Biology 321 Spring 2013   Assignment Set #4

➔ *Required Reading:*
Coat Variation in the Domestic Dog is Governed by Variants in Three Genes
http://fire.biol.wwu.edu/trent/trent/dogthreegenes.pdf

➔➔ ***Required Reading and Problem Assignments in 10th edition of text***
Chapter 1 *pgs 12-14 Change at the DNA Level & Figure 1-17*
Chapter 2 *Read pgs. 44-46 Alleles at the molecular level. Work problems 38 & 41*
Chapter 6 ***Read the entire chapter.*** *[Note—you are not responsible for the section on synthetic lethals on pg 233].   Work solved problems 2 and 3 and problems 4, 13, 14, 16, 17, 22, 25, 28, 32, 34, 36, 38, 60, 62, 72, 77*   ***OPTIONAL challenge problem for those who like to work puzzles:*** *68*
➔ *You are responsible for knowing about **lethal mutations** (but not synthetic lethals) even though we will not explicitly cover this topic in class.*

Required Problem Assignments in 9th edition of text
http://fire.biol.wwu.edu/trent/trent/assignmentset4.9.pdf

☞ Fun web site on cat genetics (optional)
http://www.fanciers.com/other-faqs/color-genetics.html

Problems sorted by type

Use of new terminology:
Additional problems: 3, 4, 5, 6, 7, 16

Complications to Mendel: Multiple alleles
Text problems: Solved problem 6.2, problem 22.   Additional problems: 1, 9,

Complications to Mendel: interpreting dominance
Text problems: 2.38, 6.25, 6.28, 6.34   Additional problems: 1, 2, 3, 4, 5, 6, 7, 16, 18

Comparing predictions of competing interpretations
Additional problems: 9, 18

Identifying Gene Interactions & pathway analysis (see also below which sorts problems according to type of gene interaction)
Text problems: Solved problem 6.3, problems 6.4, 6.13, 6.14, 6.16, 6.17, 6.32, 6.36, 6.38, 6.62, 6.72
Additional problems: 8, 10, 11, 13, 14, 15, 17, 18, 19, 20, 21, 22

Sorting a collection of mutants into complementation groups (different genes)
Text problems: 6.60, 6.77
Additional problems: 12, 17, 19

Knowing the category of complication or gene interaction that a given problem is in will undoubtedly bias your assessment of the data in these problems.

Complications to Mendel: lethal alleles
Text problems: 6.25, 6.28

Additive gene interactions
Text problems: 6.14, 6.16   Additional problems: 8, 9, 14, 21

Complementation
Text problems: 2.41b, 6.36, 6.60, 6.72   Additional problems: 10, 11, 12, 16, 17, 19

Epistatic gene interactions:
Text problems: Solved problem 6.3, problems 6.4, 6.13, 6.17, 6.32, 6.38
Additional problems: 13, 14, 15, 18, 20, 21, 22

Supressors and Modifiers: *See Assignment Set 5*

Problems from old Quizzes and Exams

❖ **Problem 1** In the human ABO blood type, A and B are codominant alleles and O is a recessive allele of the ABO gene. M and N are codominant alleles of a second gene. The Rh gene is a third gene involved in blood type specification. The Rh^+ allele is dominant to Rh^-.

Examine the data shown below. From this evidence, can the paternity be established?

Person	Blood Group		
Husband	O	M	Rh^+
Wife's lover	AB	MN	Rh^-
Child 1	O	MN	Rh^+
Child 2	A	N	Rh^+
Child 3	A	MN	Rh^-
Wife	A	N	Rh^+

❖ **Problem 2** The *palomino* horse has a golden yellow coat with flaxen mane and tail, the *cremello* is almost white, and the *chestnut* is brown. These horses are identical with respect to the basic color genotype: all are homozygous for a recessive allele (*b*) for *brown*. The table shows the results obtained upon mating these types in various combinations.

Parents			Offspring
cremello	x	*cremello*	all *cremello*
chestnut	x	*chestnut*	all *chestnut*
chestnut	x	*cremello*	all *palomino*

palomino	x	*palomino*	1 *chestnut*: 1 *cremello*: 2 *palomino*
palomino	x	*chestnut*	1 *palomino*: 1 *chestnut*
cremello	x	*palomino*	1 *cremello*: 1 *palomino*

a. Describe the genetic control of coat color as revealed by these results, including the allelic relationships.
b. Diagram the last three matings.
c. If you raised *palominos*, how would you set up your breeding program? Why?
d. If a *chestnut* individual is mated with a *cremelllo* one what is the chance of getting: (i) a *chestnut* animal? (ii) a *cremello* animal? (iii) a *palomino* animal?

