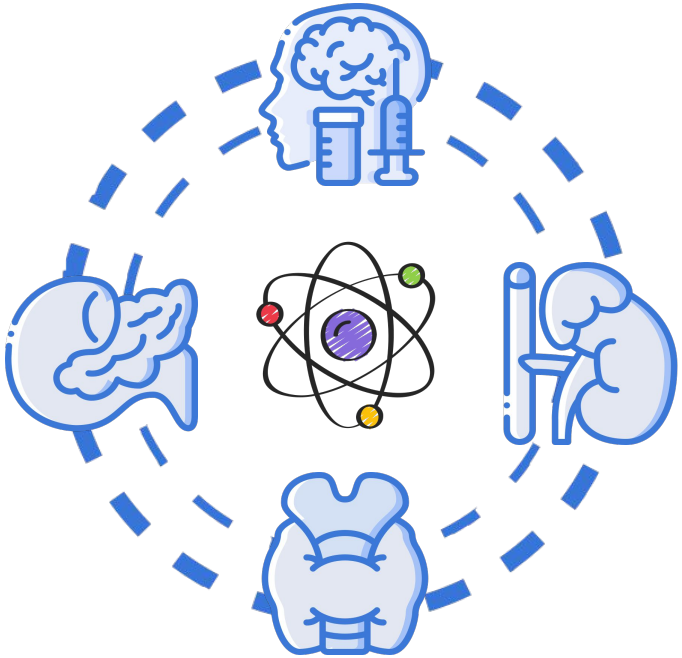
Obesity



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Objectives:

- Define and characterize obesity in terms of BMI and risk factors.
- Compare the anatomic and biochemical differences in fat deposition.
- Understand the role of adipocytes in fat storage and release of hormones.
- Discuss the hormonal control of obesity by leptin, adiponectin and other hormones.
- Discuss the management and treatment options for obesity.

Overview:

- Introduction & Body Mass Index (BMI).
- Types of fat deposition in the body & Metabolic changes in obesity.
- Adipocytes (fat cells) and weight gain & Hormones in obesity & Treatment options.

Obesity

A disorder of body weight regulatory systems

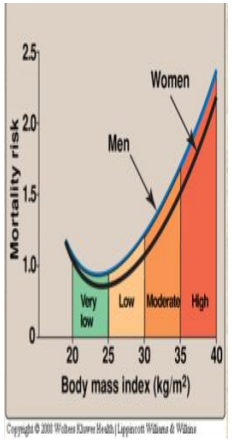
Causes accumulation of excess body fat

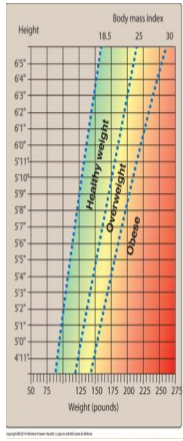
>20% of normal body weight

Body Mass Index

- BMI is an indirect measure of obesity
- Correlates height, weight and amount of body fat in an individual
- High BMI is associated with increased mortality risk

BMI	GRADE
UNDERWEIGHT	≤ 18.5	
NORMAL	18.5 - 24.9	
OVERWEIGHT	25.0 - 29.9	
OBESE	30.0 - 34.9	I
OBESE	35.0 - 39.9	II
HIGHLY OBESE	≥ 40	III





Obesity is associated with a high risk of:

- Diabetes mellitus
- Hypercholesterolemia
- High plasma triglycerides
- Hypertension
- Heart disease
- Cancer
- Gallstones, arthritis, gout
- Mortality

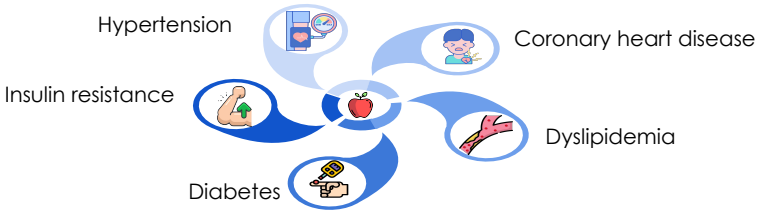
Anatomic Differences in Fat Deposition:

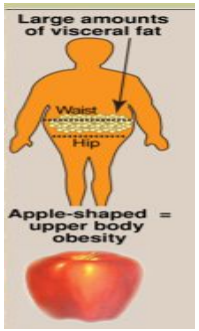
(Health risks depend on the pattern of fat deposition)

