Physiology'Endo''6''Pancreatic'Hormones&Blood'Glucose'Regulation

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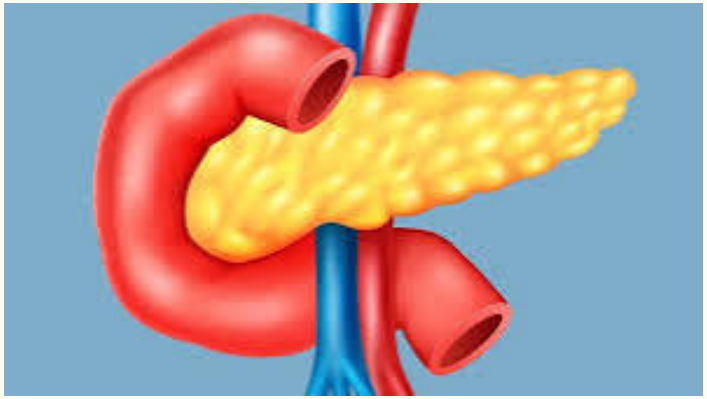
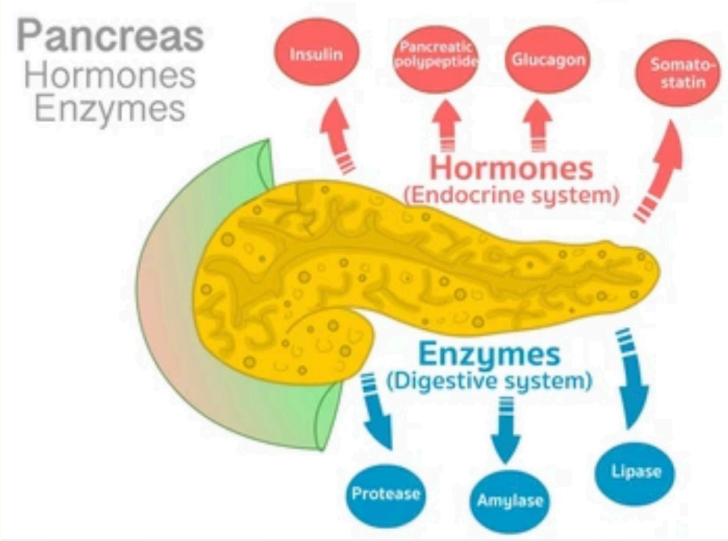




الطب والجراحة لجنة

Done by Raghad Mrayat

This paper contains an explanation of Lecture No. 6, additional information, AI questions, and questions from approved references.

Detailed Explanation:

This slide compares two groups of endocrine glands based on how they are controlled.

Group 1: Glands that obey the hypothalamus and anterior pituitary

- **These glands depend on hormonal signals from the hypothalamus and the anterior pituitary to function.**
- **Includes:**

1. **Thyroid gland: Controlled by TSH (Thyroid Stimulating Hormone) from the anterior pituitary.**
2. **Adrenal cortex: Controlled by ACTH (Adrenocorticotropic hormone).**
3. **Gonads (ovaries/testes): Controlled by LH and FSH (Luteinizing Hormone, Follicle-Stimulating Hormone).**

Example:

- **The hypothalamus releases TRH → stimulates anterior pituitary to release TSH → stimulates thyroid to produce T3/T4 hormones.**

Group 2: Glands that do NOT obey the hypothalamus/anterior pituitary

- **These glands work independently of hypothalamic or pituitary control.**
- **Includes:**

1. **Parathyroid gland: Releases PTH (Parathyroid Hormone) directly in response to low calcium levels.**
2. **Adrenal medulla: Releases adrenaline/noradrenaline in response to sympathetic nervous system signals.**
3. **Pancreas: Releases insulin, glucagon, and other hormones based on blood glucose levels, not pituitary hormones.**

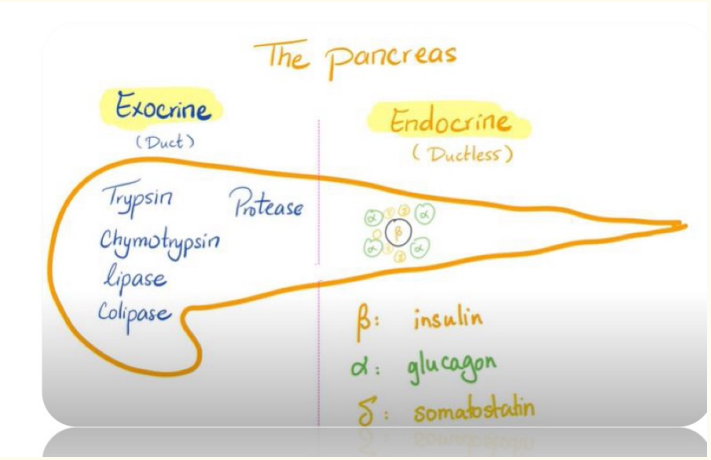
Example:

- **When blood glucose is high, the pancreas directly senses this and releases insulin — no brain or pituitary involvement.**

Summary of Slide 2:

- **Some glands are under control of the hypothalamus and pituitary (thyroid, adrenal cortex, gonads).**
- **Others act independently, responding directly to changes in the body (parathyroid, adrenal medulla, pancreas).**

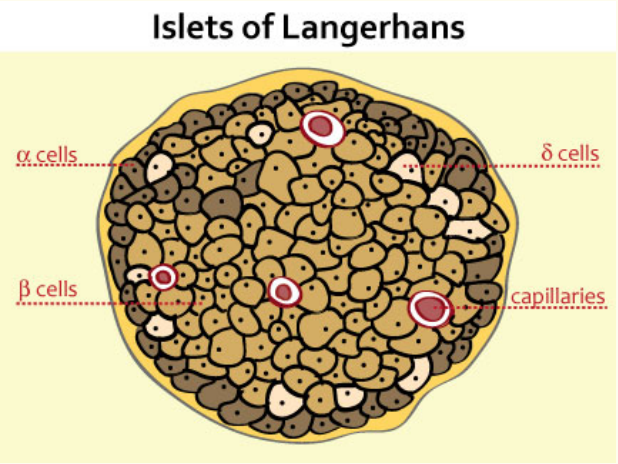
Slide 3



Content on the Slide:

Endocrine pancreas
Consists of Islets of Langerhans
Contain 4 types of cells:
- **Alpha cells → secrete glucagon**
- **Beta cells → secrete insulin**
- **Delta cells → secrete somatostatin**
- **F cells → secrete pancreatic polypeptide**

Detailed Explanation:



What is the endocrine pancreas?
- **The pancreas has two functions:**
1. **Exocrine (produces digestive enzymes).**
2. **Endocrine (produces hormones that go into the blood).**
- **The endocrine part is made of small clusters of cells called Islets of Langerhans.**

The Four Types of Islet Cells:

1. Alpha cells
- **Secrete: Glucagon**
- **Function: Increases blood glucose by promoting breakdown of glycogen in the liver.**
- **Mnemonic: "Alpha = Add glucose"**

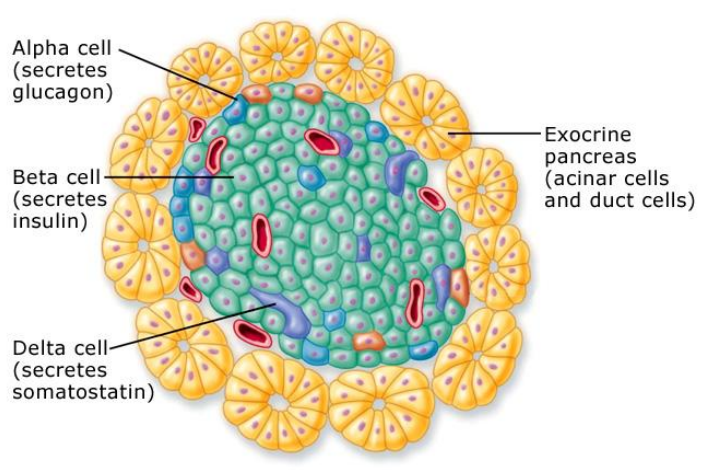
2. Beta cells
- **Secrete: Insulin**
- **Function: Decreases blood glucose by helping cells absorb glucose from the blood.**
- **Most abundant in the islets (about 60–70%).**
- **Mnemonic: "Beta = Bring glucose into cells"**