❖ **Problem 3** A loss-of-function mutation in a ***haplosufficient*** gene
(circle all correct answers):
    a. would be recessive to the wild-type allele at the organismal level
    b. would show incomplete dominance at the organismal level
    c. would be dominant to the wild-type allele at the organismal level
    d. could show one type of dominance relationship with the wild-type allele at the organismal level and a different type of dominance at the molecular level

❖ **Problem 4** Proposed gene therapy techniques include a "gene addition" strategy that is based on the idea that a disease phenotype could be corrected by the addition (to a mutant organism's cells) of a *single wild-type copy* of the mutated gene. Ignoring potential technical problems, which scenerio(s) would be instructive in exploring the ability of gene therapy to restore a wild-type phenotype. Circle all correct answers,
a. Addition of a wild-type gene copy to homozygotes for a recessive, loss-of-function mutation
b. Addition of a wild-type gene copy to heterozygotes for a recessive, loss-of-function mutation
c. Addition of a wild-type gene copy to homozyotes for a loss-of-function mutation in a haploinsufficient gene
d. Addition of a wild-type gene copy to heterozygotes for a loss-of-function mutation in a haploinsufficient gene.
Briefly defend your choice(s) in two sentences. Be sure to indicate why you did not choose the other possibilties

❖ **Problem 5** Individuals who are heterozygous for a loss-of-function mutation in the pax-6 gene exhibit aniridia, a minor eye malformation with a population frequency of 1 in 60,000–100,000 in the US. In one rare instance two individuals with aniridia married and produced a stillborn offspring with severe malformation of the brain and no eye development.
Circle True or False or I (not enough info to decide)
T F I This loss-of-function mutation represents a polymorphism for the pax-6 gene.
T F I The pax-6 gene is haploinsufficient.
T F I The pax-6 mutation is pleiotropic and completely dominant to the wild-type   allele.

❖ **Problem 6** Circle True or False**. If there are two statements, the first statement is true and you are to decide if the second statement is True or False. Consider a statement false *if any part of it is false*.**

Examine the Table below. For each statement below choose T F or I (not enough info to decide)

T F I   The gene that codes for the apoB-100 protein is haploinsufficient.

T F I   The gene that codes for the ARH protein is haploinsufficient.

T F I   In the general population, the frequency of the mutant LDL receptor allele is 0.1%. This mutant allele would be considered a polymorphism in this population.

T F I   This data in this table indicate that hypercholesterolemia (high plasma cholesterol levels) is genetically hetereogeneous.

FOUR MONOGENIC DISEASES THAT ELEVATE PLASMA LDL AND CAUSE HEART ATTACKS				
Human disease	**Prevalence in population**	**Typical plasma LDL-cholesterol level***	**Mutant gene product**	**Mechanism for decreased LDL receptor function**
		mg/dl		
Familial hypercholesterolemia			LDL receptor	Nonfunctional receptors
Heterozygous	1 per 500[†]	300		
Homozygous	1 per million[†]	650		
Familial ligand defective apoB-100			apoB-100	Decreased binding of LDL to receptors
Heterozygous	1 per 1000[‡]	270		
Homozygous	<1 per million[‡]	320		
Autosomal recessive hypercholesterolemia	<1 per 10 million[§]	470	ARH	? Altered location of receptors in liver
Sitosterolemia	<1 per 10 million	100 to 600 depending on diet	ABCG5 and/or ABCG8	Suppression of receptor gene transcription

*Typical adult plasma LDL-cholesterol is 120 mg/dl in the United States (6). †All populations. ‡ Primarily in individuals of European descent. §Primarily in individuals of Italian and Middle Eastern descent.

