

LEC 13 META Q:

1. Which enzyme is primarily responsible for the phosphorylation of glucose in low glucose conditions?

- a) Glucokinase
- b) Hexokinase
- c) Phosphofructokinase-1
- d) Pyruvate kinase

2. What is the characteristic feature of Hexokinase in terms of glucose affinity?

- a) High K_m , low affinity
- b) High V_{max}
- c) Low K_m , high affinity
- d) Activated by insulin

3. Which enzyme becomes active when glucose concentrations are high?

- a) Phosphofructokinase-1
- b) Glucokinase
- c) Hexokinase
- d) Pyruvate kinase

4. During the fasting state, which of the following occurs?

- a) Glucokinase becomes active
- b) Hexokinase operates at its V_{max}
- c) Blood glucose levels decrease immediately after eating
- d) Insulin levels increase significantly

5. In the well-fed state, which factor increases glucokinase activity?

- a) High glucose concentrations
- b) Low insulin levels
- c) High F6P concentrations
- d) Low glucose concentrations

6. What role does fructose-6-phosphate (F6P) play in regulating glycolysis?

- a) Activates PFK-1
- b) Inhibits glycolysis by re-sequestering glucokinase into the nucleus
- c) Increases blood glucose levels
- d) Increases fructose-2,6-bisphosphate production

7. What is the function of fructose-2,6-bisphosphate (F-2,6-BP) in glycolysis?

- a) Inhibits glycolysis
- b) Activates PFK-1, promoting glycolysis
- c) Inhibits PFK-1, slowing glycolysis
- d) Inhibits gluconeogenesis

8. Which of the following is a positive regulator of PFK-1?

- a) ATP
- b) Citrate
- c) AMP
- d) Protons (H^+)

9. Which of the following inhibits PFK-1 activity?

- a) Fructose-2,6-bisphosphate
- b) AMP
- c) ATP
- d) Pyruvate

10. How does the presence of AMP or fructose-2,6-bisphosphate affect the reaction curve of PFK-1?

- a) Shifts the curve to the right
- b) Reduces the V_{max}
- c) Shifts the curve to the left, enhancing enzyme activity
- d) Makes the curve linear

11. At high ATP concentrations, ATP acts as a:

- a) Positive regulator of PFK-1
- b) Negative regulator of PFK-1
- c) Substrate for pyruvate kinase
- d) Positive regulator of pyruvate kinase

12. What is the effect of fructose-1,6-bisphosphate (F-1,6-BP) on pyruvate kinase?

- a) Inhibits pyruvate kinase activity
- b) Activates pyruvate kinase through feedforward regulation
- c) Promotes gluconeogenesis
- d) Decreases glycolysis

13. Which hormone promotes glycolysis by increasing the levels of fructose-2,6-bisphosphate?

- a) Glucagon
- b) Insulin
- c) Cortisol
- d) Norepinephrine

14. During fasting, which hormone inhibits glycolysis and promotes gluconeogenesis by reducing fructose-2,6-bisphosphate levels?

- a) Insulin
- b) Cortisol
- c) Glucagon
- d) Epinephrine

15. What is the result of pyruvate kinase deficiency (PKD) in red blood cells?

- a) Increased ATP production
- b) Chronic hemolytic anemia due to low ATP
- c) Improved RBC shape and function
- d) Enhanced glycolysis in RBCs

16. Which of the following is a potential effect of arsenic poisoning on glycolysis?

- a) Increased ATP synthesis
- b) Inhibition of glyceraldehyde-3-phosphate dehydrogenase
- c) Enhanced pyruvate dehydrogenase activity
- d) Stimulation of glycolysis

17. How does fluoride inhibit bacterial glycolysis?

- a) By inhibiting glyceraldehyde-3-phosphate dehydrogenase
- b) By inhibiting enolase
- c) By increasing the V_{max} of glycolytic enzymes
- d) By activating pyruvate kinase

18. Which of the following is NOT a function of insulin in glucose metabolism?

- a) Facilitates glucose uptake into cells
- b) Activates glycolysis
- c) Promotes gluconeogenesis
- d) Increases fructose-2,6-bisphosphate levels

19. Which allosteric regulator is primarily responsible for activating PFK-1 during times of low energy?

- a) Citrate
- b) AMP
- c) ATP
- d) Alanine

20. What is the role of fructose-2,6-bisphosphate in the regulation of glycolysis and gluconeogenesis?

- a) It promotes both glycolysis and gluconeogenesis simultaneously
- b) It activates glycolysis and inhibits gluconeogenesis
- c) It inhibits both glycolysis and gluconeogenesis
- d) It inhibits glycolysis and promotes gluconeogenesis

ANSWERS

- 1. b
- 2. c
- 3. b
- 4. b
- 5. a
- 6. b
- 7. b

8. c
9. c
10. c
11. b
12. b
13. b
14. c
15. b
16. b
17. b
18. c
19. b
20. b

21. What happens to glucokinase activity during low glucose conditions?

- a) It is inactive and sequestered in the nucleus
- b) It is active in the cytosol
- c) It binds to hexokinase regulatory protein (HKRP)
- d) It converts glucose to fructose-6-phosphate

22. What is the role of GLUT-4 in glucose metabolism?

- a) It helps transport glucose into the mitochondria
- b) It facilitates glucose uptake into cells, especially during fasting
- c) It is a transporter induced by insulin for glucose uptake
- d) It inhibits glucose uptake during the well-fed state

23. Which of the following is true about insulin's effect on glucose metabolism?

- a) It decreases fructose-2,6-bisphosphate levels
- b) It inhibits glucokinase activity
- c) It enhances glycolysis by activating PFK-1
- d) It inhibits glucose uptake into cells

