



Acknowledgements

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Presenters





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Course Description

Obesity is a major health problem that affects every part of the body, including the brain. In fact, it is among the top risk factors for dementia. This course provides strategies to address obesity and use that information to help individuals prevent and treat obesity to build cognitive resilience.

Learning Objectives



Participants will be able to list 6 or more modifiable risk factors for dementia.



Participants will be able to summarize the link between obesity and dementia.



Participants will be able to identify effective interventions and strategies to address obesity



Participants will be able to identify special considerations for high-risk populations.

Alzheimer's & Dementia Facts

Scope of the Epidemic (U.S.)

6.5 million adults INCREASING 1 in 9 adults age ≥ 65 1 in 3 adults age \geq 85 +1.1% +1.8% 2/3 are women Alzheimer's deaths increased 145% from -7.3% 2000-2019, while DECREASING -10.5% other top causes of death -65.2% have declined Heart Cause Alzheimer's Breast Stroke Prostate HIV Cancer Cancer Disease of Death Disease (BAR GRAPH NOT TO SCALE) Nutrition and Dementia

145.2%

Inequities in Brain Health^{2,3,4}

African American people are **2X AS LIKELY** to have Alzheimer's

Latino people are **1.5X AS LIKELY** to have Alzheimer's



Less likely than White patients to receive a timely diagnosis;



More likely to report experiencing racial discrimination along their patient and caregiver journeys;



Less likely to be enrolled in cuttingedge Alzheimer's and brain health research.



Modifiable Risk Factors for Dementia

Alzheimer's: Non-Modifiable Risk

Factors 6, 1, 7, 8

Age

Number one risk factor is advancing age.

Risk doubles every 5 years after age 65.

Family History

Genetics vs environmental factors.

Education

Fewer years of formal education and lower levels of cognitive engagement may be risk factors.

Sex

2/3 of those with Alzheimer's are women. 16% of women age \geq 71 (11% of men). After age 65, have more than 1 in 5 chance (1 in 11 for men).

Obesity and Dementia

Modifiable Risk Factors⁹



INCREASE

DECREASE

- Healthy Diet
- Physical Activity
- Mental Activity
- Cognitive and social activity
- Hypertension High cholesterol
- Uncontrolled
 diabetes
- Obesity
- Smoking
- Depression
- Excessive Alcohol
 Intake
- Head Injury
- Air Pollution
- Hearing Loss

Brain Health Academy

Obesity and Dementia

The Lancet Report

• Updated 12 risk factor life-course model of dementia prevention

The Lancet Commissions

Dementia prevention, intervention, and care: 2020 report of \mathcal{O} the *Lancet* Commission

Gill Livingston, Jonathan Huntley, Andrew Sommerlad, David Ames, Clive Ballard, Sube Banerjee, Carol Brayne, Alistair Burns, Jiska Chen-Mansfield, Claudia Cooper, Sergi G Costaffeda, Amit Dias, Nick Fox, Laura N Gitlin, Robert Howard, Helen C Kales, Mika Kivimaki, Eric B Larson, Adesola Ogunniyi, Vasiliki Orgeta, Karen Ritchie, Kenneth Rockwood, Elizabeth L Sampson, Quincy Samus, Lon S Schneider, Gei's Yelback, Linda Teri, Maaheed Mukadam

	Relative risk for dementia (95% CI)	Risk factor prevalence	Communality	Unweighted PAF	Weighted PAF*
Early life (<45 years)					
Less education	1·6 (1·3–2·0)	40.0%	61.2%	19.4%	7·1%
Midlife (age 45–65 years)				
Hearing loss	1.9 (1.4-2.7)	31.7%	45.6%	22.2%	8.2%
TBI	1·8 (1·5–2·2)	12.1%	<u>55</u> ·2%	9.2%	3.4%
Hypertension	1·6 (1·2–2·2)	8.9%	68.3%	5.1%	1.9%
Alcohol (>21 units/week)	1.2 (1.1–1.3)	11.8%	73.3%	2.1%	<mark>0·8%</mark>
Obesity (body-mass index ≥30)	1.6 (1.3–1.9)	3.4%	58.5%	2.0%	0.7%
Later life (age >65 years)					
Smoking	1·6 (1·2–2·2)	27.4%	62.3%	14.1%	5.2%
Depression	1·9 (1·6–2·3)	13.2%	69.8%	10.6%	3.9%
Social isolation	1·6 (1·3–1·9)	11.0%	28.1%	4.2%	3.5%
Physical inactivity	1·4 (1·2–1·7)	17.7%	55.2%	9.6%	1.6%
Diabetes	1.5 (1.3–1.8)	6.4%	71.4%	3.1%	1.1%
Air pollution	1.1 (1.1–1.1)	75.0%	13.3%	6.3%	2.3%

Data are relative risk (95% CI) or %. Overall weighted PAF=39-7%. PAF=population attributable fraction. TBI=traumatic brain injury. *Weighted PAF is the relative contribution of each risk factor to the overall PAF when adjusted for communality.

