Name _____

Biology 411 - Developmental Biology
Winter Quarter 2010

Midterm 1

100 Total Points
Open Book

Choose ***20*** out the 25 questions to answer (4 pts each). Only the first 20 questions that are answered will be graded. Cross out answers that you do wish to be graded.
Provide answers using full sentences, unless instructed otherwise.

Chapters 1 and 2

1. Evolution of Body Forms (yes or no) (p. 43)

__**NO**____ a. Are all schizocoelous animals diploblastic?

__**YES**____ b. Are all protostomes triploblastic?

__**YES**____ c. Are species in the Radiata diploblastic?

__**NO**____ d. Do all animals that molt have embryos that undergo spiral cleavage?

2. (p. 33). Draw your prediction of phenotype of a genetically engineered *Volvox carteri* colony that has a null mutation of the *gls* gene. Briefly explain your reasoning.

The colony will be composed of cells that fail to undergo asymmetric division. Neither gonidia or somatic cells will form because *gls* fails to function.

3. Describe the difference between the embryological terms "prospective" and "presumptive." Give embryological examples of prospective and presumptive to illustrate their proper use, i.e. describe embryological processes using these terms.

Prospective means "capable of becoming"something.
Presumptive means "about to become"something.

Presumptive endoderm and presumption mesoderm are internalized during vertebrate gastrulation.
Prospective lens ectoderm is induced to become the lens by the optic vesicle.

4. (p. 38) Draw the phenotype of a *Dictyostelium* colony that has a loss-of-function mutation in gp24.

The amoebae will be unable to adhere to each other. The amoebae may still be able to aggregate without adhering to each other, but they will not be able to form a fruiting body.

5. (p. 17) Is the quadrate bone in the skull of alligator "homologous" to the incus of the human inner ear? Explain the reasoning of your answer.

Yes, the quadrate bone of the alligator is homologous to the incus of the human inner ear. It is homologous because it is derived from a common ancestry.

6. (p. 19) Are malformations caused by the teratogen thalidomide a consequence of disrupted allometric growth, or not? Explain your reasoning.

Thalidomide does not result in a change in differential growth. Rather, it results in the loss of specific structures, depending on the time of development. Thus, thalidomide is not acting through a shift in allometric growth.

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Chapters 3 and 4

7. (p. 50) What is the difference between a "morp" and a "mutant" phenotype?

A "morp" is a phenotypic form of a species, which is expressed under a given set of environmental conditions. A "mutant" is an aberrant phenotypic form of a species that arises from the mutation of a gene.

8. (p. 67) Is a *myeloid progenitor cell* a "stem cell" or not? Explain your reasoning.

Even thought a myeloid progenitor cell is pluripotent, the myeloid progenitor cell is not a stem cell because it does not give rise to copies of itself.

9. (p. 72) Explain what would happen if a mutation in beta-catenin resulted in its inability to bind actin? What would happen to intercellular adhesion mediated by cadherins?

Cadherins would still be able to bind to each other. However, no stable linkages would be able to form without beta-catenin interlinking cadherins to actin.

10. (pp. 65-66) Is Experiment (D) a "positive control" or a "negative control". Explain your reasoning using full sentences.

Experiment (D) is a negative control. It is designed to test whether the effect of injection, or the effects of inserting a different mRNA, would produce similar effects to the injection of *Nodal* mRNA.

11. (p. 85) Using your knowledge about chromatin, explain why the clone "CC" kitten is morphologically different than her progenitor, "Rainbow".

CC has an obvious different coloration pattern than Rainbow. This occurs because of random X chromosome inactivation during the blastula stage. Even though CC and Rainbow are genetically identical, they are not morphological the same because of differences in epigenetics.

12. (p. 97) Using RNA interference technology, explain how you could make a transgenic mouse that has the same phenotype as the Bmp7 knockout mouse in Figure 4.20.

Insert a gene that encodes an antisense version of *Bmp7* into the female pronucleus of a fertilized mouse egg. The inserted gene will replicate after being inserted into the genome. All cells in the embryos will contain the transgene. The antisense version of *Bmp7* will "knockdown" *Bmp7* expression by triggering *Bmp7* transcript degradation.

13. (p. 113) Describe how you would use a gel mobility phase-shift assay to determine whether a transcription factor necessary for speech in *Homo sapiens* interacts with a DNA fragments isolated from a *Homo floresiensis* bone. What technique would have been used to obtain enough *Homo floresiensis* DNA for the gel mobility assay? Why would scientists be interested in performing this type of study?

Polymerase chain reaction (PCR) could be used to amplify DNA from the bones. Scientists would be interested in knowing whether the Hobbits (*Homo floresiensis*) had the capacity for human speech.

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Chapter 5 and Chapter 21

14. (p. 122) What would the coloration pattern of a XY male kitten be, if it had a XXY father of the calico cat breed, and a calico cat for a mother? Explain your reasoning.

