

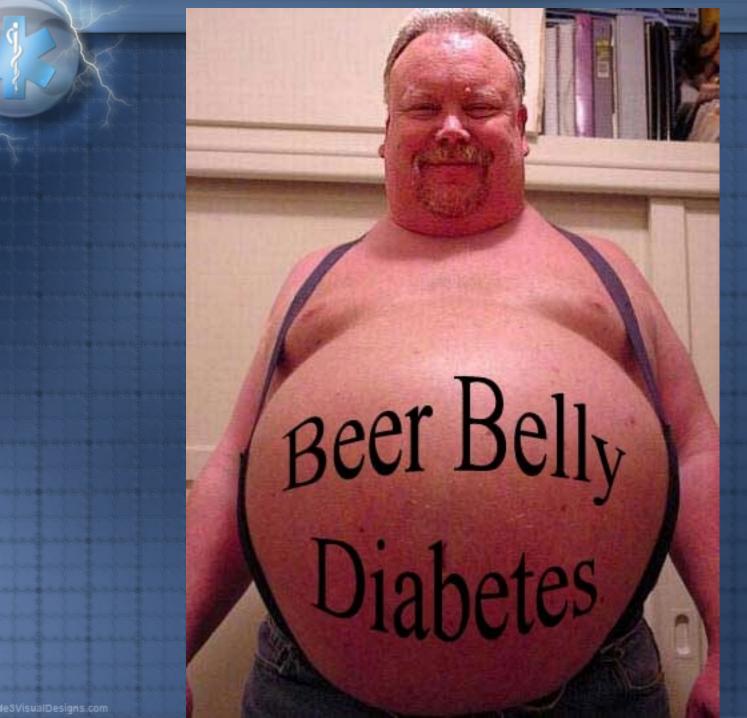
Bradley Dean, MA, NRP
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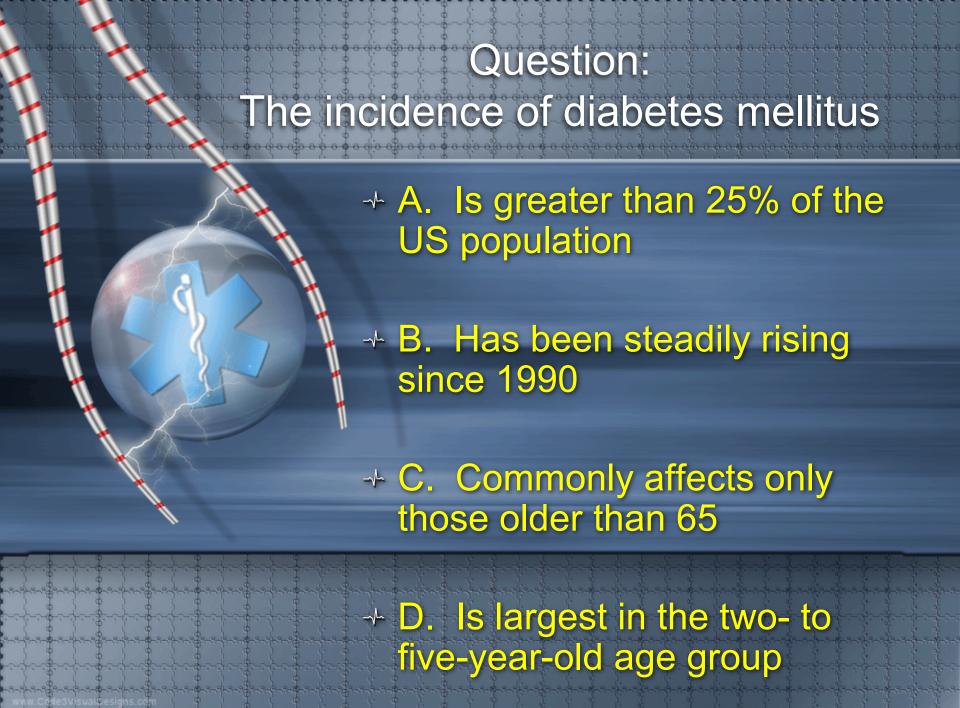
Being A Sugar Daddy doesn't mean you have Diabetes

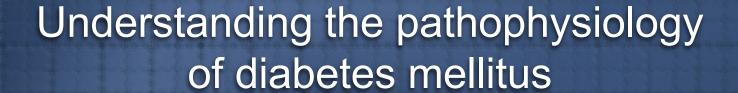












Approximately 30.3 million Americans have diabetes, which translates to about 9.4% of the US population.