Table 1: PAF for 12 dementia risk factors

Understanding Obesity

Obesity is not a Behavior/Lifestyle Factor 12

- BMI is a physical characteristic and a marker for metabolic disease
- BMI itself is neither a behavior nor a lifestyle, even though health behaviors and lifestyle factors can influence BMI
- Treating BMI as a lifestyle behavior obscures the complex etiologies that contribute to the metabolic disease of obesity
- Obesity is a fundamentally heterogeneous process with numerous underlying mechanisms and etiologies, each requiring unique prevention and treatment modalities



Viewing obesity as behavior can be harmful 11, 12, 13

- Viewing obesity as a health behavior rather than as a complex disease can contribute to weight bias and stigma
 - Promotes a **mistaken notion** that obesity is a condition that **patients choose for themselves through behaviors they elect**
 - People with obesity **already face bias and discrimination** in employment, health care, and interpersonal relationships
 - Belies the very **real struggle** that people with obesity face in **addressing actual health behaviors** and seeking the appropriate tailored approaches to manage their weight.
 - Weight bias is well documented to harm both health and quality of life for people living with obesity
 - Can promote the progression of obesity and discourage patients from seeking appropriate medical care
- Viewing BMI as a health behavior also promotes a very narrow view of obesity and strongly limits public knowledge of the myriad treatments that exist and are yet to come

Understanding Obesity 14

What is Obesity & Severe Obesity?

Obesity is a treatable disease that is a worldwide health concern associated with having an excess amount of body fat. It is caused by genetic and environmental factors and can be difficult to control through dieting alone. Obesity is diagnosed by a healthcare provider and is classified as having a body mass index (BMI) of 30 or greater. Nearly 40 percent of Americans have obesity.

Obesity Is

Obesity Is Not

- A disease.
- A worldwide health concern.
- Caused by many factors.
- Treatable and manageable.

- · Your fault.
- Yours to manage alone.
- Just about food.
- · Cured by a miracle treatment.



Obesity Classification ¹⁵

- For the general population, **BMI > 25 kg/m² is considered** overweight; BMI > 30 is considered obesity
- BMI has **limitations in assessing adiposity** in individuals with increased or decreased muscle mass, men vs women
- For individuals, accurately determining **% body fat**, android fat, and visceral fat is a better assessment of adiposity compared to BMI alone
- **Central obesity** is defined as **waist circumference > 40 in** • for men and > 35 in for women
 - Waist circumference is correlated with the risk of metabolic and cardiovascular disease

Body Mass Index $> 30 \text{ kg/m}^2$

Percent Body Fat Women: \geq 32% Men: ≥ 25%

Abdominal Obesity: Women > 35 inches

> 88 centimeters

Abdominal Obesity: Men > 40 inches

> 102 centimeters

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Obesity Prevalence Statistics 16



Racial/Ethnic Disparities in Obesity ¹⁶

- NHANES (2017-2020) BMI:
 - 41.9% of Adults
 - 49.9% Black
 - 45.6% Hispanic
 - 41.4% White
 - 16.1% Asian
- Obesity has disparate effects on racial/ethnic minorities
 - Weight bias amplifies health disparities (more obesity, more complications, less access to care)

- Generally, adults with lower incomes and lower education levels were more likely to have obesity
 - In individuals with incomes just above the federal poverty level, the adult obesity rate was 42.6%, compared with 29.7% in people with income levels at or above 400% of the federal poverty level
 - Similarly, the adult obesity rate was 35.6% in adults who had less than a high school education, but only 22.7% in college graduates.

