The Weigh Of Maturity: Evaluation and Management of Obesity in the Geriatric Population

Jeanne Ortiz, MD, FAAFP, Diplomat of the American Board of Obesity Medicine
Director, Medical Weight Management
Objectives

- Review of Obesity as a chronic disease
- Define Obesity Medicine and Principles for Weight Regulation
- Review framework for initial evaluation of the patient struggling with obesity
- Best practices for the treatment of the aging patient with obesity
Medical Definition of OBESITY

- Chronic, relapsing condition
- Multifactorial, neurobehavioral disease
- Promotes:
  - Adipose tissue dysfunction (adiposopathy)
  - Abnormal fat mass physical forces (Fat mass disease)
- Results in:
  - Adverse metabolic, biomechanical, psychosocial health consequences
Weight REGULATION
The old Paradigm

Energy balance is SIMPLE

↑ Calories ↓ Activity = Weight Gain

↓ Calories ↑ Activity = Weight Loss

CALORIES INGESTED THROUGH DIET

CALORIES BURNED THOUGH ACTIVITY
It’s Hard to Lose Weight!

Weight is controlled by a feedback system

Weight loss provokes counter-regulatory responses

Afferent Signals

- Ghrelin
- PYY
- CCK
- GLP-1
- Vagus Nerve

Hypothalamus, Endocannabinoids (ECS)

Efferent Signals

- Autonomic Nervous System

External Factors
- Food availability, palatability

ECS
- Gut and Liver
- Meal Size
- Food Intake

Energy Balance and Adipose Stores
- Energy Expenditure
- Adiponectin

Insulin
- Pancreas
- Adipose Tissue

Leptin
- Adipose Tissue
- Adrenal Cortex

Adrenal Steroids

Leptin and ghrelin have counterbalancing effects on energy balance

<table>
<thead>
<tr>
<th>Role</th>
<th>Leptin</th>
<th>Ghrelin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Anorexigenic (Reduce weight)</td>
<td>Orexigenic (Gain weight)</td>
</tr>
<tr>
<td>Timing</td>
<td>Long term energy balance</td>
<td>Acute appetite stimulatory hormone</td>
</tr>
<tr>
<td>Outcome</td>
<td>↓ Energy balance</td>
<td>↑ Energy balance</td>
</tr>
<tr>
<td>Released from</td>
<td>Adipocytes</td>
<td>Stomach</td>
</tr>
<tr>
<td>Site of action</td>
<td>1) Hypothalamus (AN) 2) Other?</td>
<td>1) Hypothalamus (AN) 2) Vagal nerve then nucleus tract solitarius</td>
</tr>
<tr>
<td>Activates</td>
<td>POMC, CART, CRH</td>
<td>NPY, AgRP, Orexin</td>
</tr>
<tr>
<td>Inhibits</td>
<td>NPY, AgRP, Orexin</td>
<td>POMC, CRH</td>
</tr>
</tbody>
</table>

Weight Regulation Principles

Satiety Pathway
- Mouth
  - Substrate -> food
- Stomach
  - Leptin
  - GLP-1
- Fat Cell
- Small Intestine

Hunger Pathway
- Brain
  - Appetite suppressants work here
- Pancreas
  - Insulin
  - Insulin -> Hunger
- Glucose-dependent Insulinotropic Polypeptide (GLP-1)
  - Blocks ghrelin

GLP-1 blocks ghrelin, leading to hunger suppression and weight regulation.
Energy Balance is "Simple"

COMPLEX!!!

↑ Calories ↑ Activity = Weight Gain

↓ Calories ↓ Activity = Weight Loss

CALORIES INGESTED THROUGH DIET

CALORIES BURNED THROUGH ACTIVITY
Biological Adaptation
Body Weight is Under Biological Control

Physiological adaptations antagonize weight change

Hunger

Energy Expenditure

Baseline body weight

Body Weight

Biological Adaptation
WHAT ABOUT SET POINTS AND PLATEAUS?

RE-SETTING Set Points

Starting Weight

Weight Regain Without plateaus

EMBRACING Plateaus

Goal weight

New set point

Weight

Time →
Challenge of Maintenance

- Homeostatic Drive
- Cravings
- Genetics
- Stress
- Behavioral
- Motivation
- Health
- Hedonic/Reward
- Environment
- Decreased Energy expenditure
  - NREE
  - REE
- Habits
What do we know?