3. Delta cells

- **Secrete: Somatostatin**
- **Function: Inhibits both insulin and glucagon secretion. Also slows digestion.**
- **Acts as a regulator to balance the system.**

4. F cells (or PP cells)

- **Secrete: Pancreatic polypeptide**
- **Function: Helps regulate pancreatic secretions and possibly appetite.**

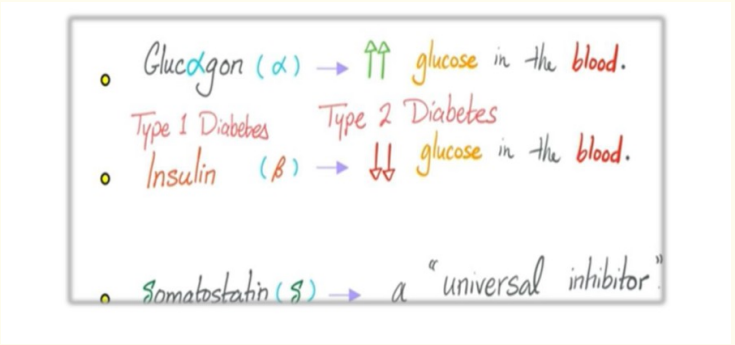


Example Scenario:

- **After a meal:**
- **Blood sugar rises → Beta cells release insulin to lower it.**
- **During fasting:**
- **Blood sugar drops → Alpha cells release glucagon to raise it.**

Summary of Slide 3:

- **The endocrine pancreas is made up of Islets of Langerhans, which contain four types of hormone-secreting cells:**
- **Alpha (glucagon), Beta (insulin), Delta (somatostatin), F cells (pancreatic polypeptide).**
- **These hormones help maintain glucose balance in the body.**



Slide 4

Content on the Slide:

Insulin

- **Small protein hormone, consists of 51 amino acids**
- **Produced by β-cells**
- **Synthesized as preproinsulin → proinsulin → insulin**
- **C-peptide is a byproduct and used as a marker for insulin secretion**

Detailed Explanation:

1. What is insulin?

- **Insulin is a small protein hormone made of 51 amino acids.**
- **It is secreted by beta (β) cells in the pancreas.**
- **Its main job is to lower blood glucose levels by helping cells take in glucose.**

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2. How is insulin produced? (Synthesis pathway)

Insulin isn't made in its active form right away. It goes through three stages:

1. **Preproinsulin (initial form):**
   - **This is the first version made inside the beta cells.**
   - **It includes a signal sequence that helps it enter the endoplasmic reticulum.**
2. **Proinsulin (intermediate form):**
   - **After the signal sequence is removed, it becomes proinsulin.**
   - **Proinsulin is inactive, like a folded-up version of insulin.**

3. **Insulin + C-peptide (final step):**
- **Enzymes cut proinsulin into two parts:**
- **Active insulin (which lowers blood glucose).**
- **C-peptide (a connecting piece, not active, but very useful in medicine).**

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3. What is C-peptide and why is it important?
- **C-peptide is a byproduct of insulin production.**
- **It has no effect on glucose, but it is released in equal amounts with insulin.**
- **Doctors use C-peptide levels as a marker to:**
- **Measure how much insulin the body is naturally producing.**
- **Differentiate between type 1 diabetes (low C-peptide) and type 2 (normal or high C-peptide).**

Example:

If a patient has high blood glucose and low C-peptide, it means their pancreas isn't producing insulin → likely Type 1 diabetes.

Summary of Slide 4:
- **Insulin is a 51-amino-acid hormone made by β-cells.**
- **It is produced in steps: Preproinsulin → Proinsulin → Insulin + C-peptide.**
- **C-peptide is useful as a diagnostic marker for insulin production.**

Type 1 Diabetes (T1D):

- **Cause: Autoimmune destruction of beta cells in the pancreas → no insulin production.**
- **Onset: Usually in children or young adults (but can happen at any age).**
- **Insulin levels: Very low or zero → patients need insulin injections.**
- **C-peptide: Low or absent.**
- **Key point: The body cannot make insulin at all.**

Type 2 Diabetes (T2D):

- **Cause: Body becomes resistant to insulin, and/or pancreas doesn't make enough insulin.**
- **Onset: Usually in adults, often linked to obesity and lifestyle.**
- **Insulin levels: Normal or high (early), may decrease later.**
- **C-peptide: Normal or high (especially early in the disease).**
- **Key point: The body makes insulin, but can't use it properly.**

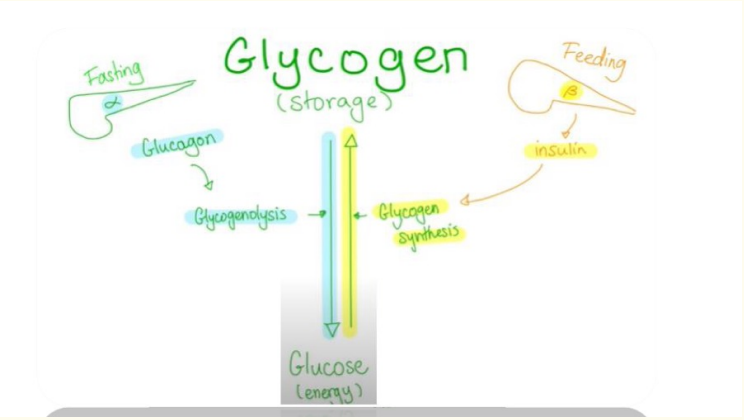
Type 2	Type 1	Feature
Present (but less effective)	Absent	Insulin
Normal or high	Low	C-peptide
Older age	Young age	Onset
Insulin resistance	Autoimmune	Cause
Lifestyle, pills, insulin	Insulin only	Treatment

Slide 5

Content on the Slide:

Insulin functions:

- **Stimulates glucose uptake by cells**
- **Stimulates glycogenesis**
- **Inhibits glycogenolysis**
- **Inhibits gluconeogenesis**
- **Promotes fat and protein synthesis**



1. Stimulates glucose uptake by cells

- **Meaning: Insulin helps body cells (especially muscle and fat) take in glucose from the blood.**
- **Result: Blood sugar levels decrease.**
- **Example: After you eat, insulin rises and helps muscles absorb glucose for energy or storage.**

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2. Stimulates glycogenesis

- **Meaning: Insulin promotes conversion of glucose to glycogen in the liver and muscles.**
- **Glycogen = stored form of glucose.**
- **Example: Extra glucose after a meal is stored in the liver as glycogen for later use.**

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3. Inhibits glycogenolysis

- **Meaning: Prevents the breakdown of glycogen back into glucose.**
- **Why? Because the body doesn't need extra glucose when insulin is high (after eating).**

4. Inhibits gluconeogenesis

- **Meaning: Stops the liver from making new glucose from non-carbohydrate sources (like amino acids).**
- **Important during: fasting or starvation — but insulin suppresses this when food is available.**

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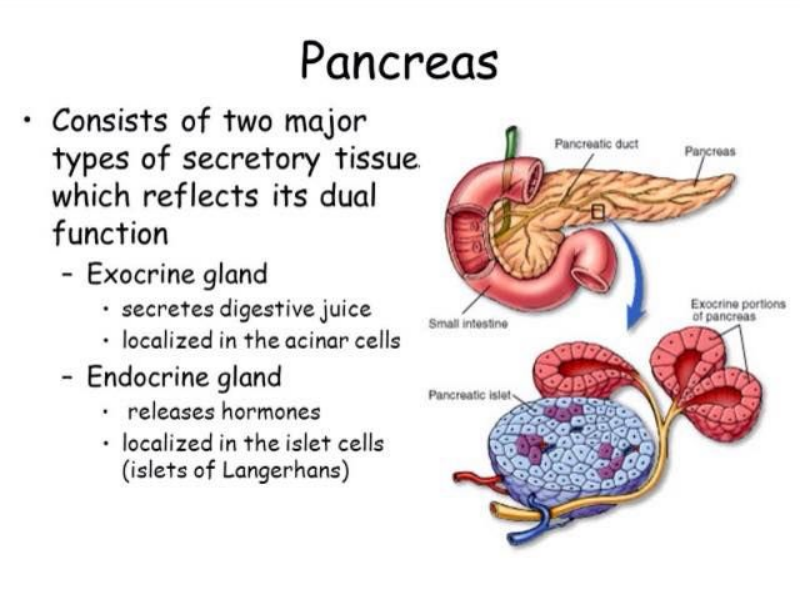
5. Promotes fat and protein synthesis