The link between Obesity to Alzheimer's and related dementias

Increased BMI and central obesity increase risk for developing dementia ¹⁷

- In a representative sample of English older adults >50 yo (N=6582), 6.9% developed dementia over 15 years
- Obesity (BMI>30) and central adiposity (WC> 35 in women, >40 in men) were associated with increased dementia incidence independent of demographics, lifestyle behaviors, apolipoprotein E-e4, hypertension and diabetes
 - People with **BMI>30** had **35% greater risk** of dementia vs BMI <25
 - Women with **central obesity** (WC>35 in) had a **39% greater risk** of dementia compared with women without central obesity (WC<35 in)
 - Group with BMI>30 and WC>35/40 had 28% higher risk of dementia than the group with BMI<30 and WC<35/40

Midlife obesity is associated with late-life dementia ²⁰

- Systematic review of 19 longitudinal studies including 589,649 people aged 35 to 65 years, followed up for up to 42 years
- Obesity (BMI ≥30) but not overweight (BMI 25–30) in midlife was associated with latelife dementia
- Across cohort studies there was a significant and consistent 47% higher dementia risk associated with obesity in midlife compared with normal BMI
- Excess body weight in midlife may contribute to vascular and neurodegenerative damage that underpins dementia through vascular and dysmetabolic pathways and directly through cell-signaling proteins secreted by the adipose tissue (e.g., leptin and adiponectin)



Obesity and cognitive deficits

- Obesity has been linked to cognitive deficits, impaired long-term potentiation and synaptic plasticity, and a smaller brain volume, increasing the probability of developing Alzheimer's disease (AD) and other dementias
- People with obesity have greater risk for developing:
 - Age-related cognitive decline
 - Vascular dementia
 - Mild cognitive impairment (MCI)
 - Alzheimer's disease
 - Parkinson's disease
 - Huntington's disease

Inflammation 18

- Alzheimer's Disease can be defined as a progressive neurodegenerative disease whose distinctive histopathological characteristics are the extracellular amyloid plaques and intracellular neurofibrillary tangles
- Obesity and diabetes are significant risk factors for developing the disease because a key mediator of the disease progression is insulin resistance within the brain that contributes to the creation of the neurofibril tangles and necrosis
- The immune system plays a role as well through the central nervous system inflammation, Toll-like Receptor (TLR) activation, and certain gut microbiota
- The hypothalamus regulates metabolic homeostasis and determines energy intake stimulation (e.g., hunger) through hormones like leptin and ghrelin

Adipose tissue 19

- Obesity causes a state of lowgrade chronic inflammation in adipose tissue that leads to the dysregulation of homeostatic systems, which in turn leads to the development of various diseases, including those related to neurodegeneration
- Adipose tissue as an endocrine organ secreting pro-inflammatory adipokines and cytokines that can trigger inflammation, oxidative stress and insulin resistance

Visceral white adipose tissue

Insulin resistance Type 2 diabetes Metabolic syndrome Increased mortality High Resistin, RBP4 High expression of T-box15 and miRNA-145 Increased macrophages, T cells, MCP1, PAI-1, IL-6, IL-8, IL-10

Brown adipose

Energy expenditure Thermogenesis Low body fat Increased insulin sensitivity Expression of UCP1

Subcutaneous white adipose tissue

Insulin sensitive Low blood glucose, insulin, and triglycerides High MW Adiponectin High expression of Glypican-4 and miRNAs: 92,95,181a,311 Increased responsiveness to TZDs

Visceral White Adipose Tissue (vWAT) and Alzheimer's Disease (AD)²¹

- The visceral WAT has the potential to influence amyloid beta metabolism, tau metabolism, microglial activation and neuroinflammation via several pathways including:
 - vWAT-derived fatty acids
 - immunological properties of vWAT
 - retinoic acid derived from vWAT
 - vWAT-regulated insulin resistance
- Adults with higher vWAT index have more pronounced memory loss, subcortical gray matter atrophy and hippocampal atrophy



Skeletal Muscle Mass and Aging ²²

- Skeletal muscle mass loss occurs during aging and is caused by atrophy of muscle fibers and a decrease in the number of myofibers
- After age 30, muscle mass declines at a rate of approximately 3%–8% per decade and accelerates from 60 years onwards
- Aging is linked to alterations in protein synthesis, degradation, folding, and trafficking
- Low skeletal muscle mass is associated with cognitive impairment and dementia in older adults

- Purported molecular mechanisms that connect loss of muscle mass with cognitive decline include:
 - altered myokine secretion
 - Inflammation
 - insulin resistance
 - abnormal protein accumulation
 - oxidative stress
 - mitochondrial dysfunction

