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Subject	Final Exam - Chapter Eighteen

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Storage Mechanisms and Control in Carbohydrate Metabolism

☒ How Glycogen is Produced and Degraded?

- Glucose is stored in our bodies as glycogen which is similar to starch found in plants, but differs from starch in the degree of chain branching; so sometimes glycogen is called animal starch.
- In the degradation of glycogen several glucose residues can be released simultaneously; one from each end of a branch rather than one at a time as would be the case in linear polymer.
- The structure of glycogen with an average chain length of branches 13 residues was found that it is **optimized** for its ability to deliver energy quickly and for the longest amount of time possible.

☒ Breakdown of glycogen (Glycogenolysis):

- Low level of glucose in blood is the triggering factor for the release of stored glycogen in the liver.
- **Liver**: glycogen → glucose-6-phosphate→ glucose → blood.
- **Muscle**: glycogen→glucose-6-phosphate →glycolytic pathway.

- Conversion of Glycogen to glucose-6-phosphate takes place in 3 reactions:

1. **Phosphorolysis**: each glucose residue cleaved from glycogen reacts with phosphate to give glucose-1-phosphate by ***glycogen phosphorylase*** {cleaves α (1→ 4) linkages in glycogen}.

   Glycogen + phosphate ion → remainder of glycogen + glucose-1-phosphate.

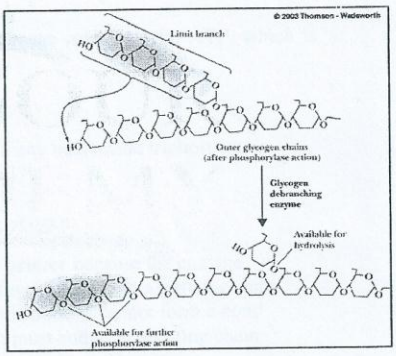
   Note: no ATP is hydrolyzed in this reaction.

   Remmber: in glycolysis, we saw another example of phosphorylation of substrate directly by phosphate without involvement of ATP; the phosphorylation of glyceraldehyde-3-phosphate to 1,3-bisphosphoglycerate. This is an alternative mode of entry to glycolytic pathway that "saves" an ATP for each glucose in the 1st step in glycolysis

2. **Isomerization** of glucose-1-phosphate to give glucose-6-phosphate by **phosphoglucomutase**.

3. **Debranching reaction**: by what is called ***debranching enzymes*** which transfer a "limit branch" of three glucose residues to the end of another branch and degrades the α (1→ 6) glycosidic bond of the last glucose residue remaining at the branch point.

- When glycogen rather than glucose is the starting material for glycolysis, there is a net gain of three ATP molecules for each glucose monomer, rather than two molecules. So, glycogen is a more effective energy source than glucose.



- Muscle tissue can mobilize glycogen more easily than fat and can do so anaerobically.

- In low intensity exercise fat is the preferred fuel, but as the intensity increases muscle and liver glycogen become more important.

☒ Formation of Glycogen from Glucose:

- The formation of glycogen from glucose **is not the exact reversal** of the breakdown of glycogen to glucose.

- Glycogen synthesis needs the hydrolysis **of UTP** as the source of energy.

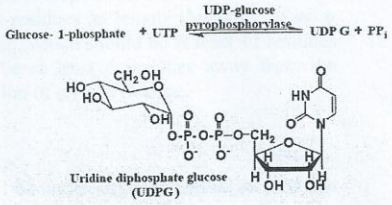
- **Stages of glycogen synthesis:**

1. Glucose-1-phosphate + UTP → uridine diphosphate glucose (UDPG) + pyrophosphate (PPi)

Note: Glucose-1-phosphate is obtained from glucose-6-phosphate by isomerization reaction

- The enzyme ***UDP-glucose pyrophosphorylase*** catalyzes this reaction.