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❖ **Problem 7** Factor V Leiden is the most common hereditary clotting disorder in the United States. This disorder results from a mutation in the factor V gene which codes for a protein that participates in forming clots in response to an injury. Without two fully functional factor V genes, the body's ability to put a brake on clot formation is inhibited. Someone who inherits one loss-of-function allele faces a five- to seven-fold increased risk of developing a serious and potentially life-threatening clot. Someone with two of the damaged genes has a 25- to 50-fold increased risk. Approximately one person in 5,000 in the United States and Europe has two of the mutated genes, corresponding to an allele frequency of 1.4%. *Pick the best restatement of these data.*
a. This pleiotropic mutant allele is incompletely dominant and haplosufficient.
b. This polymorphic mutation is incompletely dominant to the haploinsufficient wildtype allele.
c. The wildtype allele of this gene is haplosufficient since heterozygotes have a decreased risk of clots relative to homozygotes for the mutant allele.
d. None of these statements are an accurate reflection of the content of the paragraph.

❖ **Problem 8** The various colors of bell peppers that we see in markets are determined by various mixtures of yellow or red (lycopene) carotenoids and chlorophyll (green). All bell peppers start out green, but the final color of the pepper involves the interaction of genes that control the *production of the pigments* and the *loss of chlorophyll* from the pepper. For example, a pepper will appear yellow if it contains yellow carotenoid pigment but no lycopene or chlorophyll. (Modifier genes that control the amount of pigment made also come into play but are not addressed in this problem.)   You do a series of crosses with various strains of bell pepper. In both crosses the parental strains are true-breeding.

Cross #1: Red X Brown ---> F1 Red --------> F2 3/4 red 1/4 brown

Cross #2   Yellow X Brown ---> F1 Red ------>
F2   9/16 red   3/16 yellow   3/16 brown   1/16 yellowish-green

Fully explain cross #2:
• Define allele designations explicitly.
• Indicate the genotype of each F2 phenotypic class
• Explain the role of each gene in the final color of the pepper. Hint: green plus red = brown

⛨ **Problem 9** Recall that the I^A and I^B alleles of the ABO blood group are codominant and that the i^o allele is recessive. In 1910, it was (mistakenly) proposed that the ABO blood group traits were specified by two unlinked genes:

| Genotype	Blood Group (Phenotype)
A- B-	AB
aa B-	B
A- bb	A
aabb	O

Sorting out whether one or two genes determined this trait would have been easy in a model organism, but, of course, was not as straightforward with humans. You are a genetics graduate student in 1910 and are anxious to determine if the two gene interpretation is correct. In order resolve this issue, you start surveying two generation pedigrees in which the blood groups of the parents and children are known. You have a ton of short pedigrees to sort through and want to target the one that will be the most informative. **Which pedigrees do you focus on?**

• Assuming that you will not be able to collect data on large numbers of progeny from any mating, which of the following matings **can** provide useful information? Indicate YES or NO by each mating. For one YES and one NO mating, provide a brief explanation.

• For this question, ignore the issue of questionable parentage (ie. ambiguities concerning the identify of the father)

Blood groups (phenotypes) of parents:

AB X AB
A   X B
AB X O
O   X   O

⛨ **Problem 10** Albinism results from the absence of the pigment melanin. Melanin is synthesized by a series of biochemical reactions catalyzed by several different enzymes. Consider the following part of the biosynthetic pathway:

tyrosine --------------> Dihydrophenylalanine --------------> melanin
            enzyme a   enzyme b
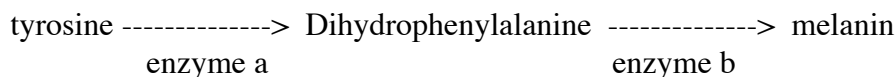
Enzyme A is coded for by gene A, which has two alleles:
 A = normal allele; a = mutant allele with zero residual enzyme activity.

The same situation exists for gene B.   For both enzymes, 50% of normal activity is sufficient for a normal phenotype. (NOTE: this question is not related to the polygenic inheritance of the degree of pigmentation observed in different races.)
a. For each genotype, give the expected phenotype (normal or albino):
AaBb, aabb, aaBB, Aabb, AaBB
b. Two albino individuals mate and have 20 children, all of whom are normally pigmented. Explain this observation using proper terminology. What are the genotypes of the parents?