24. Which of the following is associated with the presence of high F6P levels?

- a) Activation of glucokinase
- b) Downregulation of glycolysis
- c) Promotion of PFK-1 activity
- d) Activation of AMP

25. In the fasting state, which of the following is true regarding glucokinase activity?

- a) It is activated due to high blood glucose
- b) It is sequestered in the nucleus and inactive
- c) It is upregulated by high fructose-2,6-bisphosphate levels
- d) It is inhibited by low insulin levels

26. Which of the following allosteric regulators signals a high energy state in the cell and inhibits glycolysis?

- a) AMP
- b) ATP
- c) Fructose-2,6-bisphosphate
- d) Pyruvate

27. Which enzyme is primarily responsible for the conversion of glucose to glucose-6-phosphate?

- a) Hexokinase
- b) Pyruvate kinase
- c) Glucokinase
- d) Phosphofructokinase-1

28. What effect does high AMP concentration have on glycolysis?

- a) It inhibits glycolysis
- b) It activates PFK-1 and promotes glycolysis
- c) It inhibits fructose-1,6-bisphosphate
- d) It deactivates pyruvate kinase

29. Which of the following is true about pyruvate kinase regulation?

- a) ATP acts as a positive allosteric regulator
- b) Fructose-1,6-bisphosphate inhibits pyruvate kinase activity
- c) Insulin promotes pyruvate kinase activity
- d) Pyruvate kinase activity is inhibited by AMP

30. Which of the following hormones acts to reduce glycolysis and promote gluconeogenesis during fasting?

- a) Cortisol
- b) Insulin
- c) Glucagon
- d) Epinephrine

31. Which of the following is a known effect of chronic stress or synthetic cortisol use on metabolism?

- a) Increased glucose storage
- b) Enhanced glycolysis and decreased glucose metabolism
- c) Weight loss due to increased glucose uptake
- d) Weight gain due to altered glucose and fatty acid metabolism

32. How does fructose-2,6-bisphosphate (F2,6BP) act on the bifunctional enzyme PFK-2/FBPase-2?

- a) It activates the phosphatase activity, reducing F2,6BP levels
- b) It deactivates the kinase component of PFK-2/FBPase-2
- c) It inhibits the phosphatase activity, increasing F2,6BP levels
- d) It binds to the enzyme, promoting gluconeogenesis

33. What happens when glucagon levels are high in the fasting state?

- a) Glycolysis is activated and gluconeogenesis is inhibited
- b) Pyruvate kinase is phosphorylated and inactivated
- c) Fructose-2,6-bisphosphate levels increase
- d) Glycogen storage is enhanced

34. Which of the following describes the role of alanine in the regulation of pyruvate kinase?

- a) Activates pyruvate kinase to promote glycolysis
- b) Inhibits pyruvate kinase, signaling sufficient pyruvate availability
- c) Increases glucose uptake by cells
- d) Inhibits glucokinase to prevent glucose phosphorylation

35. What is a major feature of pyruvate kinase deficiency in red blood cells (RBCs)?

- a) Excessive ATP production
- b) Impaired Na^+/K^+ pump leading to disrupted ion gradients
- c) Increased glucose metabolism in RBCs
- d) Enhanced RBC flexibility for movement through capillaries

36. Which of the following is a consequence of arsenate poisoning?

- a) Inhibition of glycolysis by blocking pyruvate kinase
- b) Disruption of ATP production by competing with phosphate during glycolysis
- c) Activation of the Krebs cycle leading to excess ATP production
- d) Increased glucose metabolism due to the activation of hexokinase

37. Which compound competes with phosphate during glycolysis and disrupts ATP synthesis?

- a) Fluoride
- b) Citrate
- c) Arsenate
- d) Arsenite

38. How does fluoride contribute to the prevention of dental caries?

- a) It enhances the activity of enolase in bacteria
- b) It inhibits bacterial enolase, reducing glycolysis in bacteria
- c) It promotes glucose uptake by oral bacteria
- d) It increases the ATP production in oral bacteria

39. Which of the following is the main effect of high glucose concentrations on glucokinase?

- a) It increases the affinity of glucokinase for glucose
- b) It causes glucokinase to dissociate from its regulatory protein and become active
- c) It inactivates glucokinase by phosphorylating it
- d) It reduces glucokinase's V_{max}

40. Which of the following best describes the behavior of PFK-1 in the presence of high ATP?

- a) ATP inhibits PFK-1, reducing the glycolytic flux
- b) ATP activates PFK-1 to promote glycolysis
- c) ATP has no effect on PFK-1
- d) ATP shifts the PFK-1 curve to the left, enhancing its activity

Answer:

- 21. a
- 22. c
- 23. c
- 24. b
- 25. b
- 26. b
- 27. c
- 28. b
- 29. c
- 30. c
- 31. d
- 32. c
- 33. b
- 34. b
- 35. b
- 36. b
- 37. c
- 38. b
- 39. b
- 40. a

41. Which of the following is true regarding the mechanism of glucose uptake during the well-fed state?

- a) GLUT-4 activity decreases in muscle cells
- b) Insulin promotes the translocation of GLUT-4 to the plasma membrane
- c) GLUT-4 is not affected by insulin in adipose tissue
- d) GLUT-4 translocation occurs in liver cells under fasting conditions

42. What role does fructose-6-phosphate (F6P) play in the regulation of glucokinase activity?

- a) F6P increases glucokinase activity by inhibiting GKRP binding
- b) High F6P levels signal the activation of glucokinase in the cytosol
- c) F6P binds to glucokinase, promoting its sequestration in the nucleus
- d) F6P inhibits glucokinase by promoting its re-sequestration in the nucleus

43. How does fructose-2,6-bisphosphate (F-2,6-BP) regulate phosphofructokinase-1 (PFK-1)?

- a) F-2,6-BP inhibits PFK-1 by decreasing the enzyme's affinity for its substrate
- b) F-2,6-BP promotes PFK-1 activity by shifting its reaction curve to the right
- c) F-2,6-BP activates PFK-1 by increasing its affinity for fructose-6-phosphate
- d) F-2,6-BP inhibits glycolysis and promotes gluconeogenesis via PFK-1 inhibition

44. Which statement correctly describes the effect of high ATP concentrations on glycolysis in relation to PFK-1 activity?