Android, "apple-shaped", or upper body obesity

- excess body fat deposited in the central abdominal area

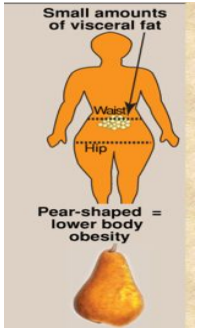
Associated risk factors are:





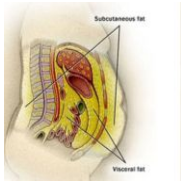
Gynoid, "pear-shaped", or lower body obesity

- Fat deposited around the hips or gluteal region
- Associated risks are lower



Different Fat Depots in The Body

Subcutaneous Fat	Visceral Fat
The fat stored just under the skin in the abdominal and gluteal-femoral region	Composed of omental and mesenteric fat
Constitutes 80-90% of the total fat in the body	Present in close association with digestive tract



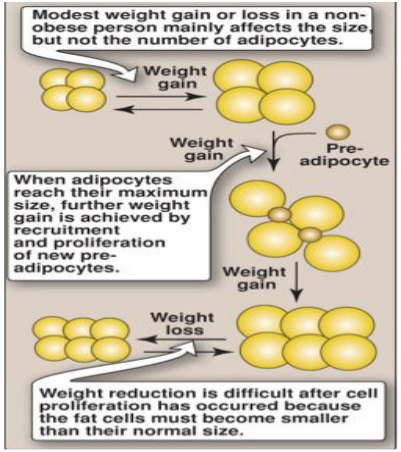
Biochemical Differences in Fat Deposits

Abdominal Fat	Gluteal Fat
Smaller cells	Larger cells
More responsive to hormones (both visceral and subcutaneous)	Less responsive (subcutaneous)
Release substances via portal vein to the liver	Release substances to circulation with no effect on the liver

Adipocytes

Prolonged overnutrition stimulates → Pre-adipocytes in adipose tissue → Proliferation / differentiation into mature fat cells → Increases adipocyte number

- Triacylglycerols (fats) are deposited in adipocytes (fat cells) which can increase in size up to a limit.
- Thus obesity is due to a combination of increased fat cell size (hypertrophy) and number(hyperplasia).
- Fat cells, once gained, are never lost
- Reduction in weight causes adipocytes to reduce in size but not in number



Ectopic fat

Excessive calories that cannot be stored in adipose tissue "spill over" into other tissues such as muscle and liver
- It is called "ectopic fat" that is strongly associated with insulin resistance

Factors contributing to obesity

Genetic: familial tendency

Environmental and behavioral
- Sex: women more susceptible
- Activity: lack of physical activity
- Psychogenic: emotional deprivation/depression
- Alcohol: problem drinking
- Smoking: cessation of smoking

Drugs: e.g. tricyclic derivatives

Causes of Weight Gain

More in and less out = weight gain

More out and less in = weight loss

Hypothalamus: control center for hunger and satiety

Endocrine disorder: Hormonal imbalance

Energy imbalance

calories consumed not equal to calories used over a long period of time due to a combination of several factors:

- Individual behaviors
- Genetics
- Social interactions
- Environmental factors

Hormonal Control

Appetite is influenced by:

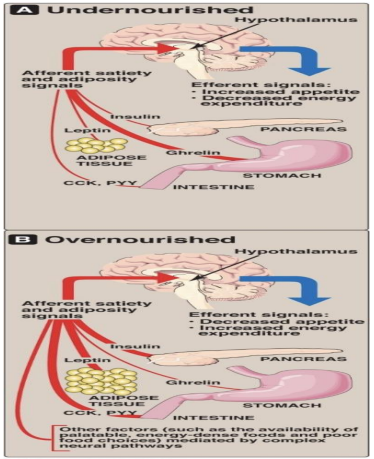
- Afferent neural signals
- Circulating hormones
- Metabolites

These signals cause the release of hypothalamic peptides and activate efferent neural signals

Adipocytes also function as endocrine cells

They release many regulatory molecules:

- Leptin
- Adiponectin
- Resistin



Explanation of the picture:

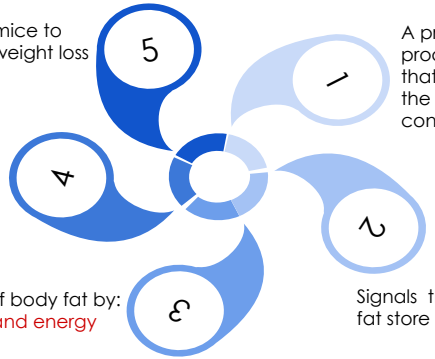
A) Undernourished state: afferent signals are sent to satiety center in hypothalamus by:
- Stomach which increase the secretion of Ghrelin to increase the appetite.
- Pancreas which decreases the secretion of Insulin so there'll be decrease in metabolism.
- Intestine which decreases the secretion of CCK.
- Adipocyte which decreases the secretion of Leptin so there'll be decrease in weight-loss.

Hypothalamus then send efferent signals to increase appetite & decrease energy expenditure.

B) Overnourished: the opposite happens.

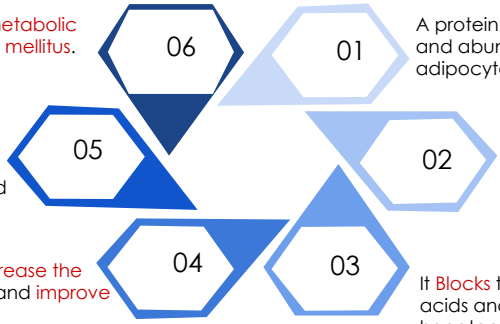
Adipocyte hormones

Leptin hormone



1. A protein hormone produced by adipocytes that is required to keep the body weight under control
2. Signals the brain about fat store level
3. Regulates the amount of body fat by: Controlling appetite (↓) and energy expenditure (↑)
4. Leptin secretion: Suppressed in starvation (depletion of fat stores) Enhanced in well-fed state (expansion of fat stores)
5. Leptin causes overweight mice to lose weight and maintain weight loss

Adiponectin hormone



01. A protein hormone exclusively and abundantly secreted from adipocytes
02. It promotes the uptake and oxidation of fatty acids and glucose by muscle and liver.
03. It Blocks the synthesis of fatty acids and gluconeogenesis by hepatocytes.
04. It net effect is to increase the sensitivity to insulin, and improve glucose tolerance.
05. Adiponectin levels are inversely correlated with body fat percentage and parallels with the HDL level.
06. Low levels are seen in metabolic syndrome and diabetes mellitus.

Leptin Resistance

Leptin resistance may have some role in human obesity:
- Dieting decreases leptin level
- Reducing metabolism, stimulating appetite

1. Leptin ↑ metabolic rate and ↓ appetite in human
2. Plasma leptin level in obese humans is usually normal for their fat mass
3. Resistance to leptin has been found in obese humans
4. The receptor for leptin in the hypothalamus is produced db gene
5. Mutation in db gene causes leptin resistance in mice
6.

1: Obese people have the normal amount of Leptin, but they have a delayed Leptin response, It's helpful to tell the patient to take a small break in the middle of eating a meal to allow leptin secretion.
2: The person either can't produce enough leptin due to genetic abnormality and they become obese, so if you give them leptin they will respond and lose weight **OR** the receptors are defected, so the person has enough leptin but no usage and this is called "leptin resistance".

Other Hormones

Ghrelin	A peptide hormone is secreted by stomach, the secretion increases just before meals and drops after meals. It **stimulates appetite** & **increases food intake** & **decreases energy expenditure and fat catabolism**. Levels in dieters are higher after weight loss: the body steps up ghrelin production in response to weight loss. the higher the weight loss, the higher the ghrelin levels.
Cholecystokinin	Peptides released from the gut after a meal, sends satiety signals to the brain.
Insulin	Promotes metabolism (& suppresses the appetite immediately).

Metabolic Changes in Obesity

Adipocytes send signals that cause abnormal metabolic changes such as:

01 Dyslipidemia

02 Glucose Intolerance

03 Insulin Resistance

Benefits of Weight Loss in Obesity

Slow weight loss is more stable &
Weight loss decreases risk factors for obesity leading to:

- ↓ Lower blood pressure.
- ↓ Serum triacylglycerols.
- ↓ Blood glucose levels.
- ↓ mortality.
- ↓ Energy requirement.
- ↑ HDL levels.
- Beneficial changes in BMR.

Treatment Options

Physical activity combined with healthy diet decreases the level of obesity & reduces risk for CVD and DM.

