

Potential Final Exam Questions

1. What's an explant? What's a callus? And what is their relationship in the context of plant cloning?
2. What are the affects of treating plants or plant tissues with auxin or cytokinin?
3. Briefly, what is the procedure for cloning a whole plant from a small explant?
4. How are plant cells converted to protoplasts? Why are protoplasts more amenable to Transgenesis than normal plant cells?
5. What plant molecule signals agrobacterium to express vir genes?
6. How does agrobacterium get its DNA into the chromosomes of plant cells?
7. What are opines and how do they benefit agrobacterium?
8. What modifications are made to the agrobacterium Ti plasmid to convert it to a vector capable of delivering a specific gene to plant cells?
9. Explain how microprojectile bombardment functions to deliver genes into plant cells.
10. What is the rationale for producing pest-resistant transgenic crop plants?
11. The *B. thuringiensis* bacterium is a natural insecticide that can be sprayed directly on to plants, where it inhibits growth of butterfly and moth larvae. Why is *B. thuringiensis* toxic to these bugs?
12. What's the difference between a protoxin and a toxin? How and where is the *B. thuringiensis* protoxin converted to a toxin?
13. What essential plant process is inhibited by glyphosate (round-up)?
14. Briefly, how are transgenic plants engineered to be glyphosate resistant?
15. What is the natural origin of PHB (poly 3-hydroxybutyric acid) and how is it related to plastic?
16. List the phases of the cell cycle and briefly describe what happens in each phase.
17. What is the function of the cell cycle control system?
18. Checkpoints regulate progression of cells through the cell cycle. What conditions must be met before cells are allowed to progress through the G1 checkpoint? G2 checkpoint? M checkpoint?
19. What is the general role of kinases and phosphatases in cell cycle control?
20. Describe how a cyclin-dependent kinase becomes fully activated.
21. How do changes in intracellular cyclin concentrations regulate progression through the cell cycle?
22. Are different phases of the cell cycle regulated by the same cyclin-cdk complex? Explain.
23. What regulates the rise and fall of cyclin levels in the cell?
24. How does DNA damage inhibit progression of cells through the cell cycle? What is the role of p53 in that process?
25. How does S-cdk trigger DNA replication?
26. What holds sister chromatids together prior to anaphase?
27. What is the role of condensins during mitosis?
28. How does M-cdk cause the fragmentation of the nuclear envelope during prophase?
29. In the transition from metaphase to anaphase, sister chromatids separate from each other. How is this separation accomplished?
30. Match the appropriate phase of mitosis (PMAT) with the following events.

- a. Nuclear envelope breaks
 - b. Sister chromatids separate
 - c. Chromosomes line up on the metaphase plate
 - d. Microtubules (spindle fibers) bind to the kinetochores of chromosomes
 - e. The nuclear envelope reforms
 - f. The chromosomes condense
 - g. Condensins organize DNA
 - h. Cohesins are degraded by proteolysis
 - i. Chromosomes decondense
 - j. Nuclear laminins are phosphorylated by M-cdk
31. Define cytokinesis
32. List the basic characteristics of budding yeast, *Sacchomyces cerevisiae*.
- a. Kingdom?
 - b. Unicellular or multicellular?
 - c. Eukaryote or Prokaryote?
 - d. Anaerobic growth or aerobic growth?
 - e. Industrial applications?
33. Describe the life cycle of the budding yeast, *Sacchomyces cerevisiae*
34. Are budding yeast diploid or haploid or can they proliferate in culture as either?
35. What happens when haploid Mata yeast are combined with haploid Mat α yeast?
36. What condition stimulates diploid yeast to undergo meiosis and sporulate?
37. How is sporulation under adverse conditions advantageous to the yeast?
38. Why are budding yeast ideal for studying the cell cycle?
39. How does the morphology of budding yeast change as they progress through the cell cycle?
40. Define the term Conditional Mutant. Explain how a temperature sensitive mutant is a conditional mutant.
41. Assume you are a scientist trying to identify cell division cycle control genes (cdc genes). Your approach is to produce random mutants in hopes of generating finding a temperature-sensitive cdc mutant. You plate out mutants and grow them at the permissive temperature. Next you produce 2 replica plates and incubate one at the permissive temperature and the other at the non permissive temperature. What observation would lead you to believe that you have identified a candidate cdc mutant? Explain.
42. Assume you have identified a temperature sensitive yeast mutant that grows at the permissive temperature, but arrests growth at the non-permissive temperature. You reason that the mutation could be in a cdc gene or in some essential gene unrelated to the cell cycle. What experiment could you perform to distinguish between these two possibilities?
43. Assume you are a scientist who has successfully identified a temperature sensitive cdc mutant. Next you wish to identify the gene that has been mutated and then clone it. You have a yeast library available for use. Describe the experiment you would perform to clone the cdc gene.
44. What is the procedure for producing a yeast genomic library?
45. Describe the general concept of cellular signaling.