More than 7.2 million people likely have the condition but remain undiagnosed (23.8% of population).

Many patients are hyperglycemic for as long as six years before the disease is detected.

The CDC reports the incidence of DM has risen by 70% in adults between the ages of 30-39; by 40% in those 40-49; and by 31% in those 50-59 years of age.

PREDIABETES

84.1 MILLION



84.1 million people —
more than 1 out of 3 adults —
have prediabetes



g out 10 don't know they have prediabetes



If you have prediabetes, losing weight by:





can cut your risk of getting type 2 diabetes in

HALF







\$245 BILLION

Total medical costs and lost work and wages for people with diagnosed diabetes

Risk of death for adults with diabetes is



50%



than for adults without diabetes

Medical costs
for people with
diabetes are
more than
twice as high



\$ as for people without diabetes

2X



People who have diabetes are at higher risk of serious health complications:











- - → NY 9.7% of the population has Diabetes
 - → NJ 8.1% of the population has Diabetes
 - → NC 11% of the population has Diabetes

- → Education Does it make a difference?
 - The population of those with less than a high-school education soars to 16%



Halle Berry



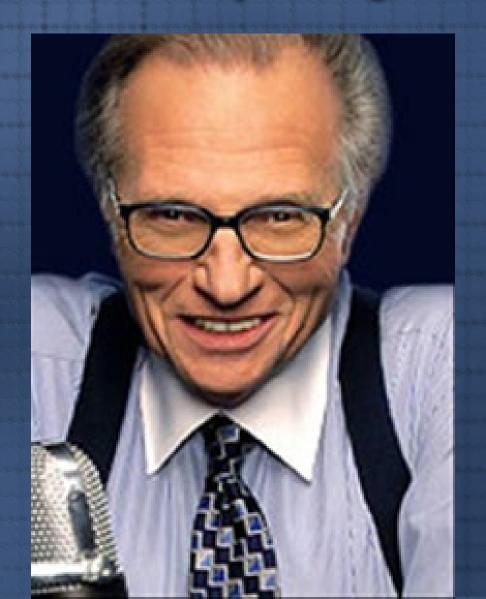


Gary Hall Jr.