- The exchange of one phosphoric anhydride bond for another has a free-energy change close to zero. The release of energy comes about when the enzyme inorganic pyrophosphatase catalyzes the hydrolysis of pyrophosphate into two phosphates (strongly exergonic reaction):



$$H_2O + PPi \rightarrow 2Pi$$

- It is common to see the energy released by the hydrolysis of pyrophosphate combined with the free energy of hydrolysis of nucleoside triphosphate. The coupling of these two exergonic reactions to a reaction that is not energetically favorable allows an otherwise endergonic reaction to take place.

- The supply of UTP is replenished by an exchange reaction with ATP, which is catalyzed by ***nucleoside phosphate kinase***:

$$UDP + ATP \leftrightarrow UTP + ADP$$

- This exchange reaction makes the hydrolysis of any nucleoside triphosphate energetically equivalent to the hydrolysis of ATP.

2. The addition of UDPG to a growing chain of glycogen:

- The initiation of glycogen synthesis requires a **primer** because the enzyme ***Glycogen synthase*** {which catalyzes the addition of UDPG to a growing chain of glycogen by formation of α (1→ 4) glycosidic bonds} cannot form a bond between two isolated glucose molecules, but it must add to an existing chain with α (1→ 4) glycosidic linkages.

- The **glycogenin** with its tyrosine hydroxyl group serves as a primer for glycogen synthesis.

- **The stages:**

  a. Glucose residue is linked to the hydroxyl group on glycogenin.

  b. Other glucose residues are added to the previous glucose residue and the **glycogenin** acts as a catalyst, until there are about **8** of them linked together, at that point **glycogen synthase** takes over.

  c. Synthesis of glycogen requires the formation of α (1→ 6) as well as α (1→ 4) glycosidic linkages. A ***branching enzyme*** is responsible for this, and it does so by transferring a segment about seven residues in length {breaking of an α (1→ 4)} from the end of a growing chain, which should be at least 11 residues long, to a branch point, which must be at least 4 residues away from the nearest existing branch point, by formation of α (1-6) linkage.

☒ Control of Glycogen metabolism:

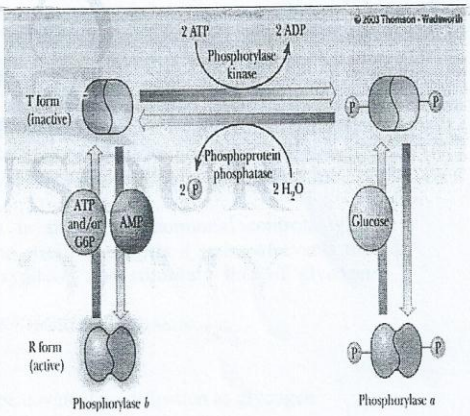
- Glycogen synthesis and breakdown should be under strict control to be sure that they don't operate at the same time, because if it happens, the net result will be losing chemical energy stored in phosphoric anhydride bonds by the hydrolysis of UTP.

- **Glycogen phosphorylase:**

  - The behavior of Glycogen phosphorylase is a major controlling factor. This enzyme is under allosteric and covalent modification control.

  - Is a dimer that exists in two forms, the inactive (T form) and the active (R form).

  - The T form (and only the T), it can be modified by **phosphorylation** of a specific serine residues on each of the two subunits.



  - The esterfication of the serines to phosphoric acid is catalyzed by the enzyme phosphorylase kinase, and the dephosphorylation is catalyzed by phosphoprotein phosphatase.

  - The phosphorylated form of Glycogen phosphorylase is called **Phosphorylase a** and the dephosphorylated form is called **phosphorylase b**.

- The switch from ***phosphorylase b to phosphorylase a is*** the major form of control over the activity of phosphorylase.

- The response time of the changes is on the order of seconds to minutes.
- Phosphorylase is also controlled more quickly in times of urgency by allosteric effectors, with a response time of milliseconds.