- a) ATP stimulates PFK-1 activity, driving glycolysis forward
- b) ATP inhibits PFK-1 by binding to an allosteric site, decreasing glycolytic flux
- c) ATP enhances the formation of fructose-2,6-bisphosphate, activating PFK-1
- d) ATP has no effect on PFK-1 activity as it is not involved in the regulation of glycolysis

45. What is the primary mechanism by which glucagon reduces glycolysis in the liver during fasting?

- a) Deactivation of pyruvate kinase via phosphorylation
- b) Activation of the PFK-2/FBPase-2 phosphatase activity to reduce F2,6BP levels
- c) Increase in fructose-2,6-bisphosphate levels to activate glycolysis
- d) Direct inhibition of PFK-1 through phosphorylation

46. Which of the following describes the impact of pyruvate kinase deficiency (PKD) in red blood cells (RBCs)?

- a) RBCs exhibit excessive glucose uptake, causing increased ATP production
- b) PKD leads to a lack of ATP, disrupting Na^+/K^+ pump activity and resulting in premature RBC death
- c) RBCs compensate for ATP depletion by increasing fatty acid oxidation
- d) PKD leads to enhanced glycolytic flux, increasing RBC lifespan

47. How does high protons (H^+) in the cell affect glycolysis?

- a) H^+ enhances PFK-1 activity by decreasing the need for ATP
- b) High proton concentration promotes glycolysis to generate more ATP
- c) High H^+ levels inhibit PFK-1 activity to prevent excessive glycolysis and reduce cellular acidosis
- d) Protons bind to PFK-1, increasing its activity under low energy conditions

48. What is the role of AMP in regulating PFK-1 activity during a low energy state?

- a) AMP inhibits PFK-1 activity, slowing down glycolysis to conserve energy
- b) AMP activates PFK-1 by increasing the enzyme's affinity for fructose-6-phosphate
- c) AMP stimulates glycolysis by activating pyruvate kinase
- d) AMP decreases PFK-1 activity by inhibiting the formation of fructose-2,6-bisphosphate

49. What effect does insulin have on the glycolytic pathway in the liver?

- a) Insulin inhibits PFK-1 activity by decreasing fructose-2,6-bisphosphate levels
- b) Insulin activates gluconeogenesis while inhibiting glycolysis
- c) Insulin promotes glycolysis by increasing fructose-2,6-bisphosphate levels, activating PFK-

1

d) Insulin does not influence the activity of PFK-1 or glucokinase

50. How does the presence of arsenite (trivalent arsenic) affect cellular energy metabolism?

- a) Arsenite enhances the conversion of pyruvate to acetyl-CoA, increasing energy production
- b) Arsenite inhibits pyruvate dehydrogenase, disrupting the link between glycolysis and the Krebs cycle
- c) Arsenite enhances ATP synthesis by reducing glycolysis and promoting oxidative phosphorylation
- d) Arsenite competes with glucose during glycolysis, increasing ATP production in cells

51. Which of the following best describes the regulatory relationship between glucagon and insulin in relation to glycolysis?

- a) Glucagon and insulin both activate glycolysis in the liver
- b) Glucagon inhibits glycolysis, while insulin activates glycolysis in the well-fed state
- c) Insulin inhibits glycolysis, while glucagon activates it during fasting
- d) Glucagon and insulin both inhibit glycolysis to prevent excess energy production

52. What is the primary effect of elevated fructose-1,6-bisphosphate (F-1,6-BP) on pyruvate kinase?

- a) F-1,6-BP inhibits pyruvate kinase by binding to the enzyme's allosteric site
- b) F-1,6-BP activates pyruvate kinase by acting as a feedforward activator
- c) F-1,6-BP reduces the V_{max} of pyruvate kinase, slowing down glycolysis
- d) F-1,6-BP inhibits pyruvate kinase, preventing excessive ATP production

53. What does high citrate concentration indicate in the regulation of glycolysis?

- a) Citrate inhibits PFK-1, signaling sufficient ATP and energy reserves
- b) Citrate activates PFK-1, increasing glycolytic flux
- c) Citrate activates glucokinase in the liver, enhancing glucose uptake
- d) Citrate has no effect on glycolysis, as it is only relevant to the Krebs cycle

54. Which statement is true about the effect of fructose-6-phosphate on the activity of glucokinase?

- a) F6P increases glucokinase activity by dissociating it from GKR
- b) F6P decreases glucokinase activity by promoting its re-sequestration in the nucleus
- c) F6P activates glucokinase in a feedforward manner to increase glucose phosphorylation
- d) F6P inhibits the activity of glucokinase by enhancing its binding to GKR

55. What is the impact of pyruvate kinase deficiency on the Na^+/K^+ pump in RBCs?

- a) PKD causes overactivation of the Na^+/K^+ pump, increasing ATP consumption
- b) PKD leads to a failure in maintaining ion gradients, resulting in premature RBC death
- c) PKD increases RBC membrane flexibility, enhancing the movement of Na^+ and K^+
- d) PKD stabilizes the Na^+/K^+ pump, preventing ion gradient disruption in RBCs

