Problem set 4 answers

1. The map of the left arm of maize chromosome 9 is shown below. C is the colorless gene, which is required for pigmentation of the kernel's aleurone layer, Bz is the bronze gene and is also required for pigmentation of the aleurone, and Wx is the waxy gene, which is required for production of starch in the endosperm of the kernel. The telomere is at the left end of the map and the circle represents the centromere.



In a cross the male parent was $C^+ Wx^+/C^+ Wx^+$; Ac/Ac and the female parent c wx/c wx; Ac⁺/Ac⁺. Remember the nomenclature: Ds and Ac are active dissociation and activator elements and Ds⁺ and Ac⁺ lack these activities.

a) 50% of the kernels from this cross are variegated for C but not Wx (the aleurone layer contains unpigmented spots and the endosperm is uniformly Wx^+), and 50% of the kernels are variegated for both C and Wx (the aleurone layer contains unpigmented spots and the endosperm contains sections that are mutant for Wx). Which chromosomes contain Ds and where do(es) the Ds element(s) map?

On one chromosome Ds is between C and Wx, and on the other it is between Wx and the centromere.

b) You find a kernel among the progeny of this cross that you believe contains an unstable allele of C (c^u). What phenotype suggests that the kernel contains an unstable allele of C? Can you determine from the phenotype of the kernel containing c^u if chromosome breakage occurred at this unstable allele? Explain.

Colorless with purple sectors. No, you cannot determine from the phenotype of the kernel containing c^u if chromosome whether breakage occurred at this unstable allele because breaks would result in a mutant C gene.

2. The following data are from McClintock's maize experiments. She scored the kernel phenotypes that resulted from two crosses. In both crosses the female parent was C⁺ bz wx / C⁺ bz wx and the male parent

was C-I $Bz^+ Wx^+ Ds / C^+ Bz^+ wx Ds$. The male parents in the two crosses were different plants. She scored the phenotype from the two crosses. Below are her results.

<u>Cross I</u>

Kernel phenotype <u>at Wx and C loci</u>	<u>Variegated</u>	<u>Not variegated</u>
C-I Wx ⁺	268	255
C ⁺ wx	248	242
C-I wx	88	91
C ⁺ Wx ⁺	84	83

Cross II

Kernel phenotype		
at Wx and C loci	<u>Variegated</u>	<u>Not variegated</u>
C-I Wx+	928	246
C ⁺ wx	164	893
C-I wx	62	387
$C^+ Wx^+$	344	100

a. Describe the phenotypes for each marker of each class of kernel.

CI Wx+, variegated-colorless with bronze sectors, waxy sectors.

CI Wx+, not variegated-colorless, starchy

C⁺ wx, variegated-purple with bronze sectors, waxy

C⁺ wx, not variegated-purple, waxy

CI wx, variegated-colorless with bronze sectors, waxy

CI wx, not variegated-colorless, waxy

C⁺ Wx⁺, variegated-purple with bronze sectors, waxy sectors

C⁺ Wx⁺, not variegated-purple, starchy

b. In cross I, how do kernels exhibiting the C-I wx and $C^+ Wx^+$ phenotypes arise? What does their frequencies tell you?

They arise in the male parent from recombination between C and Wx and their frequency can tell you the distance between these genes.

c. In cross I, why are the frequencies of variegated and not variegated kernels within a phenotypic approximately equivalent?

Because Ac is heterozygous in one of the parents and unlinked to these markers.

c. In cross II, why are the frequencies of variegated and not variegated kernels within a phenotypic class not equal. What does the frequency of variegated and not variegated in a phenotypic class tell you?

Ac is linked to the CI Wx+ markers, and their frequencies tell you that Ac is to the right of Wx and you can use the numbers to calculate the distance between Wx and Ac.

3. A wild-type *ry*+ (*rosy*) gene was introduced into a *ry* mutant using P element-mediated gene transformation, and after several additional crosses a strain containing a <u>stable</u> *ry*+ gene was established.

If a transformed male is mated to an M strain female, would you expect the P element construct used in this experiment to transpose to another site in the genome of the F1?

No, because there is no transposase present.

If a transformed male is mated to an P strain female, would you expect the P element construct used in this experiment to transpose to another site in the genome of the F1?

No, because expression of transposase in suppressed in the F1 germline.

4. In a screen for P element mutations in the miniature wings gene on X, you cross a male to a female that has a recessive lethal mutation on one X chromosome. The other chromosome is a balancer X chromosome containing an inversion, a recessive miniature wings mutation, a different recessive lethal mutation and the dominant Bar

mutation. How would you have generated the male?

Cross P cytotype males to M cytotype females to generate hybrid dysgenic progeny.

What will be the phenotypes most of progeny from the cross to generate new alleles of miniature wings?

The progeny will be females with the Bar phenotype and normal wings.

What is the phenotype and genotype of the animals that you will be screening for?

You will screen for females that have miniature wings. These should contain a new P-element-induced miniature wing mutation derived from the hybrid dysgenic male.

5. You cross two Drosophila strains of unknown origin. Strain A females are crossed to Strain B males. The progeny are partially sterile. Two ideas spring to mind—hybrid dysgenesis or a mitochondrial mutation that leads to the gonadal defects.

a. If the cause is hybrid dysgenesis, what type of strains did you cross and what is causing the sterility?

Strain A females were M cytotype and strain B males were P cytotype. The sterility is caused by the mobilization of P elements in the germline of the progeny, which leads to high rates of mutagenesis.

b. If the cause is a mitochondrial mutation, which strain contained the mutation? Why?

The A strain contained the mutation because the progeny will have inherited the defective mitochondria maternally.