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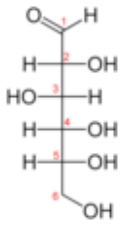
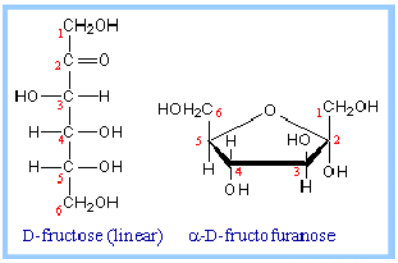
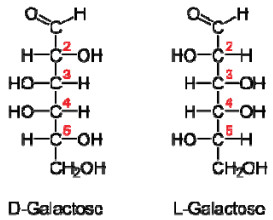
Dietary Carbohydrates

Monday, January 07, 2008
11:00 AM

5. What are the dietary source of glucose, fructose, and galactose?
    a. Starch --> glucose
    b. Sucrose --> glucose and fructose
    c. Glycogen --> glucose
    d. Lactose --> galactose and glucose
6. What are the structural features of glucose, fructose, and galactose?
    a. Glucose:
    b. 
    c. Fructose
    d. 

    e. Galactose
    f. 

7. How is glucose transported into hepatocytes? Does insulin stimulate transport?
   Sugars go directly to liver via portal blood flow and enter glycolytic pathways. Enter hepatocytes via GLUT2 transporter (Km = 15 mM, not influenced by insulin); high Km means only active when blood glucose is high
8. What are the kinetic properties of glucokinase?
    a. Only in liver and pancreatic B-cells
    b. Converts glucose --> glucose-6-P
    c. High Km (8 mM), at 10-15mM near max
    d. Low rate at blood glucose levels (5 mM)
    e. Not inhibited by glucose-6-P
9. How is the activity of glucokinase regulated? What is the effect of insulin? How does glucokinase regulatory protein control levels of glucokinase?
    a. Insulin
        i. Causes rapid increase in glucokinase mRNA
        ii. Leads to increased glucokinase production
        iii. cAMP turns off transcription
        iv. Type I diabetics do not produce glucokinase
    b. Glucokinase Regulatory Protein
        i. @low glucose levels --> GRP is bound to glucokinase (inactive)
        ii. High glucose (post-meal) --> GRP dissociates and glucokinase is activated

iii. Fructose-6-P promotes GRP binding, Fructose-1-P reverses binding

10. How is fructose metabolized in liver? What enzyme is specified to the liver? What are the products of aldolase B reaction? How does fructose metabolism affect glucokinase activity?
    a. Metabolism
        i. Phosphorylated to F-1-P by fructokinase
        ii. Aldolase B converts F-1-P to Glyceraldehyde and DHAP
        iii. Glyceraldehyde converted to Glyceraldehyde-3-P via glyceraldehyde kinase
        iv. DHAP --> G3P via triose phosphate isomerase
        v. G3P+DHAP can go to gluconeogenesis
        vi. G3P can continue on to glycolysis
    b. Aldolase B is specific to the liver and produces glyceraldehyde and DHAP
    c. F-1-P can decrease binding of GRP to glucokinase thus preserving glucokinase activity
11. How is galactose converted to glucose-1-P? What are the key enzymes? What is the role of UDP-glucose? What enzyme is defective in most cases of Galactosemia? What reactions are involved in cataract formation?
    a. Metabolism
        i. Galactokinase makes galactose-1-P
        ii. Galactose-1-P Uridylyl Transferase: Galactose-1-P + UDP-glucose --> Glucose-1-P + UDP Galactose
        iii. UDP-Galactose-4-epimerase : UDP-Galactose NAD+ --> UDP-Glucose
        iv. Phosphoglucomutase: Glucose-1-P --> G6P
        v. G6P to glycolysis
    b. Key enzymes are transferase and epimerase
    c. Galactosemia
        i. Genetic
        ii. Mutated transferase
        iii. Failure to thrive, MR
        iv. Cataracts
            1) Aldose reductase uses NADPH: Galactose --> Galactitol; Glucose --> Glucitol (sorbitol)
            2) Sugar alcohols cause cloudy whiteness to form
            3) Glucitol can be converted to fructose
            4) Seen in patients with galactosemia, diabetes
            5) Km of aldose reductase is 200 mM; cataracts only form in uncontrolled diabetes/galactosemia