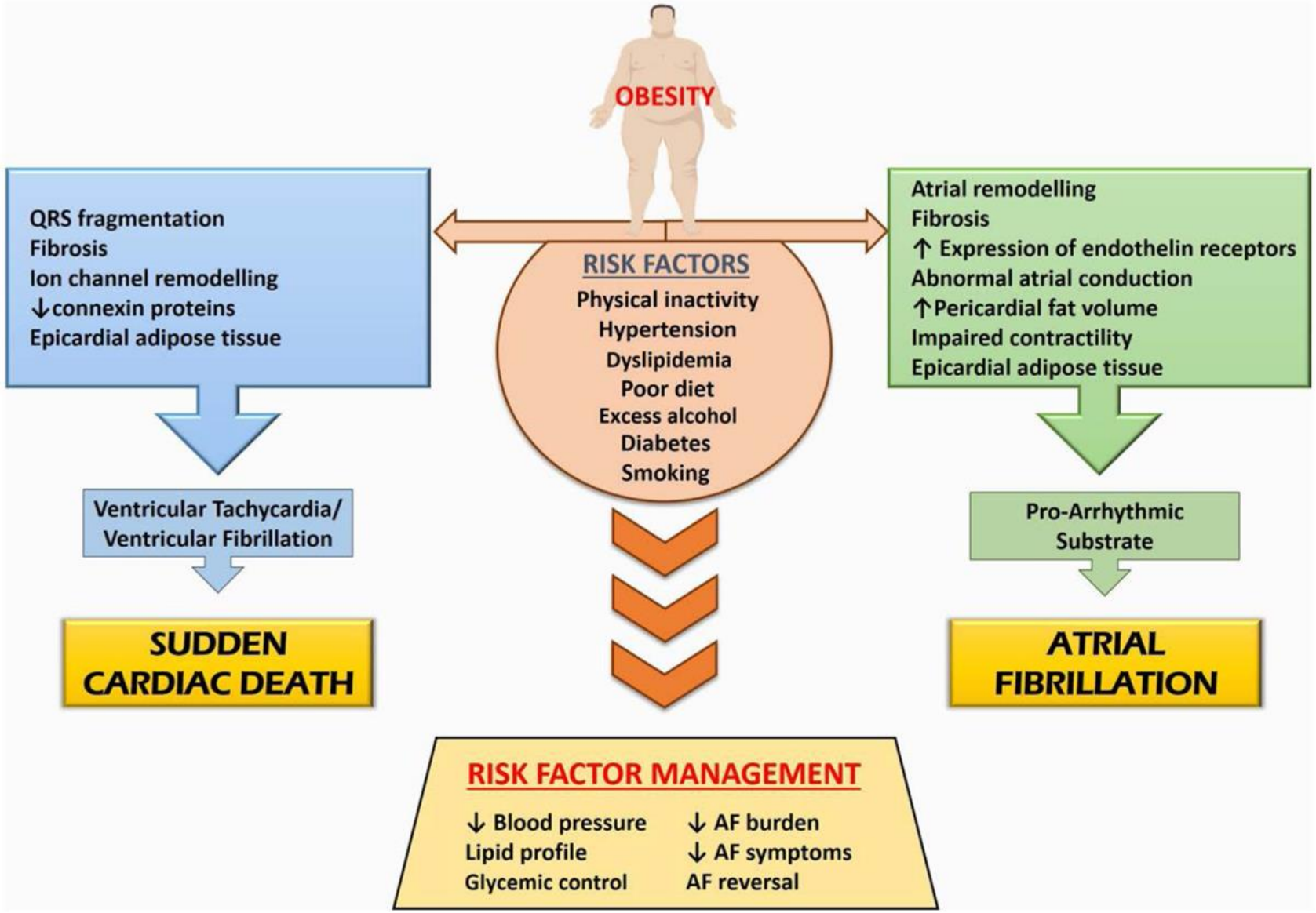
- Every 5-unit increment in BMI confers a 16% higher risk of SCD
- Obesity has been identified as the most common nonischemic cause of SCD
- Potential mechanisms may include LVH, QT prolongation, premature ventricular complexes, and autonomic imbalance.
- Both mild obesity and severe obesity are reported to be associated with greater risk of ventricular tachycardia (VT)/ventricular fibrillation (VF)
- Epicardial adipose tissue was reported to be associated with higher occurrence of premature ventricular contractions, VT/VF and all-cause long-term mortality and mortality from SCD.
- “Given that SCD is responsible for approximately half of all deaths resulting from CVD, obesity represents a modifiable target to reduce the public health burden of SCD in our society”.

