Two hormones, opposite effects

Glucose is a respiratory substrate. Negative feedback keeps its blood concentration within a suitable range as food intake and energy demands change.

1. THE PANCREAS DETECTS AND SIGNALS

BLOOD GLUCOSE RISES

↓

Beta cells release insulin

↓

Muscle: greater glucose uptake.
Liver and muscle: more glycogen formation (glycogenesis).

↓

Blood glucose falls

↓

Secretion of this hormone declines towards basal levels.

BLOOD GLUCOSE FALLS

↓

Alpha cells release glucagon

↓

Liver: glycogen breakdown and glucose production increase; glucose is released into blood.

↓

Blood glucose rises

↓

Secretion of this hormone declines towards basal levels.

Alpha and beta cells are in the pancreatic islets of Langerhans.

2. HORMONES TRAVEL; TARGET CELLS RESPOND

Insulin and glucagon are secreted into the blood. They bind to specific receptors on target cells and alter enzyme activity. They are signals, not enzymes that directly convert glucose and glycogen. Their opposing actions make them antagonistic hormones.

3. CLOSE THE FEEDBACK LOOP

A fall in glucose reduces the stimulus for insulin secretion; a rise reduces the stimulus for glucagon secretion. Explain the return towards the normal range, not an unlimited fall or rise. Basal hormone secretion still contributes to regulation.

Hormonal feedback: OpenStax, Biology 2e 37.3.
Basal secretion and hepatic glucose output: Endotext, Glucagon Physiology.

Scope: Cambridge 9700, 14.1 outcomes 9-10, p. 38; diabetes is context.

From glucagon to a cell response

Glucagon binds at the liver-cell surface. The signal is relayed inside the cell; the hormone does not need to enter the cytoplasm.

1. FOLLOW THE SIGNAL IN ORDER

RECEPTOR
Glucagon binds; the cell-surface receptor changes shape.

G-PROTEIN
The activated receptor activates a G-protein.

ADENYLYL CYCLASE
The G-protein stimulates this membrane-associated enzyme.

SECOND MESSENGER
Adenylyl cyclase converts ATP to cyclic AMP (cAMP).

PROTEIN KINASE A
cAMP activates protein kinase A, initiating an enzyme cascade.

AMPLIFICATION
Successive phosphorylation steps activate more enzymes.

CELLULAR RESPONSE
Activated glycogen phosphorylase promotes glycogen breakdown.

2. A SMALL SIGNAL CAN HAVE A LARGE EFFECT

Each activated component can activate many molecules at the next stage. This amplification produces a substantial metabolic response to a small hormone signal. The products of glycogen breakdown are processed to glucose, which liver cells can release into blood.

Distinguish the messengers: glucagon is the extracellular first messenger; cAMP is the intracellular second messenger. ATP is the precursor used to make cAMP.

Required signalling sequence: Cambridge Biology 9700, 14.1 outcome 9.
Glucagon receptors, cAMP and metabolic effects: Endotext.

Scope: Cambridge 9700, 14.1 outcomes 9-10, p. 38; diabetes is context.

Keep the terms and targets clear

Glucose is a soluble sugar, glycogen is a storage polysaccharide, and glucagon is a peptide hormone. Similar names do not mean similar roles.

1. THREE DIFFERENT METABOLIC PROCESSES

Glycogenesis	Glucose is used to build glycogen for storage.
Glycogenolysis	Stored glycogen is broken down.
Gluconeogenesis	Glucose is made from non-carbohydrate precursors, such as lactate, glycerol and glucogenic amino acids.

2. LIVER AND MUSCLE ARE NOT IDENTICAL

Insulin increases GLUT4 transporter availability in muscle-cell membranes and stimulates glycogen formation in muscle and liver. Liver glucose transport is not switched on by GLUT4 insertion. Liver glycogen can support blood glucose; muscle glycogen mainly supplies the muscle itself.

3. DIABETES MELLITUS - CONTEXT

Type 1	Immune destruction of beta cells causes little or no insulin production.
Type 2	Target tissues respond less effectively to insulin. Insulin production may eventually become insufficient for the body's needs.

Both can result in persistently high blood glucose.

WHY HIGH GLUCOSE CAN CAUSE WATER LOSS

If the kidney cannot reabsorb all filtered glucose, glucose remains in urine. It retains water osmotically, increasing urine volume and potentially causing dehydration and thirst.

Tissue-specific insulin effects: NCBI Bookshelf, The endocrine pancreas.
Diabetes types: NIDDK; kidney effects: OpenStax 37.3 (page 1 link).

Scope: Cambridge 9700, 14.1 outcomes 9-10, p. 38; diabetes is context.