LEC 17 META Q:

1. Which of the following statements accurately describes ethanol's chemical structure?

A) It consists of three carbon atoms and two hydroxyl groups.
B) It is amphipathic, with one polar and one nonpolar side.
C) It is composed of two nitrogen atoms and one hydroxyl group.
D) It is a nonpolar molecule that cannot easily pass through biological membranes.

2. Where does the majority of ethanol absorption occur in the gastrointestinal tract?

A) Stomach
B) Upper small intestine
C) Large intestine
D) Esophagus

3. Which organ is primarily responsible for the metabolism of ethanol?

A) Stomach
B) Brain
C) Liver
D) Kidney

4. What is the first enzyme involved in the metabolism of ethanol in the liver?

A) Acetaldehyde Dehydrogenase (ALDH)
B) Alcohol Dehydrogenase (ADH)
C) Cytochrome P450 2E1 (CYP2E1)
D) Acetyl-CoA Synthetase (ACS)

5. What does Alcohol Dehydrogenase (ADH) convert ethanol into?

A) Acetate
B) Acetyl-CoA
C) Acetaldehyde
D) Pyruvate

6. After acetaldehyde is formed in the liver, what happens to it next?

A) It enters the bloodstream immediately.
B) It is converted into acetate by Acetaldehyde Dehydrogenase (ALDH).

C) It is converted into pyruvate.
D) It undergoes anaerobic metabolism.

7. What is the primary fate of acetate after it is released from the liver?

A) It is stored in adipose tissue.
B) It is excreted in urine.
C) It enters the bloodstream and is utilized by muscle cells.
D) It remains in the liver for further oxidation.

8. What is the major product of muscle cells after they convert acetate?

A) Pyruvate
B) Acetyl-CoA
C) Glucose
D) NADH

9. What effect does high ethanol consumption have on NADH and NAD+ ratios?

A) It increases NAD+ levels and activates the Krebs cycle.
B) It decreases NADH levels, promoting fatty acid oxidation.
C) It increases NADH levels, disrupting the Krebs cycle.
D) It increases NAD+ levels, improving glucose production.

10. Which of the following occurs when the NADH/NAD+ ratio is high due to ethanol consumption?

A) Enhanced fatty acid oxidation
B) Inhibition of gluconeogenesis
C) Increased glucose production
D) Activation of the Krebs cycle

11. What is the primary metabolic pathway for ethanol at high concentrations?

A) Alcohol Dehydrogenase pathway
B) Microsomal Ethanol Oxidizing System (MEOS)
C) Catalase pathway
D) Glycolysis pathway

12. Which enzyme is involved in the Microsomal Ethanol Oxidizing System (MEOS) pathway?

A) Acetaldehyde Dehydrogenase (ALDH)
B) Cytochrome P450 2E1 (CYP2E1)
C) Alcohol Dehydrogenase (ADH)
D) Acetyl-CoA Synthetase (ACS)

13. What is a significant byproduct generated by the Microsomal Ethanol Oxidizing System (MEOS)?

A) Glucose
B) Reactive oxygen species (ROS)
C) Acetyl-CoA
D) Lactic acid

14. Which enzyme system is responsible for the minor pathway of ethanol metabolism that occurs in peroxisomes?

A) Alcohol Dehydrogenase (ADH)
B) Cytochrome P450 2E1 (CYP2E1)
C) Catalase
D) Acetaldehyde Dehydrogenase (ALDH)

15. Which of the following is an effect of acetaldehyde accumulation in the bloodstream?

A) Improved liver function
B) Reduced cellular damage
C) Characteristic alcohol odor and potential carcinogenic effects
D) Increased glucose production

16. What impact does high NADH levels have on gluconeogenesis during alcohol metabolism?

A) It enhances glucose production.
B) It promotes the conversion of pyruvate to glucose.
C) It inhibits gluconeogenesis due to reduced pyruvate concentration.
D) It has no effect on glucose metabolism.

17. Which of the following best describes the role of Acetyl-CoA Synthetase (ACS) in alcohol metabolism?

A) It converts ethanol into acetaldehyde.
B) It helps convert acetate into acetyl-CoA in muscle cells.
C) It activates the NADH/NAD+ ratio.
D) It is involved in the conversion of acetaldehyde to acetate.