Obesity and Cardiovascular Disease: A Scientific Statement From the American Heart Association. Tiffany M. Powell-Wiley, Paul Poirier, Lora E. Burke, Jean-Pierre Després et al
Originally published22 Apr 2021https://doi.org/10.1161/CIR.0000000000000973. Circulation. 2021;143:e984–e1010

Obesity and Atrial Fibrillation

- Every 5-unit increment in BMI confers an ~29% greater risk of incident AF
- Class I obesity: 54% increase in the likelihood of progression from paroxysmal to permanent AF.
- Class II obesity: 87% increase in risk
- Obesity elevates the risk of AF through numerous mechanisms, including structural and electric remodeling
- Epicardial adipose tissue has emerged as an important proarrhythmic substrate that may explain the excess risk of AF in obesity
- Mechanisms by which adiposity may lead to a susceptible electrophysiological substrate in the atria include fatty infiltration, adipokine-mediated fibrosis, LVDD, and inflammation, among many possibilities.

Obesity and Cardiovascular Disease: A Scientific Statement From the American Heart Association. Tiffany M. Powell-Wiley, Paul Poirier, Lora E. Burke, Jean-Pierre Després et al
Originally published22 Apr 2021https://doi.org/10.1161/CIR.0000000000000973. Circulation. 2021;143:e984–e1010



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Originally published22 Apr 2021https://doi.org/10.1161/CIR.0000000000000973. Circulation. 2021;143:e984–e1010

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Certified Cardiometabolic Health Professional (CCHP)

Initiating the Conversation

Karli Burridge, PA-C, MMS, FOMA

Gaining Health, Enara Health

5 As: Framework for Teaching and Practicing the Art of Behavior Change

ASK

ASSESS

ADVISE

AGREE

ARRANGE/
ASSIST

Vallis M, Piccinini-Vallis H, Sharma AM, Freedhoff Y. Modified 5 As. Minimal intervention for obesity counseling in primary care. Can Fam Physician. 2013;59:27–31.

