

**Cell Physiology Final Exam
Fall 2008**

Guys,

The average on the test was 69.9. Before you start reading the right answers please do me a favor and remember till the end of your life that **GLUCOSE TRANSPORT NEVER EVER USES ATP!!!!!!!!!!!!!!**

1. Which transporter was responsible for absorbing glucose into the intestinal epithelium (on the apical side)?
 - A. SGLT – sodium glucose cotransporter

2. If this transporter was defective blood glucose level after the meal would be
 - A. Normal blood glucose level

These people will not get too much of the nutrients but their blood glucose level will be normal (in fact blood glucose will always be the same, it will not go up after the meal either). The only signaling molecule capable of lowering blood glucose levels is insulin. Insulin is secreted **ONLY** when blood glucose goes up, so if their glucose never goes up it will not go down either. If you missed this question come to Endo class next semester.

3. Which type of transport made it possible for glucose to enter the neurons in the brain?
 - A. One of the GLUT uniporters
4. What happened to the molecule of glucose after it entered the neuronal cell?
 - A. It was broken to pyruvate

It was stored in the form of glycogen. No, no, no!!! Neurons do not have ability to make glycogen and they rely on blood glucose. That's why they have this "hungry" GLUT2

5. If this molecule of glucose was radioactively labeled with C14 where would you be able to find highest level of radioactivity couple of minutes later?
 - A. Cytoplasm
6. What are the final products of glucose metabolism in neurons?
 - A. CO₂ and water
7. Most enzymes of Krebs cycle are coded by nuclear DNA. Translocation of these proteins into their final destination in the cell requires
 - A. Cytosolic chaperones
 - B. A number of outer and inner membrane translocons
 - C. Both of the above**
8. A completely folded and functional protein can enter the
 - A. Peroxisomes (+ nucleus)

9. ATP that was produced from glucose is necessary for
 - A. Establishing resting membrane potential (for pump to separate Na and K across the membrane)
10. ATP was produced in
 - A. Cytoplasm
 - B. Mitochondria
 - C. Both places
11. KCN (potassium cyanide) blocks the mitochondrial cytochrome c-oxidase (one of the complexes in Electron Transport Chain). What is the cellular mechanism of death?
 - A. Lack of ATP production
12. During neuronal signaling (action potential) ions will flow through the channels in neuronal membrane. What channels are responsible for depolarization phase of action potential?
 - A. Voltage gated Na⁺ channels
13. Is ATP used during action potential?
 - A. No

Channels NEVER use ATP, only pumps do.

What molecules can move into and out of cells WITHOUT using energy of ATP? Make sure to identify type of transport first.

No ATP necessary

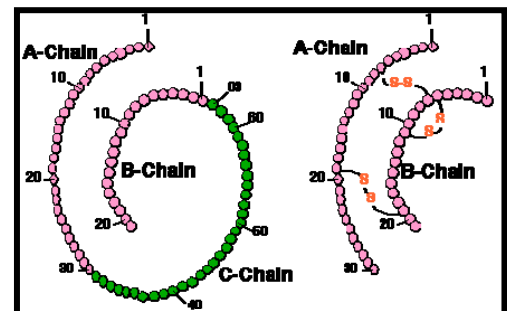
14. Glucose from the liver cell to blood
15. Oxygen into the cells
16. Glucose from primary filtrate back to blood
17. Transport of glucose into fat cells for storage in form of triglycerides

ATP is used during this transport

18. K⁺ into the cell
19. H⁺ into stomach lumen

20. Where are channel proteins synthesized?
 - A. In association with ER membranes
21. Where in the cell do they assemble in multi-subunit complexes?
 - A. In the ER membrane
22. Assembly of the multiunit complexes such as channels
 - A. Is based on the protein-protein interactions
23. What happens if channels do not assemble in the proper way?
 - A. They will be ubiquitinated and destroyed in proteasomes
24. Three polypeptides form a
 - A. Trimer

