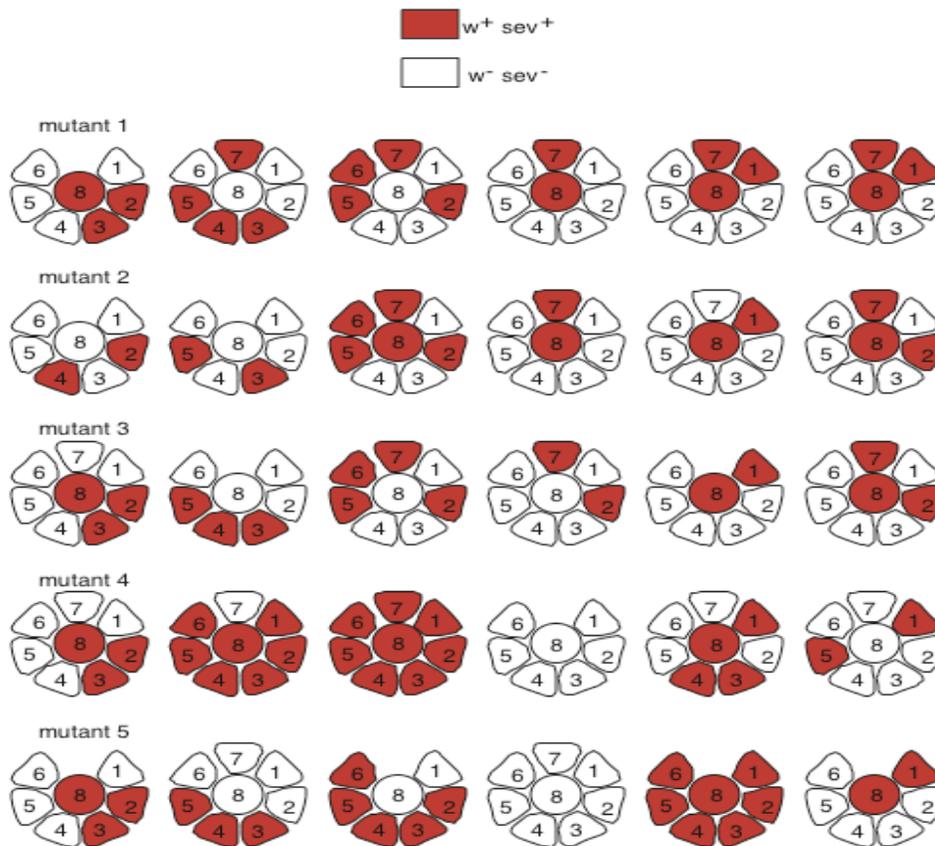


## Problem set 7

1. In a screen, you identify several new recessive mutations that lead to a loss of R7. You are interested in defining where the genes function, and carry out a mosaic analysis using *white* as a cell autonomous marker. The results for mosaic ommatidia are shown below for each mutant. What can you say about the site of function for each gene?



2. (A tricky question) The *Drosophila* MP2 neuroblast divides to produce two daughter cells, known as vMP2 and dMP2. The Numb protein is produced and distributed asymmetrically in MP2 so that it is segregated into dMP2. In the absence of *numb* function, dMP2 is transformed into an

additional vMP2 neuron. You conduct a mosaic analysis of *numb* function and generate both types of mosaics. Those where dMP2 is wild type for *numb* and vMP2 is mutant, and those where vMP2 is wild type for *numb* and dMP2 is mutant. From what you know about Numb, what do you think will be the phenotypes of the two types of mosaic animal.

3. Although the mechanisms for generating mosaic animals in *C. elegans* and *Drosophila* are different, the principle is the same: a mosaic animal is generated, and a cell autonomous marker (like *white* in the fly eye), is used to determine which cells are wild-type and which cells are mutant for the gene being studied. Acetylcholine is the neurotransmitter that is released from motor neurons in *C. elegans* and stimulates muscles that control movement to contract by binding to acetylcholine receptors on the muscle cells. Acetylcholinesterase is the enzyme that breaks down acetylcholine in the synapse to ensure that the neurotransmitter acetylcholine does not accumulate. *ace-1* is the gene that encodes the acetylcholinesterase enzyme and recessive mutations in the gene lead to animals that move abnormally. This abnormal or uncoordinated movement is caused by the buildup of acetylcholine in the neuromuscular synapse. You are interested in determining whether *ace-1* gene function is required in motor neurons, in muscles, or in both motor neurons and muscles. You generate mosaic animals that have the recessive *ncl-1* mutation and the *ace-1* mutation linked, so that when cells lose the wild-type *ncl-1* gene, they also lose the wild-type *ace-1* gene. The loss of wild-type *ncl-1* function is cell autonomous and leads to cells with large nucleoli. Thus, *ncl-1* can be used as a cell autonomous marker in this strain for the loss of *ace-1* in specific cells.

The division of the *C. elegans* zygote produces the AB and P1 cells. All of the neurons in *C. elegans* are derived from the AB cell and all of the muscles are derived from the P1 cell. Describe the results from your mosaic analysis if *ace-1* acetylcholinesterase is produced by motor neurons; by muscle; by both neurons and muscles. Be sure to include which cells contain normal nucleoli (Ncl<sup>+</sup>) and which cells contain enlarged nucleoli (Ncl<sup>-</sup>) in the different mosaic animals and which animals are uncoordinated (Unc).