Exams

Fall 1998

answer Key

7.03 Exam 1

Name: _____

Section: _____ TA: _____

Exam starts at 11:05 and ends at 11:55

There are six pages including this cover page
Please write your name on each page.

Please...

- Look over the entire exam so you don't spend too much time on hard questions leaving easy questions unanswered.
- Check your answers to make sure that they make sense.
- To help us give partial credit, show your work and any assumptions that you make.

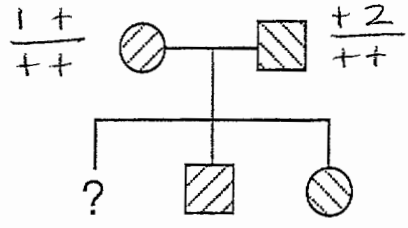
Question 1	30 points
Question 2	35 points
Question 3	35 points

Name: _____

1. The genes for two human autosomal dominant traits are 10 cM apart (as determined by meiosis in females). In the following pedigrees the traits are indicated as follows:

 = individual with trait 1 = individual with trait 2 = individual with trait 1 and trait 2

(a 15 pts.) For each of the pedigrees shown below, calculate the probability that the individual designated by "**?**" will have either dominant trait 1, dominant trait 2, or both traits.



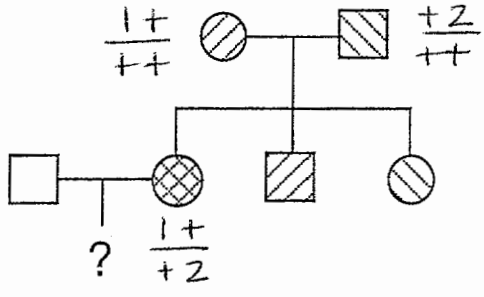
Probability

Dominant trait 1 only 1/4

Dominant trait 2 only 1/4

Both trait 1 and trait 2 1/4

(b 15 pts.)



Probability

Dominant trait 1 only (.9)(1/2) = .45

Dominant trait 2 only (.9)(1/2) = .45

Both trait 1 and trait 2 (.1)(1/2) = .05

Name: _____

2. Wild-type *Drosophila* have red eyes, and white eyes are the result of an X-linked recessive mutation. A new recessive mutation that gives apricot colored eyes is isolated. A female from a true-breeding apricot strain is crossed to a male from a true-breeding white strain, and all of the F1 females have pale-apricot eyes and all of the males have apricot eyes.

(a 5 pts.) Are the white and apricot mutations in the same gene or in different genes? Explain your answer.

Same gene. (The two genes do not complement.)
If they did complement, there would be progeny with red eyes in the F_1.

A collection of F1 females from the cross described above (all with pale-apricot eyes) are crossed to males from a true breeding white eyed strain and 1000 progeny are examined. Among these progeny, 6 flies have normal red eyes.

(b 5 pts.) What is the measured distance between the white and apricot mutations in cM?

There are 2 recombinant classes: red eyes + double mutant.
Total recombinants: 2(6)

$$\text{Distance (cM)} = \frac{2(6)}{1000} \times 100 = 1.2\,\text{cM}$$

A new recessive eye color mutation known as peach is isolated. A female from a true-breeding peach strain is crossed to a male from a true-breeding white strain, and all of the F1 females have normal red eyes and all of the males have peach eyes.

(c 5 pts.) Is the peach mutation on an autosome or on the X-chromosome? Explain your answer.

X-chromosome (because the recessive trait is expressed in males)

(d 5 pts.) Are the white and peach mutations in the same gene or in different genes? Explain your answer.