- Weight loss is hard to maintain
- Individual response to treatment(s) varies greatly and is unpredictable
- The state of maintenance is dynamic with multiple psycho-physiologic modifiers and a complex biology.
- Requires long-term integration of structural changes to one’s life, often with ongoing intermittent support/intervention.
- We don’t need to sustain a lot of weight loss for positive impact on QOL, metabolic and structural conditions.
Obesity is a Disease of Hypothalamic Signaling, NOT WILLPOWER:

• Weight regulating systems favor weight gain and prevent weight loss
• Hypothalamic and other neuron injury and inflammation may be source of the “ratchet” and problems of glucose homeostasis.
• Thus, Obesity is NOT A DISORDER of WILLPOWER but it CAN be TREATED with CHANGES in BEHAVIOR
• You are on the side of the patient against the disease. Support and empathy are appropriate.
The Good News: Efficacy of 5-10% Weight Loss

- Prevent T2DM\(^1\)
- Increases life expectancy\(^2\)
- Improves glycemic control\(^3\)
- Lowers blood pressure\(^4\)
- Improves serum lipid profile\(^5\)
- May decrease cancer risk\(^6\)
- Improves lower back pain, reflux, lower extremity arthralgias, sleep apnea\(^7\)
- Can decrease drugs for obesity-related conditions\(^7\)

Obesity is a CHRONIC Disease

“Thermogenic adaptation reminds us that the metabolic vulnerability of individual with obesity persists even after their condition has supposedly been cured by weight loss.”

Angelo Tremblay
Health Care, Education and Research

State of Health

Lifestyle/Behavioral Level

Psychological/Motivational Level

Spiritual/Being/ Meaning Realm

© 1978, 1988, 2004 by John W. Travis, MD
Obesity in the Mature Population (Special Considerations)
Obesity Evaluation of the Elderly

Limitations of BMI calculations in the Elderly

- Height decreases → Overestimates BMI
- Body composition changes → Underestimates BMI
- BMI gradually increases most of adult life and peaks at 50-59 y/o
- Fat free Mass (muscle, organ tissue, skin, bones)

Health consequences of changing body composition

- Central distribution of fat → increased risk stroke, CAD, DM HTN, high lipid panel
- Redistribution of fat into muscles and liver → increase risk insulin resistance/DMT2

Best method: Waist Circumference, Waist to hip ratio, and Body composition

- Decrease in fat free mass (FFM)
- Increase in distribution of FM
- Decrease (up to 40%) with increasing years starting at 40-50 y/o
Natural Weight Gain as We Age

Due to Energy Imbalance

1. Lower basal metabolic rate
   a. Weight increase usually 1 kg/yr

2. Increased sedentary lifestyle
   a. Changes in life (e.g., retirement)
   b. Decreased PA (injuries, OA)
   c. Co-morbidities (COPD, CAD)

3. Medications
   a. Dizziness
   b. Fatigue

4. Other factors
   a. Behavioral
   b. Psychological
   c. Social
   d. Metabolic
Obesity increases:
  → disability and poor outcomes reducing QOL
  → increases demand for health care services

Co-morbidities:
1. HTN/DMT2/CVD
2. OSA
3. OA
4. Urinary incontinence
5. ED
6. Chronic pain
7. Frailty due to reduce mobility → Reduced ADL’s

Benefits of Elderly Obesity (older than 65)

1. May be protective for Dementia
2. Energy Reserves in case of illness
3. May be associated with reduced mortality (obesity paradox) *a
   1. Possible association with increased CHF (temp)
   2. Increased osteoporosis (possible)
   3. May or may not reduce stroke risk
4. Overweight (BMI 25-29) and Obesity category 1 (BMI 30-35) may be associated with lower risk of mortality.*b
5. HF patients → lower mortality in obese patients


Intentional Weight loss in the Elderly

Risks

• Malnutrition
• Loss of weight → loss of muscle mass and bone density
• Loss of metabolic reserves against disease

Benefits

• Improvement on metabolic disturbances
• Improve physical function and QOL
• Beneficial or neutral effect on mortality
Things to Remember when managing weight in the Elderly population

- 25% of diet induced weight loss will be FFM
- Adding endurance or resistance training will help preserve FFM (25% → 12% reduction in FFM)
- No loss of FFM after only 10% weight loss in 65-80y/o when regular PA was added.
- Bone density reduction of 1-2% if 10% WL over 4-18 months period.
- Exercise can attenuate bone loss if:
  - Calcium is supplemented and vitamin D is at goal (>40)
- Fitness can alter obesity paradox
Weight Loss Goals for the >65 yrs old population

Disability-free life expectancy greater \( \rightarrow \) BMI 25-30

Lowest rate of decline of physical function \( \rightarrow \) 23-30

Emphasize Exercise \( \rightarrow \)
Improve strength, balance, flexibility, endurance, minimize bone/muscle loss, and prevent decline

Modest weight reduction \( \rightarrow \) 5-10% WL

TARGET BMI \( \rightarrow \) 25-30

Al Snih S et al., The effect of obesity on disability vs mortality in older Americans. Arch Inter Med 2007; 774-780.