56. Which of the following enzymes does arsenite directly inhibit, leading to metabolic dysfunction?

- a) Hexokinase
- b) Pyruvate kinase
- c) Pyruvate dehydrogenase
- d) Phosphofructokinase-1

57. What is the effect of high F2,6BP on gluconeogenesis?

- a) It activates gluconeogenesis by promoting phosphatase activity in PFK-2/FBPase-2
- b) It inhibits gluconeogenesis by activating PFK-1 and increasing glycolysis
- c) It has no effect on gluconeogenesis as it only affects glycolysis
- d) It increases glucose storage by inhibiting glucose uptake

Answer:

- 41. b
- 42. d
- 43. c
- 44. b
- 45. b
- 46. b
- 47. c
- 48. b
- 49. c
- 50. b
- 51. b
- 52. b
- 53. a
- 54. b
- 55. b
- 56. c
- 57. b

58. Which of the following is the primary mechanism by which high blood glucose levels activate glucokinase in hepatocytes?

- a) Glucokinase is activated by high concentrations of fructose-6-phosphate (F6P), which promotes its dissociation from GKRP
- b) Glucokinase is translocated to the cytosol after dissociating from the glucokinase regulatory protein (GKRP) upon an increase in glucose levels
- c) Glucokinase activity is directly phosphorylated by insulin to activate it in the cytosol
- d) High glucose levels inhibit glucokinase's sequestration into the nucleus, allowing it to bind to PFK-1 and enhance glycolysis

59. In the context of glucose metabolism, how does fructose-2,6-bisphosphate (F2,6BP) contribute to the coordination between glycolysis and gluconeogenesis?

- a) F2,6BP inhibits both glycolysis and gluconeogenesis to prevent futile cycling
- b) F2,6BP activates gluconeogenesis by inhibiting phosphofructokinase-1 (PFK-1)
- c) F2,6BP activates glycolysis by activating PFK-1 and inhibits gluconeogenesis
- d) F2,6BP inhibits both pathways equally, maintaining energy balance during the fasting state

60. What effect does AMP have on phosphofructokinase-1 (PFK-1) during a low-energy state in cells?

- a) AMP inhibits PFK-1 by reducing the enzyme's affinity for fructose-6-phosphate
- b) AMP activates PFK-1 by increasing its affinity for fructose-6-phosphate, promoting glycolysis
- c) AMP reduces the activity of PFK-1 and shifts glycolysis toward gluconeogenesis
- d) AMP binds to PFK-1 and decreases the enzyme's activity, halting glycolysis

61. In a cell with high ATP concentration, what is the role of ATP as an allosteric regulator of PFK-1?

- a) ATP activates PFK-1 to enhance glycolytic flux, promoting energy production
- b) ATP inhibits PFK-1 by binding to an allosteric site, signaling sufficient cellular energy and reducing glycolysis
- c) ATP has no effect on PFK-1 activity as it is a substrate, not a regulator
- d) ATP binds to the active site of PFK-1, preventing the binding of fructose-6-phosphate

62. Which of the following best describes the physiological effect of glucagon on hepatic metabolism during fasting?

- a) Glucagon promotes glycolysis by increasing fructose-2,6-bisphosphate (F2,6BP) and stimulating PFK-1
- b) Glucagon inhibits glycolysis by decreasing fructose-2,6-bisphosphate levels and activating gluconeogenesis
- c) Glucagon activates glycolysis by stimulating the translocation of GLUT-4 to the plasma membrane
- d) Glucagon increases glycolytic flux by activating pyruvate kinase in the liver

63. What is the consequence of pyruvate kinase deficiency (PKD) in red blood cells (RBCs) with respect to their ability to handle oxidative stress?

- a) PKD leads to reduced levels of reactive oxygen species (ROS) within RBCs due to ATP depletion
- b) PKD impairs RBCs' ability to neutralize reactive oxygen species (ROS), making them vulnerable to oxidative damage
- c) PKD enhances the RBCs' ability to neutralize ROS, increasing their resistance to oxidative stress
- d) PKD results in an excess of ATP production, which shields RBCs from oxidative damage

64. In the well-fed state, which of the following is the major driver for the activation of glycolysis and inhibition of gluconeogenesis?

- a) High AMP levels
- b) Elevated fructose-2,6-bisphosphate (F2,6BP) levels
- c) High ATP levels
- d) Increased glucose-6-phosphate (G6P) levels

65. How does the phosphorylation of pyruvate kinase in the liver by protein kinase A (PKA) during fasting affect glycolysis?

- a) Phosphorylation of pyruvate kinase promotes glycolysis by increasing its activity
- b) Phosphorylation of pyruvate kinase inhibits glycolysis, preventing glucose consumption during fasting
- c) Phosphorylation of pyruvate kinase has no impact on glycolysis in the liver during fasting
- d) Phosphorylation of pyruvate kinase activates gluconeogenesis by enhancing pyruvate conversion to glucose

66. What is the role of fructose-1,6-bisphosphate (F1,6BP) in the regulation of pyruvate kinase?

- a) F1,6BP inhibits pyruvate kinase by reducing its affinity for its substrate
- b) F1,6BP activates pyruvate kinase through a feedforward mechanism, enhancing glycolysis
- c) F1,6BP directly inhibits pyruvate kinase activity to reduce glycolytic flux
- d) F1,6BP prevents the formation of pyruvate, thus inhibiting pyruvate kinase

67. What effect does an increase in citrate concentration have on glycolysis in muscle cells?

- a) Citrate activates PFK-1, stimulating glycolysis
- b) Citrate inhibits PFK-1, slowing down glycolysis to prevent excessive ATP production
- c) Citrate binds to AMP, promoting glycolysis in the muscle cells
- d) Citrate has no significant effect on glycolytic enzymes in muscle cells

68. How does arsenate (pentavalent arsenic) interfere with glycolysis, specifically at the level of glyceraldehyde-3-phosphate dehydrogenase (GAPDH)?