- **In liver:**

- ***Glucose*** is an allosteric inhibitor of **phosphorylase a** by:

1. Binding to the substrate site and favoring the transition to the T state.

2. Exposing the phosphorylated serines so that the phosphatase can hydrolyze them. And this will shift the equilibrium to **phosphorylase b**.

- **In muscle**:

- **ATP**, **AMP** and **glucose -6-phosphate (G6P)** are the primary allosteric effectors.

- When the muscles use ATP to contract, AMP levels rise, this increase in AMP stimulates formation of the R state of ***phosphorylase b***, which is active.

- When ATP is plentiful or glucose -6-phosphate builds up, these molecules act as allosteric inhibitors shifting the equilibrium back to the T form.

These differences ensure that glycogen will be degraded when there is a need for energy, as is the case with high [AMP], low [G6P], and low [ATP]. But when low [AMP], high [G6P], and high [ATP], the energy need and glycogen breakdown is less. Shutting down glycogen phosphorylase activity is the appropriate response.

- Glycogen Synthesis and breakdown is also under hormonal control. When ***epinephrine*** is released in response to stress, it triggers a series of events that **suppress** the activity of glycogen synthase and **stimulate** that of glycogen phosphorylase.

- **Glycogen synthase**:

- Its activity is subjected to the same type covalent modification as glycogen phosphorylase, but the difference is that the response is opposite.

- The **phosphorylated form** is the **inactive** form and the **dephosphorylated** form is the **active** form.

- Hormonal signals (**epinephrine** or **glucagon**) stimulate the phosphorylation of glycogen synthase via an enzyme called ***cAMP-dependent protein kinase***, and by this phosphorylation it becomes inactive at the same time the hormonal signal is activating phosphorylase.

- The phosphorylation of glycogen synthase is also more complicated because there are multiple phosphorylation sites. 9 different amino acids have been found to be phosphorylated. As the progressive level of Phosphorylation increases, the activity of the enzyme decreases.

- Glycogen synthase can be phosphorylated by other enzymes like: ***phosphorylase kinase***, and ***glycogen synthase kinases***.

- Glycogen synthase is dephosphorylated by the same phosphoprotein phosphatase that removes the phosphate from phosphorylase.

- Glycogen synthase is under allosteric control; it is inhibited by **ATP** and activated by **glucose-6-phosphate**. The 2 forms of this enzyme responds very differently to G6P:

– The phosphorylated (inactive form) is called **glycogen synthase D** (for glucose-6-phosphate dependent) because it is only active under very high concentration of glucose-6-phosphate.

– The nonphosphorylated (active form) is called **glycogen synthase I** (for glucose-6-phosphate independent) because it is active even with low concentrations of glucose-6-phosphate.

☺ So, even though purified enzymes can be shown to respond to allosteric effectors, the true control over the activity of glycogen synthase is by it phosphorylation state, which, in turn, is controlled by hormonal states.

- The modifying enzymes of glycogen synthase and phosphorylase are themselves subject to covalent modification and allosteric control.

☒ **How does Gluconeogenesis produce glucose from pyruvate?**

- **Gluconeogenesis** is the conversion of puruvate to glucose.

- **Pyruvate** is a product of glycolysis, but it can arise from other sources to be the starting point of anabolism of glucose.

- Gluconeogenesis is not the exact reversal of glycolysis, because there are 3 irreversible steps in glycolysis which are bypassed in gluconeogenesis, and those are:

1. The production of **pyruvate (and ATP) from PEP**. And because reaction is exergonic, the reverse reaction is endergonic.

2. The production of **fructose-1, 6-bisphosphate** from **fructose-6-phosphate**.

3. The production of **glucose-6-phosphate from glucose**.

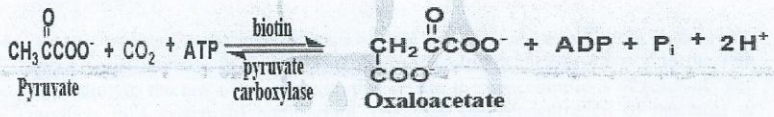
Reversing the last two reactions would require the production of ATP from ADP, which is also **endergonic reaction**. The net result of gluconeogenesis includes the reversal of these 3 glycolytic reactions, but with different pathway, reactions, and enzymes.