Larry King



Nick Jonas





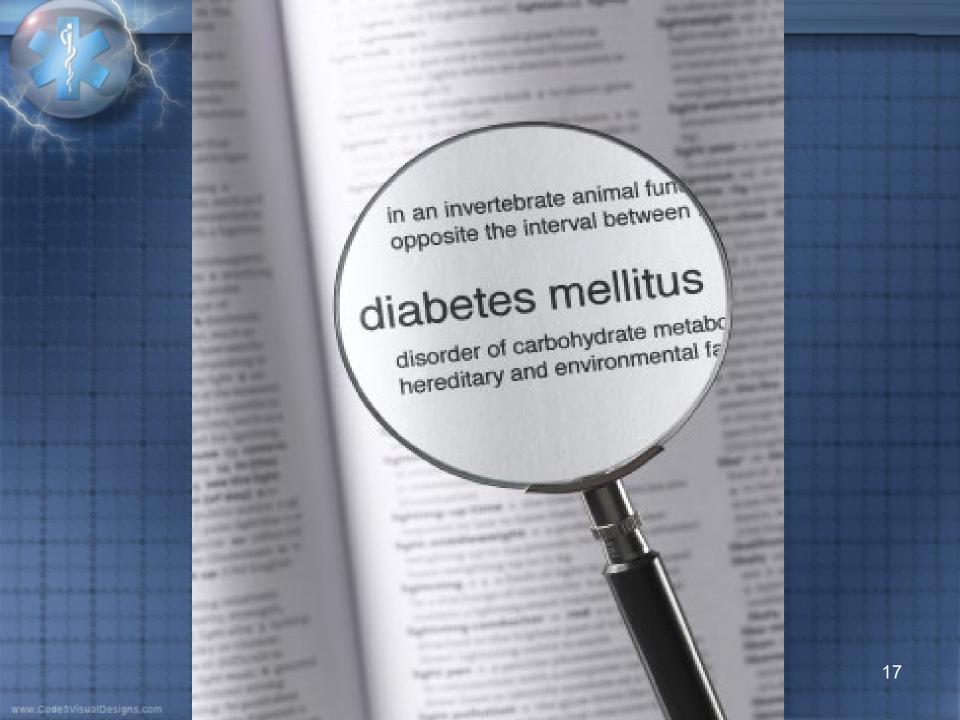
Mikhail Gorbachev





Morgan Freeman







DM

→ DM was named in ancient times by Greek physicians who observed that affected persons produced large amounts of urine that attracted bees and other insects.

→ Diabetes means "to siphon" or "to pass through" and mellitus means "honey sweet," a reference to the sugar in the urine.



DM

Defined as a group of chronic metabolic diseases characterized by hyperglycemia

Resulting from defects in insulin secretion, and metabolism function

Resulting in abnormal carbohydrate, fat, and protein metabolism

Glucose metabolism

Sources of glucose have to be available and ready to meet cellular metabolic demands, and we readily get this from our diet.

Liver

Blood glucose

Insulin

Pancreas

- Two additional methods of maintaining constant glucose levels:
 - Gluconeogenesis
 - → Glycogenolysis

Stuconegensis (liver alucose (blood) 0 - esp active after 24 acute rea -· hormones (VFZGBP) insulin infasting quicose 6 9 G6 Pase = alucagon (transcription) long term · Enzyne levels altered glucagon extrate Zenergy CAMP s charge Muscle F26,8P F1,6BP alucagon Palucocorticoid Ketogluk Ala glutamak ox-ketoy Adipocute glucagon 4 gluca gon transcription by Adyna (rate limiting TCAMP NADH NADT from muscle) NAL Malate 4DKA WADH MA ATP, CHO, ADP TAG FFA Ac COA PC (bidin) MADH NADT FA COA (transcript ALCOA NADH

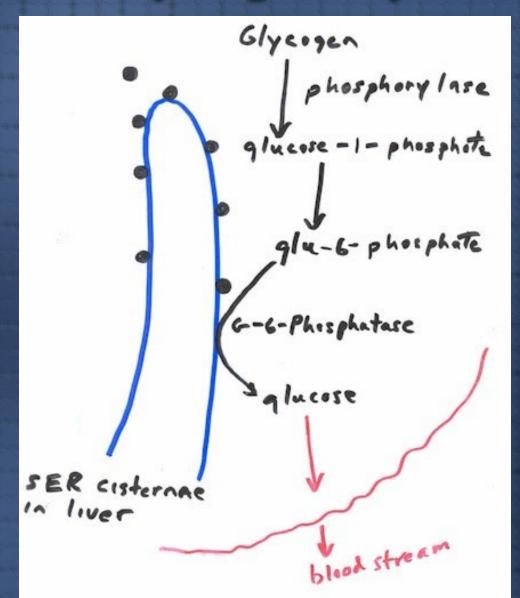


Glucose metabolism

- → Gluconeogenesis:
 - New glucose molecules are produced from non-sugar sources in the liver

