

Exercise walk-through

# 2.5

## Glycogen and gluconeogenesis

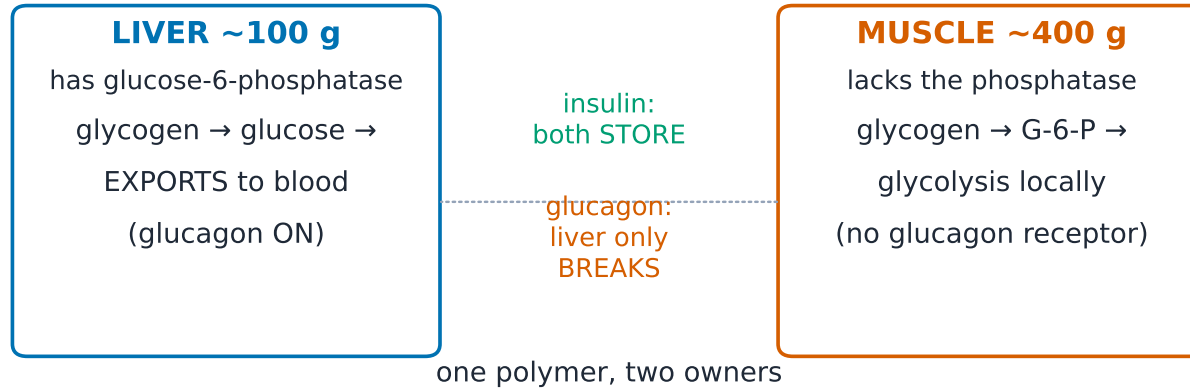
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University Medicine

## Two banks, one polymer

- 1 **Banks** Liver ~100 g exports glucose (has the phosphatase); muscle ~400 g keeps it (lacks the enzyme).
- 2 **Switch Insulin:** both store. **Glucagon:** liver only breaks down - muscle has no receptor.
- 3 **Cost Gluconeogenesis:**  $4 \text{ ATP} + 2 \text{ GTP} + 2 \text{ NADH}$  per glucose (6 high-energy bonds) - storage in reverse costs.

# Liver feeds, muscle keeps



Liver has glucose-6-phosphatase and exports; muscle traps G-6-P for itself - **insulin** stores both, **glucagon** opens only the liver.

## Exercises 2.1-2.3

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**2.1** Why can muscle glycogen not raise blood glucose? [3]

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**2.2** Name the hormone that activates liver phosphorylase and its tissue specificity. [3]

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**2.3** Gluconeogenesis from 2 pyruvate: count the high-energy phosphates spent. [3]

## Exercises 2.1-2.3

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**2.1** Why can muscle glycogen not raise blood glucose? [3]

**2.1**

- Muscle lacks glucose-6-phosphatase, so G-6-P cannot become free glucose for export.
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**2.2** Name the hormone that activates liver phosphorylase and its tissue specificity. [3]

**2.2**

- Glucagon; liver only (no muscle receptors).
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**2.3** Gluconeogenesis from 2 pyruvate: count the high-energy phosphates spent. [3]

**2.3**

- $4 \text{ ATP} + 2 \text{ GTP} = 6 \text{ high-energy phosphates (plus 2 NADH)}$ .

## Exercise 3.1

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**3.1** A 24-hour fast (fictional healthy volunteer).

Predict the fuel sequence hour by hour. Compute how long liver glycogen alone covers the brain. State the three gluconeogenic substrates and their origins. Explain why prolonged fasting spares muscle protein. [8]

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### 3.1

- Hours 0–4 absorbed fuels; 4–12 hepatic glycogenolysis; 12–24 gluconeogenesis dominant; day 3+ ketones cover  $\sim 2/3$  brain needs.
- $100 \text{ g} \div \sim 5 \text{ g/h} \approx \text{up to } 24 \text{ h}$ ; strictly, glycogen alone  $\approx 1$  day of brain use (400 vs 480 kcal).
- Substrates: alanine (muscle), glycerol (fat), lactate (Cori).
- Ketones substitute for glucose  $\rightarrow$  less gluconeogenesis  $\rightarrow$  protein sparing.

## Exercise 3.2

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**3.2** (Fictional) McArdle-pattern patient: sprint cramps, no lactate rise, myoglobinuria.

Localise the defect. Explain the absent lactate. Distinguish from a PFK-1 deficiency using the same test. State the long-term exercise advice implied by the mechanism. [7]

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### 3.2

- Muscle glycogen phosphorylase.
- No glycogen  $\rightarrow$  no extra G-6-P flux  $\rightarrow$  no pyruvate surge  $\rightarrow$  no lactate.
- PFK-1 deficiency: glycogen BREAKS down but flux dies at the gate – also low lactate, but with phosphate intermediates accumulating.
- Advice: aerobic, low-intensity exercise (fat/fatty acids still usable); avoid sprints.