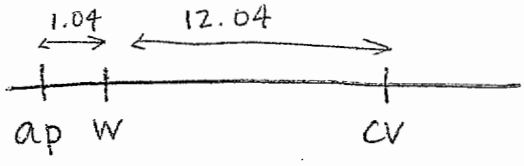
different genes (The two genes do complement)
↳ the red-eyed females

Name: _____

The recessive mutation for crossveinless wings lies on the X-chromosome. A female from a true-breeding strain with apricot eyes and crossveinless wings is crossed to a male from a true breeding strain with white eyes and normal wings. As expected, all of the F1 females from this cross have pale apricot eyes and normal wings. A large collection of these F1 females are crossed to wild-type males and 10,000 male progeny are examined. The observed phenotypes are as follows:

Phenotype		Number	
normal wings	white eyes	4,418	Parental
cv wings	apricot eyes	4,330	
normal wings	apricot eyes	610	single crossover between w and cv
cv wings	white eyes	590	
normal wings	red eyes	2	double crossover
cv wings	red eyes	50	single crossover between a : w

(e 15 pts.) Draw a genetic map showing the relative order and distances in cM between the crossveinless, apricot and white mutations.



ap-w distance: $\frac{2(2)+2(50)}{10,000} \times 100 = \frac{104}{10,000} \times 100 = 1.04\,cM$

w-cv distance: $\frac{4+610+590}{10,000} \times 100 = 12.04\,cM$

Name: _____

3. (a 5 pts.) In a yeast cross the segregation of two mutations are followed. ~~Among the 30~~

Part a omitted

(b 5 pts.) In another cross involving two different mutations, PD = 40, T = 8, NPD = 2. Give as much information as you can about the chromosomal location of these two mutations.

linked (PD > NPD)

$$cM = 100 \times \frac{T + 6NPD}{2(\text{4 tetrads})} = 100 \times \left(\frac{8 \times 12}{100}\right) = 20\ cM$$

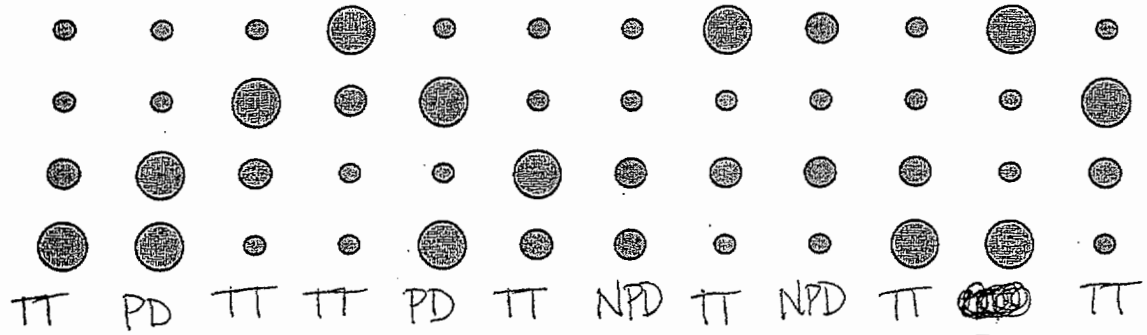
You have isolated two new yeast mutations. **big1** is a mutation that produces colonies that are larger than normal, and **sml1** is a mutation that produces colonies that are smaller than normal.

= wild-type colony

= **big1** colony

= **sml1** colony

A **big1** mutant is mated to a **sml1** mutant of the opposite mating type, the resulting diploid is sporulated and 12 tetrads are dissected. The tetrad dissection plate looks like this:



Name: _____

(c 5 pts.) What is the phenotype of a **big1 sml1** double mutant? (Does it look like wild-type, a **big1** mutant, or a **sml1** mutant?)

The NPD class is big1 sml1 } these spores are small, like the sml1 mutant
big1 sml1 }
\+ +
\+ +

(d 10 pts.) Among the 12 tetrads shown above, how many tetrads are there of each type?

PD = 3 T = 7 NPD = 2

What is the linkage relationship between **big1** and **sml1**? Give distances in cM if applicable. unlinked (PD ≈ NPD)

Part e omitted

7.03 Exam 2

ANSWER KEY

Name: _____

Section: _____ TA: _____

Exam starts at 11:05 and ends at 11:55

There are seven pages including this cover page
Please write your name on each page.

Please...

- Look over the entire exam so you don't spend too much time on hard questions leaving easy questions unanswered.
- Check your answers to make sure that they make sense.
- To help us give partial credit, show your work and any assumptions that you make.