☺ **Oxaloacetate is an intermediate in gluconeogenesis:**

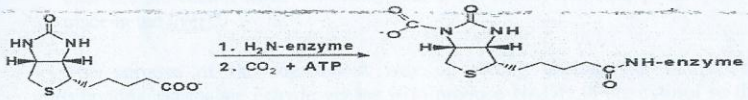
- The conversion of pyruvate to phosphoenolpyruvate in gluconeogenesis takes place in two steps:

1- Pyruvate + ATP + CO_2 + H_2O ↔ **Oxaloacetate** + ADP + Pi + 2H

- This step requires energy which is available from the hydrolysis of ATP.
- It is catalyzed by the enzyme ***pyruvate carboxylase*** (an allosteric enzyme found in mitochondria).

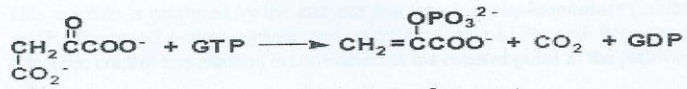


- **Acetyl-CoA** is an allosteric effector that activates pyruvate carboxylase.
- If high levels of acetyl CoA are present (i.e if there is more acetyl CoA than is needed to supply the citric acid cycle), **pyruvate** (a precursor of acetyl CoA) can be diverted to gluconeogenesis. (Oxaloacetate from the citric acid cycle can be frequently a starting point for gluconeogenesis as well). **Magnesium ion (Mg^{+2})** and **biotin** are also required for effective catalysis.
- Biotin is a carrier of CO_2, the carboxyl group of biotin forms an **amide bond** with the ε-amino group of a specific lysine side chain of pyruvate carboxylase. The CO_2 is attached to the biotin, which in turn covalently bonded to the enzyme, and then the CO_2 is shifted to pyruvate to form oxaloacetate. Note that ATP is required for this reaction.



2. **Oxaloacetate** + GTP → **Phosphoenolpyruvate** + CO2 + GDP

- This reaction is catalyzed by the enzyme **phosphoenolpyruvate carboxykinase (PEPCK)**, which is found in the mitochondria and the cytosol.
- It involves hydrolysis of GTP rather than ATP.
- Mg^{+2} also is needed for effective catalysis.



Oxaloacetate **Phosphoenolpyruvate**

- The successive carboxylation and decarboxylation reactions are both close to equilibrium; as a result the conversion of pyruvate to phosphoenolpyruvate is also close to equilibrium.
- A small increase in the level of oxaloacetate can drive the equilibrium to the right, and a small increase in the level of phosphoenolpyruvate can drive it to the left. According to the law of mass action that relates the concentrations of reactants and products in a system of equilibrium, changing the concentration of reactants or products will cause a shift to reestablish equilibrium.

Pyruvate + ATP + GTP → PEP + ADP + GDP

- The oxaloacetate formed in the mitochondria can have two fates with respect to gluconeogenesis:

1. It can continue to form PEP, which can then leave the mitochondria via a specific transporter to continue gluconeogenesis in the cytosol.

2. The other possibility is that oxaloacetate can be turned into **malate** via mitochondrial ***malate dehdrogenase***, a reaction that uses NADH.

Malate can then leave the mitochondria and have the reaction **reversed** by cytosolic malate dehydrogenase. The reason for this two step process is that oxaloacetate cannot leave the mitochondria, but malate can (this pathway takes place in the liver).

- The purpose of the roundabout way of getting oxaloacetate out of the mitochondria via malate dehydrogenase is to produce NADH in the cytosol so that gluconeogenesis can continue, because the cytosol has low ratio of NADH to NAD^+.