- **Fat synthesis (lipogenesis): Insulin helps store excess energy as fat.**
- **Protein synthesis: Insulin helps build muscle and repair tissues by promoting amino acid uptake.**

Summary of Slide 5:

Insulin:

- **Lowers blood sugar by helping cells take in glucose.**
- **Stores energy by making glycogen and fat.**
- **Prevents the liver from making or releasing more glucose.**
- **Also helps in protein building.**



Slide 6

Content on the Slide:

Glucagon

- **29 amino acids**
- **Produced by α-cells of the pancreas**
- **Stimulated by:**

• Low blood glucose
• Amino acids
• Sympathetic stimulation

- **Inhibited by:**

• High blood glucose
• Insulin
• Somatostatin

Detailed Explanation:

1. What is Glucagon?

- **A peptide hormone made of 29 amino acids.**
- **Secreted by alpha (α) cells in the pancreatic islets.**
- **It works opposite to insulin: raises blood glucose levels.**

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2. What stimulates glucagon secretion? (When does the body release it?)

- **Low blood glucose (hypoglycemia):**

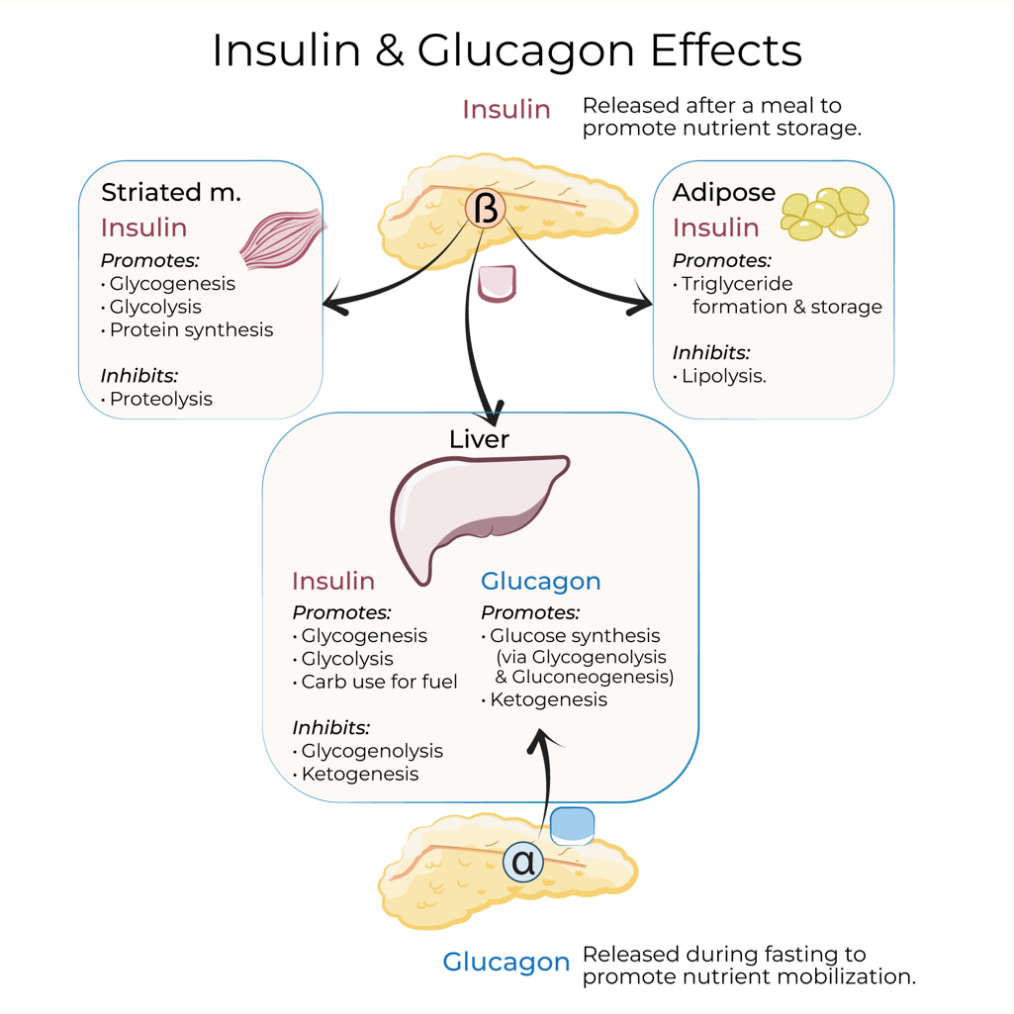
→ Main trigger. The body releases glucagon to bring glucose levels back up.

- **Amino acids (especially after high-protein meals):**

→ To prevent a drop in glucose when insulin is also released.

- **Sympathetic stimulation (fight-or-flight):**

→ Body needs more energy (glucose), so it activates glucagon.



3. What inhibits glucagon secretion? (What stops it?)

- **High blood glucose:**

→ No need to raise glucose, so glucagon is turned off.

- **Insulin:**

→ Signals that there is already enough glucose in the blood.

- **Somatostatin:**

→ A regulatory hormone that suppresses both insulin and glucagon to balance the system.

Example Scenario:

You haven't eaten for 6 hours:

- **Blood sugar drops → glucagon is released → liver breaks down glycogen → glucose enters the blood.**

After eating:

- **Blood sugar rises → insulin increases, glucagon decreases.**

Summary of Slide 6:

- **Glucagon is a 29-amino-acid hormone from α-cells.**
- **It is released when blood sugar is low, during fasting, or sympathetic stress.**
- **It is inhibited by high glucose, insulin, and somatostatin.**

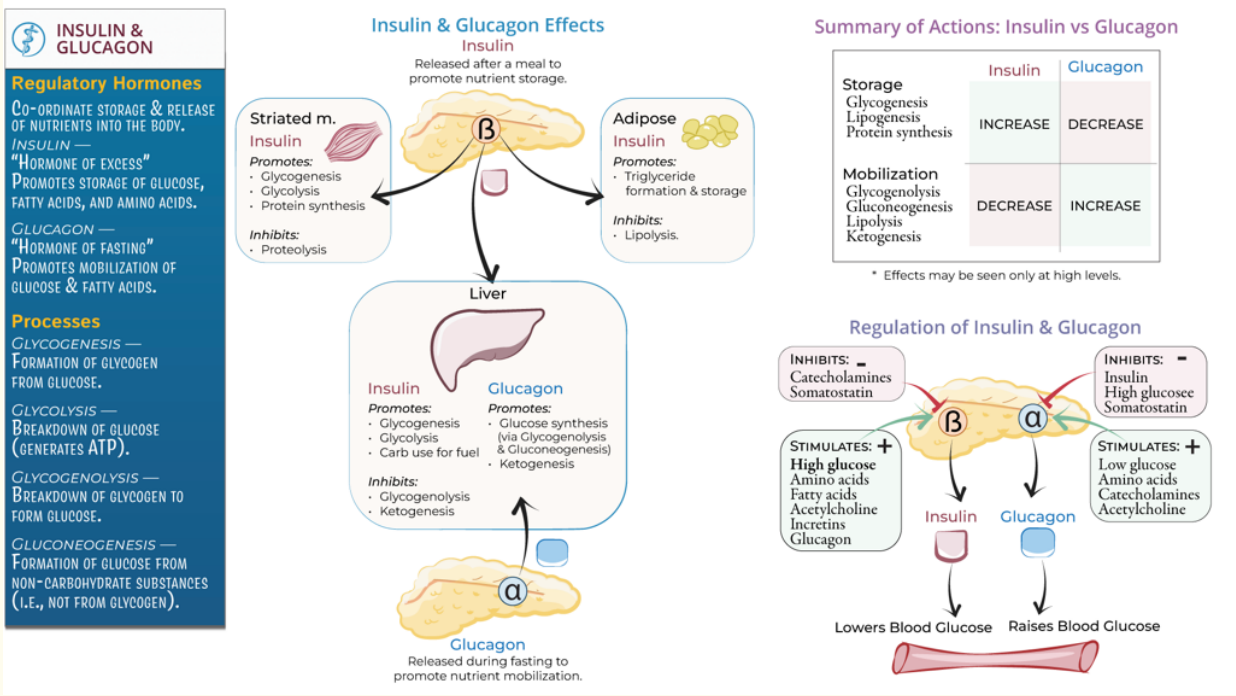
Slide 7

Content on the Slide:

Functions of glucagon:

- **Stimulates glycogenolysis**
- **Stimulates gluconeogenesis**
- **Inhibits glycogenesis**
- **Enhances lipolysis**
- **Ketogenic (increases ketone bodies)**

Detailed Explanation:



1. Stimulates glycogenolysis

- **Definition: Breaks down glycogen into glucose in the liver.**
- **Purpose: Quickly increases blood glucose during fasting.**
- **Example: If you haven't eaten in hours, the liver breaks down stored glycogen to release glucose.**

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2. Stimulates gluconeogenesis

- **Definition: Creates new glucose from non-carbohydrate sources, like amino acids and lactate.**
- **Where? Mainly in the liver.**
- **Why? To maintain blood sugar when glycogen is depleted.**

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3. Inhibits glycogenesis

- **Definition: Prevents the formation of glycogen from glucose.**
- **Why? Because the body needs glucose in the blood, not stored.**

4. Enhances lipolysis

- **Definition: Breaks down fats (triglycerides) in adipose tissue into fatty acids.**
- **Purpose: Provides energy when glucose is low.**
- **Note: Fatty acids can be used by many tissues (especially during fasting).**

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5. Ketogenic (increases ketone bodies)

- **Definition: Promotes the production of ketone bodies in the liver from fatty acids.**
- **Purpose: An alternative fuel for the brain and muscles during prolonged fasting or low-carb diets.**

Example Scenario:

You are fasting:

- **Glucagon levels rise → liver breaks down glycogen (glycogenolysis)**
- **Starts making glucose (gluconeogenesis)**
- **Breaks down fat (lipolysis) → produces ketones for brain energy**

Summary of Slide 7:

Glucagon works to increase blood glucose and provide energy during fasting by:

- **Breaking down glycogen**
- **Creating new glucose**
- **Breaking down fat**
- **Producing ketones**

1. Blood glucose level

- **Main regulator for both insulin and glucagon.**
- **High glucose → stimulates insulin release, inhibits glucagon.**
- **Low glucose → stimulates glucagon release, inhibits insulin.**
- **Example: After eating: ↑ glucose → ↑ insulin, ↓ glucagon**

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2. Amino acids

- **Stimulate both insulin and glucagon.**
- **Why both? Because:**
- **Insulin helps cells absorb amino acids.**
- **Glucagon prevents blood sugar from dropping too much (especially after high-protein meals with low carbs).**
- **Example: You eat steak (high protein, low carb) → amino acids ↑ → both insulin and glucagon released.**

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3. Autonomic nervous system

- **Sympathetic (fight-or-flight):**
- **Stimulates glucagon → increases blood glucose for energy.**
- **Slightly inhibits insulin.**
- **Parasympathetic (rest-and-digest):**
- **Stimulates insulin → promotes storage of nutrients.**

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4. Gastrointestinal hormones

- **Hormones like GIP and GLP-1 are released after eating.**
- **They stimulate insulin secretion even before glucose enters the blood — this is called the incretin effect.**
- **Help the body prepare to handle the upcoming glucose.**

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5. Somatostatin

- **Secreted by delta cells in the pancreas.**
- **Inhibits both insulin and glucagon.**
- **Acts as a local regulator to prevent over-secretion.**

Summary of Slide 8:

The secretion of insulin and glucagon is finely controlled by:

- **Blood sugar levels**
- **Amino acid levels**
- **Nervous system input**
- **Gut hormones (like GLP-1)**
- **Somatostatin (a local inhibitor)**

Slide 8

Content on the Slide:

Insulin and glucagon secretion is regulated by:

- **Blood glucose level**
- **Amino acids**
- **Autonomic nervous system**
- **Gastrointestinal hormones**
- **Somatostatin**

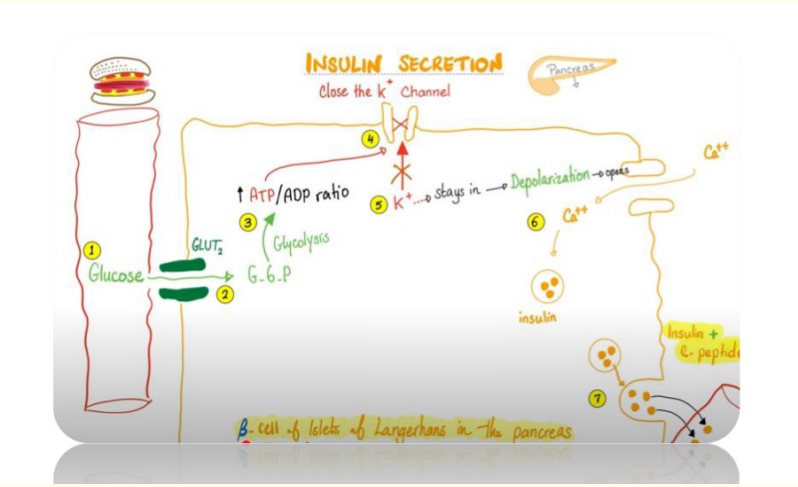
Slide 9

Content on the Slide:

Insulin/glucagon ratio is important

- **High ratio → favors storage (well-fed state)**
- **Low ratio → favors mobilization of fuels (fasting state)**

Detailed Explanation:



What is the insulin/glucagon ratio?

- **It's the balance between insulin and glucagon levels in the blood.**
- **This ratio helps determine whether the body is in a storage mode or energy release mode.**

High insulin/glucagon ratio: "Fed state"

- **Happens after eating when glucose is high.**
- **More insulin, less glucagon**
- **Body focuses on:**
- **Storing glucose as glycogen**
- **Converting glucose into fat**
- **Building proteins**
- **Example: After eating rice → insulin ↑, glucagon ↓ → energy is stored.**

Low insulin/glucagon ratio: "Fasting state"