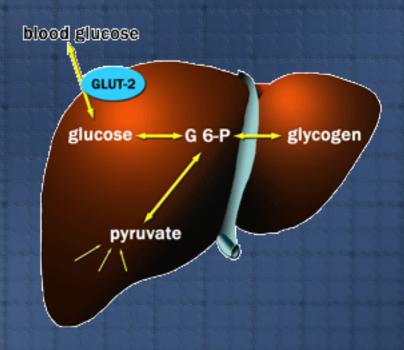


Glycogenolysis



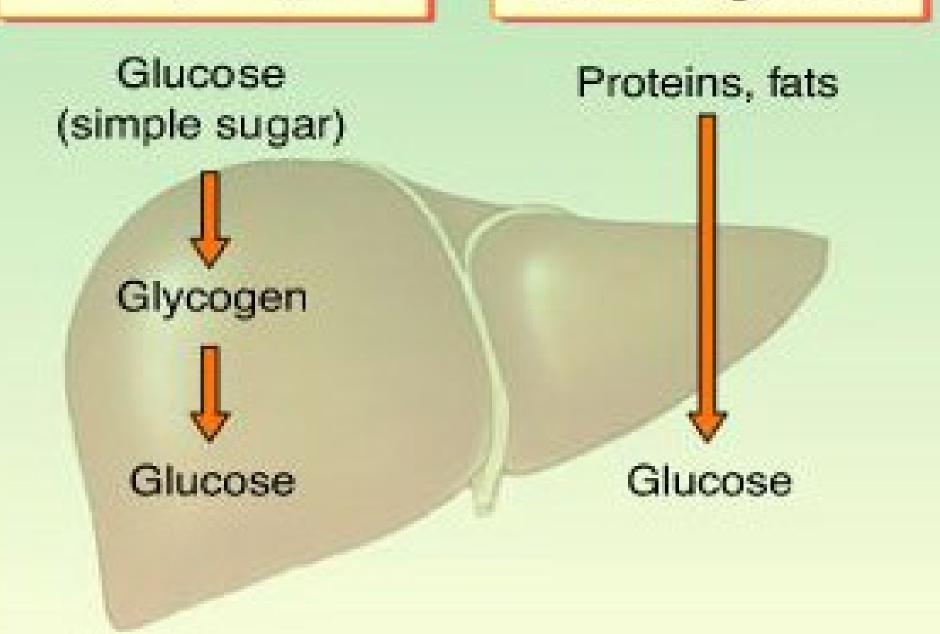
→ Glycogenolysis:

The liver breaks down stored glycogen to its component glucose molecules



Glycogenolysis

Gluconeogenesis





Question

- Glycogen breakdown into glucose molecules by the liver is called:
 - → Metabolism
 - → Glycogenolysis
 - → Glycosis
 - → Gluconeogenesis

Glucose metabolism

Serum glucose fluctuates based on the time of day, food or beverage ingested, stress level, exercise and hormone activites

→ Glucose homeostasis is achieved through the interaction of insulin, glucagon, cortisol, catecholamines, growth and other hormones and their subsequent effects on hepatic, fat and muscle cells.

Glucose metabolism

→ 70 – 120 mg/dL

→ The lowest levels occur when food has not been eaten for a number of hours

→ The highest levels occur one to two hours after eating...especially if a meal contained a high carbohydrate load



Mechanisms governing metabolism

→ Pancreas

- Located behind the stomach and between the duodenum and spleen
- Largely responsible for maintaining a specific blood glucose
- Regulates glucose metabolism through the release of three hormones from Islets of Langerhans

Pancreas Hormone Secretions

→ Islet cell type and %

→ Alpha 25%

→ Beta 60%

→ Delta 10%

Hormone secreted

Glucagon: secreted in response to decreased blood glucose levels to increase blood sugar

Insulin (antagonist of glucagon): secreted in response to increased blood glucose levels to decrease blood sugar

Somatostatin: inhibits secretion of insulin and glucagon

→ Insulin

- Carbohydrates convert to simple sugars stimulating insulin secretion (high-insulin/low-glucagon state)
- Glucose must enter the cell large molecule needing a facilitator (facilitated diffusion)
- → The glucose attaches to the receptor site insulin opens the gate glucose enters
- Cardiac, skeletal, and fat tissues need insulin to actively transport glucose into the cell – used by the mitochondria to produce adenosine triphosphate (ATP)

→ The brain uses more glucose than any other organ system....but its uptake of glucose is not insulin-dependent

- Insulin prompts liver cells to stop producing glucose and convert excess to glycogen for storage in the liver and muscle cells.
- → It is the only hormone that leads to lipogenesis.
- Insulin also stimulates the use of amino acids for protein synthesis and prevents tissues from catabolizing – anabolic hormone

Insulin has a half-life of only minutes

Healthy liver removes circulating insulin within 10-15 minutes from the time of secretion

A normal pancreas must continuously produce small amounts of insulin to control excess glucose output and keep levels constant

Factors that Control Insulin Release

- Factors that stimulate insulin release
 - Increased blood glucose levels
 - Presence of ketone bodies or free fatty acids
 - → Glucagon release
 - → Gastric secretions
 - → hyperkalemia

- Factors that inhibit insulin release
 - → Hypoglycemia
 - → Hypokalemia
 - → Hydrochlorathiazide
 - → Beta and calciumchannel blockers
 - → Phynytoin (Dilantin)
 - → Alcohol

Glucagon (gluco = glucose; agon = to drive)

In a fasting state, serum glucose drops suppressing insulin secretion and causing the release of glucagon.