18. Which group is more likely to have a faster ethanol metabolism due to specific ADH isoenzymes?

A) Southeast Asians
B) African Americans
C) Native Americans
D) Hispanics

19. How does slower ethanol metabolism in Southeast Asians primarily affect them?

A) They experience slower intoxication.
B) They metabolize ethanol more efficiently, leading to quicker sober recovery.
C) They are more prone to faster intoxication due to slower ADH activity.
D) They have no significant difference in alcohol metabolism.

20. Which of the following is a primary concern when the body is overwhelmed with excessive ethanol consumption over time?

A) Decreased oxidative stress
B) Increased gluconeogenesis
C) Accumulation of acetaldehyde and subsequent cellular damage
D) Decreased blood alcohol concentration

Answers:

1. B
2. B
3. C
4. B
5. C
6. B
7. C
8. B
9. C
10. B
11. B
12. B
13. B
14. C
15. C
16. C
17. B
18. B
19. C
20. C

21. Which of the following best describes the role of NADH in the alcohol metabolism process?

A) NADH promotes glycolysis by providing additional energy.
B) NADH is produced in the cytosol and inhibits the conversion of acetate to acetyl-CoA.
C) NADH facilitates the Krebs cycle by acting as a coenzyme.
D) NADH helps in the conversion of pyruvate to lactate, increasing lactate accumulation.

22. What is the impact of the high NADH/NAD+ ratio during alcohol metabolism on fatty acid oxidation?

A) It accelerates fatty acid oxidation by providing more NADH for energy production.
B) It inhibits fatty acid oxidation, leading to an increase in fat accumulation in the liver.
C) It enhances the oxidation of fatty acids, promoting energy generation.
D) It has no effect on fatty acid oxidation, but it accelerates gluconeogenesis.

23. Which of the following is NOT a byproduct of the Microsomal Ethanol Oxidizing System (MEOS)?

A) Hydrogen peroxide (H2O2)
B) Hydroxyethyl radical (HER•)
C) Acetate
D) Superoxide (O2−)

24. Which metabolic pathway is primarily responsible for ethanol metabolism at high ethanol concentrations?

A) Alcohol Dehydrogenase (ADH) pathway
B) Microsomal Ethanol Oxidizing System (MEOS)
C) Gluconeogenesis pathway
D) Glycolytic pathway

25. What role does the enzyme Acetaldehyde Dehydrogenase (ALDH) play in ethanol metabolism?

A) It converts acetaldehyde into acetate, a key intermediate in energy production.
B) It converts ethanol into acetaldehyde, initiating ethanol degradation.
C) It synthesizes NADH from NAD+.
D) It catalyzes the conversion of acetyl-CoA into fatty acids.

26. Which of the following best explains the carcinogenic potential of acetaldehyde?

A) Acetaldehyde accelerates cellular division, leading to tumor formation.
B) Acetaldehyde can lead to cellular mutations by interacting with DNA.
C) Acetaldehyde produces ROS, which damages DNA but does not affect cell division.
D) Acetaldehyde stimulates the production of glutathione, reducing oxidative stress.

27. What causes the characteristic odor of alcohol on the breath?

A) The presence of ethanol in the bloodstream.
B) The accumulation of acetaldehyde in the blood and its release via the lungs.
C) The presence of acetyl-CoA in the bloodstream.
D) The conversion of acetyl-CoA to glucose in the liver.

28. The MEOS pathway becomes more active at higher concentrations of ethanol due to which of the following reasons?

A) The decreased availability of NAD+ enhances the activity of CYP2E1.
B) The increased ethanol concentration activates cytochrome P450 2E1 (CYP2E1).
C) The liver cells increase glucose production, which activates the pathway.
D) The concentration of acetaldehyde increases, making MEOS the primary pathway.