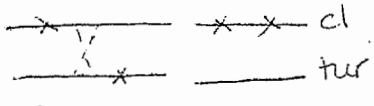
Question 1	35 points
Question 2	30 points
Question 3	35 points

Name: _____

1. You have isolated two new mutations in phage λ that produce clear plaques instead of the turbid plaques of wild-type λ phage. The two mutants are infected together into an *E. coli* host at a multiplicity of infection that ensures that each *E. coli* cell receives at least one phage of each type. The phage produced from this mixed infection are plated and of 1000 plaques examined, 997 are clear and 3 are turbid.

(**a** 10 pts.) What is the distance between the two clear plaque mutations in map units?

$\text{distance} = \frac{\#\text{recombinants}}{\text{TOTAL}} \times 100 = \frac{6}{1000} \times 100 = .6\text{mu}$



the 3 turbid plaques are recombinant, and for each turbid plaque, there is a clear plaque recombinant. Thus, there are 6 total recombinants.

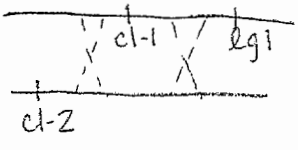
(**b** 5 pts.) If the two clear plaque mutations were found by a complementation test to be in two different closely linked genes, would this change the calculated distance between the two mutations? Explain why or why not.

No. The distance remains the same because the calculation measures the recombination between two mutations, regardless of where the genes begin or end.

(**c** 10 pts.) In fact, complementation tests show that both mutations are in the same gene known as **cI**, and therefore they are designated **cI-1** and **cI-2**. Another phage mutation designated **lg1** gives large plaques. A **cI-1 lg1** double mutant is crossed to a **cI-2** mutant as described in part **a**. From this cross, 10,000 plaques are examined and 9,970 are clear and 30 are turbid. Approximately half of the 9,970 clear plaques are large, but only 6 of the 30 turbid plaques are large. Draw a genetic map showing the relative order of the **lg1**, **cI-1**, and **cI-2** mutations. Also show the distances between the **lg1** and the **cI-1** and **cI-2** mutations in map units.

cI-2 —.6mu— cI-1 —20 mu— lg1

• Only 6 of the 30 turbid plaques are large. This suggests that these rare are the result of a double crossover. The order that requires a double crossover to get turbid large plaques is:



The distance between cI-2 : cI-1 $= \frac{30 \times 2}{10{,}000} \times 100 = .6\text{mu}$

The distance between cI-1 : lg1 $= 6/30 \times 100 = 20\text{mu}$

(of the 30 turbid plaques, 6 had an additional crossover)

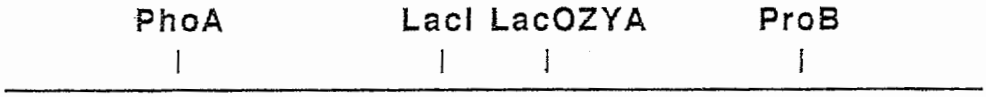
Name:

(d 10 pts.) Assume that the total genetic length of phage λ is 100 map units, and that the physical length of the phage DNA is 50,000 base pairs. From your measurement of the distance between the **cI-1** and **cI-2** mutations in part **a**, estimate the minimum number of amino acids in the protein product of the **cI** gene.

$$\frac{50{,}000\ bp}{100\ m.u.} \times .6\ mu \times \frac{1\ amino\ acid}{3\ bp} = 100\ amino\ acids$$

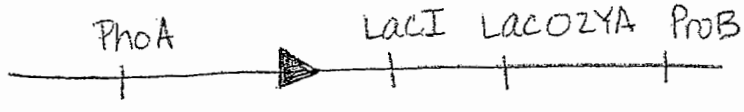
↑ we use the distance between cI-2 and cI-1 as the minimum distance

2. The region of the *E. coli* chromosome near the **Lac** operon is diagramed below:



You start with a strain that is **F$^+$ PhoA$^+$ Lac$^+$ ProB$^-$ Str^s**, and then you isolate a derivative of this strain that on mating to an **F$^-$** recipient strain can transfer **PhoA$^+$** efficiently but transfers **Lac$^+$** much less efficiently and only after long mating times.

(**a** 5 pts.) Draw a diagram of the **Hfr** that you have isolated showing where the **F** plasmid has inserted into the chromosome and the direction of the origin of transfer.