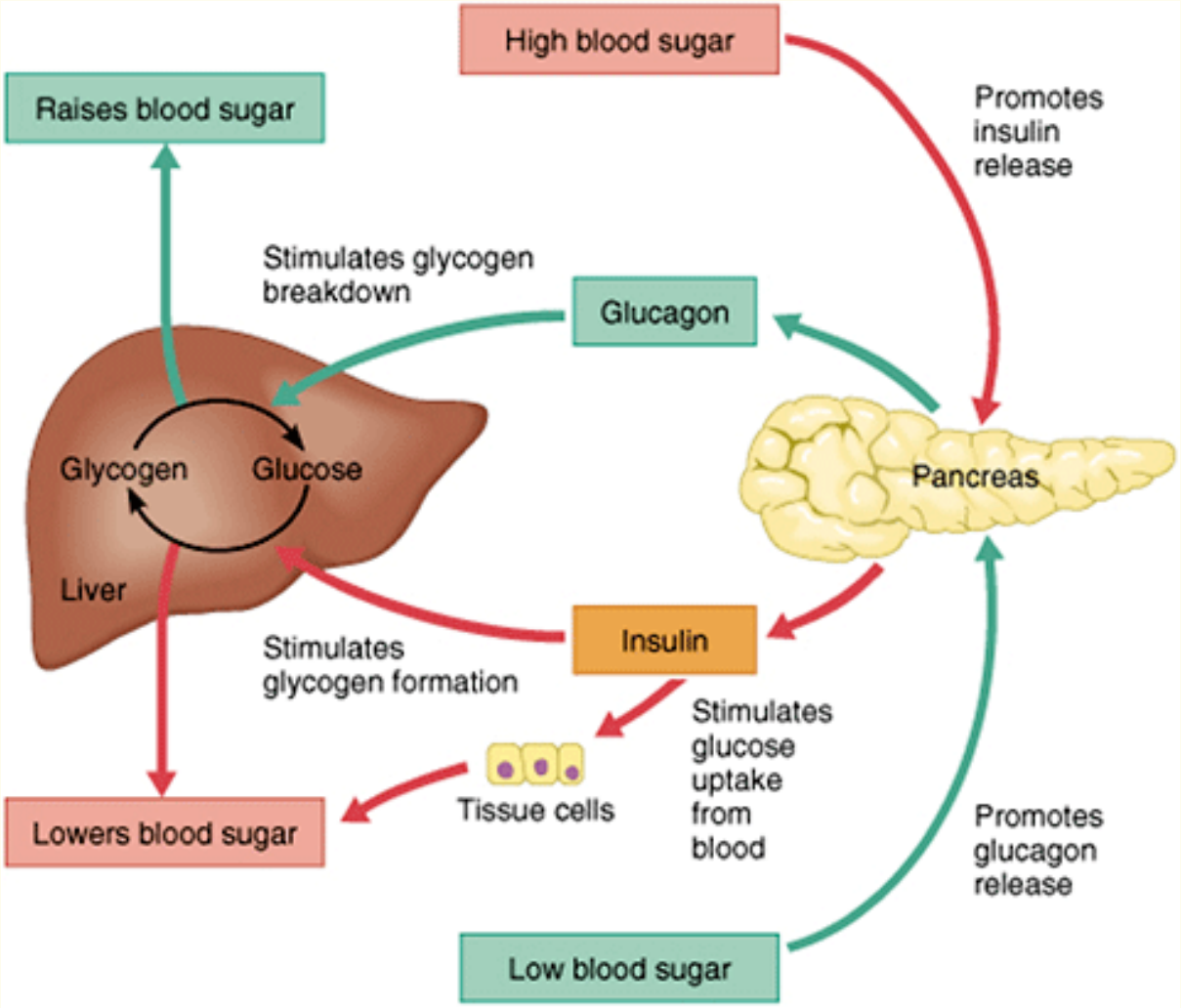
- **Happens during fasting, exercise, or stress.**
- **Less insulin, more glucagon**
- **Body shifts to:**
- **Breaking down glycogen to release glucose**
- **Producing glucose from amino acids (gluconeogenesis)**
- **Breaking down fat (lipolysis)**
- **Producing ketones for brain fuel**
- **Example: You skip breakfast → insulin ↓, glucagon ↑ → body uses stored energy.**

Key Concept:

- **The higher the ratio, the more the body is in "storage mode."**
- **The lower the ratio, the more the body is in "breakdown mode" to release energy.**

Summary of Slide 9:

- **The insulin/glucagon ratio controls how your body handles energy.**
- **High ratio = storage of glucose, fat, and protein.**
- **Low ratio = release and use of stored fuels.**



Slide 10

Content on the Slide:

Effect of insulin/glucagon ratio on liver metabolism
High ratio (insulin dominates):

- ↑ **Glycogenesis**
- ↑ **Lipogenesis**
- ↑ **Protein synthesis**
- ↓ **Gluconeogenesis**
- ↓ **Glycogenolysis**

Low ratio (glucagon dominates):

- ↓ **Glycogenesis**
- ↓ **Lipogenesis**
- ↓ **Protein synthesis**
- ↑ **Gluconeogenesis**
- ↑ **Glycogenolysis**
- ↑ **Ketogenesis**

Detailed Explanation:

In the liver:

When insulin is high (high insulin/glucagon ratio):
- The body is in the fed state — time to store energy.

1. ↑ Glycogenesis:

→ Glucose is converted into glycogen for storage.

2. ↑ Lipogenesis:

→ Extra glucose is turned into fat.

3. ↑ Protein synthesis:

→ The liver helps build proteins.

4. ↓ Gluconeogenesis:

→ The liver stops making new glucose, since there's enough already.

5. ↓ Glycogenolysis:

→ No need to break down glycogen when blood sugar is high.

When glucagon is high (low insulin/glucagon ratio):
- The body is in fasting/starving mode — time to use stored energy.

1. ↓ Glycogenesis:

→ No new glycogen made.

2. ↓ Lipogenesis:

→ Fat synthesis stops.

3. ↓ Protein synthesis:

→ Body avoids building, focuses on survival.

4. ↑ Gluconeogenesis:

→ Liver produces new glucose from amino acids.

5. ↑ Glycogenolysis:

→ Stored glycogen is broken down into glucose.

6. ↑ Ketogenesis:

→ Liver converts fats into ketone bodies for energy (especially for the brain during starvation)

Summary of Slide 10:

The insulin/glucagon ratio controls the liver's role in energy balance:

- **High ratio: promotes storage (glycogen, fat, protein).**
- **Low ratio: promotes energy release (glucose, ketones).**

What does “Ketogenic” mean?

- **Ketogenic means “promotes the production of ketone bodies”.**
- **Ketone bodies are alternative energy molecules made by the liver from fatty acids.**
- **Produced especially during:**
- **Fasting**
- **Low carbohydrate diets**
- **Uncontrolled diabetes (especially Type 1)**

Why does glucagon increase ketone bodies?

- **When blood glucose is low, and there’s not enough insulin:**
- **The body starts to break down fats (lipolysis) into fatty acids.**
- **The liver takes these fatty acids and converts them into ketone bodies.**
- **Glucagon activates enzymes that enhance this process.**

Types of ketone bodies:

1. **Acetoacetate**
2. **Beta-hydroxybutyrate**
3. **Acetone (exhaled → causes fruity breath smell in diabetic ketoacidosis)**

Purpose of ketone bodies:

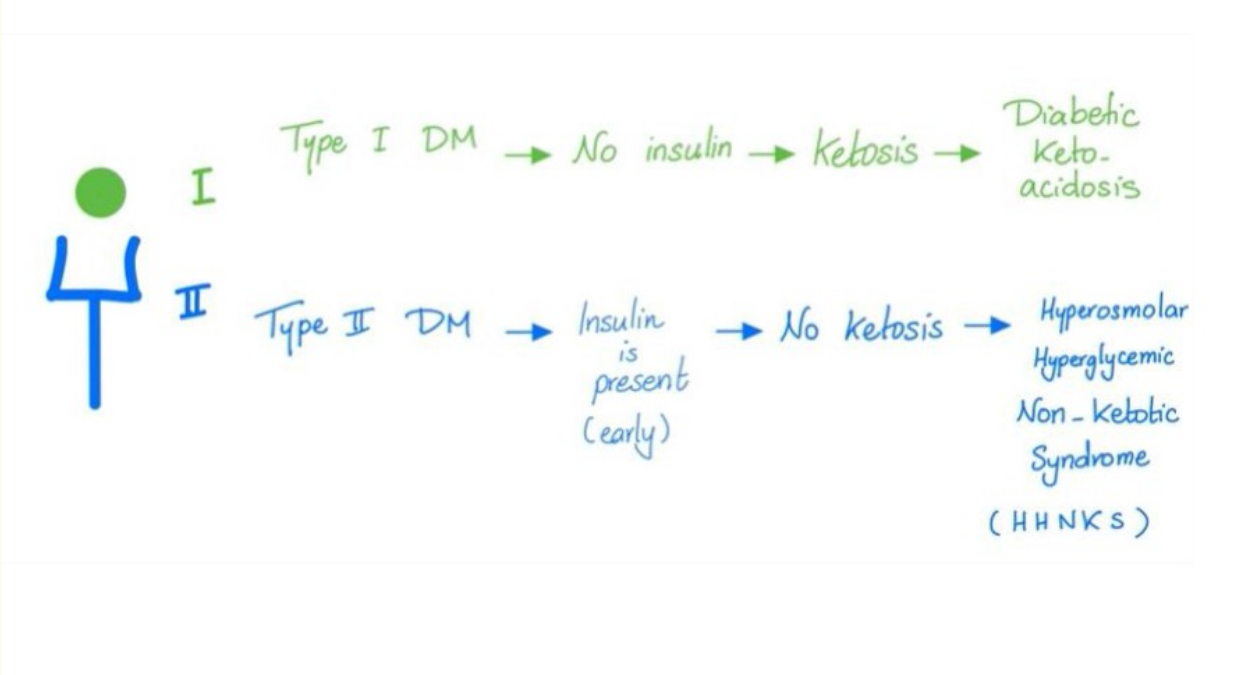
- **Serve as a backup energy source, especially for the brain and muscles when glucose is not available.**
- **The brain normally prefers glucose, but in prolonged fasting, it adapts to using ketones.**