⁜ **Problem 11** Snapdragons that can biosynthesis the pigment anthocyanin have reddish purple petals. Three true-breeding white lines of snapdragons that do not produce anthocyanin have been established. The mutation in each of these lines is recessive. *Line C* was developed by plant breeders in California, *Line H* in Holland and *Line W* in Washington. When petals from *Lines H and C* lines are ground up together in buffer in the same test tube, the solution, which appeared colorless at first, gradually turned reddish purple. When petals of either line were ground up alone, no color was produced.
a. Using **proper genetic terminology**, explain this observation. What can account for the production of anthocyanin in the test tube? Briefly explain
b. If Lines C and H are crossed, what fraction of the F2 will be pigmented?
c. The petals of lines H and W are ground up together, but no color is produced. Your lab partner suggests that the pH of the buffer is incorrect. Unhappy with the suggestion that you don't know how to make up a buffer of the correct pH, you suggest an alternative explanation. **Be sure to use proper genetic terminology.**

⁜ **Problem 12** Six mutant strains (#1 - 6) of Drosophila have been identified that have shorter wings than wild-type. Two sets of experiments are performed with the mutant strains. All strains used are truebreeding.

Experiment #1: Reciprocal crosses between mutant and wild-type:

Parent		Progeny		
Female	Male	female	male	
M#1	wt	wt	wt	wt = wild type (long wing)
wt	M#1	wt	wt	
M#2	wt	wt	wt	m = mutant (short wing)
wt	M#2	wt	wt	
M#3	wt	wt	m	
wt	M#3	wt	wt	
M#4	wt	wt	wt	
wt	M#4	wt	wt	
M#5	wt	m	m	
wt	M#5	m	m	
M#6	wt	wt	wt	
wt	M#6	wt	wt	

CONTINUES ON THE NEXT PAGE

Experiment #2: The mutant strains are crossed to each other and the F1 examined:

Female Parent

		1	2	4	6	
Male parent	1	--	+	+	+	-- means all progeny mutant
	2	+	--	+	+	+ means all progeny wild-type
	4	+	+	--	+	
	6	+	+	+	--	

(i) Choose the statement that describes experiment #2 most accurately.
a. This is a set of complementation tests which will tell you which of the genes are linked.
b. This is a set of complementation tests which will tell you how many different genes in Drosophila can be mutated to produce a short-wing phenotype.
c. This is a set of complementation tests which will tell you how many different genes are mutated in this set of short-winged mutants. It will also tell you which strains have mutations in the same gene.
d. This is a set of complementation tests which will tell you how many different genes are mutated in this set of mutants. It will also tell you whether the genes are linked or unlinked.
e. None of these statements accurately describes what you can determine from this set of crosses.

(ii) From the data in tables #1 and #2, you can conclude that this collection of short-winged strains represents mutations in at least five different genes.
**a.** True   **b.** False

(iii) The mutation in strain #5 could be allelic with the mutation in strain #3.
a. Yes, this is consistent with the data given
b. No, the data eliminate this possibility

� **Problem 13** The Christmas poinsettia produces modified leaves called bracts that may be white, pink or red. The pink pigmentation results from a two-step, enzymatically controlled biochemical process in which a colorless precursor is first converted to a red pigment that is then converted to the pink pigment in the second step. The color of the bract is determined by the final product of the pathway. Genes A and B assort independently
A= dominant wild-type allele   a = loss-of-function
B= dominant wild-type allele   b = loss-of-function

white ⟶ red ⟶ pink
    A   B
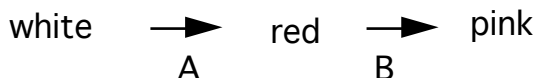
A plant of genotype AaBb is **test-crossed**. What fraction of the progeny will be white?
a. 1/4   b. 3/4   c. 1/2   d. 1   e. 0   f. cannot determine from this info

� **Problem 14** Several genes interact to determine the phenotype of the fur coat of the domestic cat. The genes listed below are all autosomal. For this question assume that all animals are <u>homozygous or hemizygous for the black pigment allele</u> of the X-linked pigmentation gene.

