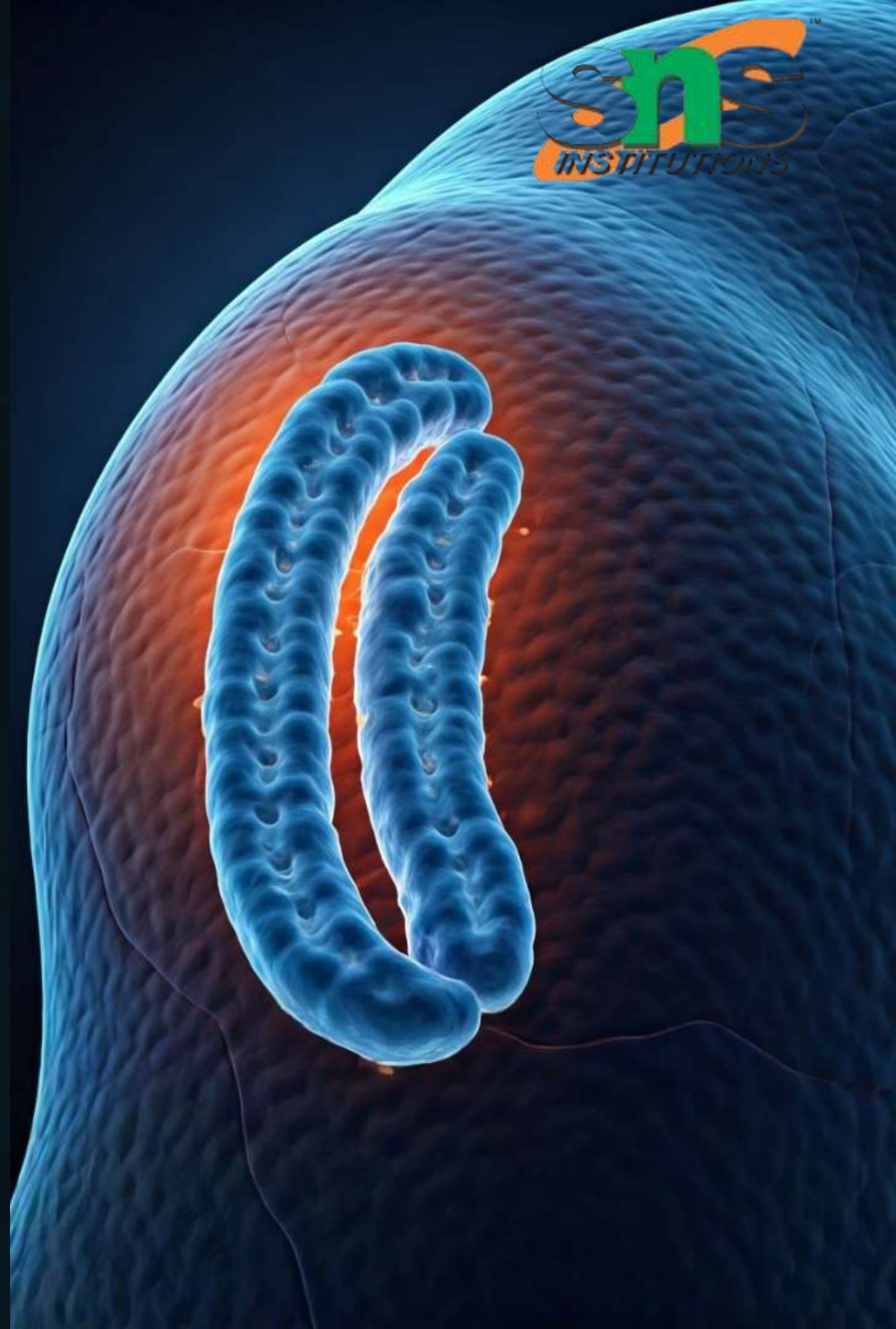
Ketone Bodies: Formation, Utilization, and Ketoacidosis

This presentation explores ketone metabolism and its clinical significance.

by SRI VIKRAM S



Ketogenesis: Formation of Ketone Bodies

Location

Occurs inside liver mitochondria.