46. If a signal molecule is large and hydrophilic, is it more likely to bind to a cell surface receptor or an intracellular receptor? Explain.
47. Briefly describe the 4 forms of cellular signaling
 - a. Paracrine
 - b. Endocrine
 - c. Synaptic
 - d. Contact-dependent
48. Cells respond to a signal molecule by either initiating new gene expression or by altering the activity of pre-existing proteins. Which pathway is faster? Why?
49. Steroid hormones such as estrogen bind to _____ receptors and alter cell activity by _____.
50. Describe the signal transduction pathway of cortisol.
51. How do ion-channel-coupled receptors respond to their signal molecules?
52. How do enzyme-coupled receptors respond to their signal molecules?
53. How do G-protein-coupled receptors respond to their signal molecules?
54. What is the relationship between GTP/GDP and G protein activity?
55. What is the role of a second messenger in signal transduction?
56. Provide 2 examples of common second messengers.
57. What enzyme converts ATP to cAMP?
58. How does cAMP affect the activity of cAMP-dependent protein kinase (PKA)?
59. Describe, in detail, the adrenaline signaling pathway that results in glycogen breakdown.
60. What enzyme converts phosphoinositol phosphate (PIP₂) to inositol triphosphate (IP₃) and diacylglycerol (DAG)?
61. How does inositol triphosphate (IP₃) stimulate an increase in intracellular calcium?
62. What effect does calcium have on protein kinase C?
63. What events are required to activate a receptor tyrosine kinase?
64. Classical genetics and Reverse genetics are 2 techniques used to determine the function of a gene. Compare and contrast these 2 techniques.
65. What is the function of interferon?
66. When do cells secrete interferon?
67. What effect does thymidine kinase have on the biological activity of gancyclovir?
68. G418 is a drug that kills mammalian cells. What gene confers resistance to G418?
69. How are the neo gene and G418 used to select for transformed cells that have taken up an allelic exchange plasmid?
70. Mouse embryonic stem cells are transformed with an allelic exchange plasmid designed to knock out gene X. In this plasmid, the neo gene is flanked by DNA sequences homologous to gene X, while the thymidine kinase gene lies outside the homologous regions. How would you select for transformed cells that have undergone a homologous recombination to knock out gene X?
71. Assume you have successfully knocked out gene X in mouse embryonic stem cells. How could you use those cells to produce a whole animal in which gene X is knocked out?
72. How does necrosis differ from apoptosis?
73. What is the function of apoptosis in animals? How does this process benefit the animal? Provide examples.