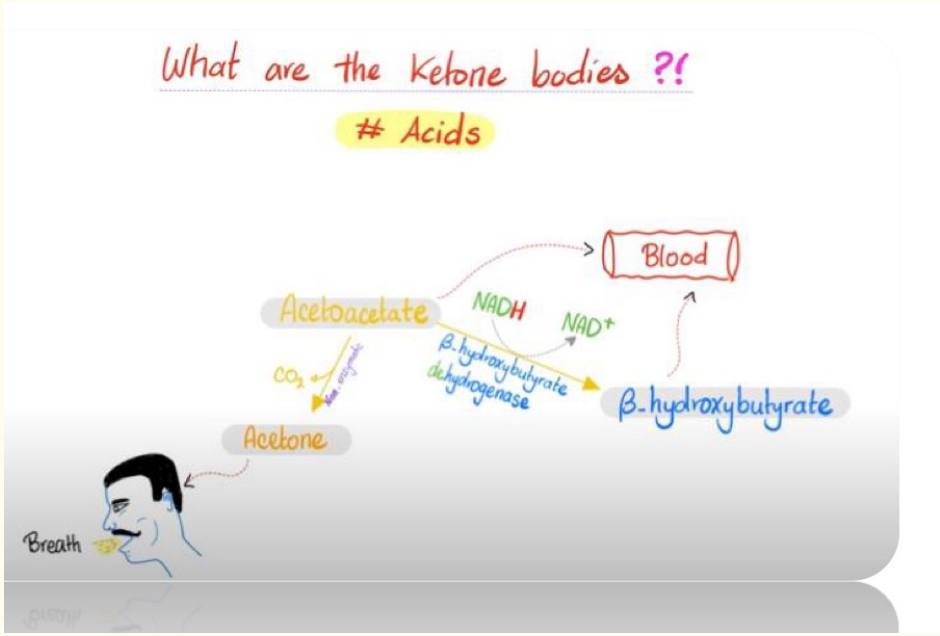
Important clinical note:

- **In uncontrolled Type 1 diabetes:**
- **No insulin → glucose can't enter cells → body thinks it's starving.**
- **Glucagon rises → massive ketone production → leads to diabetic ketoacidosis (DKA) — a dangerous condition with acidic blood and dehydration.**

Summary (Ketone Focus):

- **Glucagon increases lipolysis → liver converts fatty acids to ketones.**
- **Ketones are essential during prolonged fasting or carbohydrate restriction.**
- **Overproduction of ketones (especially without insulin) can be dangerous.**





Slide 12

1. What is cAMP?

- **cAMP (cyclic AMP) is a second messenger inside cells.**
- **It helps relay signals from certain hormones (like glucagon) to the inside of the cell, triggering metabolic changes.**

———

2. Glucagon signaling through cAMP (stimulatory pathway):

- **Glucagon binds to its G-protein coupled receptor (GPCR) on the surface of liver cells.**
- **This activates a Gs protein, which then activates the enzyme adenylyl cyclase.**

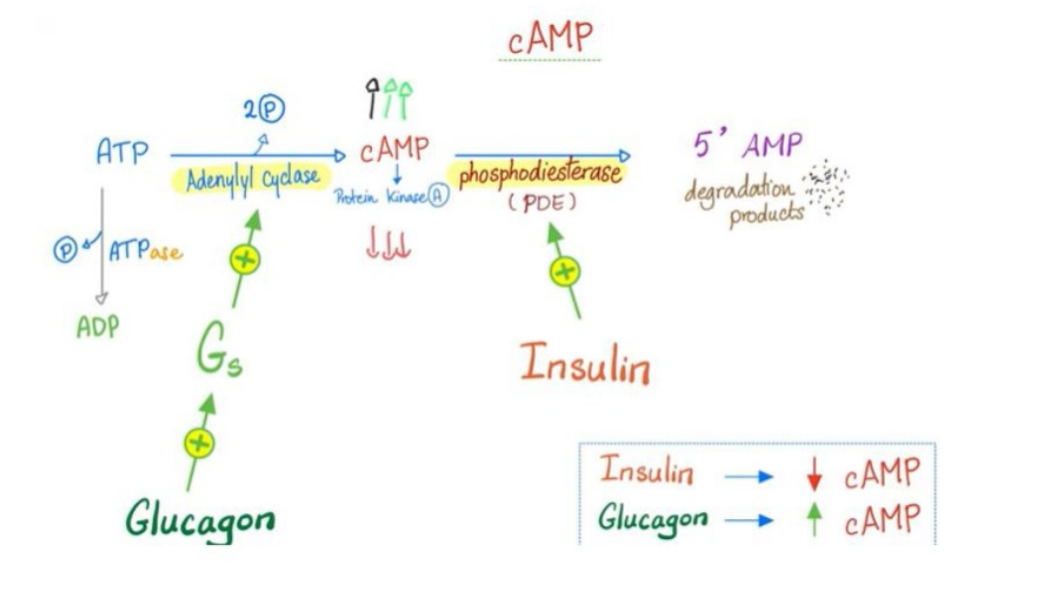
Role of adenylyl cyclase:

- **Adenylyl cyclase converts ATP → cAMP (the second messenger).**

Then:

- **cAMP activates Protein Kinase A (PKA).**
- **PKA phosphorylates target enzymes, leading to:**
- **↑ Glycogenolysis (glycogen breakdown)**
- **↑ Gluconeogenesis (new glucose production)**
- **↓ Glycogenesis (inhibited glycogen formation)**

Result: Blood glucose increases.



3. Termination of cAMP signaling – Role of phosphodiesterase:

- **To stop the cAMP signal, the enzyme phosphodiesterase (PDE) degrades cAMP.**
- **It converts cAMP → 5'AMP (inactive degradation product).**
- **This step ends the glucagon signal and prevents overstimulation.**

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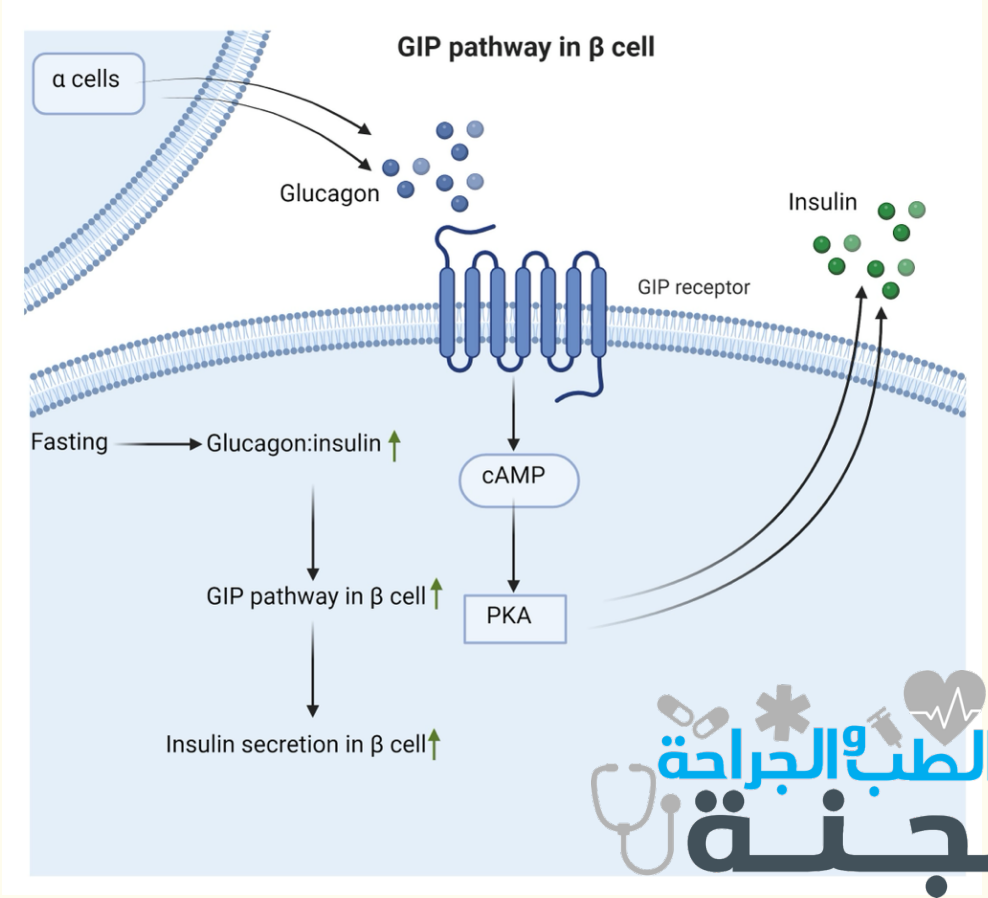
4. Insulin signaling (not via cAMP):