- a) Arsenate inhibits GAPDH by competing with phosphate, reducing ATP production
- b) Arsenate enhances GAPDH activity by increasing phosphate availability
- c) Arsenate activates GAPDH by forming a complex with its cofactor
- d) Arsenate has no effect on GAPDH activity in glycolysis

69. What is the role of the glucokinase regulatory protein (GKRP) in the regulation of glucokinase activity in the liver?

- a) GKRP binds glucokinase and promotes its translocation to the nucleus in the presence of high glucose levels
- b) GKRP binds glucokinase and sequesters it in the nucleus under low glucose conditions, inhibiting its activity
- c) GKRP activates glucokinase in the cytosol during fasting, promoting glucose phosphorylation
- d) GKRP is responsible for the phosphorylation of glucokinase, increasing its activity during the fed state

70. What is the result of the sequestration of glucokinase in the nucleus under low glucose conditions?

- a) Glucokinase activity is enhanced, facilitating glucose phosphorylation
- b) Glucokinase is unable to interact with the glycolytic pathway, reducing glucose utilization
- c) Glucokinase increases its affinity for glucose, promoting glycolysis even during fasting
- d) Sequestration in the nucleus results in the activation of the insulin signaling pathway

71. How does the presence of AMP and fructose-2,6-bisphosphate affect the reaction velocity of PFK-1 in the presence of fructose-6-phosphate?

- a) They cause a shift to the right of the reaction curve, slowing down the reaction
- b) They cause a leftward shift, enhancing enzyme activity and increasing the reaction velocity at lower substrate concentrations
- c) They cause no change in the reaction velocity, regardless of substrate concentration
- d) They cause a reduction in the V_{max} of the reaction, making it less efficient

72. Which of the following best explains the effect of high blood glucose on hexokinase and glucokinase activity?

- a) High glucose levels activate hexokinase in the liver to promote glucose utilization
- b) Glucokinase is activated at high glucose concentrations, while hexokinase becomes inactive
- c) Hexokinase becomes the primary enzyme for glucose phosphorylation at high glucose concentrations
- d) Hexokinase remains active regardless of glucose concentration, while glucokinase remains inactive

73. What is the primary role of insulin in the regulation of pyruvate kinase activity in the liver?

- a) Insulin inhibits pyruvate kinase activity by increasing glucose levels
- b) Insulin promotes pyruvate kinase activity by increasing fructose-1,6-bisphosphate (F1,6BP) levels
- c) Insulin reduces pyruvate kinase activity by decreasing F2,6BP levels
- d) Insulin activates pyruvate kinase by stimulating glucagon signaling

74. What is the consequence of high levels of fructose-6-phosphate (F6P) on glucokinase activity in the liver?

- a) High F6P levels activate glucokinase by promoting its binding to GKRP
- b) High F6P levels inhibit glucokinase by promoting its sequestration in the nucleus
- c) High F6P levels enhance glucokinase activity by increasing glucose uptake
- d) F6P does not affect glucokinase activity, as it only regulates PFK-1

Answer:

- 58. b
- 59. c
- 60. b
- 61. b
- 62. b
- 63. b
- 64. b
- 65. b
- 66. b
- 67. b
- 68. a
- 69. b
- 70. b
- 71. b

- 72. b
- 73. b
- 74. b

75. Which of the following best explains the role of glucokinase in the regulation of glycolysis during the well-fed state?

- a) Glucokinase is activated by fructose-6-phosphate to enhance glycolysis
- b) Glucokinase activity is regulated by insulin, and it becomes active in the cytosol to phosphorylate glucose when glucose levels rise
- c) Glucokinase activity is inhibited by high levels of glucose-6-phosphate (G6P), which signals sufficient glucose utilization
- d) Glucokinase is sequestered in the nucleus during fasting and cannot participate in glycolysis in the well-fed state

76. In the context of glycolysis, how does fructose-2,6-bisphosphate (F2,6BP) regulate the enzyme phosphofructokinase-1 (PFK-1)?

- a) F2,6BP inhibits PFK-1 by decreasing its affinity for fructose-6-phosphate
- b) F2,6BP activates PFK-1 by increasing its affinity for fructose-6-phosphate, promoting glycolysis
- c) F2,6BP inhibits glycolysis and promotes gluconeogenesis by activating PFK-1
- d) F2,6BP inactivates PFK-1 in the liver to maintain stable blood glucose levels

77. What happens to pyruvate kinase activity when fructose-1,6-bisphosphate (F1,6BP) levels are high in the liver?

- a) F1,6BP inhibits pyruvate kinase, reducing glycolytic flux
- b) F1,6BP activates pyruvate kinase through feedforward regulation, enhancing glycolysis
- c) F1,6BP directly phosphorylates pyruvate kinase, decreasing its activity
- d) F1,6BP acts as a competitive inhibitor of pyruvate kinase to prevent excessive glucose metabolism

78. How does insulin contribute to the activation of glycolysis and inhibition of gluconeogenesis?

- a) Insulin activates PFK-2 to increase fructose-2,6-bisphosphate levels, which activate PFK-1 and stimulate glycolysis while inhibiting gluconeogenesis
- b) Insulin inhibits PFK-2, reducing fructose-2,6-bisphosphate levels, and activating gluconeogenesis
- c) Insulin directly activates PFK-1 by phosphorylation, enhancing glycolysis in the liver
- d) Insulin prevents the activation of glucokinase by inhibiting its translocation to the cytosol

79. During the fasting state, how does glucagon influence phosphofructokinase-2 (PFK-2) and fructose-2,6-bisphosphate (F2,6BP) levels?