(**b** 5 pts.) The **Hfr** described above is mated to an **F$^-$ PhoA$^-$ Str^r** strain. After 10 minutes of mating a **PhoA$^+$ Str^r** recombinant strain is isolated. Will this new recombinant strain itself be able to transfer the **PhoA$^+$** marker to an **F$^-$ PhoA$^-$** recipient strain? Explain why or why not.

No. The Hfr transfers fertility last.

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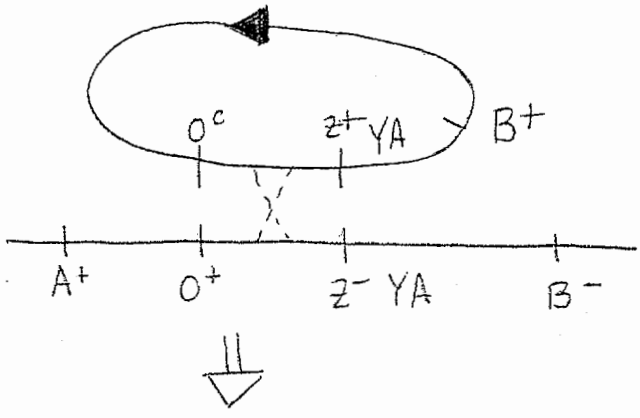
Now you would like to introduce a **LacO^c** mutation into the **Lac** operon carried by the **Hfr** strain isolated above. To do this, you grow phage P1 on a **ProB$^+$ LacO^c** host and then use the resulting phage lysate to infect the **Hfr** strain described above (genotype: **Hfr PhoA$^+$ Lac$^+$ ProB$^-$**), selecting for **ProB$^+$**.

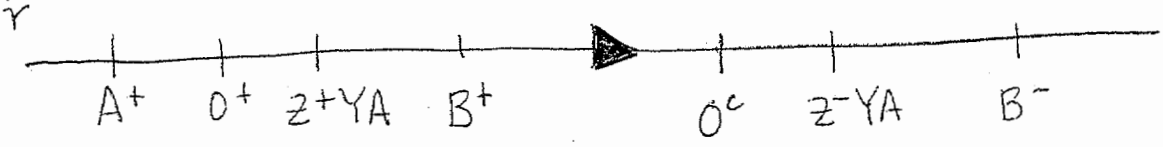
(c 10 pts.) Describe a specific test that you could use to find strains that carry **LacO^c** among the **ProB$^+$** transductants. Given that **ProB** and **LacO** show linkage of 60% cotransduction, how many **LacO^c** strains would you expect to find among 10 **ProB$^+$** transductants?

- Test ProB$^+$ transductants for constitutive β-galactosidase activity. (i.e. for β gal activity in the absence of inducer

- 60% of 10 = 6 LacO^c strains

(d 10 pts.) From the transductant isolated in part **c** (genotype: **Hfr PhoA$^+$ LacO^c ProB$^+$**) you isolate an **F'** that can transfer both **LacO^c** and **ProB$^+$** early and efficiently. This **F'** strain is mated to an **F$^-$ LacZ$^-$ ProB$^-$** recipient to produce a strain with the following genotype: **PhoA$^+$ LacZ$^-$ ProB$^-$ / F' LacO^c ProB$^+$**. This strain shows constitutive **Lac** expression but you are able to isolate a rare derivative of this strain that shows normal inducible **Lac** regulation. Draw a diagram showing how the strain with normal inducible **Lac** regulation could be produced. Your answer should show both the chromosome and **F'** in the starting strain, clearly indicating all relevant genetic markers. Any homologous recombination events should be indicated and the position of all markers in the final strain should be shown including the direction of the origin of transfer. Finally, you should indicate whether the final strain that has inducible **Lac** regulation is an **F$^-$**, **F$^+$**, **Hfr**, or **F'**.





Name: _____

3. You have identified a new strain of *E. coli* that can grow on starch. The starch degrading enzyme amylase is made only at low levels under normal growth conditions, but when starch is added to the *E. coli* culture the levels of amylase enzyme increase 100-fold. You isolate three mutants that affect amylase synthesis. The mutant $\mathbf{A}^-$ is in the structural gene for amylase and prevents the synthesis of amylase enzyme. Both the $\mathbf{B}^-$ and $\mathbf{C}^-$ mutations, which are linked to $\mathbf{A}^-$, give expression of amylase even in the absence of starch. The table below gives the amylase enzyme activities for a set of strains in either the presence or absence of the inducer starch.

