Name _____

Biology 411 - Developmental Biology
Winter Quarter 2010

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.

1. (Essner *et al*. paper, Dicussion section) A researcher using a laser beam to photoablate (i.e. kill with light) dorsal forerunner cells in a zebrafish embryo. Describe the downstream effects on the embryo.

Kupffer's vesicle will not form. Without Kupffer's vesicle, the embryo might not be able to specify left-right asymmetry correctly. There is a substantial chance the embryo will develop with an inverted left-right asymmetry.

2. Draw what you believe the phenotype of a *caudal* Drosophila mutant embryo would look like. Label the parts of the embryo and explain your reasoning.

The embryo would be mirror-symmetric around a medial plane in the AP-axis. Both halves of the embryo would contain anterior components (i.e. thorax, head, and acron). The abdomen and telson would be missing because *caudal* is needed to specify posterior body parts.

3. (pp. 244, 248-249) Explain what would happen to pharyngeal cell fate specification if the AB and P1 cells of a two-celled *C. elegans* embryo were separated? Explain your reasoning in terms of the lineages and potencies of the AB and P1 blastomeres.

Pharyngeal endoderm would fail to form in the AB cell lineage because pharyngeal precursors come from the ABa blastomere. The ABa blastomere requires GLP-1 signaling from the EMS cell, which is derived from the P1 blastomere. This signaling would not occur if the AB and P1 blastomeres were separated.

Pharyngeal endoderm would still form in the P1 lineage because it is dependent on the *skn-1* gene, which is a maternal gene.

4. (p. 221) A mutation in *Pmar1*occurs during spermatogenesis in a sea urchin, leading to a haploid insufficiency of *Pmar1* in the zygote. Draw the embryo that would result from this mutation. Explain your reasoning.

***Pmar1* is required for the transcription of veg2 endoderm specification genes. Endoderm and secondary mesenchyme would not form. Lack of *Pmar1* activity would also lead to the de-repression of a *repressor* gene that inhibits skeletogenic differentiation of primary mesenchyme cells, as well as the activation of *Delta* (needed to activate veg2 endoderm specification genes). If primary mesenchyme cells do form, they will not produce skeletal rods. The embryo would not specify endoderm, and therefore would probably not gastrulate. The embryo would probably look like the last image of Figure 8.11C.**

5. (p. 299) Using a eyelash as a scalpel (i.e. a cutting tool), you remove the dorsal marginal zone of a *Xenopus* embryo during mid-gastrulation (Figure 10.12B) and place it in an appropriate culture medium. Diagram the shape changes that you believe will occur in the "explanted" piece of the embryo. Label the NIMZ and IMZ. Explain your reasoning.

The DMZ would elongate as cells in the IMZ undergo convergence and extension in the mediolateral dimension. Cells in the NIMZ would continue to undergo radial intercalation, producing an extension of the tissue in the anterior-posterior dimension. Overall, the explant would elongate in the AP dimension. It would narrow in the mediolateral dimension in the IMZ region, but not in the NIMZ region.

6. (pp. 280-282) Draw the expression pattern of *eve* in a *fushi tarazu (ftz)* mutant *Drosophlia* embryo at the stage of development illustrated in Figure 9.32D, using a diagram like Figure 9.33A. Identify segment and/or parasemgent borders in your diagram. More points will be awarded for clarity of drawing and labeling.

***eve* expression would expand into regions that would normally be expressing *ftz*. *eve* would still be absent from segment boundary regions expressing *wingless*.**

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7. (pp. 215, 224-228) What would happen to archenteron formation if the filopodia of **primary** mesenchyme cells were ablated with a laser during sea urchin gastrulation. Explain your reasoning.

No defect would be expected to occur. The archenteron in sea urchin is initially formed by an invagination of the vegetal plate. Convergence and extension of cells in the archenteron would still take place, resulting in its elongation. Secondary mesenchyme cells would still be able to assist the archenteron in the last 1/3 of its elongation.

8. (p. 214) Draw the resulting cleavage pattern that would result if a frog zygotic nucleus was used to replace the zygotic nucleus of a snail embryo. Explain your reasoning.

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

9. (p. 234-235) Explain how you might rescue a normal wild-type phenotype in a molluscan embryo that has had its polar lobe removed. Hint: consider a cell transplantation experiment.

The wild-type phenotype might be restored if one replaced a 4D, or a 4d blastomere, in an embryo that had its polar lobe removed, with a similar blastomere from a normal embryo.

10. (p. 231) Start with two snail embryo at the 4-cell stage. One embryo is from a *DD* mother. The other embryo is from a *dd* mother. Replace the C blastomere in the embryo from the *dd* mother with the C blastomere from the *DD* mother. Predict the outcome on the coiling pattern of the adult, and explain why this would occur.

The transplated C blastomere would not rescue the wild-type phenotype in the *dd* embryo. A left-handed coiling pattern would be produced in the resulting adult individual.