- a) Glucagon activates PFK-2 through a signaling cascade, increasing F2,6BP levels to promote glycolysis
- b) Glucagon inhibits PFK-2 by activating protein kinase A (PKA), reducing F2,6BP levels, and thus inhibiting glycolysis while promoting gluconeogenesis
- c) Glucagon reduces protein kinase A activity, leading to a higher concentration of F2,6BP and stimulating glycolysis

d) Glucagon stimulates gluconeogenesis by increasing F2,6BP levels through direct activation of PFK-1

80. What is the primary physiological consequence of pyruvate kinase deficiency (PKD) in red blood cells (RBCs)?

- a) RBCs exhibit impaired ATP production, leading to the inability to maintain ion gradients, disrupting RBC shape, and causing hemolytic anemia
- b) RBCs compensate by increasing fatty acid oxidation, reducing the reliance on glycolysis for ATP production
- c) RBCs accumulate excess pyruvate, leading to increased ATP production and RBC dysfunction
- d) RBCs become more resistant to oxidative damage due to increased antioxidant activity

81. Which of the following describes the effect of high ATP levels on the activity of phosphofructokinase-1 (PFK-1)?

- a) ATP binds to PFK-1, activating it and promoting glycolysis when energy levels are high
- b) ATP acts as a substrate for PFK-1 and stimulates glycolysis in low-energy states
- c) ATP inhibits PFK-1 by binding to an allosteric site, signaling sufficient cellular energy and reducing glycolytic activity
- d) ATP increases the affinity of PFK-1 for its substrate, fructose-6-phosphate, leading to accelerated glycolysis

82. What is the effect of citrate on glycolysis, particularly on phosphofructokinase-1 (PFK-1)?

- a) Citrate activates PFK-1, enhancing glycolytic flux to meet cellular energy needs
- b) Citrate inhibits PFK-1 by binding to an allosteric site, signaling sufficient energy availability and slowing down glycolysis
- c) Citrate increases the concentration of fructose-2,6-bisphosphate, thereby promoting glycolysis
- d) Citrate activates PFK-2, increasing F2,6BP levels and enhancing glycolysis

83. Which of the following statements best describes the dual role of ATP in regulating phosphofructokinase-1 (PFK-1) during glycolysis?

- a) ATP activates PFK-1 when ATP concentrations are low, promoting glycolysis
- b) ATP serves as a substrate at low concentrations and an inhibitor at high concentrations, reducing glycolytic activity when energy is abundant
- c) ATP only serves as a substrate for PFK-1 and does not affect its allosteric regulation
- d) ATP inhibits PFK-1 only during the well-fed state to prevent unnecessary glucose breakdown

84. What role does AMP play in the regulation of PFK-1 during periods of low cellular energy?

- a) AMP inhibits PFK-1 to prevent excessive glucose breakdown and conserve energy
- b) AMP activates PFK-1, promoting glycolysis to generate ATP in response to low energy levels
- c) AMP reduces the activity of PFK-1, ensuring glycolysis does not proceed too rapidly during low-energy states
- d) AMP has no effect on PFK-1 regulation in low-energy conditions

85. What is the effect of arsenic on glycolysis, specifically with regard to glyceraldehyde-3-phosphate dehydrogenase (GAPDH)?

- a) Arsenate (pentavalent arsenic) competes with phosphate during glycolysis, reducing ATP production by inhibiting GAPDH
- b) Arsenite (trivalent arsenic) directly inhibits pyruvate dehydrogenase, disrupting the conversion of pyruvate to acetyl-CoA
- c) Arsenate enhances GAPDH activity, increasing glycolysis and ATP production
- d) Arsenic has no effect on GAPDH or glycolysis at physiological concentrations

Answer:

- 75. b
- 76. b
- 77. b
- 78. a
- 79. b
- 80. a
- 81. c
- 82. b
- 83. b
- 84. b
- 85. a

86. How does the regulation of glucokinase during the fasting state help maintain blood glucose levels?

- a) Glucokinase is active, catalyzing the phosphorylation of glucose to maintain glucose-6-phosphate levels
- b) Glucokinase is sequestered in the nucleus and inactive, preventing glucose phosphorylation and conserving energy
- c) Glucokinase activity is inhibited by glucagon to promote gluconeogenesis in the liver
- d) Glucokinase is activated by high ATP concentrations, favoring gluconeogenesis

87. What is the primary reason why hexokinase has a higher affinity for glucose (low K_m) compared to glucokinase?

- a) Hexokinase is designed for rapid glucose uptake in tissues that require constant energy, even at low glucose concentrations
- b) Hexokinase is activated by insulin, increasing its affinity for glucose to promote glycolysis
- c) Glucokinase has a higher V_{max} , making it less sensitive to low glucose concentrations
- d) Hexokinase operates primarily in muscle tissues, where energy demand is less sensitive to glucose fluctuations

88. How does fructose-6-phosphate (F6P) regulate the activity of glucokinase in the liver?

- a) F6P binds to glucokinase and directly activates it to enhance glycolysis
- b) High concentrations of F6P promote the re-sequestration of glucokinase into the nucleus, inhibiting glycolysis
- c) F6P directly inhibits glucokinase by blocking its translocation to the cytosol
- d) F6P has no effect on glucokinase activity, as it only regulates phosphofructokinase-1 (PFK-1)

89. Which statement best describes the relationship between AMP and ATP in regulating glycolysis during energy-deprived conditions?