	Amylase activity in enzyme units		
	– starch	+ starch	
$A^+ B^+ C^+$	1	100	
$A^- B^+ C^+$	0	0	
$A^+ B^- C^+$	100	100	constitutive
$A^+ B^+ C^-$	100	100	constitutive
$A^- B^+ C^+ / F' A^+ B^+ C^+$	1	100	
$A^+ B^- C^+ / F' A^+ B^+ C^+$	100	200	dominant
$A^+ B^+ C^- / F' A^+ B^+ C^+$	2	200	recessive
$A^+ B^- C^+ / F' A^- B^+ C^+$	100	100	} cis acting
$A^- B^- C^+ / F' A^+ B^+ C^+$	1	100	
$A^+ B^+ C^- / F' A^- B^+ C^+$	1	100	} trans acting
$A^- B^+ C^- / F' A^+ B^+ C^+$	1	100	

(a 5 pts.) Give as complete a description as you can of the properties of the $\mathbf{B}^-$ mutation, and propose a molecular function for the regulatory component that is affected by the $\mathbf{B}^-$ mutation.

B^-: constitutive
dominant
cis acting

operator (site of repressor binding)

Name: _____

(**b** 5 pts.) Give as complete a description as you can of the properties of the **C**$^-$ mutation, and propose a molecular function of the regulatory component that is affected by the **C**$^-$ mutation.

C$^-$: constitutive
recessive
trans-acting

repressor

You isolate a new mutation **D**$^-$ that alters amylase expression. In P1 transduction experiments **D**$^-$ is not linked to **A**$^-$, **B**$^-$, or **C**$^-$. The properties of some strains with the **D**$^-$ mutation are shown below.

	Amylase activity in enzyme units	
	– starch	+ starch
D^+	1	100
D^-	1	1 ← uninducible
$*D^-/F'\ D^+$	1	100 ← recessive
$D^-\ A^-$	0	0
$D^-\ B^-$	100	100 - constitutive
$D^-\ C^-$	100	100 - constitutive

(*Note that F' D^+ does not carry the amylase gene)

(**c** 15 pts.) Is **D**$^-$ uninducible or constitutive? uninducible

Is **D**$^-$ dominant or recessive? recessive

Is the function affected by the **D**$^-$ mutation most likely to act in trans or only in cis to the amylase gene?

In trans ... it is unlinked, and, therefore, cannot be a regulatory site.

Is the **D**$^-$ mutation most likely to act earlier or later than **B**$^-$ in the pathway for amylase regulation? earlier D$^-$ phenotype is hidden by B$^-$, meaning B$^-$ must act further downstream in the regulatory pathway.

Is the **D**$^-$ mutation most likely to act earlier or later than **C**$^-$ in the pathway for amylase regulation? earlier D$^-$ phenotype is hidden by C$^-$

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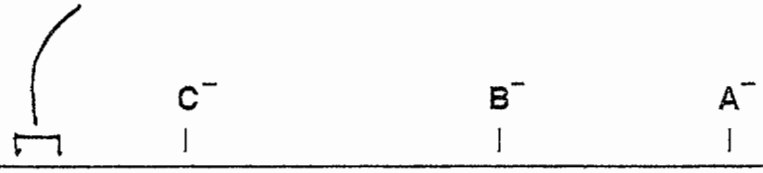
By performing biochemical experiments, you find that the protein product of the gene that is affected by the **D**$^-$ mutation binds to starch and can also bind to DNA at a site near to the **A**$^-$, **B**$^-$, and **C**$^-$ mutations as shown on the diagram below.