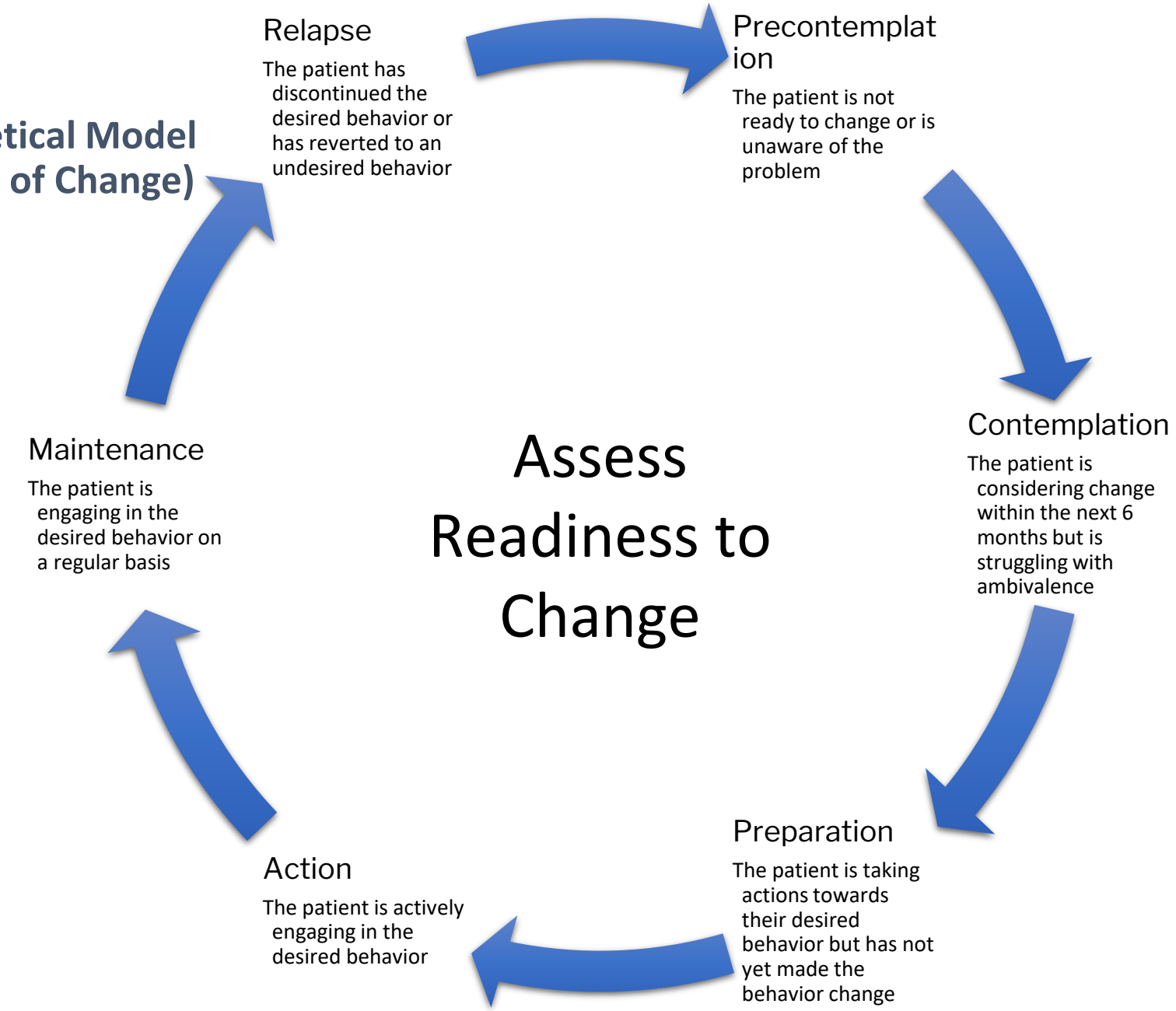
Ask for Permission to Discuss Weight

Weight is a complex medical issue that many people struggle with, and it can impact your health and wellbeing. How would you feel about working on this together?

Is now a good time to discuss how your weight and health may be affecting each other and how we can work together on it?

Vallis M, Piccinini-Vallis H, Sharma AM, Freedhoff Y. Modified 5 As. Minimal intervention for obesity counseling in primary care. Can Fam Physician. 2013;59:27–31.

Transtheoretical Model (AKA Stages of Change)



Stage of Change will determine behavioral approach

Adapted from Prochaska and DiClemente (1983)

Evidence-Based Behavioral Approaches for Obesity Management

Motivational Interviewing: Contemplation/ Preparation/ Relapse Stage (AMBIVALENCE)

Cognitive Behavioral Therapy: Preparation/ action/ maintenance stage

Motivational Interviewing

- *"Motivational interviewing is a collaborative conversation style to strengthen a person's own motivation and commitment to change"*

  ~ Miller and Rollnick

- Move a patient from ambivalence towards action by strengthening the patient's OWN motivation and commitment to change
- Everything the patient needs is already inside of them. Your job is to ask the right questions, listen, and guide with empathy and compassion

Miller WR, Rollnick S. Motivational Interviewing: Helping People Change. 3rd ed. Guilford Press; 2013.

The Spirit of MI: CAPE

Compassion: The desire to see the other free from suffering. Putting their needs before your own

Acceptance: supporting patient autonomy and accepting their right to change or not to change

Partnership: the collaborative relationship between two experts

- Avoid the "righting reflex". Avoid "let me fix this".

Evocation: Evoke "change talk" and have the patient argue for change

Miller WR, Rollnick S. Motivational Interviewing: Helping People Change. 3rd ed. Guilford Press; 2013.

Evoking Change Talk

"Why is increasing your activity important to you right now?"

"You say you want to weigh X again. How was your life different at that weight?"

"What needs to happen for you to make this change?"

"How do you think your life would look 5 years from now if you made this change, and how do you think it might look if you didn't make this change?"

Change metrics

"On a scale of 1-10, how important is this to you right now?"

"On a scale of 1-10, how confident are you that you can make this change right now?"

Follow up Questions to Change Metric Questions

Why are you at a 5 and not at a 3 or 4 (**LOWER** number)?

What would it take for you to get from a 5 to a 6 or 7 (**HIGHER** number)?