11. (pp. 268-272, 283, 286) *Ultrabithorax* gene is normally expressed in the abdomen and posterior thorax. Explain why a bithorax phenotype is produced upon mutation of the *Ultrabithorax* gene. Would a spiracle form in the Abd1 segment in the bithorax mutant? Explain your reasoning.

***Ultrabithorax* normally represses anterior thorax segment structures in parasegment 5 and 6. Upon mutation of the gene, this repression is lost. Specification of anterior thorax structures expands towards the posterior end of the embryo. A spiracle would not form in Abd1 segment in a bithorax mutant, because the AbdA protein is still there to repress spiracle specification.**

12. (p. 283) Draw the expected pattern of cuticular structures on the dorsal area of the A3 segment in a Hedgehog mutant. Assume the mutated Hedgehog gene product is null in its normal function. Explain your reasoning.

The A3 segment would be covered in cuticular hairs. Wingless expression would expand because it is no longer repressed by Hedgehog signaling, leading to cuticular hairs everywhere.

13. (p. 287) Draw the phenotype of a *Drosophila* embryo that would result if amino acid 9 on the recognition helix of the Bicoid protein was mutated from a lysine to a glycine residue. What could you do to rescue the wild-type phenotype?

The *bicoid* mutation phenotype is produced: two abdomens that are mirror-symmetric about the median plane of the AP axis. One could rescue the embryo by injecting *bicoid* mRNA into the anterior end of the oocyte.

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14. (p. 241) Draw a diagram like the right-most panel in Figure 8.39. Show what would happen if FGF signals from the tunicate endoderm failed to be produced. Show the specification cell types in the proper locations.

Notochord and mesenchyme would not be specified. Neural tube and muscle, respectively, would expand into those locations.

15. (p. 343) What would happen to the anterior-posterior axis of a chick embryo that developed in the weightlessness of outer space?

The anterior-posterior axis would no longer be determined by gravity. The posterior end of the chick blastoderm would not be associated with the lighter portions of the yolk mass.

16. If specification of pole cells in *Drosophila* failed to occur because of a gene mutation, what would the resulting phenotype of adult fly look like?

The adult would have normal external morphology. However, it would lack germ cells and would not be able to reproduce.

17. (p. 310) Describe the outcome if a frog oocyte was depleted of maternal β-catenin, then fertilized. Draw the resulting embryo, and explain your reasoning for the phenotype.

After being depleted, β-catenin would not be stabilized in a specific location. As a consequence, a Nieuwkoop Center would not be established, nor would the Spemann-Mangold Organizer. A ventralized embryo would result. The term for this embryo is a "belly piece", or "bauchstuck."

18. What is the purpose of a F2 mutagenesis screen (p. 327)?

The purpose of a F2 mutagenesis screen is to identify recessive mutations that affect embryonic development. The F2 screen is designed to identify F2 parents that are homozygous in the mutant allele.

19. In amphibians, what role does the blastocoel play in the specification of germ layers? (p. 307).

The blastocoel separates the animal pole from the vegetal prospective endoderm. Mesoderm inducing signals are not transmitted effectively through the blastocoel fluid, thus preventing the animal cap from being induced to become mesoderm.

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20. (p. 319-322) Draw the resulting frog larvae that would result if Wnt expression were <u>inhibited</u> 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 inhibitors are normally secreted by anterior mesoderm and anterior endoderm.

21. (pp. 356-357) Draw the morphology of the embryo that gave rise to the famous Siamese twins, Eng and Chang Bunker, at day 5 of their development in 1810.

The embryo looks like a normal human embryo, with an inner cell mass and a trophoblast. The partial splitting of the inner cell mass that leads to conjoined twins occurs around day 9.

22. (p. 350) What would happen to the early mouse blastocyst if *Cdx2* expression was knocked down with morpholinos?

The trophoblast would not be maintained. All cells in the embryo would become inner cell mass (ICM) cells. *Cdx2* is necessary to inhibit *Oct4*. If *Oct4* is active, trophoblast fate is inhibited.

23. Explain the molecular events in the Nieuwkoop Center, after it becomes active following the midblastula transition (p. 311).

Inhibition of beta-catenin phosphorylation results in decrease of beta-catenin degradation. Beta-catenin can then translocate to the nucleus and bind to the Tcf3/LEF transcription factor, leading to the expression of *siamois*. *Siamois* expression demarcates the locus of the Nieuwkoop Center. *Siamois* helps activate *goosecoid*, a gene that is partially responsible for the functions of the Spemann-Mangold Organizer.

24. (pp. 362-364) Most mammals have 7 cervical vertebrae. Why would a null mutation of a Retinoic Acid (RA) receptor result in a transformation of the wild-type C7 vertebral phenotype into a C6 vetebral phenotype? What is the name that is given to this type of body part transformation?

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 fate.

25. What would happen if Hensen's node from a chick embryo were transplanted into the ventral margin of a zebrafish blastoderm? Explain your answer.

A secondary body axis would be induced. Similar to the embryonic shield, Hensen's node secretes dorsalizing factors that antagonize BMP signaling, by binding to BMP molecules and preventing their access to BMP receptors.