*Site where the **D** gene product binds*

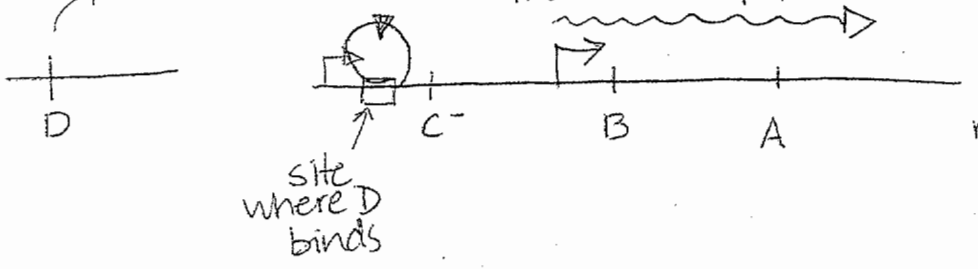


(**d** 10 pts.) Propose a molecular model that accounts for the behavior of the **A**$^-$, **B**$^-$, **C**$^-$ and **D**$^-$ mutations and that explains how starch acts as an inducer of amylase expression. Be as specific as you can.

+ starch

starch → D binds starch

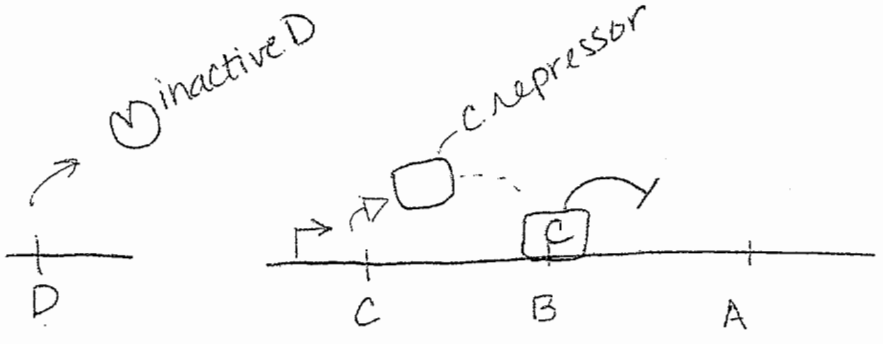
D + starch binds operator of gene C, repressing the transcription of C, allowing expression of the operon.



[No repressor is being made, so expression of the operon can proceed]

– starch

inactive D → C repressor



The D repressor is inactive, so the C gene is expressed. Now that the C repressor is being made, it binds to the operator, B, preventing expression of the operon.

Note - Exam #3 omitted

1998

7.03 Final Exam

Name: Key

Section: _____ TA: _____

There are thirteen pages including this cover page

Please write your name on each page.

Question 1	20 points
Question 2	25 points
Question 3	22 points
Question 4	20 points
Question 5	10 points
Question 6	24 points
Question 7	28 points
Question 8	26 points
Question 9	25 points

Name: _____

1. Shown below is a hypothetical scheme for the formation of eye pigment in *Drosophila*.

$$\text{Blue pigment} \xrightarrow{\textbf{Bl}} \text{Purple pigment} \xrightarrow{\textbf{Pr}} \text{Red pigment}$$

The enzyme encoded by the **Pr** gene converts a purple pigment into the normal red pigment in the eye. The **pr**$^-$ allele is recessive and homozygous **pr**$^-$ flies have purple eyes. The enzyme encoded by **Bl** gene converts a blue pigment into the purple pigment. The **bl**$^-$ allele is recessive and homozygous **bl**$^-$ flies have blue eyes. Both the **Pr** and **Bl** genes are on the X chromosome. A male from a true-breeding blue eyed strain is crossed to a female from a true-breeding purple-eyed strain.

(a 3 pts.) All of the F_1 female progeny from this cross have normal eyes. What colored eyes should the F_1 male progeny have?

$X^{Bl^+pr^-}Y \rightarrow$ purple

(b 7 pts.) An F_1 female fly (with normal eyes) is crossed to a wild-type male and a large number of male progeny from this cross are examined. Among the male progeny there are flies with normal red eyes, flies with purple eyes, and flies with blue eyes. You notice that significantly more male progeny have blue eyes than have purple eyes. Give an explanation why this should be the case.

Blue males come from both recombinant and nonrecomb. classes, purple only from non.