1. **The agouti gene:** A greyish coat coloration results from wild-type alleles **(A)** of the agouti gene, which cause a yellow band to be produced on each hair. Recessive, loss-of-function alleles **(a)** of the agouti locus are present in many cat populations. The aa genotype is epistatic to the tabby gene. Why?
2. **The tabby gene:** Superimposed on this background is a pattern of dark stripes, where the hairs have no yellow band. The pattern of stripes is controlled by a single tabby gene. You will need to determine which allele is dominant.
- *Mackeral tabby:* wild-type striped: **allele symbol t^M**
- *Blotched tabby* -- whorls of pigment: **allele symbol t^B**
3. **The albino gene:** This gene specifies the enyzme tyrosinase which catalyzes the first step in melanin biosynthesis.
       P= wild-type allele   **p =** loss-of-function allele

Your friend is a cat breeder and he mates two cats from different strains. Assume parental strains are homozygous for all genes involved.

Parental:
Blotched tabby male   X   albino female

↓

F1   all kittens   Mackerel tabby

↓

F2   2 Mackerel tabby   1 blotched tabby
                1 albino   1 solid black

a. Your friend is very surprised to see only Mackerel tabby cats in the F1 since neither parent exhibited this pattern of stripes. *Using the allele symbols designated above*, give the genotypes of the parental and F1 animals and the F2 black kitten. *Indicate the type of gene interaction that is seen in the female parent.*
b. What is the probability that a given F2 kitten will be a blotched tabby?
c. What is the probability that a given F2 kitten will be albino?

✠ **Problem 15** You have a true-breeding wild-type strain of fish. A colleague of yours gives you a mutant fish that is naked (no scales). [Another question like this is on the last page of this handout.]

You do the following crosses:

Parental   naked   X   wild-type scales
                  ⬇   (truebreeding)
F1   1/2 linear scales

                    1/2 wild-type scales

F1 Cross A   F1 linear X F1 linear

                    ⬇

          F2   9/16 linear
                3/16 wild-type
                4/16 naked

F1 Cross B   F1   linear   X   F1 wild-type

                    ⬇

          F2   3/8 linear
                3/8 wild-type
                1/4 naked

F1 Cross C   F1 wild-type X   F1 wild-type

                    ⬇

          F2   3/4 wild-type
                1/4 naked

A: Define allele symbols; indicate dominance; and indicate the trait that each gene controls

B What type of gene interaction is operating here? Use proper terminology (one word; no explanation) _____

C. Indicate genotypes for all crosses.
❖ **Problem 16** As discussed in lecture, the hormone gibberellin (GA) is an important determinant of plant height. A friend of yours has two dwarf strains of rice: strain A and strain B. Each strain is true breeding and carries mutations in just one gene. Strain A has a recessive mutation in a gene required for GA biosynthesis. Strain B has a loss-of-function mutation in the gene that codes for the GA receptor protein. The wild-type allele of the GA receptor gene is haploinsufficient. Your friend crosses the two strains and is surprised to see a semi-dwarf F1.
a. Your friend expected a completely wild-type F1. *Using **proper genetic terminology**, explain why your friend expected to see a wild-type F1. Two sentences maximum.*
b. What factor did your friend overlook that explains the F1 phenotype? *Two sentence explanation.*

⌗ **Problem 17** In the life cycle of wasps and bees, <u>unfertilized eggs develop as haploid males while fertilized eggs develop as diploid females</u>.
        Wild-type wasps have purple eyes, which contain both a "black" pigment and a "scarlet" pigment, which are the products of independent biosynthetic pathways. An investigator isolated four strains with recessive mutations resulting in a black-eyed phenotype (called mutants 1-4). He crosses true-breeding females of each mutant strain with males of each mutant strain and observes the female cross progeny. The results are indicated in the table.
(+) =purple eyes   (-) = black eyes

Mutant	#1	#2	#3	#4
#1	—	—	+	—
#2		—	+	—
#3			—	+
#4				—

(i) Choose the best restatement of these data

a. This is a complementation test, which shows that mutants 1, 2 & 4 are suppressors of each other.

b.   This is a complementation test, which shows that mutant 3 suppresses each of the other mutations.

c. This is a complementation test, which shows that mutations 1, 2 & 4 are in different genes and mutations 1 and 3 are allelic.

d. This is a complementation test, which shows that mutations 1, 2 & 4 are alleles of the same gene. Mutation 3 is in a different gene.

(ii) The F1 female progeny from the cross of mutants 1 X 3 are left unmated and their haploid male offspring examined. Designate the genotype of the F1 females. Assuming independent assortment, predict the genotypic and phenotypic ratios of the male progeny.
(iii) Generate a hypothetical model for eye pigment biosynthesis in the wasp and indicate where the genes discussed in this problem might act. Be sure to indicate the end-product of the pathway.