☒ The role of Sugar phosphates in Gluconeogenesis:

- The other two reactions in which gluconeogenesis differs from glycolysis are ones in which phosphate-ester bond to a sugar-hydroxyl group is hydrolyzed. Both reactions are catalyzed by phosphatases and both are exergonic.

- The **first reaction** is the hydrolysis of fructose-1,6-bisphosphate to produce fructose -6- phosphate and phosphate ion.

This reaction is catalyzed by the enzyme ***fructose-1, 6-bisphosphatase*** (inhibited by AMP, fructose-2,6-bisphosphate and stimulated by ATP), and because of this allosteric control this reaction is considered as the **control point** in the pathway.

When the cell has an ample supply of ATP, the formation rather than the breakdown of glucose is favored.

- The **second reaction** is the hydrolysis of glucose-6-phosphate to glucose and phosphate ion.

- The enzyme that catalyzes this reaction is ***glucose-6-phosphatase***, with Mg^{+2} ions for efficient catalyses. This reaction happens in the smooth endoplasmic reticulum only.

☒ How carbohydrate metabolism is controlled?

We have seen many aspects of carbohydrate metabolism:

- **Gluconeogensis,** and **glycogen break-down** where the glucose is obtained.
- **Glycolysis** and **glycogen synthesis** where the glucose is the starting point. So how all these related pathways are controlled?

☒ Control of phosphofructokinase and fructose-1,6-bisphosphatase:

We mentioned that fructose-2, 6-bisphosphate (F2,6P) is an allosteric activator of **phosphofructokinase (PFK) in glycolysis** and allosteric inhibitor of **fructose bisphosphate phosphatase (FBPase) in gluconeogenesis.**

- ↑ fructose-2,6-bisphosphate → stimulate glycolysis
- ↓ fructose-2,6-bisphosphate → stimulate gluconeogensis

The concentration of F2,6P in a cell depends on the balance between its synthesis, catalyzed by phosphofructokinase-2 (PFK-2), and its breakdown, catalyzed by fructose-bisphosphatase-2 (FBPase-2).

The enzymes that control the formation and the break down of fructose-2,6-bisphosphate are themselves controlled by **phosphorylation/dephosphorylation** mechanism similar to glycogen phosphorylase and glycogen synthase. How?

- Phosphorylation of the dimeric protein leads to an increase in the activity of **fructose bisphosphate phosphatase-2 (FBPase-2)** and a decrease in concentration of **F2,6P,** ultimately stimulating gluconeogenesis.
- Dephosphorylation of the dimeric protein leads to an increase in **Phosphofructokinase-2 (PFK-2)** and an increase in the concentration of **F2, 6P,** ultimately stimulating glycolysis.

☒ **Mechanisms of metabolic control:**

Type of control	Mode of operation	Examples
Allosteric	Effectors (substrates, products, or co-enzymes) of the pathway inhibit or activate the enzyme	ATCase, phosphofructokinase
Covalent modification	Inhibition or activation of the enzyme depends on the formation or breaking of a bond, frequently by Phosphorylation or Dephosphorylation	Sodium-potassium pump, glycogen phosphorylase, glycogen synthase.
Substrate cycles	2 opposing reaction such as formation and breakdown of a given substance are catalyzed by a different enzymes which can be activated or inhibited separately	Glycolysis and gluconeogenesis
Genetic control	The amount of enzyme present is increased by protein synthesis.	Induction of β-galactosidase

☺ **Substrate cycling** refers to the fact that opposing reactions can be catalyzed by different enzymes. So, they can be independently regulated and have different rates. We will take an example of conversion of fructose-6-phosphate to fructose-1,6-bisphosphate, and breakdown of fructose-1,6-bisphosphate to fructose-6-phosphate.