29. What is the primary function of Acetyl-CoA Synthetase (ACS) in muscle cells during alcohol metabolism?

A) It converts acetate into acetyl-CoA for entry into the Krebs cycle.
B) It facilitates the conversion of acetaldehyde into acetate.
C) It generates NADH from NAD+.
D) It inhibits the Krebs cycle by reducing acetyl-CoA synthesis.

30. Which of the following best describes the function of Catalase in ethanol metabolism?

A) Catalase oxidizes ethanol to acetaldehyde, contributing to alcohol degradation.
B) Catalase converts acetaldehyde into acetate, enhancing alcohol metabolism.
C) Catalase converts hydrogen peroxide (H_2O_2) into water and oxygen, with a minor role in ethanol metabolism.
D) Catalase acts as a major enzyme in the conversion of ethanol to acetyl-CoA.

31. How does the Microsomal Ethanol Oxidizing System (MEOS) contribute to liver damage during chronic alcohol consumption?

A) It increases NAD+ availability, promoting oxidative phosphorylation.
B) It generates reactive oxygen species (ROS) that cause oxidative stress in hepatocytes.
C) It converts ethanol into glucose, overloading liver function.
D) It accelerates the production of acetyl-CoA, leading to fatty liver disease.

32. Which of the following best explains why Southeast Asians are more likely to experience greater intoxication after consuming alcohol compared to other populations?

A) They have mutations in ADH that speed up ethanol metabolism.
B) They lack Acetaldehyde Dehydrogenase (ALDH), causing acetaldehyde accumulation.
C) They have faster CYP2E1 activity, leading to faster ethanol breakdown.
D) They metabolize ethanol to acetyl-CoA more rapidly, resulting in lower blood ethanol levels.

33. What is the role of Reactive Oxygen Species (ROS) in the metabolism of ethanol via the MEOS pathway?

A) ROS enhance ATP production and cellular respiration.
B) ROS cause oxidative stress and damage to hepatocytes.
C) ROS act as coenzymes in the oxidation of ethanol.
D) ROS facilitate the conversion of acetaldehyde to acetate.

34. How does a high NADH/NAD+ ratio in the liver impact glucose production from pyruvate?

A) It stimulates gluconeogenesis by enhancing pyruvate conversion to glucose.
B) It prevents pyruvate from entering gluconeogenesis, reducing glucose production.
C) It accelerates glycolysis, leading to higher glucose levels in the blood.
D) It increases the conversion of lactate to pyruvate, boosting glucose output.

35. What effect does the accumulation of acetate in muscle cells have during alcohol metabolism?

A) It leads to the conversion of acetate into glucose.
B) It is converted into acetyl-CoA, which enters the Krebs cycle for energy production.
C) It inhibits the Krebs cycle by decreasing NAD+ availability.
D) It decreases the oxidation of fatty acids and glucose.

36. What is the effect of excessive NADH produced during alcohol metabolism on the Krebs cycle?

A) It accelerates the cycle, producing more ATP.
B) It inhibits the Krebs cycle, decreasing energy production and leading to metabolic disorders.
C) It stimulates the oxidation of fatty acids, increasing energy yield.
D) It enhances glucose production, providing more substrates for the cycle.

37. In individuals with a faster ADH isoform, what is the likely effect on alcohol intoxication and recovery?

A) Faster metabolism of ethanol, leading to quicker intoxication.
B) Faster removal of ethanol from the system, leading to quicker recovery.
C) Slower ethanol metabolism, prolonging intoxication effects.
D) No significant effect on alcohol metabolism or recovery.

38. Why is acetaldehyde considered to have a higher carcinogenic potential compared to ethanol itself?

A) Acetaldehyde has direct mutagenic effects by binding to DNA.
B) Acetaldehyde is less toxic than ethanol and does not cause cellular mutations.
C) Acetaldehyde promotes increased ethanol metabolism, which damages liver cells.
D) Acetaldehyde inhibits the function of enzymes in the Krebs cycle, leading to cell death.