- **Insulin binds to its tyrosine kinase receptor.**
- **This receptor autophosphorylates and activates protein phosphatases.**
- **These phosphatases remove phosphate groups from metabolic enzymes.**

Effects of insulin:

- **↑ Glycogenesis**
- **↑ Glucose uptake**
- **↓ Gluconeogenesis**
- **↓ Glycogenolysis**

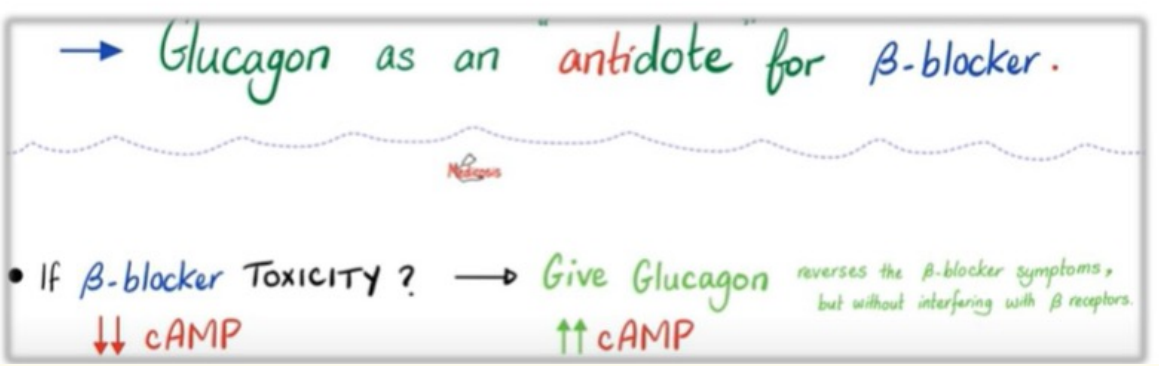
Result: Blood glucose decreases.



Slide 14: Glucagon as an Antidote for Beta Blocker Toxicity

Slide Content Summary (based on standard medical education):

Glucagon can be used as an antidote for β-blocker overdose
If β-blocker toxicity, give IV glucagon



Detailed Explanation:

What are β-blockers?

- **β-blockers (beta blockers) are medications used to:**
- **Lower blood pressure**
- **Treat arrhythmias**
- **Reduce heart workload (e.g., in heart failure)**

β-blocker toxicity:

- **In overdose, β-blockers cause:**
- **Severe bradycardia (slow heart rate)**
- **Hypotension (low blood pressure)**
- **Heart block or even cardiac arrest**
- **This happens because they block β-adrenergic receptors, preventing the heart from responding to adrenaline.**

How does glucagon help?

Glucagon's unique role:

- **Glucagon activates a different receptor than β-adrenergic receptors.**
- **It binds to its own receptor, which activates adenylyl cyclase → increases cAMP → increases heart rate and contractility, even when β-receptors are blocked.**

So even though β-blockers block the usual adrenaline pathway, glucagon bypasses it and restores cardiac function.

Dose (clinical context):

- **In case of β-blocker overdose, IV glucagon is given.**
- **Typical initial dose: 3–10 mg IV bolus, followed by continuous infusion if needed.**

Why not just use adrenaline?

- **In β-blocker overdose, β-receptors are blocked, so adrenaline becomes ineffective.**
- **Glucagon works independently of those receptors.**

Summary of Slide 14:

- **In β-blocker toxicity (bradycardia, hypotension), glucagon is an effective antidote.**
- **It acts via its own receptor to increase cAMP, improving heart rate and contraction, bypassing β-receptor blockade.**

Slide 15: Growth Hormone (GH / Somatotropin)

Insulin-like actions & Anti-insulin actions

Detailed Explanation:

1. What is Growth Hormone (GH)?

- **Also called Somatotropin.**
- **Secreted by the anterior pituitary gland.**
- **Has dual actions: sometimes it acts like insulin, and other times it acts against insulin.**

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2. Insulin-like actions (via IGF-1):

These effects are indirect, mediated by IGF-1 (Insulin-like Growth Factor-1), which is produced by the liver in response to GH.

- **↑ Protein synthesis:**

→ Promotes muscle growth and tissue repair.

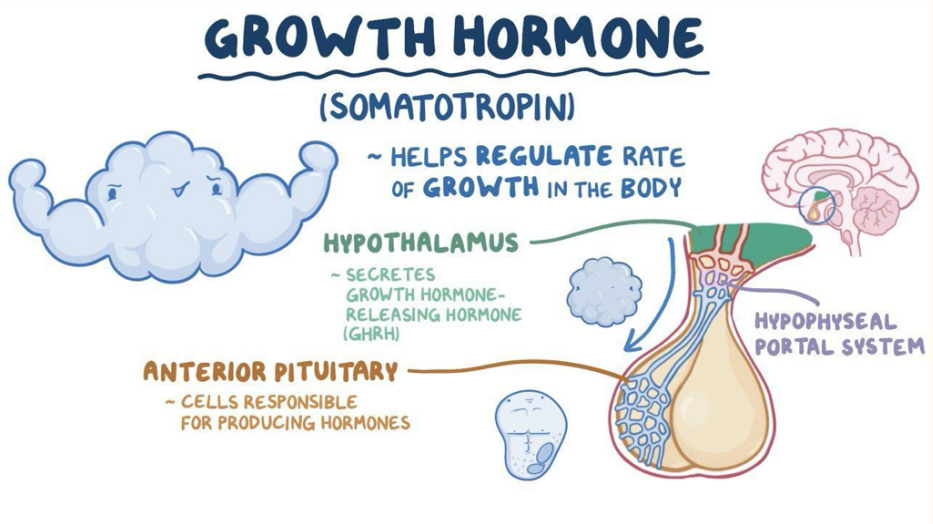
- **↑ Cell growth and division:**

→ Especially in bone and cartilage (important for height in children).

- **↑ Amino acid uptake into cells:**

→ Helps build proteins, similar to insulin.

Result: Promotes anabolism (building tissues), like insulin does.



3. Anti-insulin actions (direct effect of GH):

These effects oppose insulin, especially in metabolism:

- **↓ Glucose uptake by cells (especially muscle and fat):**

→ Causes insulin resistance.

- **↑ Lipolysis (fat breakdown):**

→ Provides free fatty acids for energy.

- **↑ Gluconeogenesis in liver:**

→ Increases blood glucose levels.

Like or Opposite Insulin?	Effect of GH	Action Type
Like insulin	Promotes growth via IGF-1	↑ Protein synthesis
Opposite	Gluconeogenesis	↑ Glucose production
Opposite	Causes insulin resistance	↓ Glucose uptake
Opposite	Increases energy from fat	↑ Fat breakdown

Result: These effects raise blood sugar, opposite of insulin.

How does the balance work?

- **GH promotes growth and repair (via IGF-1) but also ensures energy availability by raising glucose and fatty acids.**
- **That's why it can cause hyperglycemia and insulin resistance in excess.**

Example Clinical Relevance:

- **Acromegaly (GH excess in adults):**

→ Can lead to insulin resistance and even Type 2 diabetes due to GH's anti-insulin effects.

