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Biology 411 - Developmental Biology
Winter Quarter 2011

Midterm 2 KEY

100 Total Points
Open Book

Read the Following Instructions:

* Answer 20 questions out of the available 25 questions - (5 pts each)

* Cross out answered questions that you do not want graded. We will grade the first 20 answered questions that are not marked out.

* Provide answers using **full sentences**, unless instructed otherwise.

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1. In the Essner *et al*. paper that you read for Discussion section, researchers used a laser beam to photoablate (i.e. kill with light) dorsal forerunner cells in a zebrafish embryo. Kupffer's vesicle did not form.

Draw a **normal** zebrafish embryo at the tailbud stage or (Figure 7.37). Diagram where Kupffer's vesicle is normally located.

Kupffer's vesicle forms in the posterior part of the tail rudiment, beneath the posterior limit of the notochord.

2. (pp. 220-225) Draw what you believe the phenotype of a *bicoid*; *torso* double mutant (i.e. two mutations) *Drosophila* embryo would look like. Label the parts of the double mutant embryo and explain your reasoning.

The embryo would have only abdominal structures that are mirror-symmetric about the embryonic midline. *Bicoid* mutants are mirror-symmetrical about the embryonic midline, and have only an abdominal region with telsons at either end. *Torso* is necessary for the generation of terminal structures, these being the acron and the telson. Without *torso*, our double mutant could not form telsons, so it would have only abdominal structures.

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3. (p. 198) What would happen to cell fate specification in *C. elegans* if POP-1 was mutated to a null phenotype?

Excessive numbers of endodermal cells would be specified.

4. (p. 170) If *Pmar1* become activated in a Veg2 cell, which was isolated from the rest of the sea urchin embryo, would the isolated cell express non-skeletogenic mesenchyme genes? Explain your reasoning.

***Par1* would repress *HerC*, which would de-repress *Delta*. Delta protein would be secreted. Delta would then bind to the Notch receptor. This interaction would activate the expression of non-skeletogenic mesenchyme genes.**

5. (pp. 176-177) Draw and label the 5 concentric circles of presumptive endoderm and presumptive mesoderm of the sea urchin vegetal plate shown in Figure 5.19. Indicate (i.e. mark with numbers) the most likely original locations of cells 1 and 7 in Figure 5.20 on the vegetal plate.

Cell 1 is in the foregut endoderm. Cell 7 is in the hindgut endoderm.

6. (pp. 229-232) Draw the parasegment boundaries in a ftz (fushi tarazu) Drosophlia mutant at the embryonic stage shown in Figure 6.32B.

The mutant will be missing several segments, as seen in 6.32B. The segment boundaries correspond to the furrows in the blastoderm. The parasegment boundaries contain the posteriormost 1/3 (or so) of a given segment, and the anteriormost 2/3 (or so) of the segment immediately behind it.

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7. (pp. 191-192) **Diagram** how the cells in the ascidian notochordal rudiment rearrange to create the notochord. What is this process called?

The cells undergo mediolateral intercalation, to produce convergence and extension of the notochord.

8. (pp. **159-160**, 179-181, **and 242-244**) Draw the resulting cleavage pattern that would result if a snail zygotic nucleus was used to replace the zygotic nucleus of a fertilized frog egg. Explain your reasoning.

The cleavage pattern of the frog embryo would be unchanged. Spiral cleavage patterns in snail embryos are determined by maternal gene products, not zygotic gene products. Points were awarded for completeness, as well as writing clarity.

9. **(pp. 309-310)** The famous Siamese twins Eng and Cheng probably formed from a partial splitting of a bilayer germ disk after the aminon had formed. If this was the case, when during development did the splitting of the embryo occur? Explain you reasoning.

The partial splitting of the blastodisc occurred after day 9, after the amnion had formed.

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10. (pp. 187-189) If cytoplasm from a b4.2 blastomere of an ascidian embryo is transplanted into the B4.1 blastomere, what effect would you expect to see with regard to B4.1's fate? Explain your reasoning.

The transplanted cytoplasm from b4.2, (an ectoderm precursor) would not change the fate of the B4.1 blastomere, which would still generate muscle tissue. Experiments have shown that when yellow crescent cytoplasm is transplanted from B4.1 to b4.2, b4.2 generates muscle instead of ectoderm-- meaning that the yellow crescent cytoplasm's effect overwhelms whatever determinants are present in b4.2. It is therefore reasonable to predict that when b4.2's cytoplasm is transplanted to B4.1, any effect it was capable of generating would be overwhelmed by muscle fate determining factors in B4.1's yellow crescent cytoplasm.

11. (pp. 213-214) Draw the nuclear localization of the *dorsal* gene product in both the wild-type *Drosophila* embryo and the *spätzle* mutant *Drosophila* embryo at the cycle 12 stage (p. 204).

The *dorsal* gene product accumulates in ventral nuclei of the wild-type embryo. It fails to accumulate in ventral nuclei of *spätzle* mutant embryos.