❖ **Problem 18** You are interested in studying the inheritance of the leaf spots in the wildflower blue-eyed Mary. Populations of blue-eyed Marys in British Columbia are polymorphic for purple leaf spots that result from patches of cells in the epidermis of the leaf that contain anthocyanin pigment. As shown below, leaves may show <u>heavy</u>, <u>medium</u> or <u>no</u> <u>spotting</u>.
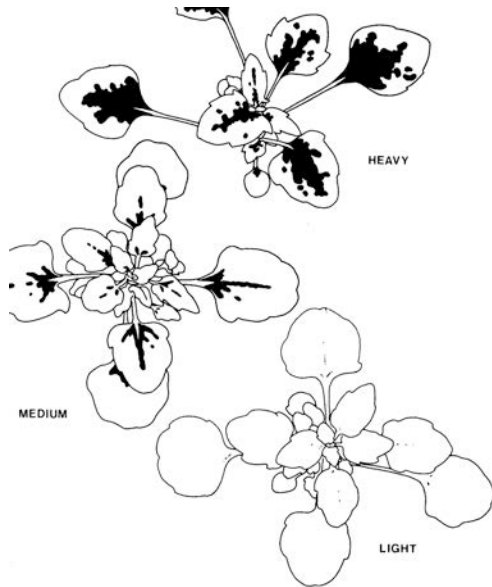

Figure 3.1   Various expressions of the leaf-spot gene in blue-eyed Mary. The existence of any leaf spot is probably deter-

You have collected some blue-eyed Marys from the wild. You have a true-breeding strain with heavy-spots and a true-breeding strain with no-spots. You want to figure out the inheritance of this trait. The following table lists three of the many possible ways that the trait could be specified genetically

GENOTYPES

	Heavy	Medium	No-spots
Hypothesis #1 One gene 2 alleles called a^1 and a^2 Incomplete dominance			
Hypothesis #2 One gene, 3 alleles a^1 = heavy a^2 = medium and a^3 = no-spots > = Complete dominance $a^1 > a^2 > a^3$			
Hypothesis #3 Two genes that interact: Gene A involved in anthocyanin biosynthesis; a is a recessive allele that produces a non-functional enzyme Gene S determines the extent of pigmentation in leaf: S- heavy ss medium			

(i). Indicate in the table what the genotype or genotypes for each phenotype would be given the scenerio in the first column. If there is more than one possibility be sure to indicate that.

(ii). Using the two true-breeding strains, design a simple experiment to differentiate between the first two possibilities. Be sure to include an F2 generation and what you would predict for both F1 and F2 given #1 or #2.

(iii). A friend of yours wants you to generate a true-breeding strain of plants with medium-spots from your original true-breeding strains.
a. If hypothesis #1 is correct, what do you tell your friend?
b. If hypothesis #2 is correct, what do you need to do?

(iv) As it turns out, the data you generate isn't consistent with either hypothesis #1 or #2, so you begin a consideration of #3.
You do the following cross. Indicate the genotypes of the parents, F1 and F2 progeny. Also give the predicted ratios of the F2 progeny classes.

Heavy-spots X No-spots ----> F1 self ----> F2 heavy, medium and no spots

(v). As it turns out, you had picked up two no-spot plants in the wild and generated two different true-breeding no-spot strains. You repeat the experiment as described in (iv) with a different no-spot strain, but this time get only heavy-spot or no-spot F2 progeny.
a. Briefly explain this observation.
b. Is this data consistent with hypothesis #3. Briefly explain

❖ **Problem 19** Rex cats have bent whiskers and very soft, mole-like fur. The Cornish Rex (C) strain of cats descended from a single rex offspring born in 1950 in Cornwall, England that was the product of a brother-sister mating. The Devon Rex (D) appeared in Devonshire in 1960 as the product of two unpedigreed animals. A third rex line (B) appeared in East Berlin in 1951. The Oregon Rex (O) was found in 1959 and the Selkirk Rex (W) made an appearance in Wyoming (USA) in the 1980s.