39. Which enzyme system contributes the least to ethanol metabolism in the body?

A) Alcohol Dehydrogenase (ADH)
B) Microsomal Ethanol Oxidizing System (MEOS)
C) Catalase system
D) Cytochrome P450 2E1 (CYP2E1)

40. What is the relationship between lactic acidosis and high ethanol consumption in the liver?

A) NAD+ depletion due to high NADH levels inhibits gluconeogenesis, causing lactate accumulation.
B) High ethanol levels activate lactic acid production, enhancing glucose output.
C) Lactate is converted to acetyl-CoA to fuel the Krebs cycle.
D) Lactic acidosis leads to enhanced ethanol metabolism, providing energy to the liver.

Answer Key:

21. B
22. B
23. C
24. B
25. A
26. B
27. B
28. B
29. A
30. C
31. B

32. B
33. B
34. B
35. B
36. B
37. B
38. A
39. C
40. A

41. In the context of alcohol metabolism, how does the accumulation of NADH affect lipid metabolism in the liver?

A) Increased NADH leads to the promotion of lipogenesis, favoring the storage of fat.
B) NADH accumulation stimulates fatty acid oxidation, aiding in fat breakdown.
C) NADH depletion reduces lipogenesis, promoting fat accumulation in the liver.
D) NADH directly regulates lipid synthesis, increasing adipose tissue storage.

42. What is the primary role of Cytochrome P450 2E1 (CYP2E1) in alcohol metabolism under chronic alcohol consumption?

A) CYP2E1 exclusively metabolizes acetaldehyde to acetate in the liver.
B) It increases the efficiency of ethanol metabolism at low concentrations.
C) CYP2E1 activates at higher ethanol concentrations and contributes to oxidative stress through ROS production.
D) CYP2E1 suppresses the MEOS pathway, preventing liver damage during high ethanol intake.

43. Which of the following statements most accurately describes the relationship between alcohol-induced oxidative stress and liver function?

A) ROS produced by the MEOS pathway have no effect on liver function.
B) Chronic oxidative stress from alcohol consumption leads to hepatic steatosis and fibrosis due to ROS.
C) ROS reduce fatty acid oxidation, leading to a healthy liver function and increased energy production.
D) Increased ROS directly enhance the function of alcohol dehydrogenase, accelerating alcohol metabolism.

44. Which factor most likely explains why African Americans metabolize ethanol faster than Southeast Asians despite having the same ADH isoenzymes?

A) African Americans have a higher expression of Acetaldehyde Dehydrogenase (ALDH) isoenzymes, enabling rapid breakdown of acetaldehyde.
B) African Americans possess a different distribution of ADH isoenzymes, leading to quicker ethanol breakdown.
C) Southeast Asians have polymorphisms in both ADH and ALDH that slow alcohol

metabolism.
D) African Americans have a more efficient MEOS pathway than Southeast Asians.

45. What would likely happen to glucose production during alcohol consumption when the NADH/NAD+ ratio is elevated in hepatocytes?

A) Glucose production would increase because of increased glycolysis.
B) Elevated NADH would inhibit gluconeogenesis, reducing glucose production.
C) NADH would increase pyruvate availability, accelerating glucose formation.
D) NADH would have no effect on glucose metabolism during alcohol consumption.

46. What is the primary consequence of the catalase enzyme system being involved in ethanol metabolism in the peroxisomes?

A) Catalase plays a significant role in ethanol detoxification by converting it to acetate, reducing liver damage.
B) Catalase has a minor contribution to ethanol metabolism and primarily eliminates hydrogen peroxide by converting it into water and oxygen.
C) Catalase is essential for the oxidation of acetaldehyde to acetate in the peroxisomes.
D) Catalase promotes the conversion of ethanol to acetyl-CoA in the mitochondria.

47. Given that acetaldehyde is a known carcinogen, how does its accumulation in tissues from alcohol consumption potentially contribute to cancer development?

A) Acetaldehyde directly stimulates cell division, increasing the likelihood of tumor formation.
B) Acetaldehyde binds to cellular DNA, forming adducts that cause mutations, leading to cancer.
C) Acetaldehyde increases the production of antioxidants, reducing oxidative damage in tissues.
D) Acetaldehyde promotes immune system suppression, making it easier for cancers to grow undetected.