Substrates

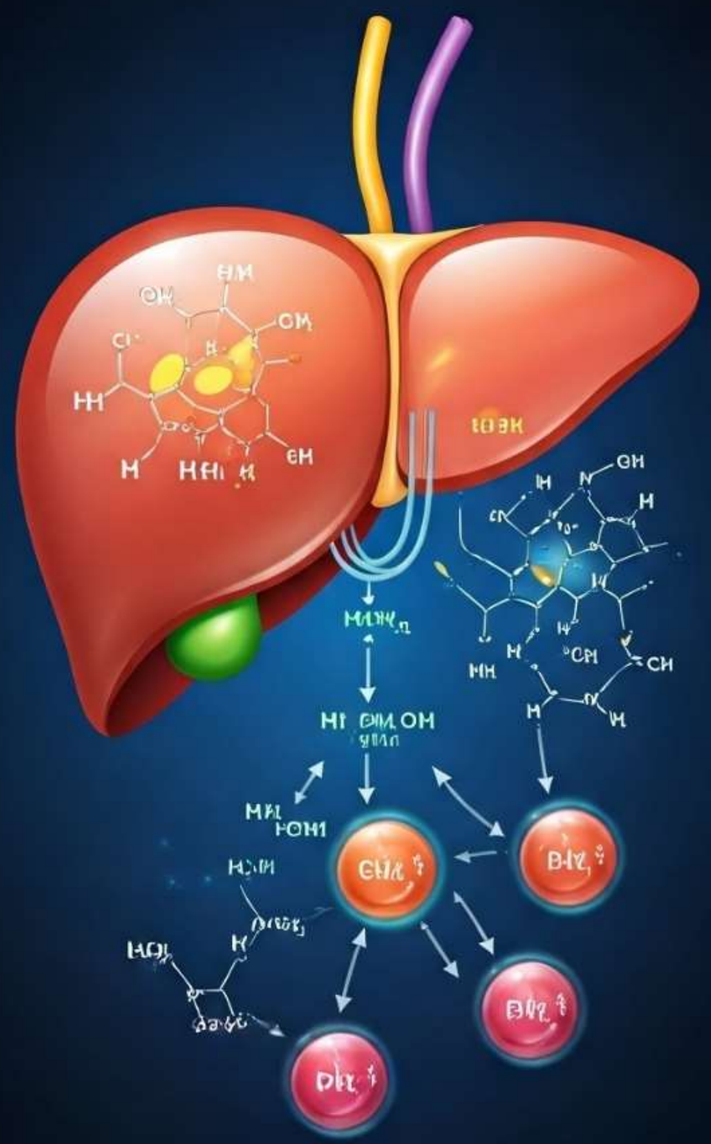
Acetyl-CoA from fatty acid oxidation.

Key Enzymes

HMG-CoA synthase and lyase drive the process.

End Products

Acetoacetate, β-hydroxybutyrate, and acetone.



Regulation of Ketogenesis

Hormonal Control

Insulin inhibits; glucagon stimulates ketogenesis.

Fatty Acid Availability

More fatty acids increase ketone production.

CPT-I Activity

Carnitine palmitoyltransferase I regulates fatty acid transport.

Starvation Effect

Fatty acid mobilization overloads the Krebs cycle.

Ketone Body Utilization

Target Tissues

Used by brain, heart, and muscle.

