Citation Key
for more information see: http://open.umich.edu/wiki/CitationPolicy

Use + Share + Adapt

{ Content the copyright holder, author, or law permits you to use, share and adapt. }

- **Public Domain – Government**: Works that are produced by the U.S. Government. (17 USC § 105)
- **Public Domain – Expired**: Works that are no longer protected due to an expired copyright term.
- **Public Domain – Self Dedicated**: Works that a copyright holder has dedicated to the public domain.
- **Creative Commons – Zero Waiver**
- **Creative Commons – Attribution License**
- **Creative Commons – Attribution Share Alike License**
- **Creative Commons – Attribution Noncommercial License**
- **Creative Commons – Attribution Noncommercial Share Alike License**
- **GNU – Free Documentation License**

Make Your Own Assessment

{ Content Open.Michigan believes can be used, shared, and adapted because it is ineligible for copyright. }

- **Public Domain – Ineligible**: Works that are ineligible for copyright protection in the U.S. (17 USC § 102(b)) *laws in your jurisdiction may differ

{ Content Open.Michigan has used under a Fair Use determination. }

- **Fair Use**: Use of works that is determined to be Fair consistent with the U.S. Copyright Act. (17 USC § 107) *laws in your jurisdiction may differ

Our determination **DOES NOT** mean that all uses of this 3rd-party content are Fair Uses and we **DO NOT** guarantee that your use of the content is Fair.

To use this content you should **do your own independent analysis** to determine whether or not your use will be Fair.
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:
      ![Glucose](image1)
   b. Fructose:
      ![Fructose](image2)
   c. Galactose:
      ![Galactose](image3)

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