Core Communication Skills of MI: OARS

Open-ended questions: allows the patient to explore how they feel, which is important in resolving ambivalence

Affirmations: recognizing a good quality in another person and using this to build their confidence in changing their behavior.

Reflections: can be simple or complex

Summaries: selectively summarize the change talk and turn this into an actionable plan

Miller WR, Rollnick S. Motivational Interviewing: Helping People Change. 3rd ed. Guilford Press; 2013.

Cognitive Behavioral Therapy

- **Self-monitoring**
  - Food logging
  - Activity logging
  - Regular self-weighing
- **Stimulus control**
  - Reduce triggers or cues that lead to undesired behaviors
  - Increase triggers that promote the desired behaviors
- **Problem solving**
  - Identifying problems or barriers and developing solutions and strategies to overcome them
  - Allow the patient to determine most of the solutions, unless they specifically ask you for your advice. In that case, provide a list of 2-4 options for the patient to choose from

Cognitive Behavioral Therapy

- **Goal setting**
  - Set SMART goals
- **Contingency management**
  - Develop strategies to overcome setbacks
  - Encourage patients to plan for lapses and relapses
- **Enlisting social support**
  - Encourage an “accountability partner,” such as a friend, coworker, or family member
- **Stress management**
  - Assist your patient in finding healthful coping strategies to manage stress

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Case Presentation – Initial Assessment, Diagnosis & Treatment of Obesity

Karli Burridge, PA-C, MMS, FOMA

Gaining Health, Enara Health

Meet Ravi

- 30-year-old American male of Southeast Asian descent
- Presents to PCP for follow up after recent ER visit
- Prior medical history: None
- Height: 5’9, weight: 183.9 lbs, BMI: **27.2**
- Other vitals unremarkable
- Abdominal pain and constipation have resolved, but Ravi hasn’t been to see a PCP for several years and wants to get established as a patient

PMH

- Medications: None
- Surgeries: None
- Drug allergies: NKDA
- Family history:
  - Mother: Age 67. Type 2 Diabetes, hypertension, hyperlipidemia
  - Father: Age 72. Type 2 Diabetes, GERD, Hyperlipidemia
  - Sister: Age 34. Gestational diabetes
- Social history:
  - Alcohol: None
  - Tobacco use: None
  - Illicit drug use: None
  - Graduate student
  - Lives with his parents, his sister and her husband and their two children

Ravi’s Recent ER Visit

Presented for abdominal pain

- Ultrasound: Fatty liver
- CT: Constipation
- Rest of work-up negative
- Discharged same day

Discharge:

- Dx: Constipation
- Plan:
  - Laxative, stool softener
  - Follow up with PCP

Initiating the Conversation

Ravi, I see here on your ultrasound that you have something called “fatty infiltration of the liver”. There can be several causes for this which we will investigate, but the most common cause is excess weight. Would it be okay if we talked about your weight today and how it may be affecting your health?

Yeah, I guess so. I mean, I have gained some weight the past few years in graduate school, but I didn’t realize it was affecting my health!

Initiating the Conversation

The causes and effects of weight gain are complex and can affect people differently. I'd like to order some additional labs and schedule a follow up with you in a few weeks to discuss all of this in more detail. How does that sound?

Okay, that sounds good. My parents both have diabetes, and I certainly don't want to go down that road.

Ravi's Initial Work-Up

- Obesity-specific physical exam
  - Waist circumference: 38 inches
  - Neck circumference: 15 inches
  - Mild acanthosis along neck
  - Otherwise, unremarkable exam
- Forms to fill out prior to next appointment:
  - Weight history (graph)
  - Nutrition history
  - Physical Activity history
  - Sleep history
  - Psych history
- Labs (fasting):
  - CBC
  - CMP
  - TSH with reflex
  - Lipid panel
  - HbA1c
  - Vitamin D
  - Insulin
- Dx:
  - Non-Alcoholic Fatty liver Disease
  - Obesity
  - Abnormal weight gain
- Scheduled for follow up in 3 weeks

Multiple Choice

According to the World Health Organization's classification of obesity, does Ravi have obesity (based on his BMI, sex, waist circumference, and his ethnic background)?