12. Explain why maternal determinants are important in early embryogenesis.

In early embryonic development, cells must expeditiously move and divide in particular patterns in order to establish the geometry and spatial relationships needed for further development. This requires a variety of proteins and transcription factors-- however, prior to the mid-blastula transition, transcription does not occur in the embryonic cells. This means that transcription factors, mRNAs, must be essentially prepackaged in the oocyte. (Often they are tethered to the oocyte's plasma membrane for correct localization.)

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13. (pp. 207-237) Name five morphogens involved in *Drosophlia* embryogenesis.

Bicoid, Nanos, Torso, Hedgehog, Caudal, Wingless, Dorsal

14. (pp. 192-193) Given that their genomes are roughly the same size, how can you account for the profound difference in complexity between *C. elegans* and *H. sapiens*?

***C. elegans* may have inflated its gene numbers by gene duplication. Gene products in *Homo sapiens* are often multi-functional, whereas *C. elegans* gene products usually have a single function. Humans use alternative splicing of mRNA to create a much larger proteome than *C. elegans*.**

15. (pp. 234-236) At Harry Potter World, you enter a chamber called the Homeotic Room. A group of wizards ask you for your most desired homeotic transformation. They cast a spell, and you undergo a homeotic transformation. Diagram your final phenotype and explain what a homeotic transformation is. Don't worry, the transformation is only temporary.

Homeosis is a transformation of one body part for another.

16. (pp. 226-227) What is the difference between a segment and a parasegment in a *Drosophila* embryo?

Segments are repeating primordial units that form along the anterior-posterior axis of the embryo and which correspond to parts of the adult body. Their boundaries are formed by furrows in the blastoderm. Parasegments are transegmental units, and are the fundamental units of embryonic gene expression in *Drosophila*.

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17. (pp. 260-263) Explain what would happen in the amphibian embryo if *siamois* was activated everywhere in the blastula. Explain your reasoning for the phenotype.

The embryo would become hyperdorsalized. *Siamois* activates Organizer genes in presumptive mesoderm. With Organizer genes activated everywhere in the mesoderm, ventral tissue specification would not occur, resulting in hyperdorsalization. The formation of multiple axes is also possible.

18. What is the purpose of making a F3 generation in a F2 mutagenesis screen (p. 274)?

The purpose of the F3 generation is to identify F2 parents that carry a recessive mutant allele.

19. (pp. 249-251) Explain how epiboly occurs in the animal cap of the amphibian embryo.

Radial intercalation of animal cap cells (presumptive ectodermal cells) drives the spreading of the ectoderm over the surface of the embryo.

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20. (p. 269) Draw the resulting frog larvae that would result if both Wnt and BMP expression were inhibited in the anterior region of the embryo. Explain your reasoning.

The embryo would be normal. Brain tissue formation is normally inhibited by Wnt and BMP signals. For a brain to be induced in its proper location, Wnt and BMP inhibitors are normally secreted by anterior mesoderm and anterior endoderm.

21. (pp. 343-345, 349-350) Draw the structure of the vertebrate neural tube if TGF-beta signaling were blocked. Hypothesize as to whether or not the sulcus limitans (p. 344) would form, and justify your hypothesis.

The sulcus limitans would not form because there would be no gradients of TGF-beta molecules to interact with the dorsal-ventral gradient of Shh. Dorsal neural fate specification would not occur. Thus, dorsal neurons and dorsal interneurons would not be specified.

22. (p. 302) What would happen to the early mouse blastocyst if *Nanog* expression was knocked down with morpholinos?

A majority of the inner cell mass cells would form hypoblast cells, rather than epiblast and embryonic stem cells. A population of ICM cells may persist owing to the actions of Stat3.

23. (pp. 290-294) Figure 8.7E. Are there notochord precursor cells located in the middle part of the primitive streak? Explain your reasoning.

No, they are all in the most anterior portion of the early primitive streak.

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24. (pp. 315-317) Most mammals have 7 cervical vertebrae. Predict the probable homeotic transformation that would occur if the RA receptor was lost AND a loss of *Hox10* expression occurred. Explain your reasoning.

Retinoic Acid (RA) suppresses anterior fate specification in vertebrates. With a loss of function in a RA receptor, a homeotic transformation of cervical vertebrae occurs. The most posterior cervical vertebra is transformed into a more anterior (thoracic) fate. Loss of *Hox10* is expressed only in the most posterior part of the embryo, so knocking down its expression would have no effect on the cervical vertebrae. However, lumbar vertebrae would be transformed into thoracic vertebrae because of the loss of *Hox10* expression.

25. (p. 342) Explain the function of the protein that underlies the mutant phenotype pictured in figure 9.11B. Why is it called the snakehead mutant?

The snakehead mutant has a dysfunctional Na+/K+ ATPase, which prevents the embryo from inflating its brain ventricles with saline fluid. The shape of the embryo's head looks like a snake.