- a) AMP activates glycolysis by enhancing the activity of PFK-1, while ATP inhibits it, signaling high energy status
- b) AMP competes with ATP for binding to PFK-1, modulating its activity in a concentration-independent manner
- c) AMP inhibits glycolysis, while ATP activates it during energy depletion
- d) AMP and ATP both activate PFK-1, ensuring maximal glycolysis even under high energy conditions

90. Why is pyruvate kinase considered a rate-limiting enzyme in glycolysis, and how does it interact with fructose-1,6-bisphosphate (F1,6BP)?

- a) Pyruvate kinase is rate-limiting due to its irreversible reaction, and it is activated by F1,6BP through feedforward regulation
- b) Pyruvate kinase operates in a feedback inhibition loop with F1,6BP, limiting glycolysis under energy-deprived conditions
- c) Pyruvate kinase is rate-limiting because it is inhibited by F1,6BP, slowing down glycolysis under high-energy states
- d) Pyruvate kinase is rate-limiting, but it does not interact with F1,6BP; instead, it relies on ATP to control its activity

91. How does the activity of PFK-2/FBPase-2 influence fructose-2,6-bisphosphate (F2,6BP) levels and the balance between glycolysis and gluconeogenesis?

- a) PFK-2 increases F2,6BP levels during fasting, promoting glycolysis and inhibiting gluconeogenesis
- b) PFK-2 decreases F2,6BP levels during the well-fed state, activating gluconeogenesis
- c) Phosphorylation of PFK-2/FBPase-2 decreases F2,6BP levels, favoring gluconeogenesis and inhibiting glycolysis
- d) PFK-2 increases F2,6BP levels, inhibiting gluconeogenesis and activating glycolysis only during times of stress

92. How does the liver use fructose-2,6-bisphosphate to prevent futile cycles between glycolysis and gluconeogenesis?

- a) F2,6BP ensures both glycolysis and gluconeogenesis occur simultaneously, maximizing ATP production
- b) F2,6BP activates glycolysis and inhibits gluconeogenesis in the well-fed state, and vice versa during fasting
- c) F2,6BP blocks allosteric regulation of enzymes, allowing both glycolysis and gluconeogenesis to run unopposed
- d) F2,6BP is inactive during the well-fed state, thus preventing any glycolytic activity

93. What happens to the activity of phosphofructokinase-1 (PFK-1) when the pH of the cell drops, causing increased proton (H^+) levels?

- a) High H^+ levels activate PFK-1 to ensure sufficient ATP production during acidosis
- b) High H^+ levels inhibit PFK-1 activity to prevent excessive glycolysis and worsening acidosis
- c) PFK-1 becomes insensitive to H^+ , allowing glycolysis to continue despite the acidotic

condition

d) Low pH enhances PFK-1 activity, promoting glycolysis as a buffer mechanism

94. In the context of glycolysis, how do the actions of insulin and glucagon balance glucose metabolism?

a) Insulin promotes glycolysis by increasing the availability of fructose-2,6-bisphosphate, while glucagon inhibits glycolysis by decreasing F2,6BP

b) Insulin activates gluconeogenesis, and glucagon promotes glycolysis to increase ATP production

c) Insulin and glucagon both stimulate glycolysis to generate ATP during fasting conditions

d) Insulin inhibits gluconeogenesis and activates glucose storage pathways, while glucagon enhances glycolysis and glucose production

95. What is the role of hexokinase in tissues with low glucose concentrations, and how does it differ from glucokinase's role in tissues with high glucose concentrations?

a) Hexokinase is active during fasting and has a high affinity for glucose to maintain basal metabolism, while glucokinase only becomes active when glucose levels are elevated post-meal

b) Hexokinase operates in the liver, while glucokinase functions in muscle and adipose tissue

c) Hexokinase has a high V_{max} to accommodate high glucose concentrations, while glucokinase has a low affinity for glucose to prevent excess phosphorylation

d) Both hexokinase and glucokinase operate equally in all tissues, depending on insulin levels

96. How does citrate inhibit phosphofruktokinase-1 (PFK-1), and what does this tell us about cellular energy status?

a) Citrate activates PFK-1 by increasing fructose-6-phosphate availability, signaling low cellular energy

b) Citrate inhibits PFK-1 as a feedback mechanism, signaling that energy levels are sufficient in the cell, thus slowing glycolysis

c) Citrate increases PFK-1 activity to rapidly break down glucose when energy is abundant

d) Citrate inhibits PFK-1 to prioritize fatty acid metabolism over glucose breakdown

Answer:

86. b

87. a

88. b

89. a

90. a

91. c

92. b

93. b

94. a

95. a

96. b

97. How does the sequestration of glucokinase in the nucleus during low glucose conditions impact glycolysis in the liver?

- a) It activates glucokinase, enhancing glycolytic flux in the liver
- b) It inhibits glucokinase, preventing glucose phosphorylation and conserving glucose for critical functions
- c) It increases the transport of glucose into the cytosol for faster glycolysis
- d) It promotes glucokinase's interaction with hexokinase to maintain energy balance

98. Which of the following best describes the function of fructose-2,6-bisphosphate (F2,6BP) in the liver during the well-fed state?