Conversion

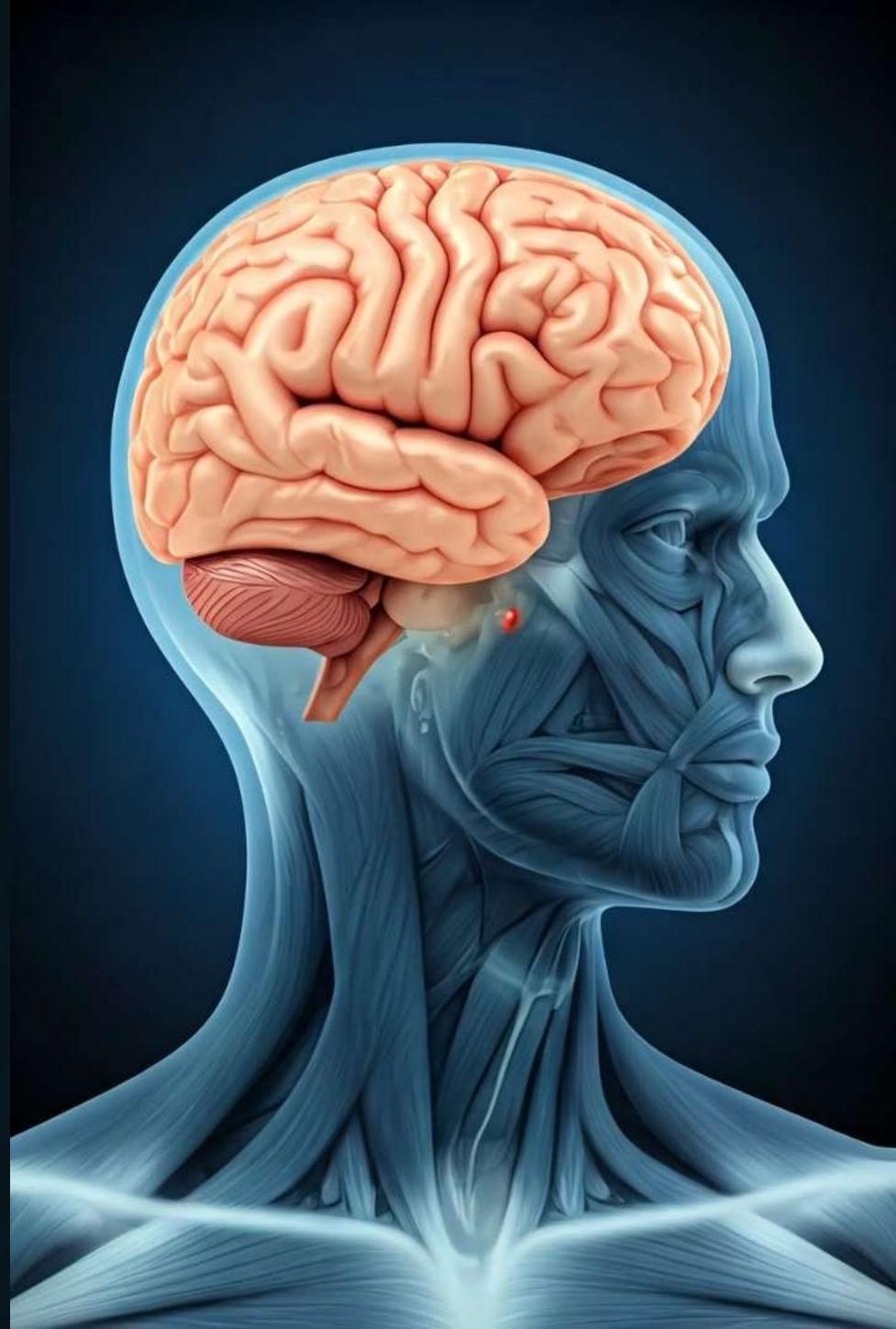
Acetoacetate converts to acetyl-CoA for energy.

Brain Adaptation

Brain adapts to ketones after days of starvation.

Key Enzyme

Thiophorase converts ketones; absent in liver.



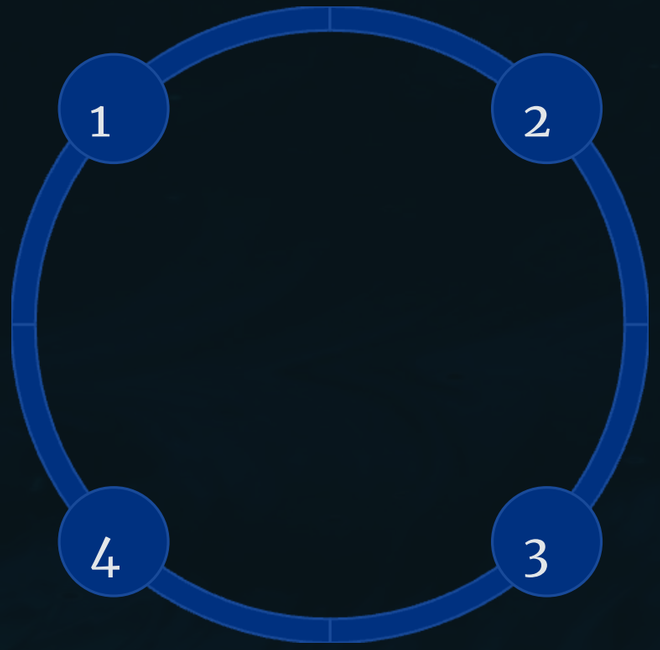
Physiological Significance of Ketone Bodies

Alternative Fuel

Provides energy during low glucose availability.

