MCDB 4650 (2020) MidTerm #1 review questions

Class 1: Introduction

What are the core components of the cell theory?

Why do we believe that LUCA exists and what can we know about LUCA?

What is kinds of evidence suggest that different types of animals share a common ancestor?

When is a cell an organism?

What does it mean that cells are social?

Can cells be social without forming a single organism?

Explain why the connection between genotype and phenotype is complex (outside of mendelian examples).

How might you identify a gene (and the information it encodes) experimentally?

Class 2: Genes, experimental methods & outcomes.

Where does genetic information come from?

What is the evidence that genetic information is encoded for in molecules (of DNA)?

How is it recognized and can be hiddenin a cell?

What are common features (parts) of a gene?

What are the common characteristics of a molecular machine?

What exactly is a polypeptide, a protein, an RNA, a macromolecular complex (what is a ribosome?)

Class 3: Stochastic gene expression: sampling phenotypic space with a single genotype

How might changing the # of lac repressor binding sites (O) or repressor molecules [R] in the cell influence the expression of the lac operon?

How might the cell benefit by turning the lac operon on randomly, even in the absence of lactose?

Why isn't the lac operon always expressed?

What (generically) determines the threshold concentration of a cellular response?

What is meant by the "half-life" of a molecule? What factors can influence it?

How does half-life of key regulatory molecules influence cellular response times?

How (and why) do stochastic effects influence molecular and cellular systems?

What is monoallelic expression and what is the evidence that it exists?

In the Elowitz et al paper, describe the processes that determine which fluorescent proteins are expressed, are these reversible or irreversible events?

How does your answer change if instead of fluorescent proteins, they used transcription factors?

How do gene expression events become (effectively) irreversible?

Class 4: Quorum sensing & aggregative multicellularity

What is meant by evolutionary "costs and benefits"?

How do social factors influence such costs and benefits?

What is quorum sensing and why is it useful?

What types of bacterial responses might be under the control of quorum sensing control and why?

Why is the type of autoinducer produced critical?

What, if anything, is the value of stochastic decision making?

How is the threshold for the quorum response set?

How might a mutation in the receptor change quorum sensing responses? For example, if they reduce ligand-receptor binding affinity?

What factors might influence whether a cell builds a DNA import machine?

What factors will determine whether a bacterial cell should commit suicide?

What forms of "altruistic self-sacrifice" make sense evolutionarily?

What is a (social) cheater, how does selection influence their prevalence in a population?

Class 5: Clonal multicellularity

What kind of behavior(s) emerged in *Chlorella vulgaris* in the response to the predator *Ochromonas vallescia and why*? (Borass et al paper)

How do new genes arise? What is the role of gene duplication?

What are the possible fates of a newly generated gene?
What is meant by the terms homolog, ortholog, paralog, and convergence?
What is a null mutation, an antimorphic, hypomorphic, hypermorphic, or a neomorphic mutation?
How could a null mutation produce a dominant phenotype?
What distinguishes a somatic cell from a germ line cell?
What processes are involved in cell differentiation?

Class 6: Cellular polarity & asymmetries

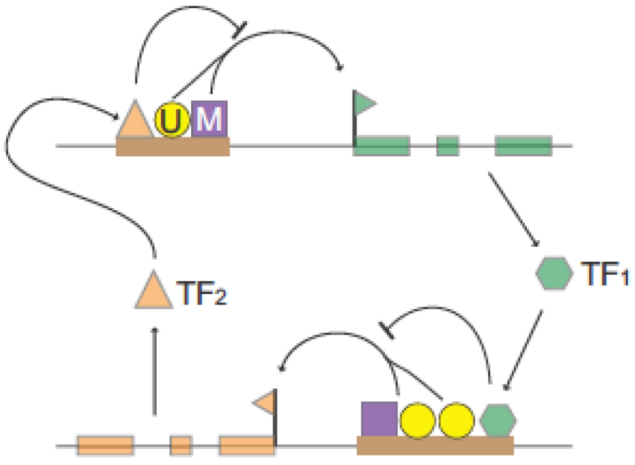
Are cells inherently asymmetric? what does that mean?
How can cellular asymmetries by generated?
What happens once they do?
How do, generally, asymmetries arise in multicellular organisms?
Why would (scientific) people come to think that humans, sponges, and unicellular choanoflagellates share a common ancestor?
What could asymmetric determinants be? In what situation(s) could there be problems with these determinants and how would that affect the early embryo?
What is chromatin diminution and how does it emerge from cellular asymmetry?
How does it influence cell behavior?
How might asymmetries arise from DNA replication? Cell division plane? Centrosome inheritance? Sperm entry cite?
What types of cellular systems display chirality? How might we visualize cellular asymmetry?

Class 7: Establishing Asymmetries / Left-Right

How might different myosins have arisen molecularly (evolutionarily)?
Why does muscle contract rather than expand (or both)?
Why is it that MTs can support movement in both directions?
How do microfilament and microtubule systems interact?
Does the microtubule system have a handedness?
What is ectopic expression and how is it done?
What does it mean that “phenotype is 100% penetrant and is specific to Myo1D”?
What does GFP expression control for?
What is a “chimera” what is a chimeric protein (polypeptide)?
What does a morpholino do? Compare to shRNA, CRSIPR Cas9.
What is the implication that myoD1 has a similar role in L/R determination in Drosophila, zebrafish and Xenopus?

Class 8: Signaling systems

How does the Notch signaling pathway differ from other signaling pathways?
What is the molecular machine behind Notch signaling?
How do feedback networks work?
Be able to generate plausible predications (and explanations) for the effects of mutations or inhibitors on the outcomes of various signaling pathways (when you are given the pathway - no memorization).
What is going on (what is described) in this picture (→).
which interactions might be cooperative? how would positive versus negative cooperative interactions influence the behavior of the system?



How many enhancers does a gene have? How would you determine which enhancers are active in a particular?
Be able to provide plausible predications (and explanations) for the effects of sequence changes on transcription factor binding affinity and gene responses to signaling gradients.

Class 8: Signaling systems, continued

How would changes in a transcription factor binding motif lead to changes in response to morphogen gradient?

How is the response of a cell to a morphogen gradient similar to or different from a bacterium's quorum sensing response?

Beside the distribution of the morphogen, what other factors might influence the behavior (read out) of a morphogen gradient?

Class 9: Establishing embryonic axes and HOX genes

Consider the role of Hox genes in anterior-posterior axis specification.

What is Hox cluster?

What evidence suggests that the common ancestor of all bilaterian animals had hox clusters.

What is a homeobox?

More questions coming - stay tuned

General:

How does "in situ hybridization work, what does it reveal?

Besides distribution of RNA, what other ways might you get a protein gradient?