A breeder obtains animals from each strain (truebreeding) and performs pairwise mating. The results are summarized below. The letter indicates the geographic location where the strain appeared. All rex strains show an autosomal mode of inheritance.   R= rex offspring   WT = wild-type offspring

females

males	C	D	B	O	W
C	Rex	WT	Rex	WT	Rex
D	WT	Rex	WT	WT	Rex
B	Rex	WT	Rex	WT	Rex
O	WT	WT	WT	Rex	Rex
W	Rex	Rex	Rex	Rex	Rex

a. This type of pairwise mating between animals with the same mutant phenotype is called a _____

b.   State the purpose of this test (one sentence)

c. First interpret the data for the C, D, B and O strains. List your conclusions below. You do not need to write out genotypes.

d. What is odd about the Rex W data? How would you interpret it? *(Two sentences maximum)*

❖ **Problem 20** In some plants, a red pigment (called cyanidine) is synthesized enzymatically from a colorless precursor. A purple pigment can be synthesized from cyanidine by the enzymatic addition of an -OH group to the cyanidine molecule.   The first step requires the product of gene A and the second step requires the product of gene B. These genes are on different chromosomes. Loss-of-function mutations of each gene are recessive to the wild-type alleles. Assume that the last pigment biosynthesized in the pathway determines the flower color.   The following crosses were performed with true-breeding parental strains:

Parental   red   X   white
                    ↓
F1   purple
                    ↓ self
F2   **??**

*What are the phenotypic ratios for the F2? There is one correct answer. Partial credit possible. In other words, some answers are more wrong **than others.** **No explanation needed.***

a.   9/16 white   3/16 red   4/16 purple
b.   9/16 white   3/16 purple 4/16 red
c.   9/16 purple 7/16 white
d.   9/16 purple   3/16 red   4/16 white
e.   9/16 purple   3/16 white 4/16 red
f.   9/16 purple 7/16 red

h. 9/16 white 7/16 purple
i. not enough info to predict

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❖ **Problem 21**   Fur color in Martian coolits is polymorphic trait --green, yellow, blue or albino Here are the results of various matings among green individuals. (Brood sizes are big enough to give reliable progeny ratios.)

  Parents   **Progeny**
1. Green X Green ➜ all green
2. Green X Green ➜ 3/4 green 1/4 blue
3. Green X Green ➜ 3/4 green 1/4 yellow
4. Green X Green ➜ 3/4 green 1/4 white
5. Green x Green ➜ 9/16 green   3/16 yellow   4/16 blue
6. Green x Green ➜ 9/16 green   3/16 yellow   4/16 white
7. Green X Green ➜
            27/64 green 16/64 white 12/64 blue 9/64 yellow

Part A: How is pigment biosynthesized in the Martian coolit?
  - *Circle the pathway(s) below that are consistent with the above data. [There may be more than one correct pathway.]*
  - *Each arrow represents an step catalyzed by the wild-type allele of a specific gene. Loss-of-function mutations are recessive. The color at the endpoint of the pathway(s) determines the color of the coolit*

a. colorless ➜ yellow ➜ blue ➜ green
b. colorless ➜ blue ➜ yellow ➜ green
c. two independent biosynthetic pathways:
   colorless ➜ yellow
    colorless ➜ blue

Part B: In three sentences or less explain why the denominators of the progeny ratios are different in crosses 3, 5 and 7 above. If you want to write out genotypes, that's fine, but not required

❖ **Problem 22** Many wild populations of foxgloves are polymorphic for flower color, which includes dark magenta, light magenta and white. Many genes function in foxgloves to specify the production and deposition of pigment in the flower. *One gene's normal function is to prevent pigment deposition in the petal*s. You have a *truebreeding* dark magenta strain which you cross with a white-flowered plant that you have collected from a nearby field. Inspect the data shown below. ***Hint: the gene interaction here is epistasis.***

Define allele symbols in this box. Indicate dominance and which trait(s) the gene(s) controls. *BELOW: Indicate all genotypes in each box.*

Parental: **dark magenta (truebreeding) X white**

→ F1　1/2　**dark magenta**　　1/2　**white**

Self *F1* dark magenta plants:

3/4 dark magenta
1/4 light magenta

Self *F1* white plants:

12/16 white
3/16 dark magenta
1/16 light magenta

Predict outcome (phenotypes & ratios) of the following cross:
F1 dark magenta plants X F1 white plants