A. Based on the WHO classifications, Ravi has obesity based on his BMI but not his waist circumference
B. Based on the WHO classifications, Ravi does not have obesity
C. Based on the WHO classifications, Ravi has obesity based on waist circumference but not BMI
D. Based on the WHO classifications, Ravi has obesity based on BMI and waist circumference

Multiple Choice Answer

According to the World Health Organization's classification of obesity, does Ravi have obesity (based on his BMI, sex, waist circumference, and his ethnic background)

A. Based on the WHO classifications, Ravi has obesity based on his BMI but not his waist circumference

B. Based on the WHO classifications, Ravi does not have obesity

C. Based on the WHO classifications, Ravi has obesity based on waist circumference but not BMI

D. Based on the WHO classifications, Ravi has obesity based on BMI and waist circumference

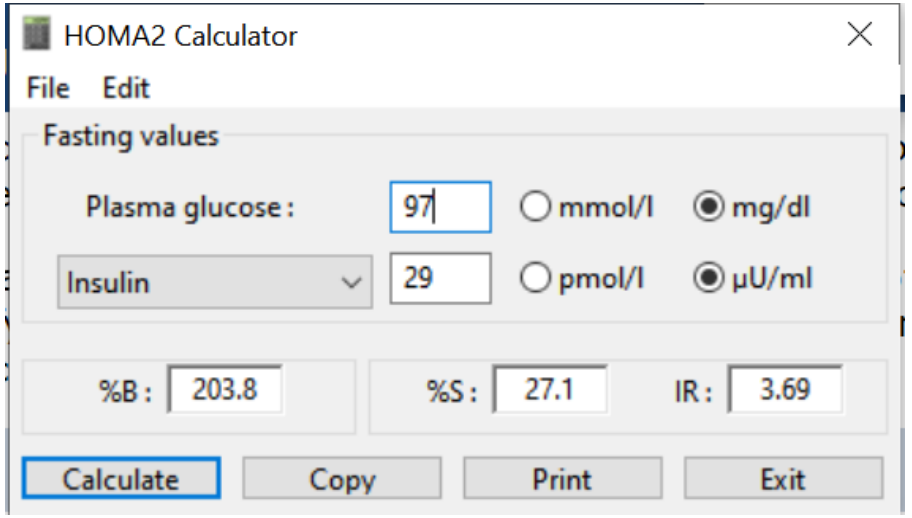
Explanation: WHO Obesity classification for Asian and South Asian population

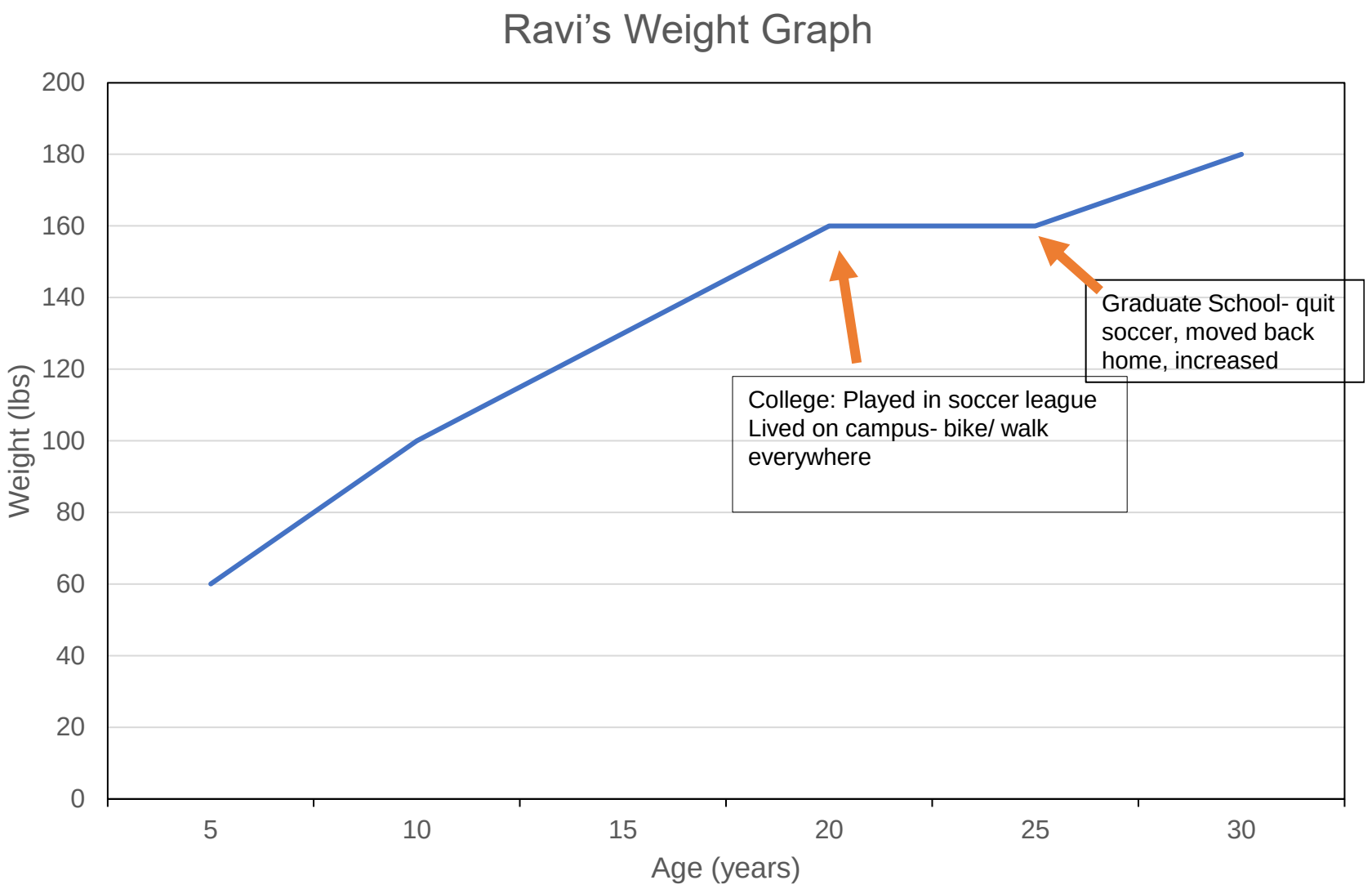
- Overweight (pre-obesity) - BMI between 23 and 24.9 kg/m^2
- Obesity - BMI greater than 25 kg/m^2
- Waist Circumference >31.5 women
- Waist Circumference >35 men