Oreopoulos, 2009
Patient Evaluation
5 A’s of Obesity Management

Ask
- Ask for permission to discuss body weight.
- Explore readiness for change.

Assess
- Assess BMI, waist circumference, and obesity stage.
- Explore drivers and complications of excess weight.

Advise
- Advise the patient about the health risks of obesity, the benefits of modest weight loss (i.e., 5-10 percent), the need for long-term strategy, and treatment options.

Agree
- Agree on realistic weight-loss expectations, targets, behavioral changes, and specific details of the treatment plan.

Arrange/Assist
- Assist in identifying and addressing barriers; provide resources; assist in finding and consulting with appropriate providers; arrange regular follow up.

Reference/s: [113] [115] [116]

Four Pillars of Weight Management
Four Pillars of Weight Management

- Metabolism
- Nutrition
- Physical Activity
- Behavior
Weight History

• Weight growing up (childhood, MS, HS, college years)
• Pregnancies (?GDM, toxemia)
• Peak Adult weight (more than one)
• Major stressors (deaths, divorce, etc)
• Longest time at certain weight
• Major weight loss, type of regimen, ?success
*Potency includes many factors, such as the amount, rate, and sustainability of weight loss, and the long-term resolution of adiposopathy and fat mass disease. Potency varies greatly for each individual (i.e., long-term adherence to a lifestyle program can be as potent as gastric bypass surgery).

Surgery
(In order of lowest risk/cost and potency):
LAGB<VSG<RNY

Very Low Calorie Diet

Lifestyle + Medication
Includes lifestyle, and anti-obesity medications

Lifestyle
Includes nutrition, physical activity, and behavioral programs

Risk/Cost

Potency*
METABOLISM
Contributing Factors to Obesity

- DIET
  - Sleep Disruption
  - Medications
  - Genetics/Epigenetics
- Obesity
  - Stress
  - Other medical problems (thyroid, depression)
  - Hormones
  - Sedentary Lifestyle
- Other medical problems (thyroid, depression)
Review of System

- Cardiovascular
  - HTN, CHF, PE
- Neurologic
  - Stroke, fibromyalgia
- Psychiatric
  - Body Image, impaired QOL, Depression
- Respiratory
  - SOB, OSA, Asthma
- Endocrinology
  - Metabolic syndrome, T2DM, PCOS, Dyslipidemia, infertility
- Musculoskeletal
  - Gout, immobility, LBP
- Integument
  - Striae, cellulitis, acanthosis, intertrigo
- Gastrointestinal
  - GERD, NAFLD, gallbladder, hernia
- Genitourinary
  - Stress incontinence, kidney stones, breast cancer
Metabolism-Related Labs

- **Thyroid** → TSH, free T4
- **Insulin resistance / Diabetes** → fasting insulin, A1C, CMP (monitor trends)
- **Hyperlipidemia** → Trig, HDL
- **Hypertension** → BUN, creat, GFR
- **NAFLD/NASH** → AST, ALT, hCRP
- **Sleep apnea** → STOP BANG, Sleep study (AHI)
- **Vitamin Deficiencies** → Vit D, Vit B12, Iron studies
Concurrent Medications
# Weight Gain Promoting Medications

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-depressants</td>
<td>Tricyclics: trazodone, nortriptyline, amitriptyline</td>
</tr>
<tr>
<td></td>
<td>SSRIs: paroxetine, citalopram, escitalopram</td>
</tr>
<tr>
<td></td>
<td>Others: mirtazapine, venlafaxine</td>
</tr>
<tr>
<td>Diabeties treatments</td>
<td>Insulin</td>
</tr>
<tr>
<td></td>
<td>Sulfonylureas: glipizide</td>
</tr>
<tr>
<td></td>
<td>Thiazolidinediones: pioglitazone</td>
</tr>
<tr>
<td>Atypical antipsychotics</td>
<td>Clozapine, olanzapine, quetiapine, risperidone</td>
</tr>
<tr>
<td></td>
<td>aripiprazole</td>
</tr>
<tr>
<td>Anti-hypertensives: Betablockers</td>
<td>Propranolol, metoprolol, atenolol</td>
</tr>
<tr>
<td>Anti-epileptics</td>
<td>Gabapentin, valproic acid</td>
</tr>
<tr>
<td>Anti-histamines</td>
<td>Diphenhydramine, hydroxyzine, cetirizine, fexofenadine</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Lithium</td>
</tr>
<tr>
<td>Glucocorticoids</td>
<td>Prednisone, methylprednisone</td>
</tr>
<tr>
<td>Other</td>
<td>Tamoxifen, HAART</td>
</tr>
<tr>
<td>Hormones</td>
<td>Depoprovera</td>
</tr>
</tbody>
</table>
## In Management of Diabetes, consider substitutions...