Dieting: use of low-calorie diet & restriction of excessive energy intake

Drugs:

- **Orlistat**: A pancreatic and gastric lipase inhibitor which decreases the breakdown of dietary fat.
- **Lorcaserin**: promotes satiety.

Surgery: Surgical procedures are designed to reduce food consumption in patients with **BMI >40.** It's used when other treatment options **fail**.

Take Home Messages

Obesity is correlated to an increased risk for a number of chronic conditions and mortality

Summary

Fat deposition (Shape/Anatomical)	
Android / Apple-shaped	**Gynoid / Pear-shaped**
Fat deposited in the central abdominal area, has more risk.	Fat deposited around the hips or gluteal region, has less risk.
Fat deposition (Type)	
Subcutaneous	**Visceral**
Fat stored just under the skin, in abdominal & gluteal regions, constitutes 80-90% of total fat.	Composed of omental and mesenteric fat, has close association with digestive tract.
Fat deposition (Location/Biochemical)	
Abdominal	**Gluteal**
Smaller cells, more responsive to hormones (both visceral and subcutaneous), release substances via portal vein to liver.	Larger cells, less responsive (subcutaneous), release substances to circulation with no effect on the liver.
Ectopic fat	
Excessive calories that cannot be stored in adipose tissue spill over into other tissues (e.g. muscle & liver), it's strongly associated with insulin resistance.	

Hormonal control

Leptin: decreases appetite, increases energy expenditure, helps in weight loss, it's enhanced in well-fed state.

Adiponectin: promotes oxidation and uptake of FA & glucose by muscle & liver, blocks gluconeogenesis & FA synthesis, increases sensitivity to insulin, improves glucose tolerance.

Ghrelin: stimulates appetite, increases food intake, decreases energy expenditure and fat catabolism, it's enhanced in starvation & weight loss.

Cholecystokinin: released after a meal, sends satiety signals to the brain.

Insulin: suppresses appetite & promotes metabolism.

Quiz

MCQs :

Q1: Which of the following is CORRECT about Leptin hormone:

a) it increases the appetite **b)** it decreases the energy expenditure
c) it's suppressed in starvation **d)** bd gene mutation cause Leptin resistance

Q2: Which of the following is CORRECT about Adiponectin hormone:

a) high level is seen in diabetes **b)** improves glucose tolerance
c) block oxidation of fatty acids **d)** promote gluconeogenesis

Q3: Which of the following hormones decrease the energy expenditure:

a) CCK **b)** Insulin **c)** Leptin **d)** Ghrelin

Q4: A man's BMI is 36.9 what grade of obese is he according to the measurement

a) I **b)** II **c)** III **d)** IIII

Q5: Modest weight gain or loss in non obese person effects:

a) the size and not the number of adipose tissue
b) the number and not the size of adipose tissue **c)** none of the above

Q6: Abdominal fat is:

a) large cells **b)** subcutaneous and visceral
c) Subcutaneous only **d)** no effect on liver

SAQs :

Q1: List 3 adipokines secreted by the adipocytes

Q2: List 3 metabolic changes that happens is obesity

Q3: List the difference between subcutaneous fat and visceral fat

Q4: List the different anatomical fat deposition and the risks associated with each one

★ **MCQs Answer key:**

1) C 2) B 3) D 4) B 5) A 6) B

★ **SAQs Answer key:**

1) Leptin, adiponectin and resistin.
2) Dyslipidemia, Glucose intolerance and Insulin resistance.
3) Slide No.4
4) Slide No.4

Team members

Girls Team:

- **Ajeed Al-Rashoud**
- Alwateen Albalawi
- Amira AlDakhilallah
- Deema Almaziad
- Ghaliah Alnufaei
- Haifa Alwaily
- **Leena Alnassar**
- Lama Aldakhil
- Lamiss Alzahrani
- Nouf Alhumaidhi
- Noura Alturki
- Sarah Alkhalife
- Shahd Alsalamah
- Taif Alotaibi

Boys Team:

- Alkassem Binobaid
- Fares Aldokhayel
- Khayyal Alderaan
- Mashal Abaalkhail
- Naif Alsolais
- Omar Alyabis
- Omar Saeed
- Rayyan Almousa
- Yazen Bajeaifer

Team Leaders

Lina Alosaimi

Mohannad Alqarni

★ **What doesn't kill you makes you stronger.**
- Kelly Clarkson

We hear you