The kitten would have not have mosaic coloration. The genotype of the kitten is XY. Two or more X chromosomes that possess the calico pigmentation gene must be present the individual in order produce the mosaic (i.e. "calico") phenotype.

15. (p. 127) What is the advantage of alternative splicing in organisms?

Different versions of proteins can be made from the same mRNA transcript. This allows different isoforms (i.e. versions) of the protein to be expressed in different tissues of the body, or at different times of an organism's life cycle.

16. (p. 118) What phenotype will probably result from a human zygote that has a methylated promoter of the Angelman Syndrome locus on paternal chromosome 15? Explain why this occurs.

One would expect a normal human phenotype. The Angelman Syndrome (AS) gene is not normally expressed on paternal chromosome 15. The AS locus is inactivated by imprinting during early embryogenesis.

17. (p. 119-120) Why is gene dosage compensation related to sex chromosomes?

Gene products from the X chromosome must be similar in the cells of male and female organisms. Because the number of X chromosomes varies between males and females, different organisms employ different mechanisms to balance the transcription of sex chromosomes genes between males and females. For instance, X chromosome inactivation is employed in vertebrates. In fruit flies, the transcription rate of the Y chromosome genes is doubled to compensate for the presence of two active X chromosomes.

18. In terms of sex-linked genetic diseases, how can cellular mosaicism result in a disease that is found predominantly in females? (Discussion Group, JAMA paper)

In human females, random X chromosomes inactivation among cells in the morula stage results in organs that are composed of cellular progeny, which possess either an active maternal or an active paternal X chromosome. If a gene involved in cell-cell recognition is located on the X chromosome, then cells with an active paternal-X chromosome will be expected to interact with cells containing an activate maternal-X chromosome. If a mutation of the gene is present on either the paternal-X chromosome, or the maternal-X chromosome, cell-cell recognition problems may occur. An example of this may be craniofrontonasal syndrome, in which the ephrinB1 gene on the X chromosome is mutated.

19. (p. 686-687) You are in a Parkinson's Disease research institute. You get the idea that co-injecting a mixture of glial stem cells and dopaminergic neurons into the brains of Parkinsonian mice might improve their motor problems. Your research team has already developed a means

of producing dopaminergic neurons using therapeutic cloning. How could you obtain glial stem cells to co-transplant along with the dopaminergic neurons?

Embryonic stem cells would be exposed to FGF and platelet-derived growth factor in cell cultures separate from the neurons. After being induced, glial stem cells would be ready to be co-injected with dopaminergic neurons into a recipient mouse.

Chapter 6

20. (p. 158) Describe why BMP homodimers, by themselves, are not active kinases. A homodimer is composed of either two type I receptors, or two type II receptors.

BMP ligand binding brings the serine/threonine kinase domain of a type I BMP receptor into contact with the GS box of a type II BMP receptor. The resulting heterodimer can autophosphorylate the cytoplasmic tails of the type I and type II receptors, resulting in a major increase in kinase catalytic activity.

21. β-catenin is mutated so that it can no longer be de-phosphorylated. What effect would this have on the output of the canonical Wnt signaling pathway? (p. 155)

β-catenin would be continuously degraded. The canonical Wnt signaling pathway would NOT be activated. β-catenin would NOT migrate to the nucleus, bind to the LEF/TCF transcription factor. Transcription of β-catenin specific target genes would NOT occur.

22. (pp. 134 and 682) Propose how a genetically engineering HIV virus vector could be used to treat a patient whose cells contain a deleterious gene (e.g. HIV reverse transcriptase) using miRNA.

A retrovirus could be used to insert a gene sequence encoding a therapeutic miRNA into a patient's cells. Once expressed in the patient's transfected cells, the miRNA would be cut by the Dicer protein. The resulting miRNA fragments would combine with RISC to block translation of targeted mRNA, as well as to degrade the targeted mRNAs of the patient's cells.

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Chapter 7

23. Would soluble bindin protein from *S. purpuratus* block fertilization from occurring in *S. franciscanus*? Explain your reasoning.

No, bindin protein from *S. purpuratus* would not bind to the binding receptor in *S. franciscanus*. Bindin proteins are highly species specific to prevent cross-fertilization between species. Thus, one would NOT expect a binding protein from one sea urchin to bind to the bindin receptor in an unrelated species.

24. (p. 182) Describe three differences between sea urchin fertilization and mouse fertilization.

(a) Fertilization is external in the sea urchin, whereas fertilization is internal in the mouse.
(b) The sea urchin sperm cell has an acrosomal process, whereas the mouse sperm cell does not.
(c) Mouse sperm require capacitation, whereas sea urchin sperm do not.

25. (p. 207) Explain what nonequivalence of mammalian pronuclei means. What is the molecular basis of nonequivalence?

Nonequivalence means that male and female pronuclei have different genetic capacities to contribute to the development of an embryo. Much of the nonequivalence between male and female pronuclei is thought to arise from differences in cytosine methylation patterns, which are established during gametogenesis.