Glucagon serves as the on switch, causing glycogen in the liver to break down to glucose (glycogenolysis)



Glucagon

- Also serves as the *on switch* for the hepatic *ketogenic pathway*, where fatty acids convert into ketoacids and ketone bodies, which are oxidized for energy (gluconeogenesis).
- → The activation of lipolysis and the ketogenic pathways serve as a feedback loop stimulus for insulin secretion.

→ The initial fall in insulin, followed by increased glucagon release, stimulates additional insulin secretion protecting the body from ketoacidosis, and hyperglycemia in non-diabetics.

→ The link between carbohydrate and lipid metabolism is of great significance in uncontrolled diabetes.



Somatostatin

Also called growth-hormone-releaseinhibiting hormone inhibits insulin and glucagon secretion keeping them in balance → Epinephrine stimulates hepatic glucose production, stimulates glucagon secretion, suppresses insulin secretion, inhibits peripheral glucose use and stimulates lipolysis.

Cortisol functions much like glucagon, but with less potency.



Diabetes classifications

- Eliminate the confusing terms IDDM and NIDDM
- → Replace IDDM with type 1 diabetes
- → Replace NIDDM with type 2 diabetes
- → Retain the term gestational diabetes mellitus (GDM)
- Add impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) as risk categories for DM.



Type 1 diabetes mellitus

- → Type 1 accounts for 5-10% of all diagnosed
- Genetic, environmental, and autoimmune factors
- Insulin-producing islet cells are depleted or destroyed in an autoimmune response to a viral or environmental insult.
- Characterized by an absolute lack of functioning insulin or insulin-secretion deficiency due to pancreatic beta cell depletion.



Type 1

→ The onset usually occurs prior to age 40 with the peak onset at 13....but can be developed as early as 1, or as late as 70.

When the cells cannot use circulating glucose they transition to using fat for energy (lipolysis) resulting in ketone formation....causing metabolic acidosis...and ultimately DKA.

Signs and symptoms of type 1

- Postprandial hyperglycemis transitioning to fasting hyperglycemia
- Weight loss (catabolic state)
- → Fatigue
- → Polyuria
- → Polydypsia
- → Polyphagia
- Abdominal pain with vomiting
- → Blurred vision
- Ketones in the urine
- → Frequent, persistent infections



Treatment of type 1 DM

- Prescribed insulin

Oral anti-hyperglycemic agents

Animal-source insulins are made from the pancreas glands of cows and pigs



Type 2 DM

- → 90% of the diabetic population has type 2 DM.
- → The incidence increases with age, usually striking adults older than 40-45 years of age.
- → A distressing new trend is the rising incidence in children who are obese, spend more than five hours per day in front of the TV or computer, rarely exercise and eat poor diets (low in fiber) – the same risk factors that lead to diabetes in adults.



Type 2 DM

- Pathophysiology
 - → Insulin resistance
 - → The pancrease produces insulin the body can not use it appropriately.
 - Glucose can not enter the cells resulting hyperglycemia
 - → As blood sugar rises, the pancreas increases its output of insulin, triggering a hyperinsulinemic state.