Muscle and cognitive function ²²

- Sarcopenia is associated with an increased likelihood of cognitive impairment and cognitive dysfunction
- Cross-sectional studies support an association between muscle strength and cognitive function in general and specific domains
- Some longitudinal studies indicate a bi-directional association between **poor muscle** strength and poor cognitive function
- Cross-sectional studies suggest that **gait speed** is associated with **cognitive function** (executive function, visuospatial ability and psychomotor function)
- Low handgrip strength and low muscle mass are independent risk factors for cognitive decline in community-dwelling older individuals
- Lifestyle factors are likely to **mediate the association between sarcopenia and cognitive impairment:** physical inactivity, poor diet, smoking, alcohol consumption,

Sarcopenia and health and dementia ^{23, 24}

- Individuals with sarcopenia or frailty frequently show a decline in physical function, associated with lower skeletal muscle mass, low muscle strength and endurance, balance dysfunction, and gait variability, as well as falls and fractures
- Diagnostic measures of sarcopenia in older adults (low skeletal muscle mass, muscle strength, and physical performance) are associated with detrimental health outcomes and morbidity (falls and fractures, caregiver dependence in activities of daily living, diabetes, and cognitive decline/impairment)
- Prevalence of **sarcopenia** in older community-dwelling individuals with **dementia** is more than **3 times higher** than individuals without dementia
- Sarcopenia and frailty may also be related to increased depression and psychologically affect quality of life

Sarcopenic Obesity 25, 26

- Sarcopenic obesity is the co-existence of low muscle mass/function and excess adiposity
- Many dementia patients not only face skeletal musculature loss (sarcopenia) but also face accumulation of body fat (excess adiposity/obesity)
- Japanese study of 1615 older adults (65-84 yo) and looked at sarcopenia (handgrip strength) and obesity (BMI) on MCI and dementia
- After multivariate adjustment, sarcopenic obesity was independently associated with increased odds of MCI and dementia compared with the control



Intervention recommendations

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Impact of the pro-inflammatory Western Diet 27,28

- Pro-inflammatory diets cause an increase in reactive oxygen species (ROS), insulin resistance (IR), and dysregulation in the activation of the sympathetic nervous system (SNS) and the renin–angiotensin–aldosterone system (RAAS) that contributes to the increase in obesity and related conditions
- Disturbances in **hypothalamic-pituitary-adrenal axis regulation** starts the cycle that creates obesity and related cognitive problems
- Hippocampus handles memory, but it also plays a role in decision making and the western diet-induced disturbances lower the ability of the hippocampus to regulate food intake, leading to overeating
- Hippocampal disturbance and the **addictive behaviors** stimulated by the high-fat, sugary foods are a powerful combination that perpetuates obesity

Diet/Lifestyle Factors 29

- Create a calorie deficit
- Cut out high-fat, processed, and sugary food
- Consume a plant-based diet with antioxidant-rich foods, omega-3 polyunsaturated fatty acids, curcumin, flavonoids, calcium, magnesium, potassium and drink caffeine to reduce inflammation
- Increase physical activity to improve glycemic control, reduce dyslipidemia, and decrease Aβ aggregates associated with cognitive decline and AD

A Comparison of the Western and Mediterranean Diets

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Western Diet	Mediterranean Diet			
-Defined by high amounts of sugar, carbohydrates, fat, salt	-Low amounts of processed sugars and foods			
-Trans fat predominates	-Unsaturated fats (e.g., olive oil)			
-Red meats	-Lean meats (e.g., chicken)			
-Sparse consumption of vegetables	-Limited red meats			
-Omega 6:3 imbalance	-Predominately made up of plant-based foods			
-Creates a pro-inflammatory environment -Highly palatable, addictive foods	-Creates an anti-inflammatory environment			
-Perpetuates obesity and diabetes	-Combats obesity and diabetes			
-Induces oxidative stress and insulin resistance	-Associated with high levels of anti- oxidants			

