Glycolysis

* GluT receptor for Glucose entry , working in bidirectional.

- GluT 1 → BBB ← Red blood cell. / Fetus. / barrier.
- GluT 2 → kidney / Liver / pancreas.
- GluT 3 → neuron / kidney.
- GluT 4 → muscle / Adipose tissue.

Insulin independent.

insulin dependent.

- Kinase → adding Ⓟ.
- mutase → changing Ⓟ position.

Glucose

Muscle ← Hexokinase / or / Liver ← Glucokinase ; ATP → ADP

Glucose-6-P (prevent it to exit).

phosphGlucoisomerase

Fructose-6-P

phospn fructo kinase-1 (Pfk1) ; ATP → ADP

step 3 rate-limiting-step.

Fructose-1,6-bisphosphate

Aldolase

num 2 since we have (2) GA3P

triose-P-isomerase

DHAP ⇌ 2GA3P (Glycer-Aldehyde-3-P).

GA3P D.H ; 2NAD+ → 2NADH ; 2ATP → 2ADP.

2 1,3-Bisphospho Glycerate

phospho Glycerate kinase (adding Ⓟ for ADP). ; 2ADP → 2ATP

2 3-phospho Glycerate

phospho Glycerate mutase

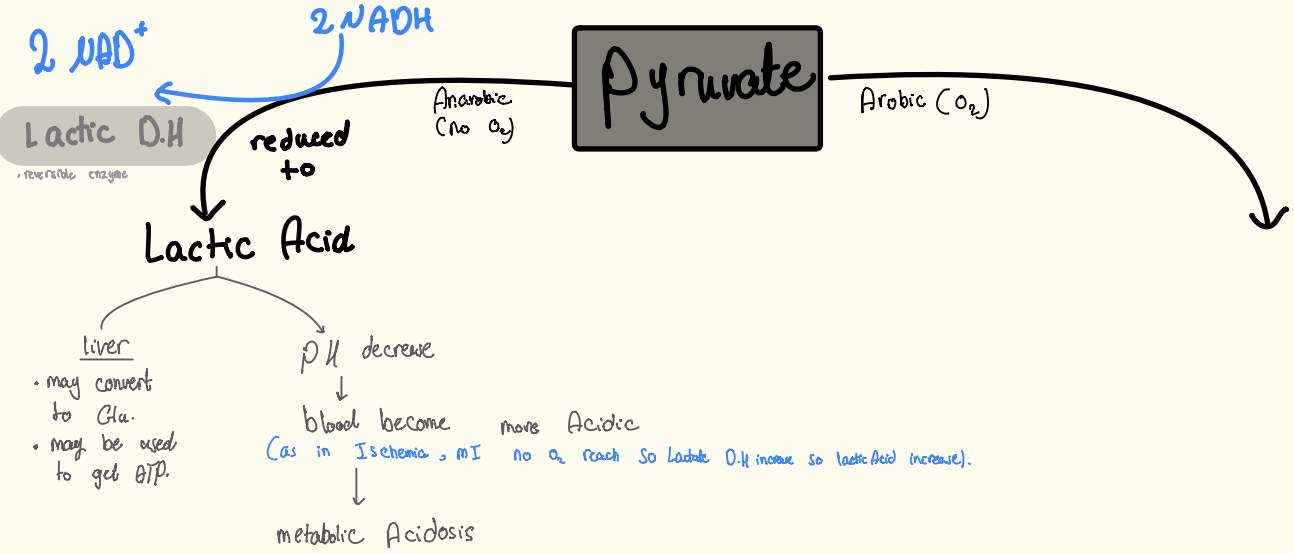
2 2-phospho Glycerate

Enolase

2 phospho enol pyruvate [PEP]

pyruvate kinase (adding Ⓟ to ADP). ; 2ADP → 2ATP

2 pyruvate



* occur in Cytoplasm.
* end product: 2 pyruvate, 2 NADH, 2 ATP (4 but we use 2)

Regulation of Glycolysis

Allosteric
molecules binding to active site and make conformational change.

Hormonal
Could do:
de/phosphorylation.

allosteric

- **Glucokinase** present in nucleus ← Glucose / Fru-6-phosphate activate it., return it into nucleus

Insulin stimulate more Glycolysis to happen so it stimulate Glucokinase.

Glucagon released when Glucose is gone, I don't need Glycolysis, so inhibit enzyme. (I need Gluconeogenesis).

- **Phosphfructo kinase 1**

Fructose 2,6 B-phosphate, ↑ADP, ↓ATP } → show low level of ATP. (stimulate enzyme).
↑Citrate } → inhibit enzyme (show high ATP level).

Fructose-6-phosphate ⟵ PFK2 | F2,6BP ⟶ Fructose 2,6-Bis phosphate
most powerful activator for PFK1

insulin Stimulate more glycolysis to happen (stimulate PFK2, inhibit F2,6-BP)

Glucagon inhibit Glycolysis to happen (inhibit PFK2, inhibit F2,6BP).

- Glucagon → وبيفعل → Phosphorylation. when PFK-2 get phosphorylated become inhibited. when F2,6BP get phosphorylated become activated

• Insulin ——stimulate——→ dephosphorylation. when PFK-2 get de-Ⓟ become activated.
when F2,6BP get de-Ⓟ become inhibited.

● Pyruvate Kinase (adding Ⓟ for ADP) generating Pyruvate when I need ATP.

by phosphorylation
Glucagon, ↑ ATP, ↑ Acetyl-CoA (result from fatty acid oxidation) inhibtor for this enzyme. } Allosteric
hormonal
more Acyl CO-A → lead to more krebs

Insulin, Fru-1,6 Bisphosphate activator for enzyme.
by dephosphorylation

* Arsenate work as Glycolysis inhibitor by inhibit < pyruvate kinase / GA3P DH.