Fructose-6-phosphate + ATP → Fructose-1,6-bisphosphate + ADP
(***phosphofructokinase***)
(This reaction occurs in glycolysis ΔG = -25.9)

fructose-1,6-bisphosphate + H2O → fructose-6-phosphate + Pi
(***fructose-1,6-bisphosphotase***)
(This reaction occurs in gluconeogenesis, ΔG=-8.6)

Note that the opposing reactions are not the exact reverse of one another. If we add 2 reaction together: ATP + H2O ↔ ADP + Pi

ATP is the price paid for independent control for these opposing reactions.

❖ By using these different control mechanisms, the organism can divide the labor among different tissue, how? **Cori cycle** (by Gerty & Carl Cori) clarifies this.

Cori cycle: - cycling of glucose due to glycolysis in muscle and gluconeogenesis in liver.

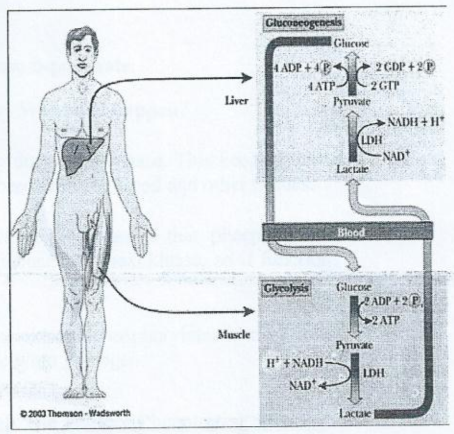
➢ Under conditions of oxygen dept, glycolysis in skeletal muscle produces lactate. (anaerobic metabolism)

➢ Lactate that is produced transferred to liver through blood.

➢ By gluconeogenesis in the liver, the lactate recycled to glucose (lactate is first oxidized to pyruvate).

➢ Glucose produced by the liver is transported back to the muscle.

➢ **The labor divided between liver and muscle.**



Note: in the same cell, glycolysis and gluconeogenesis are not highly active at the same time, when ATP needed, the glycolysis is more active, when little need for ATP, gluconeogenesis is more active.

- In glycolysis → 2ATP produced.
- In gluconeogenesis → 4ATP and 2GTP needed.

☺ So that in **Cori cycle we require the hydrolysis of 2ATP, and 2GTP.**

☒ <u>**Control of pyruvate kinase**</u>:

– Pyruvate kinase found as isozymes, with 3 different subunits, M (muscles), L (liver), A (other tissue).

– Pyruvate kinase is allosterically **<u>inhibited</u>** by:

1) **ATP** (no reason to metabolize glucose if there is already ATP).
2) **Alanin** (it is an amino version of pyruvate; it is synthesized from pyruvate by an enzyme called ***transaminase***).

- Pyruvate kinase is allosterically **activated** by **Fructose-1,6-bisphosphate**.

- **Liver** isozyme of the pyruvate kinase is subjected to covalent modification in addition to the allosteric control. **HOW?**

When glucose levels in the blood is low, glucagon production from the pancreas increases, which lead to the production of a protein kinase that **phosphorelate** the liver pyruvate kinase making it **LESS ACTIVE**, thus terminating the process of glycolysis and starting gluconeogenesis.

☒ **Control of hexokinase:**

- **Hexokinase** is allosterically inhibited by, **glucose-6-posphate**.
- If glycolysis is inhibited by phosphofrutokinase. **What will happen?**
- Glucose-6-phosphate will accumulate, shutting down hexokinase. This keeps glucose from being metabolized in the liver when it is needed in the blood and other tissues.
- However, the liver contains a second enzyme, **glucokinase** that phosphorylates glucose. This enzyme has a higher K_M for glucose than hexokinase, so it functions only when glucose is abundant.
- If there is an excess of glucose in the liver, glucokinase phosphorylates it to glucose-6-phosphate; so it can eventually be polymerized into glycogen.

