REVIEW QUESTIONS FOR MIDTERM II

1. *C. elegans* is a model organism used for studying of aging and longevity. A mutant was found that lives much longer than wild-type worms. This homozygous mutant was crossed to a pure-breeding *dpy* mutant that had a normal lifespan . Note that the pure-breeding long-lived mutant was wild type for Dpy

L = long-life span *dpy+* = wildtype body size allele

N = normal life span *dpy-* = Dpy mutant

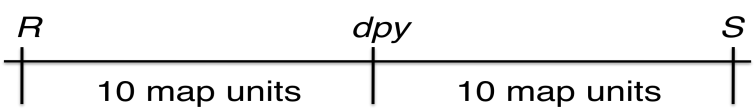
F1: All long-lived, normal body size x the tester

F2:

Long-lived, normal body size	286
Long-lived, Dpy	11
Normal life span, Dpy	290
Normal life span, normal body size	13
Total	600

a) Write out the parental and F1 genotypes above. Then show your work to indicate the relative position of the longevity gene (L) compared to the *dpy* mutation.

The following two markers R and S are both 10 map units away from *dpy* as shown. You cannot tell which side of the *dpy* gene your new longevity gene lies.



b) Explain what you would expect see in pair-wise crosses if the longevity mutation was between R and *dpy* rather than between S and dpy.

i) R- N/R- N x R+ L/R+ L

F1: R- N/R+ L x R- N/R- N (test cross)

F2: *fill in table below with approximate data set*

Long-lived, R+/R-	
Long-lived, R-/R-	
Normal life span, R+/R-	
Normal life span, R-/R-	
Total	600

ii) S- N/S- N x S+ L/S+ L

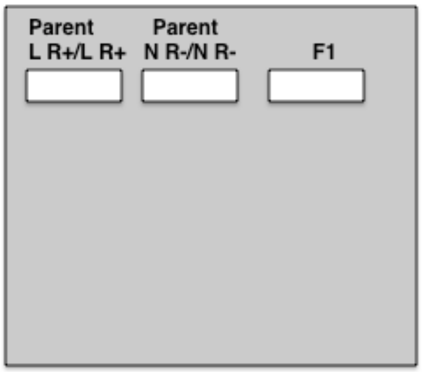
F1: S- N/S+ L x S- N/S- N (test cross)

F2: *fill in table below*

Long-lived, S+/S-	
Long-lived, S-/S-	
Normal life span, S+/S-	
Normal life span, S-/S-	
Total	600

c) The two different R phenotypes are difficult to observe when looking at the organisms. Alternatively, PCR primers are available to amplify the R- mutant vs. wildtype R+ alleles. The mutant allele R- is the result of a deletion and it yields a shorter PCR product compared to wildtype R+.

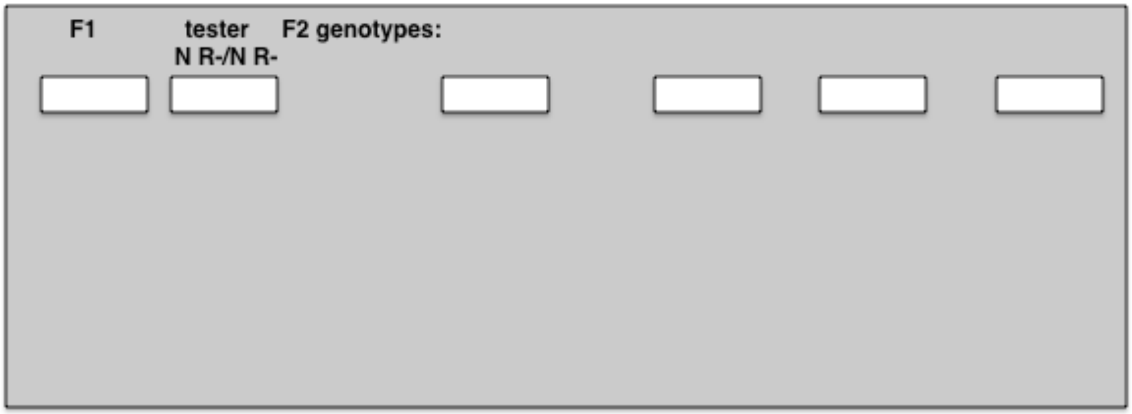
When a true-breeding long-lived mutant (L R+/L R+) is crossed to a true-breeding normal life span N R-/N R- mutant, the F1 is long-lived. Show expected banding patterns for the two parents and F1 if you amplified the R locus by PCR and ran the products on a gel.



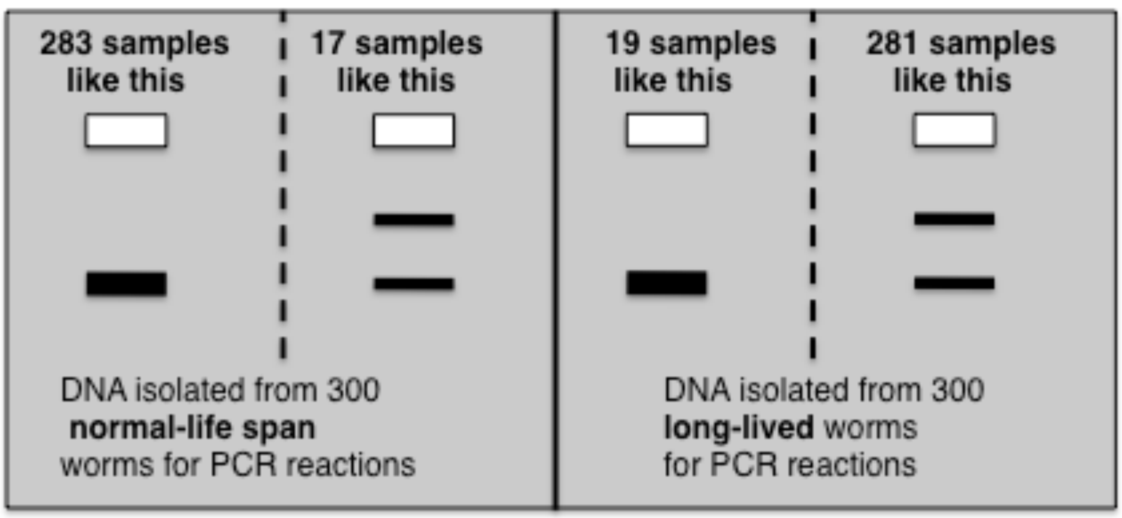
d) The F1 long-lived worms L R+/N R- worms were crossed to true-breeding normal life span R- mutants (tester).