Weir CB, Jan A. BMI Classification Percentile And Cut Off Points. [Updated 2021 Jun 29]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK541070/

Ravi's Follow Up: Lab Results

Insulin	Gluc.	AST/ALT	TG	HDL	LDL	HbA1c	Vit D.	TSH
29 mu/mL	97 mg/dL	32/ 45	160 mg/dL	35 mg/dL	98 mg/dL	5.6%	23 ng/mL	4.3 mlU/L




Ravi's Weight Graph
200
180
160
140
120
100
80
60
40
20
0
Weight (lbs)
5
10
15
20
25
30
Age (years)
College: Played in soccer league
Lived on campus- bike/ walk everywhere
Graduate School- quit soccer, moved back home, increased

Ravi's Nutrition History

24-Hour Recall

Time	Food/ Beverages	Place
11:30am	12-inch sandwich with cheese, turkey, lettuce, tomato. 1 bag chips	Chain restaurant
3:00pm	Fruit Smoothie	Campus
6:30pm	Rice, naan bread, tofu curry, green beans	Home
8:30pm	1 cup ice-cream	Home
10:30pm	1 cup Chex-mix, 8 oz apple juice	Home
12:00am	Handful gummy bears	Home

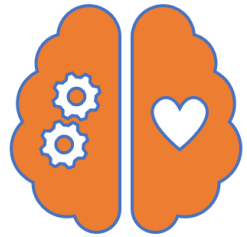
Calories	Carbs	Fat	Protein
2,992	62% (448 g)	23 % (73 g)	14% (104 g)

- Has never attempted a weight loss nutrition plan
- Eats some meals on campus, dinner at home- Mom and sister cook/ groceries
- No food allergies/intolerances
- At home: Eat traditional Indian food, mostly plant-based but he does eat meat/ animal products
- Grazes in evening when studying/ watching TV

Ravi’s Physical Activity History

- **Current activity**: None
- **Previous activities**: Played soccer growing up and throughout college. Didn’t have a car in college so he biked and walked everywhere.
- **Reason for discontinuing soccer**: Moved away from team- moved back home with parents. Got a car- drives to campus now
- **Perceived barriers**: Time, his friends don’t play soccer
- **Readiness:** 7/10. Would like to be more active but is concerned about time constraints
- **Access to safe places to be active**: Park near his house, gym on campus