48. Which of the following best explains why muscle cells convert acetate to acetyl-CoA during alcohol metabolism?

A) Acetyl-CoA is a precursor for the synthesis of fatty acids, which are then used as energy.
B) Acetyl-CoA enters the Krebs cycle, producing ATP, which muscles require for contraction and other functions.
C) Acetate is used by muscle cells to form glucose for energy.
D) Acetyl-CoA is converted into pyruvate, which is further oxidized for ATP production.

49. What potential role does the gut microbiota play in alcohol metabolism, particularly regarding the catalase enzyme system in the gastrointestinal tract?

A) Microflora produce alcohol dehydrogenase, which accelerates the conversion of ethanol to acetaldehyde in the stomach.
B) The microbiota may produce acetaldehyde through the action of catalase, which can be absorbed and affect liver metabolism.
C) Gut bacteria are involved in converting ethanol directly to acetyl-CoA in the gut.
D) Microbial catalase in the colon neutralizes acetaldehyde, reducing its carcinogenic potential.

50. Given that acetate is released into the bloodstream after alcohol consumption, which of the following best explains its significance in the peripheral tissues?

A) Acetate is used as a primary energy source by peripheral tissues, including muscle cells, by converting it into acetyl-CoA.
B) Acetate is converted to glucose in tissues, providing a significant energy source.
C) Acetate is stored in tissues to be used as fat reserves for later use.
D) Acetate is utilized by liver cells to create proteins for tissue repair.

51. Which of the following best explains the role of NAD+ in alcohol metabolism, especially regarding the shift to anaerobic pathways during ethanol degradation?

A) NAD+ acts as a cofactor in alcohol dehydrogenase, allowing the conversion of ethanol to acetaldehyde.
B) NAD+ is essential for the conversion of acetyl-CoA into fatty acids, preventing excessive fat buildup.
C) The depletion of NAD+ leads to a backup of metabolic intermediates, promoting lactate production and leading to lactic acidosis.
D) NAD+ is involved in enhancing oxidative stress, which stimulates the MEOS pathway.

52. How does the overactivation of the MEOS pathway due to chronic alcohol consumption affect liver health in the long term?

A) It causes a reduction in the generation of reactive oxygen species, promoting liver health.
B) Overactivation leads to oxidative damage and contributes to the progression of alcoholic liver disease, including cirrhosis.
C) It improves liver detoxification efficiency by enhancing alcohol breakdown.
D) It helps the liver efficiently store acetaldehyde as a form of energy for later use.

53. In what way could ethnic variations in ADH isoenzymes influence the alcohol-related health risks in different populations?

A) Faster ADH activity leads to less alcohol-related liver damage, as alcohol is metabolized more efficiently.
B) Slower ADH activity, such as in Southeast Asians, may lead to higher blood alcohol concentrations and greater risk of intoxication.

C) Ethnic variations in ADH isoenzymes have no significant impact on alcohol metabolism or health outcomes.
D) Faster ADH activity increases the production of acetaldehyde, contributing to higher carcinogenic risk.

54. Given that high NADH/NAD+ ratios can inhibit gluconeogenesis, what alternative metabolic pathways are activated to meet the body's energy needs during alcohol consumption?

A) Fatty acid oxidation is enhanced to provide acetyl-CoA for the Krebs cycle.
B) The body shifts to anaerobic glycolysis, resulting in lactate buildup to maintain ATP production.
C) Glycogen stores are rapidly converted to glucose to counteract the inhibition of gluconeogenesis.
D) Gluconeogenesis continues unhindered despite high NADH levels, ensuring glucose production.

55. What role do fatty acids play in alcohol metabolism under high NADH/NAD+ ratios in hepatocytes?

A) Excessive NADH enhances fatty acid oxidation, contributing to energy production.
B) High NADH levels suppress fatty acid oxidation, contributing to the accumulation of fatty acids in the liver.
C) Fatty acids are used as a primary energy source for the liver in the absence of glucose.
D) NADH promotes the breakdown of fatty acids to generate glucose.

Answers:

41. A
42. C
43. B
44. C
45. B
46. B
47. B
48. B
49. B
50. A
51. C
52. B
53. B
54. B
55. B

Done By: Khaled Ghanayem