25. Which of the following motifs are responsible for creating stable dimers?
A. Coiled-coil motif
26. What type of secondary structure does usually form transmembrane segments of the protein?
A. Alpha helix
27. Which cells have the ability to store excess of glucose?
A. Liver cells (+muscle +fat) NO OTHER
28. Insulin secreted in response to a meal and increased blood glucose levels binds to
A. Receptor tyrosine kinase receptors
29. Binding of insulin to insulin receptor in liver cells will
A. Increase transcription of GLUT 4 uniporters
B. Increase insertion of GLUT 4 uniporters
C. Increase glucose transport to liver cells
D. All of the above
30. What is the mechanism of transduction used by insulin receptor to stimulate glucose storage?
A. Interaction with adapter proteins
Insulin receptor DOES NOT produce second messengers! If it did it would be soooo easy to treat type II diabetes
31. Insulin is produced in
A. Pancreatic β cells
32. The biochemical synthesis of proteins is called
A. Translation
33. What is the first amino acid in preproinsulin (full peptide that emerges from ribosome when insulin mRNA is translated)?
A. Methionine
34. Insulin has following targeting sequences
A. N-terminal ER targeting sequence
35. In the absence of any signal or targeting sequence, the expected subcellular location for the protein is
A. Cytosol
36. What type of posttranslational modification is responsible for converting proinsulin into insulin (see Fig on the right)
A. proteolytic cleavage
37. Where does this modification happen?
A. In Golgi



38. Insulin has 3 disulfide bonds in its structure. Where are these bonds formed?
A. In ER
39. What amino acids form disulfide bonds?
A. Cysteine
40. What cells express the gene for insulin?
A. Pancreatic β cells
41. Sulfonylureas are used as drugs in type II diabetes to stimulate more insulin secretion. What is their mechanism of action?
A. Closing of ATP gated K^+ channels independently of blood glucose levels
42. The appearance of glucose in urine in people with high blood glucose levels is the result of
A. Transport occurring via a limited number of transporters (saturation of transporters responsible for reabsorption)
43. Decreased number of GLUT 4 transporters in cell membranes of muscle cells will cause
A. High blood glucose levels after the meal
44. In addition to being a source for ATP production what is (are) other functions of carbohydrates in the cell?
A. Posttranslatinal modifications
B. Making proteins less sticky
C. Both of the above
45. Attachment of N-linked oligosaccharides happens in
A. ER
46. Binding of epinephrine to β -adrenergic receptors
A. Leads to an increase in cAMP
47. β adrenergic receptor belongs to the following receptor family
A. G protein coupled receptors
48. If your heart is no longer pounding as hard as it was at the beginning of this exam what has happened to adrenaline signaling cascade (notice the difference with adaptation of light receptors later!)
A. Phosphorylation of adrenaline receptors
B. Binding of arrestin
C. Endocytosis of the receptors and removal from cell surface
D. All of the above
49. During endocytosis sorting of molecules that are degraded and the molecules that are recycled is based on
A. pH gradient

50. Which of the following is a second messenger involved in response to adrenaline in muscle cells? (see Fig on the right)

A. cAMP

Ca²⁺ comes through channels

51. Which of the following is a second messenger involved in response to adrenaline in liver cells?

A. cAMP

B. IP₃

C. DAG

D. All of the above

52. IP₃ comes from

A. Phospholipids in cell membranes

53. Which enzyme makes cAMP from ATP?

A. Adenylyl cyclase

54. Which amino acid is the target for phosphorylation by Protein Kinase A?

A. Serine

55. Chronic stress hormone cortisol will bind to following type of the receptor

A. Receptors in the cytosol

56. Activation of cortisol receptors will

A. Increase gluconeogenesis

57. Which statement about intracellular hormone receptors is FALSE?

A. Have multiple transmembrane domains

58. Increased number of phospholipids with unsaturated fatty acyl chains in biological membrane

A. Will increase membrane permeability to cortisol (it still goes through imperfections)

59. Cortisol is produced in the cytosol of adrenal cortex cells from cholesterol. People with mutation in the internalization sequence of LDL receptor will have