Exercise

Important during prolonged physical activity.



Glucose Sparing

Saves glucose for crucial functions like the brain.

Muscle Protection

Reduces muscle protein breakdown.

Ketoacidosis: Definition and Causes

Definition

Excess ketones cause metabolic acidosis.

Primary Cause

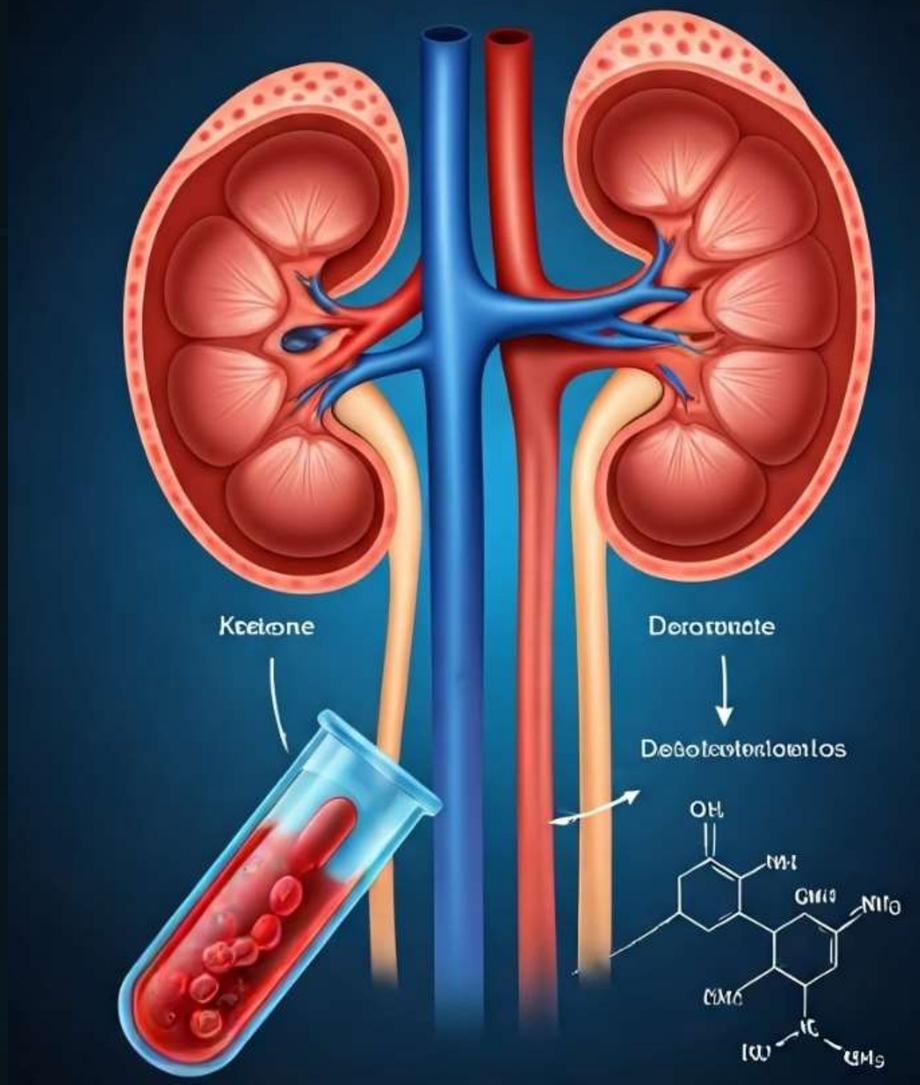
Uncontrolled type 1 diabetes due to insulin deficiency.

Mechanism

Increased lipolysis raises ketogenesis beyond buffering limits.

Other Causes

Severe starvation and alcoholism as triggers.



Pathophysiology of Diabetic Ketoacidosis

1 **Hyperglycemia**

Blood glucose above 250 mg/dL.

2 **Metabolic Acidosis**

pH below 7.3; bicarbonate under