Dietary Interventions for Dementia 31, 32, 33

Observational studies on protective factors

• folate, B vitamins, Vitamins C, D, E, selenium

Lower saturated fat

- In the Women's Health Study, saturated fat intake was associated with a faster decline in memory by 70%. Women with the lowest saturated fat intake had the brain function of women six years younger
- Whole diets/Plant-based diets to reduce cognitive decline and dementia
 - **Mediterranean diet** (high in vegetables, legumes, fruits, nuts, cereals, olive oil and low intake of saturated fat and meat)
 - WHO guidelines recommend a Mediterranean diet to reduce the risk of cognitive decline or dementia
 - A plant predominant diet such as the Mediterranean, DASH (dietary approaches to stop hypertension), and the hybrid thereof, the MIND (Mediterranean and DASH Intervention for Neurodegenerative Delay) diet has been associated with significant prevention of cognitive decline

MIND (Med + DASH)³⁴

- Rush University Memory and Aging Project (N = 923, 58-98 yo)
- Emphasizes foods to support a healthy brain and recommends limiting potentially damaging choices
- Strict adherence to the MIND diet resulted in a 53% reduction in risk for Alzheimer's
- Moderate adherence to the diet was associated with a 35% risk reduction
- Those with high adherence had cognitive functioning equivalent to a person who was 7 ¹/₂ years younger!

WHAT'S ON THE MIND DIET?



Treating Obesity as a Chronic Disease 15



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Treatment for obesity ³⁰



Obesity Treatment Pyramid ³⁵



Treatment approaches "

AHA/ACC/TOS Prevention Guideline

OPEN

2013 AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society

Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation, American Pharmacists Association, American Society for Nutrition, American Society for Parenteral and Enteral Nutrition, American Society for Preventive Cardiology, American Society of Hypertension, Association of Black Cardiologists, National Lipid Association, Preventive Cardiovascular Nurses Association, The Endocrine Society, and WomenHeart: The National Coalition for Women With Heart Disease

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Complications-Centric Model for Care of the Patient with Overweight/Obesity



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Academy of Nutrition and Dietetics Adult Weight Management 2022 Evidence-Based Nutrition Practice Guideline ³⁶

- MNT Approach for Adults with Overweight or Obesity
 - Utilize the Nutrition Care Process
 - Provide Medical Nutrition Therapy
 - Adapt Goals and Interventions
 - Minimize Weight Bias and Stigma
- Coordination of Care
 - Collaborate with Interprofessional Healthcare Team
 - Coordinate Care in a Variety of Settings
- MNT Amount
 - Number and Frequency of Interactive Contacts
 - Intervention Duration
 - Follow-Up Contacts
- Delivery of MNT
 - Telehealth and In-Person Care
 - Group and Individual Contacts
- Payment for Services
- Dietary and Lifestyle Intervention Approaches
 - Dietary Patterns
 - Components of a Comprehensive Intervention
- Special Populations
 - Co-Morbidities
 - Pharmacotherapy and Metabolic and Bariatric Surgery
 - Members of Groups Disproportionately Affected by Overweight or Obesity and Under-Resourced Communities

Key elements of medical nutrition therapy provided by dietitians for adults with overweight or obesity ³⁶



Dietary approaches to consider ³⁶

Diet	Description	Macronutrient-targeted diets ^{162,163}	15% or 25% of total calories from protein; 20% or 40% of total
Calorie reduction for weight loss, when appropriate and desired			calories from fat; 35%, 45%, 55%, or 65% of total calories from carbohydrate
Dietary Approaches to Stop Hypertension (DASH) diet ¹⁶²	SH) diet ¹⁶² Rich in fruits and vegetables, legumes and nuts, whole grains, and low-fat dairy. Limit foods high in saturated fat, sweets, and sugar-sweetened beverages. Number of servings per food group to consume is usually provided to assist with calorie reduction.	Meal replacement ¹⁶³	Liquid or bar meal replacements
		Mediterranean-style diet ^{162–164}	No agreed upon guidance, but generally higher in fruits and vegetables, low in red meats, higher use of plant-based proteins and seafood and use of olive oil as a fat source. Number of servings per food group to consume is usually provided to assist with calorie reduction.
Higher-protein diet ¹⁶³	25% of total calories from protein, 30% of total calories from fat, and 45% of total calories from carbohydrate		
Higher-protein zone—type diet ^{162—164}	5 meals/d, each with 40% of total calories from carbohydrate, 30% of total calories from protein, and 30% of total calories from fat	Moderate-protein diet ¹⁶³	12% of total calories from protein, 58% of total calories from carbohydrate, and 30% of total calories from fat
Lacto-ovo-vegetarian-style diet ¹⁶³	Fruits and vegetables, eggs, plant-based proteins, and dairy products; no meat	MyPlate ¹⁶¹	Guidance from the US Department of Agriculture to promote inclusion of all food groups and intake of a variety of
Low-carbohydrate diet ^{162,164,}	Initially ${<}20$ g/d carbohydrate , increasing to ${<}130$ g/d		nutrients. Suggests filling half of one's plate with fruits and vegetables, making half of grains whole grains, varying
Low-fat vegan-style diet ¹⁶³	10% to 25% of total calories from fat		proteins, and moving to lower-fat dairy products.
Low-fat diet ^{162,163}	20% of total calories from fat	Ornish diet ^{162,164}	<10% calories from fat
Lower-fat, high-dairy/calcium with added fiber and low-glycemic index foods $^{\rm 163}$	≤30% fat 4 servings/d dairy With or without increased fiber and/or low-glycemic-index (low glycemic load) foods	The American Heart Association-style Step 1 diet ¹⁶³	Prescribed energy restriction of 1,500 to 1,800 kcal/d, <30% of total calories from fat, <10% of total calories from saturated fat