<table>
<thead>
<tr>
<th>Potential Weight Gain</th>
<th>Weight Neutral / Potential Weight Loss or Less Gain</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Insulins</strong></td>
<td>Metformin</td>
</tr>
<tr>
<td>NPH &amp; Regular</td>
<td>Pramlintide (Symlin)</td>
</tr>
<tr>
<td>Glargine, Lispro, Aspart</td>
<td></td>
</tr>
<tr>
<td><strong>Sulfonylureas</strong></td>
<td>Exenatide (Byetta, Bydureon)</td>
</tr>
<tr>
<td>Glyburide</td>
<td>Liraglutide (Victoza)</td>
</tr>
<tr>
<td>Glipizide</td>
<td></td>
</tr>
<tr>
<td>Glimepiride</td>
<td>Sitagliptin (Januvia)</td>
</tr>
<tr>
<td><strong>Glinides</strong></td>
<td>Saxagliptin (Onglyza)</td>
</tr>
<tr>
<td>Nateglinide, Repaglinide</td>
<td></td>
</tr>
<tr>
<td><strong>TZDs</strong></td>
<td>Dapagliflozin (Forxiga)</td>
</tr>
<tr>
<td>Pioglitazone, Rosiglitazone</td>
<td></td>
</tr>
<tr>
<td><strong>GLP-1</strong></td>
<td></td>
</tr>
<tr>
<td><strong>DPP-IV</strong></td>
<td></td>
</tr>
<tr>
<td><strong>SGLT2</strong></td>
<td></td>
</tr>
<tr>
<td><strong>a-glucosidase inhib (Acarbose)</strong></td>
<td></td>
</tr>
</tbody>
</table>
Nutritional Therapy for Obesity
Choosing Nutritional Therapy for Obesity

The most appropriate nutritional therapy for weight loss should be safe, effective, and one to which the patient can adhere.

- Encourage foods that result in a negative caloric balance to achieve and maintain a healthy weight
- Consider the following:
  - Individual food preferences, eating behaviors, and meal patterns
  - Cultural background, traditions, and food availability
  - Time constraints and financial issues
  - Nutritional knowledge and cooking skills
Lifestyle Changes for Weight Management

• Lifestyle change is essential to successful weight management.
  – Eating balanced meals with lean protein, whole grains, moderate healthy fats and fresh fruits and vegetables.
  – Eating frequently during the day, not skipping meals or going long periods of time (>4-5 hours) without eating.
  – Monitoring portion sizes.
  – Drinking 64 oz of water daily
  – Getting at least 150 minutes per week of moderate physical activity.
Physical Activity
Medical Evaluation to Ensure Safety before Beginning New Exercise Program

- Assess current physical activity level
- Assess readiness
- Assess potential need for medical testing/evaluation (i.e., cardiac stress testing, pulmonary function tests, musculoskeletal assessment, etc.)
- Assess mobility, fitness, and potential equipment needs or modifications
- Potential adjustment of medications
  - Before start of physical activity plan
  - During implementation of physical activity plan
Assess Mobility

**Unable to Walk**
- Seated exercise program
- Arm exercises (i.e., arm cycling)
- Swimming/aquatic exercises (e.g., shallow or deep water exercises)
- Gravity-mediated physical activity
- Consider physical therapy evaluation
  - Recommend rehabilitation & physical therapy guided activity program
  - Set physical activity goals
  - Assess special equipment needs

**Limited Mobility, Able to Walk**
- Walking
- Swimming/aquatic exercises (e.g., shallow or deep water exercises)
- Gravity-mediated physical activity
- Assess for special equipment needs

**No Substantial Limitations to Mobility**
- Exercise/physical activity prescription plan driven by patient and guided by clinician
- Assess for special equipment needs

Reference/s: [191] [192]
Work to fill both “buckets” of activity

Exercise prescription
(FITTE)
- **F**requency
- **I**ntensity
- **T**ime spent
- **T**ype
- **E**njoyment level

**Physical Activity**
- Work outs
- Weights

**Daily Activity**
- NEAT
- Chores

**Work out**: At least 10 minutes 3 times per day, 4-5 times per week at a moderate to vigorous intensity (raising heart rate).