Ravi's Sleep and Psych History



Sleep

- **Bedtime**: 1am-2am most nights
- **Wake time**: 7am most mornings. Sleeps in on weekends until 10am
- **Average sleep per night**: 6 hours
- **Sleep quality**: Good
- **Snoring**: None

Psych/ Mental Health

- Negative for depression/ anxiety/ suicidal ideation
- Stress**: 8/10**. Sources of stress: Graduate school, living back at home with his parents, sister, her husband and their two young children.
- Coping mechanisms: Stays up late when everyone else in in bed to watch TV and have quiet time. Sometimes turns to food especially when studying at night

Ravi, I notice a lot of simple starches and sugars in your nutrition, like the Chex-mix, chips, ice-cream, bread, rice, naan, and juice. These foods can really cause your insulin to spike, contributing to your insulin resistance. Do you have any ideas on how we could put together a plan on how to cut back on these foods?

I never really paid a lot of attention to that, but you're right… I guess I could just swap out the juice with water and snack on nuts or fruit if I get hungry in the evening, and I could cut back on the rice and bread and eat more veggies. I could probably also just go to bed a little earlier too. I often just stay up because that's the only time it's quiet in my house and I can have some "me" time!

Those sound like some great ideas! It also sounds like you need some time to yourself. Is there anything else you like to do to get you out of the house for some “me” time? Maybe something active?

Sometimes it’s nice to just get out of the house and go for a walk by myself and listen to a podcast or music. I’ve also been thinking about looking into the intermural soccer team. I just don’t know if I have time for that right now though. I have been super busy with my research and my dissertation.

Let’s just start with the walking then, and maybe look into the time commitment for soccer. How much time do you think you could spare to go on a walk, and when would be the best time for you?

I could probably do 20 minutes a day, after dinner would probably be best, when the house is usually crazy!

That sounds great! So, to summarize:

1. You will reduce your simple starches and sugars by swapping out juice and other sugar-sweetened beverages with water.
2. You will replace your snacks with fruit and nuts and reduce your bread and rice by eating more veggies.
3. You will get to bed by midnight and aim for at least 7 hours of sleep
4. You will take a brisk 20-minute walk after dinner while listening to a podcast or music.
5. Lastly, I'd like for you to take a vitamin D supplement and let's get you started on 500 mg ER metformin for your insulin resistance, and I'll see you back next month.

How does that plan sound to you?

That sounds very doable. Thanks so much for your help.

Ravi's Diagnoses

- Non-alcoholic Fatty Liver Disease (NAFLD)/ Metabolic-Associated Fatty Liver Disease (MAFLD)
- Obesity
- Dyslipidemia
- Vitamin D deficiency

1 Month Later

- **PA:** Brisk walk 20 minutes 5 days a week
- **Nutrition**: Has replaced juice/ smoothies with water and snacks with fruits and nuts. Eating less bread, rice, more veggies
- **Sleep**: Is averaging 7 hours of sleep
- **Stress**: 6/10: Has improved since getting more sleep, walking
- **Medications**: Taking 5,000 iu Vit D 3 daily, metformin ER 500 mg
- **Weight**: Lost 1 pound.

Plan

1

Increase metformin ER to 500 mg BID

2

Increase walks to 30 minutes daily. Track steps.

3

Track nutrition with app:

- Aim for 30-40 g protein per meal plus 10 g with snacks (total: 100-120 g protein per day).
- Aim to keep carbs <100g per day

4

Follow up in 1 month

1 month later

- Lost 2 lbs.
- Tracking nutrition with app: ~1,900 kcals, 100-120 g protein daily, <100 g carbs per day
- PA: 30 minutes brisk walking daily and started weight training 2 times a week at the gym on campus
- Sleep: 7 hours
- Stress: 5/10
- Is frustrated with slow weight loss, complains of hunger in the early afternoon. AOMs are not covered by insurance.