Growth Hormone (GH) = Somatotropin — Full Diagram Breakdown

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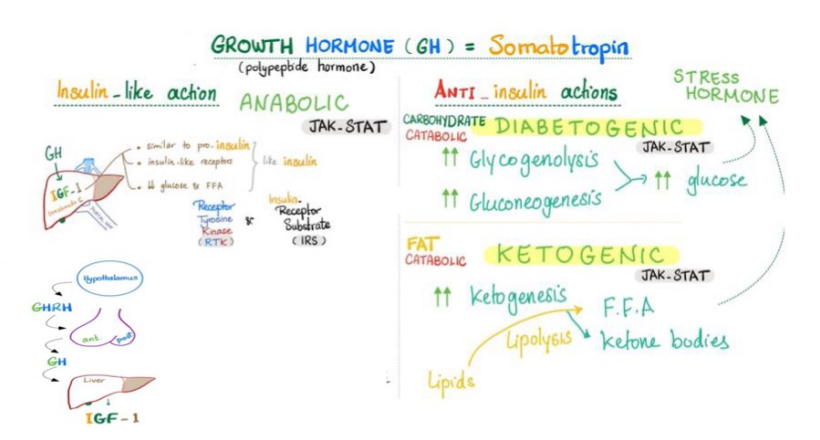
1. GH Basics:

- **GH is a polypeptide hormone secreted by the anterior pituitary.**
- **It is stimulated by GHRH (Growth Hormone-Releasing Hormone) from the hypothalamus.**
- **GH acts directly and indirectly via IGF-1 (produced by the liver).**



2. Insulin-like Action (Anabolic effects via IGF-1):

Pathway:

- **GH → liver → IGF-1 (Insulin-like Growth Factor 1)**

Effects:

- **IGF-1 is similar to pro-insulin.**
- **Binds to insulin-like receptors (Receptor Tyrosine Kinase, RTK).**
- **Activates IRS (Insulin Receptor Substrate).**
- **Promotes:**
- **↑ Protein synthesis**
- **↑ Cell growth & proliferation**
- **↑ Glucose uptake**
- **↓ Glucose-to-FFA conversion (prevents fat overproduction)**

Summary:

IGF-1 mimics insulin's anabolic functions — building tissues, especially bone and muscle — this is the "insulin-like" effect.

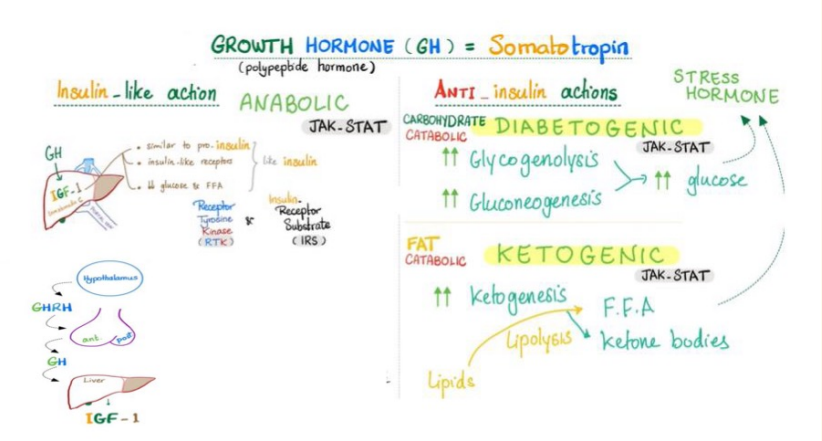
3. Anti-insulin Actions (Catabolic):

These are direct effects of GH, opposite to insulin's action:

A. Carbohydrate metabolism – "Diabetogenic"

- **↑ Glycogenolysis (breakdown of glycogen to glucose)**
- **↑ Gluconeogenesis (new glucose production in liver)**
- **↑ Blood glucose levels**
- **Leads to insulin resistance over time**
- **Works via JAK-STAT pathway**

Result: GH increases glucose availability — diabetogenic effect.



B. Fat metabolism – "Ketogenic"

- **↑ Lipolysis (fat breakdown) → increases Free Fatty Acids (FFA)**
- **FFAs go to liver → ↑ Ketogenesis → produces ketone bodies**
- **Provides energy during fasting/stress**
- **Also via JAK-STAT pathway**

Result: GH promotes fat usage → ketogenic effect

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4. Stress Hormone Role

- **GH acts as a stress hormone, especially in:**
- **Fasting**
- **Exercise**
- **Hypoglycemia**
- **Ensures energy supply by:**
- **Increasing glucose (via gluconeogenesis)**
- **Increasing fats/ketones (via lipolysis & ketogenesis)**

AI question

A 14-year-old boy with Type 1 Diabetes is brought to the ER unconscious. His blood glucose level is 34 mg/dL. The doctor gives him an injection to rapidly increase his blood sugar.

What is the most likely hormone used, and what is its second messenger pathway?

A. Insulin – Tyrosine kinase
B. Glucagon – cAMP
C. Somatostatin – IP3
D. Cortisol – JAK-STAT

Correct answer: B. Glucagon – cAMP

A 65-year-old man accidentally overdosed on his beta-blocker medication. He presents with bradycardia, hypotension, and signs of heart failure. Which of the following is the best initial antidote?

A. Atropine
B. Epinephrine
C. Glucagon
D. Dopamine

Correct answer: C. Glucagon

After a high-carbohydrate meal, which of the following metabolic changes would you expect in the liver?

A. Increased glycogenolysis
B. Increased gluconeogenesis
C. Increased glycogenesis
D. Increased ketogenesis

Correct answer: C. Increased glycogenesis

A 50-year-old man with acromegaly develops elevated fasting blood glucose levels. Which of the following explains this effect of growth hormone?

A. Increased insulin secretion
B. Increased glucose uptake in muscles
C. Increased gluconeogenesis
D. Decreased lipolysis

Correct answer: C. Increased gluconeogenesis

Which of the following hormone–second messenger pairings is correct?

A. Insulin – cAMP
B. Glucagon – Tyrosine kinase
C. Growth hormone – JAK-STAT
D. Somatostatin – Tyrosine kinase

Correct answer: C. Growth hormone – JAK-STAT

case study-style MCQs in English based on trusted physiology sources, such as Guyton & Hall, Ganong's Medical Physiology, USMLE question banks, and educational platforms like Osmosis, AMBOSS, and Lecturio.

Case:
A 19-year-old male skips breakfast and begins to feel dizzy, confused, and eventually faints. His blood glucose level is measured at 40 mg/dL.

Question:
Which hormone is most likely responsible for correcting his blood glucose in this situation?

A. Insulin
B. Glucagon
C. Epinephrine
D. Cortisol

Correct Answer: B. Glucagon
Explanation: Glucagon is secreted by α-cells in response to hypoglycemia. It increases blood glucose by stimulating glycogenolysis and gluconeogenesis.

Case:
A 64-year-old man presents with bradycardia and hypotension after an overdose of propranolol (a non-selective beta-blocker). The medical team administers a hormone that improves his heart rate and cardiac output.

Question:
Which hormone is used, and what is its mechanism?

A. Epinephrine – activates β1-receptors
B. Glucagon – increases cAMP via a separate receptor
C. Insulin – enhances cardiac glucose uptake
D. Vasopressin – increases vascular tone

Correct Answer: B. Glucagon – increases cAMP via a separate receptor
Explanation: Glucagon bypasses the β-adrenergic receptor pathway and stimulates cAMP production through its own receptor, improving heart function.

Case:
A 47-year-old male with acromegaly is found to have elevated fasting blood glucose levels. He is not diabetic and has no history of metabolic syndrome.

Question:
What is the most likely reason for his hyperglycemia?

A. IGF-1 causes insulin resistance
B. GH decreases insulin secretion
C. GH directly opposes insulin action
D. GH increases insulin binding to receptors

Correct Answer: C. GH directly opposes insulin action
Explanation: GH reduces glucose uptake in tissues, increases gluconeogenesis and lipolysis, and contributes to insulin resistance.

Case:
After eating a carbohydrate-rich meal, which of the following changes would you expect in the liver?

A. Increased glycogenolysis
B. Increased ketogenesis
C. Increased glycogenesis
D. Increased gluconeogenesis

Correct Answer: C. Increased glycogenesis
Explanation: High insulin after a meal promotes glycogen formation (storage of glucose) in the liver.

Case:
A research study investigates how different hormones exert their effects. One hormone is found to increase cAMP, activate protein kinase A, and stimulate gluconeogenesis in liver cells.

Question:
Which hormone is being studied?

A. Insulin
B. Glucagon
C. Growth hormone
D. Somatostatin

Correct Answer: B. Glucagon
Explanation: Glucagon activates adenylate cyclase → ↑ cAMP → activates PKA → increases gluconeogenesis and glycogenolysis.