Major causes of insulin resistance

→ Obesity – truncal obesity

→ Risk increases by 20% for each 5% gain in weight from ages 20-53

→ Fat tissue decreases the number of insulin

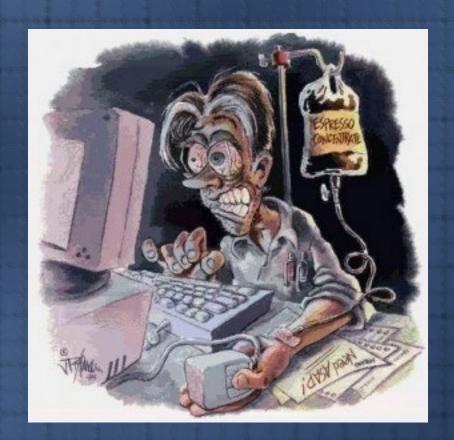
receptors



Hyperglycemia – greater than 300 mg/dL triggers insulin resistance



Stress increases release of insulin-neutralizing, counter-regulatory hormones



- Later in the disease, beta cells fail and patients experience insulin deficiency due to pancreatic beta cell dysfunction.
- The combination of insulin resistance and alteration in beta cell function leads to impaired glucose tolerance (IGT).
- Only one of four patients with IGT will progress to type 2 DM.
 - Hyperinsulinemia, hypertension, high triglycerides, low HDL (good), and a change in the size and density of LDL (bad) cholesterol.



Damaging effects of hyperinsulinemia

- → Insulin acts as an oxidant
 - Vascular beds increase production of plasminogen activator inhibitor-1 (PAI-1)
 - Microthrombi and endothelial inflammation
 - → Plaque formation and atherosclerosis



Damaging effects of hyperinsulinemia

- Increase in plasma calcium
 - → Increases vascular tone HTN
 - → CHF
 - → Stroke
- → Insulin is also an important salt retaining hormone
 - → Weight gain from fluid retention
 - → Electrolyte imbalances



Signs & Symptoms of type 2 DM

- Polyuria, polydypsia, and polyphagia (as in type 1 DM, but may be more subtle)
- → Blurred vision
- → Muscle cramps
- Non-healing infections
- → Fatigue that progresses to exhaustion
- → Dry, itchy skin
- → Impotence
- → Nighttime diarrhea

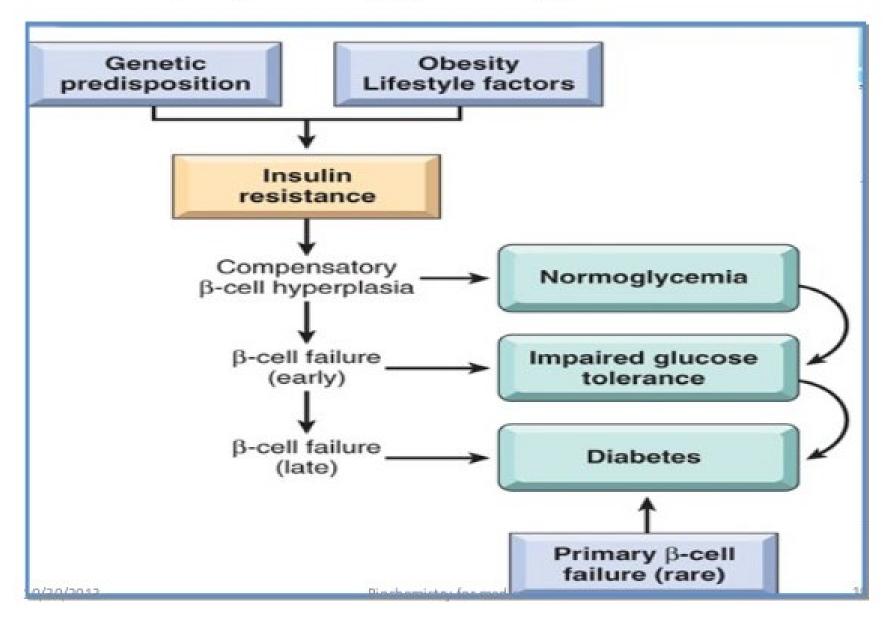


Treatment of type 2 DM



- → Healthy eating
- Exercise and weight loss
 - Even a 5-10% loss of total body weight may lead to better blood glucose
 - Surgical options have all but eliminated some patients issues with DM
- Oral antihyperglycemia agents
- → Physicians prescribe insulin for about 40%

Pathophysiology of Type 2 DM





Summary

Watch yourself!! Good diet, exercise, and a healthy lifestyle

- → Keep an eye on your glucose / a1c
 - → So you don't have to continually prick your finger!!



Glucose -- Oh, sugar sugar --You are my favorite fuel From the blood-borne substrate pool.