i) Draw the chromosomes of the original parents, the F1 and tester. Then draw the four possible gametes (showing their chromosomes too). Indicate which gametes are parental and which are recombinant.

ii) In the gel shown below draw in the expected banding pattern for the F1, tester, and the four possible F2 genotypes. Write the 4 F2 genotypes above their respective lanes on the gel.



As mentioned, the F1 long-lived worms were crossed to true-breeding normal life span R- mutants. F2 worms were first phenotyped as either long-lived mutants or having a normal life span. DNA was then isolated from the two groups and PCR was performed to amplify the R locus. The results are shown below.



e) i) On the gel showing PCR reactions from the normal life span identify which banding pattern shows a parental phenotype and which banding pattern shows a recombinant phenotype.

ii) On the gel showing PCR reactions from the long-lived worms identify which banding pattern shows a parental phenotype and which banding pattern shows a recombinant phenotype.

f) Show your work to indicate the distance between the longevity gene and the R gene and draw a complete map showing all genes.

2. You have 6 populations of mice, each one displaying symptoms associated with congenital indifference to pain (can't feel pain). Two of the mutants are caused by mutations in the same gene, a third is caused by a dominant mutation in a different gene, a fourth is a recessive mutation in the same gene affected by the dominant mutation. The fifth population is a recessive mutation in yet another gene, and the sixth population may be due to environmental effects. Note: the reason these are "populations" rather than just 6 mutants is because we don't know that they are true-breeding yet.

a) If you crossed these mice with wild-type, what would you expect?

b) Can you perform a complementation test with all of these strains of mice? If you cannot do the test with a particular strain explain why? Predict the results of the complementation test for those strains you can use.

	Strain 1	Strain2	Strain3	Strain 4	Strain 5	Strain 6
Strain 1						
Strain 2						
Strain 3						
Strain 4						
Strain 5						
Strain 6						

3. Two true-breeding individuals (ABC/ABC x abc/abc) are crossed to create an organism that is heterozygous at three loci. The triple heterozygote is testcrossed, and the resulting 2000 progeny phenotypes are as follows. *Note that A>a, B>b, C>c*

A	B	C	455
a	b	c	470
A	b	C	35
a	B	c	33
A	B	c	37
a	b	C	35
A	b	c	460
a	B	C	475
		total	2,000

a. Based on your analysis of the above results what do you conclude about linkage between the three loci? **Show all of your work including calculations and the parental and recombinant types**.

b. Draw the chromosome(s) of the triple heterozygote, labelled with the genes/alleles and map distances.

c. Imagine that B and C are so tightly linked that *no* crossing-over occurs between them. What phenotype classes, and in what proportions, would you observe in the progeny of the testcross?

d. Now, Assume the A locus is responsible for a trait you are interested in, A – increased vitamin D production in the plant, and aa – wild-type. The B and C loci are different molecular markers. How would you determine if the vitamin D locus (A) is linked to the molecular marker loci (B and C)? Add hypothetical molecular marker genotypes to the plants involved in the crosses above, and predict what the data (banding patterns) would look like for the linkage you determined. You can assume that you analyze linkage between A and B and A and C separately.

e. What if you performed an F1 self? What plants would you harvest DNA from? What banding patterns would you observe if you did PCR on the microsatellite? What frequency would each banding pattern be expected (based on the linkage you calculated above)? Draw gels to aid in your explanation.

4. Ch 6. #17

In sweet peas, the synthesis of purple anthocyanin pigment in the petals is controlled by two genes, B and D. The pathway is

$$\text{white intermediate} \xrightarrow{\text{gene } B \text{ enzyme}} \text{blue intermediate} \xrightarrow{\text{gene } D \text{ enzyme}} \text{anthocyanin (purple)}$$

a. What colour petals would you expect in a pure-breeding plant unable to catalyze the first reaction?

b.

c. What colour petals would you expect in a pure-breeding plant unable to catalyze the second reaction?

d. If plants in parts a and b are crossed, what colour petals will the F1 plants have?

e. What ratio of purple: blue: white plants would you expect in the F2?

5. (Ch 6 #35) The petals of the plant *Collinsia parviflora* are normally blue, giving the species its common name, blue-eyed Mary. Two pure-breeding lines were obtained from color variants found in nature; the first line had pink petals, and the second line had white petals. The following crosses were made between pure lines, with the results shown.

Parents	F_1	F_2
blue × white	blue	101 blue, 33 white
blue × pink	blue	192 blue, 63 pink
pink × white	blue	272 blue, 121 white, 89 pink

a. Explain these results genetically. Define the allele symbols that you use, and show the genetic constitution of the parents, the F1, and the F2 in each cross

b. A cross between a certain blue F2 plant and a certain white F2 plant gave progeny of which 3/8 were blue, 1/8 were pink, and 1/2 were white. What must the genotypes of these two F2 plants have been?