**NEAT**: Non-exercise activity of thermogenesis (calorie burning) – adding steps to your day during your regular activities.
Behavior Therapy
YouCan
STAGES OF CHANGE

Relapse
Falling back into old patterns, actions and behaviours. Each relapse is met with new insights and knowledge leading to less frequency in setbacks.

Pre-Contemplation
Not thinking about or has rejected change.
Living in Harms Way

Contemplation
Thinking and talking about change. Seeks out support.
Tired of Living in Harms Way

Maintenance
Achieving positive and concrete developments with continuing and potentially little support.
Living Out Of Harms Way

Action
Taking positive steps by putting the plan into practice.
Gradually Moving Out of Harms Way

Planning
Planning what it would take to make change happen.
Strategizing How to Move Out of Harms Way

Adapted from Prochaska & DiClemente and Ignacio Pacheco | YOUCAN 2012
Why Do People Eat Like They Do?

Environment
- Others are eating
- Food is available
- Offers of free food
- Perceived obligations
  - Family gatherings
  - Business meetings
  - Clean-plate syndrome

Reward
- Eating as a remuneration for a good accomplishment
- Eating as compensation for a bad day
- Eating for pleasure, not because of hunger
- Over-consumption of palatable food may affect the brain’s reward system
  - Stimulates opioid release
  - Decreases biologic stress response
  - May ultimately simulate addiction-like reward deficits, which promotes compulsive eating
Why Do People Eat Like They Do?

**Timing and Emotions**

- **Timing**
  - It’s mealtime
  - Special occasions
  - Holidays
- **Emotions**
  - Celebrate happiness
  - Soothe sadness
  - Surrogate for love and/or affection
  - Treat:
    - Boredom
    - Fatigue
    - Stress

**Physiologic**

- Hunger before meals
- Lack of satiety after meals
- Five senses:
  - Sight of food
  - Smell of food
  - Hear talk of food or sound of cooking food
  - Taste of food
Behavior Therapy Techniques: Elements for Optimal Success

**Doable**
- Practical
- Accessible
  - Frequency
  - Consistency

**Efficacious**
- Evidence-based

**Measurable**
- Feedback
- Trackable
- Verifiable

**Self-ownership**
- Autonomous stakeholder
- Personal stakeholder
  - Positive reinforcement
  - Negative reinforcement

Obesity Pharmacotherapy
When to consider use of Anti-Obesity Medications

1. BMI indications: **BMI ≥ 30 or BMI ≥ 27 + CoM**
2. **Maximize** behavioral modification and stabilize weight prior to medication trial
3. Continue medications only in responders
4. Use combinations if monotherapy does not give desired results
5. **Long term use**
High Responders lose >5% in 3-months

Start low dose

F/U 1 month

F/U 3 months

Adverse effects

DC or ↓ dose

<3-5%
<12 lbs
@max dose

>3-5%
>12 lbs

Weight Loss

Drugs with Long term Use Criteria
• Lorcaserin
• Phentermine/Topiramate
• Liraglutide
• Naltrexone/Bupropion

D/C
Use alternative med

Continue
Same dose

F/U 3 mo
Current Anti-Obesity Medications

- **Adrenergic**
  - Phentermine
  - Phentermine/Topiramate
  - Diethylpropion
  - Phendimetrazine
  - Bupropion
  - Naltrexone/Bupropion

- **Anti-epileptic**
  - Topiramate
  - Zonisamide

- **Anti-diabetic**
  - Metformin
  - Exenatide
  - Canagliflozin (Dapa-, Empa-)
  - Pramlintide
  - Liraglutide

- **Other**
  - Orlistat
  - Lorcaserin
Treatment of Obesity
It takes time to make the right choice
HAVE TO TRY THE SHOE ON TO SEE IF IT’S COMFORTABLE”
What Should We Do?

- Practice “Obesity Medicine,” not “Weight Loss”
- Recognize the complexities of Obesity as a Disease
- Acknowledge the difficulty of achieving significant weight loss and maintenance
- Recognize that small weight loss can result in meaningful health gains and manage expectations
- Reinforce healthy behaviors, rather than over-focusing on the number on the scale (pounds)