☒ **Why is Glucose Sometimes Diverted Through The Pentose Phosphate Pathway?**

- Pentose phosphate pathway is an alternative to glycolysis, but here the production of ATP is not our main concern.
- What are the importances of this pathway:
  - Production of **five carbon sugars** like ribose (important in RNA, DNA)
  - Production of **NADPH** a compound that is the reducing agent.

What is the difference between NADH and NADPH?

1) Structure: **NADPH** has one extra phosphate group esterified to the ribose ring of adenine nucleotide portion of molecule.
2) Function: NADH is produced in the oxidative reactions that give rise to ATP. NADPH is a reducing agent in biosynthesis (reduction processes).

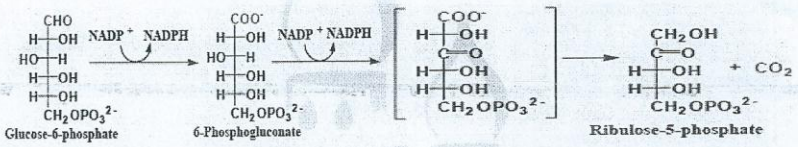
- The pentose phosphate pathway can be divided into 2 phase:

1. **Oxidative phase:** - result in production of NADPH and five carbon sugars.
2. **Nonoxidative phase**: - which involve nonoxidative reshuffling of the carbon skeletons of the sugars, to produce compounds that connect this pathway with glycolysis.

☺ **Oxidative reactions of the pentose phosphate pathway:**

Step 1: the **glucose-6-phosphate** is oxidized to **6-phosphogluconate** by the enzyme called ***glucose-6-phosphate dehydrogenase.*** The **NADPH** is produced by this reaction.

Step 2: involve oxidative decarboxylation of **6-phosphogluconate** to produce **ribulose-5-phosphate** (ketose), the enzyme used is ***6-phosphogluconate dehydrogenase,*** and **NADPH** is produced once again. **CO_2** is produced as well.



☺ **Nonoxidative reactions of the pentose phosphate pathway:**

- In the remaining of this pathway, several reactions involve transfer of two- and three-carbon units.
- There are 2 different reactions in which **ribulose-5-phosphate** isomerizes:

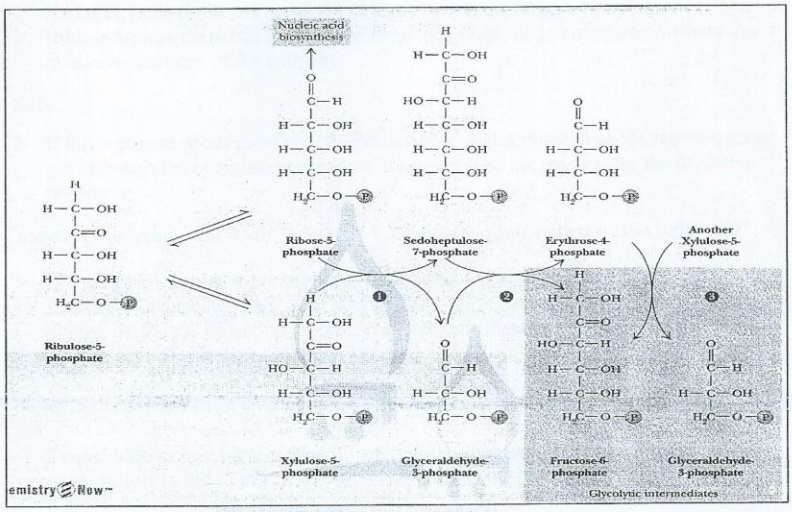
1) To produce **xylulose-5-phosphate** (ketose) by enzyme ***phosphopentose-3-epimerase,*** here, there is inversion of configuration around C3.

2) To produce **ribose-5-phosphate** (aldose) by enzyme called ***phosphopentose isomerase.*** Ribose-5-phosphate is an important building block for the synthesis of nucleic acids and coenzymes such as NADH.