74. Why do healthy neutrophils (white blood cells) die by apoptosis when they are only 2 days old? What is the consequence to the animal if they don't?
75. Why do severely DNA-damaged cells undergo apoptosis? What is the consequence to the animal if they don't?
76. List several important caspase substrates. Describe how cleavage of these substrates causes programmed cell death?
77. How do caspases cause fragmentation of the nuclear DNA?
78. How do caspases cause blebbing?
79. How do caspases cause fragmentation of the nucleus?
80. What signals neighboring cells to engulf apoptotic cells by phagocytosis?
81. What is the difference between a procaspase and a caspase?
82. How, in general are caspases activated?
83. Describe how the extrinsic pathway of apoptosis is triggered in virus-infected cells
84. What is the role of the mitochondrion in the intrinsic pathway of apoptosis?
85. How does the Bcl2 family of proteins (Bcl2, BH123, and BH3) regulate cytochrome c release from the mitochondrion?
86. Survival factors tell cells to avoid apoptosis and continue living. Some survival factors upregulate expression of Bcl2. How does that inhibit apoptosis?
87. List the 3 ways that microRNAs can repress the expression of their target mRNAs.
88. What types of processing do microRNAs undergo before they exit the nucleus?
89. How does the enzyme Dicer modify microRNAs?
90. What is the role of the RISC complex in the function of microRNAs?
91. What is the fate of an mRNA if a microRNA binds to its 3'UTR with perfect complementarity? What if the microRNA binds with imperfect complementarity?
92. How can microRNAs silence the transcription of a gene?
93. How does RNA interference (RNAi) function to inhibit replication of mammalian viruses?
94. How does RNA interference (RNAi) function to inhibit the jumping of transposons from one chromosomal site to another?
95. Compare and contrast Benign and Malignant tumors
96. Can cancer be caused by a single mutation
97. What is the evidence for the multi-hit hypothesis of cancer?
98. Why are cancerous cells genetically unstable?
99. What is the relationship between natural selection and the development of a cancerous tumor?
100. Mutations in what classes of genes cause cancer?
101. What are some of the changes cancer cells undergo that allow them to metastasize?
102. Compare and contrast cancer stem cells with cancer transit amplifying cells
103. List the most common cancer-causing agents.
104. What is the normal role of a protooncogene?
105. What is the difference between a protooncogene and an oncogene?

106. The conversion of a protooncogene to an oncogene requires what type of mutation; a dominant, gain of function mutation or a recessive, loss of function mutation?
107. What is the normal function of Ras?
108. How is Ras activated?
109. How could mutations in Ras cause cancer?
110. What is the normal role of a tumor suppressor gene?
111. What is the historical significance of Rous sarcoma virus in the study of cancer?
112. How did Francis Rous prove that rous sarcoma virus caused tumors in chicken?
113. What is the normal role of a tumor suppressor gene?
114. What chromosomal defect causes the hereditary form of retinoblastoma?
115. How does the retinoblastoma (Rb) gene suppress cell division in normal, healthy cells?
116. What is the relationship between Rb, E2F, cyclin-dependent kinase and cell division?
117. How are levels of p53 kept low in normal, healthy cells?
118. What causes p53 levels to rise in cells?
119. What is the relationship between p53, p21, Cdk and cell division?
120. List the most commonly mutated genes in colorectal cancer? Which of these genes is mutated first, second, third during tumor development?
121. What type of cancer is caused by human papilloma virus (HPV)?
122. What is the relationship between HPV proteins E6 and E7 and the development of cervical cancer?
123. List the most common cancer treatment strategies.
124. Taxol is a drug that disrupts the formation of microtubules. Why is this drug useful in cancer treatment?
125. Why are anti-angiogenesis factors useful in cancer treatment?

Potential Essay Questions

1. Describe the infection of a plant by *Agrobacterium tumefaciens*. For full credit, discuss the roles played by the Ti plasmid, T-DNA, vir genes, acetosyringone, auxin, cytokinin, opines. Also discuss DNA transfer, integration and gene expression.
2. Describe the genetic engineering of transgenic *Arabidopsis* that express high levels of PHB (poly 3-hydroxybutyric acid).
3. You are a researcher studying the cell cycle in eukaryotes. As a means of furthering your understanding of the molecular mechanisms of the cell cycle you decide to identify genes involved in cell cycle control. You choose yeast as a model system and you take a classical genetic approach. Explain, in detail, how you would make and screen for temperature-sensitive (ts) mutants that are defective in cell cycle control (cdc mutants).
4. Assume you are a molecular biologist working on epidermal growth factor (EGF) in mice. EGF is encoded by a single gene. You wish to generate a knockout mouse that lacks the EGF gene. Describe in detail the how you would perform this task.
5. Describe in detail the interferon/STAT/Jak signal transduction pathway