Physical Activity 15

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Takeaway Messages Obesity and Physical Activity

- 1. Routine physical activity may improve body composition
- Routine physical activity may improve adiposopathic endocrine and immune body processes
- Physical activity may improve metabolic, musculoskeletal, cardiovascular, pulmonary, mental, sexual, and cognitive health
- 4. Dynamic training promotes weight loss and may help prevent weight gain or regain
- 5. Resistance training may improve body composition, prevent muscle loss during weight loss, and increase resting energy expenditure

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Physical Activity 15

- In addition to physical exercise, increased energy expenditure can be achieved via increased leisure time physical activity and non-exercise activity thermogenesis (NEAT)
- A common physical exercise prescription (FITTE) includes frequency, intensity, time spent, type, and enjoyment
- Metabolic equivalent tasks (METS) are used to assess the intensity of physical exercise, with one MET equal to the amount of energy expended for one minute while lying down at rest [equal to 3.5 milliliters of oxygen consumption per kilogram of bodyweight per minute (3.5 ml/kg/min)]
- Standing = 2 METS; walking 4 miles per hour = 4 METS; running 10 miles per hour = 16 METS
- 10. Progress can be measured through tracking activity patterns over time via various activity logs, or can be measured by using a reliable technique to measure body composition

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Behavioral Therapy 15

- Eating behavior in patients with increased body fat often reflects the imbalance in physiologic forces that strongly resist weight reduction and weakly resist weight gain.
- Eating behavior can be affected by:
 - genetic predisposition
 - mental stress
 - Emotions
 - habitual time cues
 - Environment
 - information gap
 - reward factors
 - psychiatric disease
 - eating disorders (e.g., binge-eating disorder, bulimia nervosa, sleep-related eating disorder and night-eating syndrome)

Behavioral Therapy 15

- Behavior related to weight regain may be related to personal and physiologic priority imbalances (i.e., "lack of time") as well as physiologic changes of a weight reduced state
- Behavior therapy elements for optimal success include **promoting behaviors that are doable, efficacious, measurable, and which engage self-ownership**
- Behavior therapy implementation optimally includes frequent encounters with qualified medical professionals, education, stimulus control, cognitive restructuring, goal setting, self monitoring, behavioral contracting, problem solving, social support, etc.
- For patients ready for change, healthful nutrition and physical activity may be aided by weight management technologies, access to healthful nutrition and physical activity resources and/or knowing the existence of social media resources
- The relationship between **obesity and depression is bidirectional**. Obesity is a risk factor for mood disorders; mood disorders are a risk factor for obesity

Pharmacotherapy 15

- Adjunct to Nutritional, Physical Activity, and Behavioral Therapies
- Objective is to treat disease of excess adiposity
- Facilitate management of eating behavior
- Slow progression of weight gain/regain
- Improve the health, quality of life, and body weight of the patient with overweight or obesity

Anti-obesity /medications Approved for Short-term Use

- Phentermine
- Diethylpropion
- Phendimetrazine
- Benzphetamine

Anti-obesity Medications Approved for Chronic Use

- Phentermine HCl/topiramate
 extended release
- Naltrexone HCI/bupropion HCI extended release
- Liraglutide
- Orlistat
- Semaglutide

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Surgical Weight Loss 15



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Other

patients

Higher BMI,

Type 2 DM

Metabolic

Lower BMI;