A. Low levels of cholesterol transport into the cells

60. When you close your eyes the Na⁺ current (dark current) in the rods and cones of your retina will

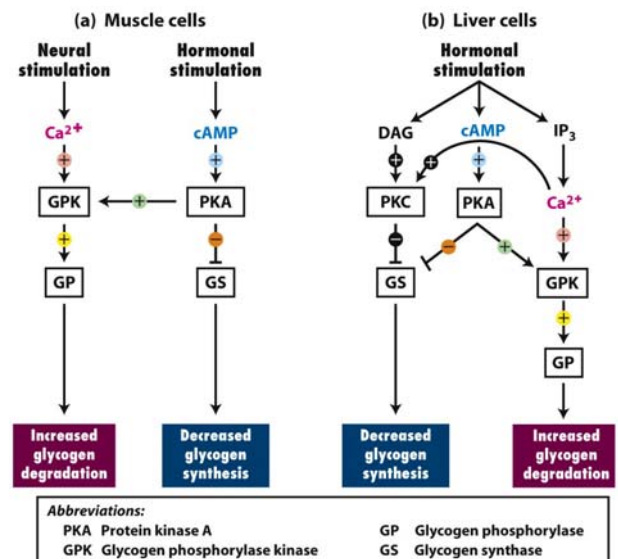
A. Increase

61. How will it affect membrane potential of rods and cones?

A. Closing of the eyes depolarizes cell membrane

62. The level of cGMP in rods in the dark is

A. High



63. When you walk out from this room to the bright light outside what would be a mechanism of the adaptation in the rods?
A. Phosphorylation of the light receptors (see difference with adrenaline receptors)
64. In visual signal transduction cascade
A. Receptor activation is caused by light
65. In which step of actin polymerization subunits are added and lost at the same rate?
A. Steady state
66. Actin capping proteins
A. Are controlled by second messenger pathways
67. The region of a motor protein that interacts with the motor's cellular cargo is the
A. Tail domain
68. Which portion of myosin interacts with actin filaments?
A. The head domain
69. A head domain of myosin molecule
A. Binds actin and hydrolyses ATP
B. Is responsible for generating force
C. Is the most conserved region in myosin
D. All of the above
70. Which protein motif do you expect to find in Ca^{2+} sensitive proteins such as tropomyosin?
A. Helix-loop-helix
71. Myosin II
A. Powers muscle contraction and cytokinesis
72. During contraction of striated muscles
A. Myosin pulls actin filaments together
B. "Walking" of myosin causes sarcomere to shorten
C. Myosin "walks" toward the barbed end of actin
D. All of the above
73. Stiffness of bodies after death is caused by
A. Myosin being bound to actin filaments
74. The functional unit of a muscle cell is
A. Sarcomere
75. Muscle contraction is initiated by
A. An increase in cytosolic Ca^{2+} concentration

76. Recovery of calcium after skeletal muscle contraction is accomplished by
A. A pump
77. Anticancer drug vinculin blocks polymerization of microtubules. Which processes in the cell are affected?
A. Formation of mitotic spindle
78. Which is the property of microtubules?
A. Go through random periods of shortening and lengthening
79. Universality of the genetic code means that
A. None of the above – it means that ALL organism on earth have the same universal genetic code
80. What determines the direction of the vesicle delivery within a cell?
A. Type of coat protein
81. What are the mechanisms for exclusion of resident Golgi and/or ER proteins from transport into plasma membrane?
A. Lack of recruitment signals in protein sequence
82. What organelles play a vital role in protein degradation?
A. Lysosomes
83. Proteasomes are
A. Tunnel-like macromolecule with protease activity
84. Cellular stress such as starvation will result in degradation of cell's own material. Which of the choices below is most likely to describe the process?
A. Autophagy
85. Activation of cell surface receptors
A. Can change cell metabolism
B. Can affect gene transcription
C. Can cause the cell to die
D. All of the above
86. Pertussis (whooping cough) toxin blocks activation of G protein by GPCReceptors. In whooping cough the levels of IP₃ in the cell are
A. Lower than normal

TRUE (A)

Multiunit complexes form by self-assembly

Membrane grow only by expansion of pre-existing membranes

All living cells are enveloped by plasma membrane

Every cell expresses GLUT 1 uniporter (I removed this one from scoring as some of you were fancy enough to think about plant cells)

G protein coupled receptors are intrinsic membrane proteins
Receptors can be located in the nucleus
GLUT2 returns glucose back into the blood during glycogenolysis in liver
Adaptation of the receptor might include removal of the receptor from the cell membrane

FALSE (B)

Hereditary information is stored in tertiary structure of proteins
Coat proteins polymerize after vesicle is pinched from the membrane
G proteins have endogenous GTPase activity and can synthesize cGMP as a second messenger.
Peptide bond formation is enzymatic reaction

Mark C on your scantron (I have no clue how 2 of you managed to mess this one up)