- a) It inhibits glycolysis and activates gluconeogenesis
- b) It increases the activity of PFK-1 to promote glycolysis and inhibits gluconeogenesis
- c) It activates pyruvate kinase to increase ATP production
- d) It inhibits PFK-2, blocking the formation of F2,6BP

99. Which of the following describes the effect of elevated AMP levels in a cell during periods of low energy?

- a) AMP inhibits glycolysis to preserve energy stores
- b) AMP activates PFK-1 to enhance glycolysis and ATP production
- c) AMP promotes gluconeogenesis by inhibiting glycolysis
- d) AMP has no effect on glycolysis and only regulates oxidative phosphorylation

100. What is the effect of high ATP levels on PFK-1, and what does this indicate about cellular energy status?

- a) High ATP levels activate PFK-1 to promote glycolysis and energy production
- b) High ATP levels inhibit PFK-1, signaling a high energy state and reducing unnecessary glucose breakdown
- c) High ATP levels activate glycolysis, even in the presence of sufficient energy
- d) High ATP levels have no effect on PFK-1 activity, as PFK-1 is primarily regulated by F2,6BP

101. How does insulin influence the activity of glucokinase in the liver during the well-fed state?

- a) Insulin inhibits glucokinase to conserve glucose for other tissues
- b) Insulin activates glucokinase by promoting its translocation to the cytosol, enhancing glycolysis
- c) Insulin reduces glucokinase activity by increasing its sequestration in the nucleus
- d) Insulin reduces the expression of glucokinase in the liver to favor gluconeogenesis

102. Which of the following statements about hexokinase and glucokinase is correct regarding their V_{max} and affinity for glucose?

- a) Hexokinase has a high V_{max} and low affinity for glucose, making it effective at high glucose concentrations
- b) Glucokinase has a low V_{max} and high affinity for glucose, making it effective at low glucose concentrations
- c) Hexokinase has a low V_{max} and high affinity for glucose, ensuring efficient glucose phosphorylation at low concentrations
- d) Glucokinase has a high V_{max} and low affinity for glucose, making it active only at high glucose concentrations

103. What is the effect of citrate on glycolysis, and how does it reflect the metabolic status of the cell?

- a) Citrate inhibits PFK-1, signaling a high energy state and reducing the need for glucose breakdown
- b) Citrate activates PFK-1, indicating that cellular energy levels are low and additional glucose breakdown is required
- c) Citrate has no effect on glycolysis as it primarily regulates fatty acid metabolism
- d) Citrate activates glycolysis by binding to pyruvate kinase, increasing ATP production

104. How does the interaction between AMP and ATP regulate PFK-1 during periods of energy depletion?

- a) AMP inhibits PFK-1 to reduce glucose breakdown, while ATP activates PFK-1 to promote glycolysis
- b) AMP activates PFK-1, while ATP inhibits PFK-1, thus promoting glycolysis when energy is low
- c) AMP and ATP both inhibit PFK-1, reducing glycolysis and promoting glucose storage
- d) AMP activates PFK-1, but ATP has no effect on glycolysis

105. Which of the following best describes the role of pyruvate kinase in the context of energy production during glycolysis?

- a) Pyruvate kinase is inhibited by fructose-1,6-bisphosphate, promoting energy storage
- b) Pyruvate kinase is activated by ATP and alanine to produce excess pyruvate
- c) Pyruvate kinase catalyzes the final step of glycolysis, producing pyruvate and ATP
- d) Pyruvate kinase activity is increased during gluconeogenesis to generate glucose

106. What is the primary impact of insulin on blood glucose levels during the post-meal state?

- a) Insulin increases blood glucose levels by promoting gluconeogenesis in the liver
- b) Insulin decreases blood glucose levels by enhancing glucose uptake into tissues and stimulating glycolysis
- c) Insulin maintains stable blood glucose by inhibiting glycolysis and promoting glucose storage
- d) Insulin decreases blood glucose by inhibiting glucose uptake in muscle and adipose tissues

107. How does high fructose-6-phosphate (F6P) act as a feedback inhibitor in the regulation of glycolysis?

- a) F6P inhibits glucokinase activity, preventing glucose phosphorylation during high glucose conditions
- b) F6P increases the activity of PFK-1 to promote glycolysis and energy production
- c) High F6P concentrations promote the re-sequestration of glucokinase into the nucleus, inhibiting glycolysis
- d) F6P stimulates pyruvate kinase to ensure efficient ATP production in glycolysis

108. What effect does glucagon have on the activity of PFK-2/FBPase-2 during the fasting state?

- a) Glucagon increases F2,6BP levels, promoting glycolysis and inhibiting gluconeogenesis
- b) Glucagon activates PFK-2 to increase glycolysis in the liver, reducing blood glucose levels
- c) Glucagon decreases F2,6BP levels, inhibiting glycolysis and promoting gluconeogenesis
- d) Glucagon has no effect on PFK-2/FBPase-2 during the fasting state

109. How does the defect in pyruvate kinase function contribute to the pathophysiology of pyruvate kinase deficiency (PKD)?

- a) PKD increases the rate of glucose breakdown, causing excessive ATP production in red blood cells
- b) PKD reduces the activity of pyruvate kinase, leading to low ATP production and impaired cell function in RBCs
- c) PKD activates gluconeogenesis to maintain RBC shape during low glucose conditions
- d) PKD leads to excessive pyruvate accumulation, inhibiting other metabolic pathways

110. What is the primary cause of hemolytic anemia in pyruvate kinase deficiency (PKD)?

- a) Increased ATP production in RBCs, causing cell rupture
- b) Insufficient glucose breakdown due to inhibited pyruvate kinase activity, leading to energy deficiency in RBCs
- c) Elevated glucose-6-phosphate (G6P) levels, disrupting RBC membrane stability
- d) Excess pyruvate accumulation in RBCs, causing oxidative damage

Answer:

- 97. b
- 98. b
- 99. b
- 100. b
- 101. b
- 102. c
- 103. a
- 104. b
- 105. c
- 106. b
- 107. c
- 108. c
- 109. b
- 110. b

Done By: Khaled Ghanayem