Higher BMI,

Type 2 DM

disease

disease

with:

GERD.

comments

Largest data set

Currently most

common

procedure

performed

Performance

and removal

rate of at least

25 percent at

Most technically

challenging

five years

no metabolic has declined

Other considerations when addressing Obesity

People First Language

- "People-first" language recognizes the potential hazards of referring to or labeling individuals by their disease
- "Patient who has obesity" or "patient with overweight or obesity" is preferred over "obese patient"
- Standard with other diseases, such as cancer, wherein "patient with cancer" is preferred over "cancerous patient"
- Respectful communication using 'People-First Language' creates positive, productive discussions in health care settings about weight and health

Encouraged Terms	Discouraged Terms	
Weight	Morbidly obese	
Excess weight	Obese	
Unhealthy weight	Fat	
Overweight	Heavy	
Body mass index	Large size	
Affected by obesity		
Living with obesity		

Social Determinants of Health (SDoH)

- Understand your own position and role of implicit bias
- Patient-centered care means looking at all perspectives
- Consider **feasibility of recommendations** for eating, moving, and access to care
- Ask about safety and availability
- Consider **social/community context** and support for lifestyle recommendations
- Inquire about financial implications and stress



Evaluating Obesity Treatment for Outcomes that matter

- Weight/BMI change
- Body composition
 - Central obesity
 - Visceral adiposity
 - Fat mass/body fat %
- Sarcopenia
 - Skeletal Muscle Mass
- Other health conditions (diabetes, heart disease)
- Mental Health
 - Quality of life

Address modifiable risk factors and obesity

- Consider **complex problems** exist in many domains
- Provide interventions that are **individualized and holistic**
- Focus on wellbeing, quality of life, mental health
- Tailor psychosocial interventions to the **patient's needs**
- Consider treating **co-occurring conditions** such as diabetes, hypercholesterolemia, hypertension
- Monitor for sarcopenia, sarcopenic obesity, frailty and other muscle-related parameters
- Check in about **physical activity**, **cognitive and social activity**

Higher weight protective later in life?

- While much of midlife is where interventions take place, consider the research showing higher weight may be protective later in life
 - Higher BMI later in life (>65 yo) is associated with better cognition
 - Weight loss in old age, are related to cognitive decline and increased risk of developing AD
- As the disease develops before the appearance of cognitive symptoms, it is suggested that low weight in old age may be a manifestation of this early stage of the disease and a sign of premature brain dysfunction
- Consider focus on **protecting and building lean muscle mass during midlife**, and less focus on weight/BMI/adiposity during older age?

Conclusions

- Obesity is a fundamentally heterogeneous process with numerous underlying mechanisms and etiologies, each requiring unique prevention and treatment modalities
- Treating the disease of obesity and the intersection with inflammation, adipose tissue, cognitive functioning, dementia and Alzheimer's Disease is nuanced and complex
- Treatment should focus on reducing weight during midlife with a focus on preserving/building muscle mass to protect against sarcopenia later in life
- Obesity treatment modalities require a comprehensive approach with nutrition, physical activity, behavior, and pharmacology/surgery when indicated
- Assessment and monitoring should include body composition assessment of central obesity, visceral adiposity, fat mass, muscle mass, and metrics to assess co-occurring health conditions

Tools and resources for health professionals

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Resources for Providers & Patients

- Obesity Action Coalition https://www.obesityaction.org/
- Obesity Medicine Association https://obesitymedicine.org/
- Academy of Nutrition and Dietetics <u>https://www.eatright.org/</u>
- American Board of Obesity Medicine https://www.abom.org/
- Stop Obesity Alliance <u>https://stop.publichealth.gwu.edu/resources</u>
- Communicating Brain Health Messaging with the African American and Latino Communities -<u>https://www.usagainstalzheimers.org/sites/default/files/2022-</u> 04/BrainHealthEquity_PracticalGuide_Final_Digital.pdf
- BrainGuide by UsAgainstAlzheimer's <u>https://mybrainguide.org/</u>

Thank you!





This presentation and related resources are available at: <u>https://www.usagainstalzheimers.org/nutrition-and-dementia</u>

Please register for additional courses at: https://www.usagainstalzheimers.org/brain-health-academy

> For more information, contact: Kelly O'Brien UsAgainstAlzheimers <u>kobrien@usagainstalzheimers.org</u>

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