Plan

01

Add phentermine 15 mg QD

02

Increase metformin as tolerated to 2,000mg daily

03

Continue tracking nutrition

04

Gradually continue to increase exercise as tolerated- consider joining Saturday soccer league

1 Year Later

- Ravi continued to follow up monthly for the first 6 months, then every 3 months
- Typical Nutrition:
  - 8am: Eggs, plain Greek yogurt with berries or a protein shake
  - 12:00pm: Salad with chicken/ salmon, unsweet iced tea
  - 4:00pm: Almonds and apple
  - 6:30pm: Veggies with tofu, fish, or chicken, cauliflower rice
- Physical Activity: Daily 30-minute walk/ run. Strength training twice weekly x 45 minutes. 90 minutes soccer in Saturday mornings (~ 390 min/ week)
- Sleep: 7-8 hours
- Medications: Metformin 2,000 mg, phentermine 15mg PRN, Vit D3 2,000 iu daily

1 year later

	Weight (lbs.)	BMI Kg/m²	Total Weight change (lbs.)	% Total Body Weight Loss	Insulin (mu/mL)	NAFLD	TG (mg/dL)	HDL (mg/dL)	Vit D. (ng/mL)
Initial	183.9	27.2	-	-	29	Yes	160	35	23
1 year	159.3	23.5	-24.6	13.4%	8	No	130	40	41

Resources: Weight Bias and Stigma

- *Why Weight? A Guide to Discussing Obesity & Health With Your Patients*: http://whyweightguide.org/
- Tool kit modules to help providers or health care students prevent obesity bias http://www.uconnruddcenter.org/weight-bias-stigma
- Weight Bias in Healthcare – A Guide for Healthcare Providers Working with Individuals Affected by Obesity http://www.obesityaction.org/weight-bias-and-stigma/weight-bias-guides/weight-bias-in-healthcare-a-guide-for-healthcare-providers-working-with-individuals-affected-by-obesity
- People-First Language for Obesity -http://www.obesityaction.org/weight-bias-and-stigma/people-first-language-for-obesity

Resources: Initiating the Conversation, Motivational Interviewing

- *Weight Can't Wait: Guide for the Management of Obesity in the Primary Care Setting (STOP Obesity Alliance: https://stop.publichealth.gwu.edu/)*
- Sandra Christensen. *A Clinician's Guide to Discussing Obesity with Patients* Springer, 2021ed. Guilford Press;
- Centre for Collaboration Motivation & Innovation. www.centreCMI.ca
- WR, Rollnick S. *Motivational Interviewing: Helping People Change*. 3rd ed. Guilford Press; 2013

MC Questions

Multiple Choice

Tracking nutrition with an app is an example of:

A. Stimulus Control
B. Self Monitoring
C. SMART Goal Setting
D. Contingency Management

Multiple Choice Answer

Tracking nutrition with an app is an example of:

A. Stimulus Control
B. Self Monitoring
C. SMART Goal Setting
D. Contingency Management

Explanation: The term “self-monitoring” is used to describe the systematic recording by the client of his or her own behavior.

Ref: Claudia Avina. The Use of Self-Monitoring as a Treatment Intervention. *Evidence-Based Adjunctive Treatments*, 2008

Multiple Choice:

With non-ketogenic dietary energy restriction, the circulating levels of which appetite-regulating gut hormone tends to increases?

A. Leptin
B. GLP-1
C. Ghrelin
D. CCK

Multiple Choice Answer:

With non-ketogenic dietary energy restriction, the circulating levels of which appetite-regulating gut hormone tends to increase?

A. Leptin
B. GLP-1
C. Ghrelin
D. CCK

Explanation: Circulating levels of ghrelin increase with energy restriction (with the exception of ketogenic diets, which may decrease ghrelin or result in so changes in ghrelin). This increase in ghrelin can lead to increased appetite. Levels of GLP-1, CCK, and leptin generally decrease with energy restriction and weight loss.

Busetto et al. Mechanisms of Weight Regain. European Journal of Internal Medicine Jan 2021

Multiple Choice

Which hormone stimulates lipogenesis and inhibits lipolysis?

A. Ghrelin
B. Leptin
C. Oxyntomodulin
D. Insulin

Multiple Choice

Which hormone stimulates lipogenesis and inhibits lipolysis?

A. Ghrelin
B. Leptin
C. Oxyntomodulin
D. Insulin

Explanation: Insulin stimulates lipid synthesis and adipogenesis and inhibits lipolysis. Ghrelin, leptin, and oxyntomodulin affect appetite but not lipolysis or lipogenesis.

Ref: Saponaro et al. The Subtle balance between lipolysis and lipogenesis: A critical point in metabolic homeostasis. *Nutrients.* 2015.