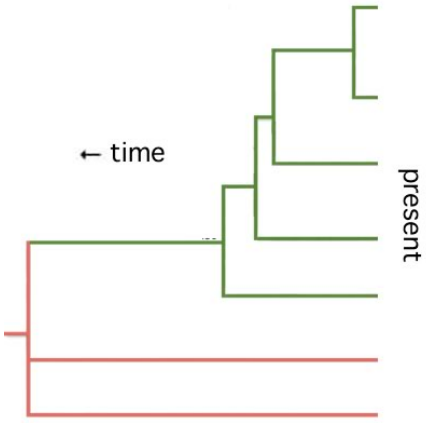
DEVO Midterm 2 review questions **March 2020**

http://virtuallaboratory.colorado.edu/DEVO@CU/index.html

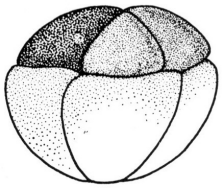
class 10:



- What features of embryonic development make various model systems (frog, fly, worm, fish, chick, mouse, human) useful and what are their limitations (for understanding human development)?
- Why is the Xenopus embryo easier to manipulate that either the *C. elegans* or mouse/human embryos.
- Provide a plausible model for why different organisms differ egg size and behavior and patterns of early development.
- Draw a model in which you place these organisms in a rough phylogenetic (evolutionary) relationship; explain your reasoning.
- Xenopus laevis has twice as many genes as humans, while Drosophila has about half as many genes as *C. elegans.* Explain the relationship between gene number and organismic complexity.
- Explain why mice and primates are not perfect model systems for studying humans (development or pathologies.

class 11/12 Xenopus + Zebrafish

- What does the term "essential" mean when it comes to a gene? How can a "non-essential" gene be conserved?
- Amphibian embryos were used to demonstrate that in vertebrates, genes are not lost during development and cellular differentiation. What types of experiments were used to support this conclusion?
- Such experiments also indicate that nuclei of differentiated cells can be "reprogrammed" to support pluripotent and totipotent behaviors - what kinds of processes are involved in the transition from differentiated to totipotent.
- Why are genetic markers needed to characterize the ability of a nucleus from a differentiated cell to generate a normal embryo (adult)?



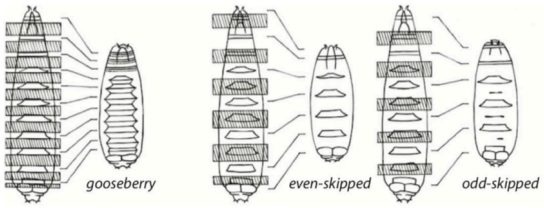
- How would you prove that the nuclei of blastomeres from an 8 cell Xenopus embryo can support totipotent cellular behavior.
- How is the generation of iPSCs similar to and different from the reprogramming of nuclei in using Xenopus?
- Map out the processes that lead from an oocyte to an embryo with an anterior-posterior and dorsal ventral axis in Xenopus. Which asymmetries are present before fertilization.
- How is it that sperm entry leads to later molecular asymmetries associated with embronic (dorsal/anterior-ventral/posterior)l axis formation

- How could you "dorsalize" or "ventralize" a Xenopus embryo?
  - Predict how mutations in the Wnt / Dsh (and Huluwa) pathway influence embryonic axes and target gene expression.
  - What one can learn from single cell RNA SEQ studies of cells at various stages during (Xenopus) embryogenesis.

- How might mitosis give rise to an asymmetry in the resulting daughter cells?
- What type of experiment could let you know whether there was mixing of cells during embryo formation?
- Explain the types of processes likely to be involved in the switch from maternal to zygotic gene expression.
- How do antibodies morpholinos, miRNAs, CRISPR CAS9 work (in general)?
- How are zebrafish and xenopus similar in terms of early development, how are they different?
- How are cellular interactions involved in "smoothing" the effects of noise within the zebrafish wnt signaling systems. Why is such a system important?

class 13/14 - Drosophila

- What made Drosophila a good system studying early embryonic patterning
- Predict and explain outcomes associated with mutations in the Bicoid/Nanos system.
  - Why carry out a screen looking at larval rather than adult phenotypes?
  - Why did they not study dominant mutations?
  - Why was the use of balancer chromosomes useful to them? what is a balancer chromosome?
  - How can you tell that a genetic screen is "saturated", what does that mean in practical terms?
- How would you identify a gene that acted maternally?
- How can you tell (using a graph) whether a mutational screen in a particular process (such as early embryonic patterning) is near or has reached saturation?
  - What does "saturation" mean exactly.
- If the early Drosophila embryo were patterned by a single simple gradient would you expect the classes of mutant phenotypes observed?



- What does it mean that two mutant alleles complement each other? Are they in the same or different genes?
- What if cells on the posterior side of the signaling cell respond, but those on the anterior side do not; what can you conclude?
- Given a signaling pathway predict the outcome of null mutations. How might an amorphic (null) allele have a dominant phenotype?
- Would you expect an anti-morphic or a neomorphic allele to be dominant or recessive (explain)?
- How does a enhancer-GAL4 / UAS-target shRNA system work?

class 15 - *C. elegans*

- What features of *C. elegans* development facilitate genetic/developmental studies. How is this similar to the bristle / segment pattern in Drosophila larvae?
- What does it mean to be a hermaphrodite?
- How did features of *C. elegans* development faciliate studies of programmed cell death (apoptosis)?
- How could you tell whether a gene is involved in a process (such as apoptosis)?
  - Why is it that you could not identify all genes that might be involved in such a process?.

class 16 -- Early mouse embryo asymmetries

- How is the mouse/human egg different from the eggs of frog, fish, worm, and fly.
  - How does this difference influence axis formation

- Draw a schematic of the process driving the specification of cell types (trophectoderm, epiblast, primitive endoderm) - where and how do these asymmetries arise?
- Given a signaling network / pathway predict outcomes of mutations.
- When a mouse (mammalian) embryo undergoes compaction, what types of cellular processes are likely to be involved? Justify your suggestion(s).