- In rearrangement of carbons atoms, **2** molecules of xylulose-5-phosphate, and **1** molecule of ribose-5-phosphate (**total # of carbons = 15**) rearrange to give 2 molecules of fructose-6-phosphate, and 1 molecule of glyceraldehydes-3-phosphate (**total # of carbons = 15**).

- Two enzymes are responsible for this rearrangement:

1) ***Transketolase*** → transfers a two-carbon unit. (Catalyze the first and the third reaction)
2) ***Transaldolase*** → transfers a three-carbon unit. (Catalyzed the second reaction)



The rearrangement process involves 3 reactions which are summarized in the following table:

	reactant	Enzyme	Product
Two-carbon shift	C5 + C5	Transketolase	C7 + C3
Three-carbon shift	C7 + C3	transaldolase	C4 + C6
Two-carbon shift	C4+C5	Transketolase	C3+C6
Net reaction	3C5	↔	2C6 + C3

The C7→ sedoheptulose-7-phosphate
The C3→ glyceraldehydes-3-phosphate
The C6→ fructose-6-phosphate
The C4→ erythrose-4-phosphate

- **Pentose phosphate pathway = hexose monophosphate shunt.** This is because in the pentose phosphate pathway, G6P can be converted to fructose-6-phosphate and glyceraldehydes-3-phosphate by a means other than the glycolytic pathway.

☒ Control of Pentose Phosphate Pathway:

As we know the important of pentose phosphate pathway is to produce NADPH, and ribose-5-phosphate.

- **NADPH**, to be produced needs the oxidative portion of the pathway
- **Ribose-5-phosphate** can be obtained from the glycolytic intermediates without the oxidative reactions of the pathway.

So;

- If the organism needs more NADPH than ribose-5-phosphate, then the reaction series goes through the complete pathway as discussed. The net reaction for the oxidative portion is:

6 glucose-6-phosphate + 12 $NADP^+$ + 6H2O → 6ribose-5-phosphate + 6CO2 + 12NADPH + $12H^+$

- If the organism has a greater need for ribose-5-phosphate than for NADPH, then fructose-6-phosphate, and glyceraldehydes-3-phosphate can give rise to ribose-5-phosphate by the operation of transketolase and transaldolase reactions without the need to get through the oxidative reactions.

Note: the reactions catalyzed by transaldolase and transketolase are reversible.

- **Transaldolase** has many common features with the enzyme **aldolase**, which we met in glycolysis (what step?)

- **Transketolase** resembles **pyruvate decarboxylase**, both needs **Mg^{+2}**, and **thiamine pyrophosphate (TPP);** and carbanion play a crucial role in reaction machanism as well.

Questions

1-The preferred fuel source in low intensity exercises:
a- fat
b- glycogen
c- all of the above
d- none of the above

2-Glycogen phosphorylase under control by:
a- allosteric
b- covalent modification
c- Genetic control
d- a+b

3-Epinephrin cause:
a- suppres the activity of glycogen synthase
b- stimulate glycogen phophorylase
c- a+b
d- suppress phosphorylase

4-The hydrolysis of glu-6p to glucose and phosphate ion occur:
a- ER
b- cytosol
c- Mitochondria
d- all of the above

5-Transketolase enzyme transfer:
a- 2 carbon unite
b- 3carbon unite
c- 1 carbon unite
d- 4 carbon unite

6-Which of the following enzyme under genetic control:
a- ATCase
b- β-galactosidase
c- Na^+-K^+ pump
d- all of the above

7-Glycogen phosphorylase under allosteric inhibitor in liver by:
a- glucose
b- AMP
c- ATP
d- all of the above

Answer sheet

Question #	answer
1-	a-fat
2-	d-a+b
3-	c-a+b
4-	a-ER
5-	a-2 carbon unite
6-	b-β-galactosidase
7-	a-glucose

نعتز بثقتكم