$X^{Bl^+pr^-} X^{bl^-Pr^+} \times X^{Bl^+Pr^+}Y$

$\downarrow$

non-recomb. $\begin{cases} X^{Bl^+pr^-}Y \rightarrow \text{purple} \\ X^{bl^-Pr^+}Y \rightarrow \text{blue} \end{cases}$ recombinants $\begin{cases} X^{Bl^+Pr^+}Y \rightarrow \text{red} \\ X^{bl^-pr^-}Y \rightarrow \text{blue} \end{cases}$

(c 10 pts.) Given that the **Pr** and **Bl** genes are 16 cM apart on the X chromosome, determine the number out of 100 male progeny from the cross in part **b** that should have purple eyes, blue eyes, or normal red eyes.

$16 = 100 \times \frac{\#\text{recomb}}{100}$

	Number
Purple-eyed males:	42
Blue-eyed males:	42+8=50
Red-eyed males:	8
Total =	100

16 recombinants
1/2 red
1/2 blue

84 non recombinants
1/2 purple
1/2 blue

Question 2 omitted

3. A deletion of the yeast **URA9** gene gives an intermediate level of growth without uracil (Ura+/-). From the **ura9Δ** strain, you isolate a robust Ura+ derivative (strain 1) which you then cross to wild type (**URA9+**). The tetrads from this cross are as follows:

4 Ura+	2 Ura+ : 2 Ura+/-	3 Ura+ : 1 Ura+/-
101	98	414

(a 6 pts.) What genetic event occurred to give robust growth without uracil in strain 1?

extragenic suppressor

(since the suppressor suppresses a deleted allele, it is bypass)

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Next, starting with the **ura9Δ** strain, you isolate a completely Ura- derivative, which does not grow without uracil (strain 2) which you then cross to wild type (**URA9+**). The tetrads from this cross are as follows:

2 Ura- : 2 Ura+	4 Ura+/-	2 Ura+/- : 1 Ura- : 1 Ura+
94	97	385

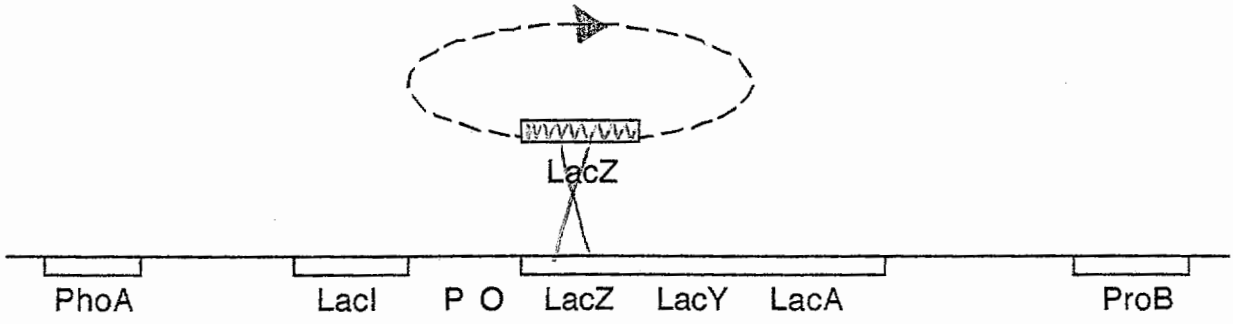
(b 6 pts.) What genetic event occurred to give the Ura- phenotype in strain 2?

synthetic lethality
unlinked

Parts c+d omitted

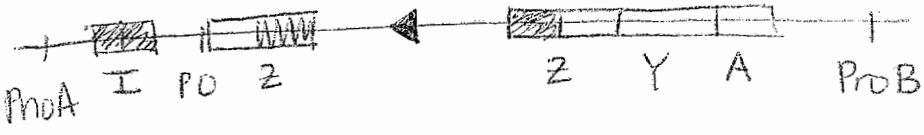
Name: _____

4. You have constructed an **F'** that carries the **LacZ** gene. Because the **F'** carries only the coding sequence of the **LacZ** gene, the **Lac** promoter, operator, and **LacY** and **LacA** genes are not included on the **F'**. A diagram of the **F'** as well as the **Lac** operon and flanking markers is shown below.



(a 2 pts.) The **F'** carrying **LacZ** is transferred into a strain with a wild type copy of the **Lac** operon on the chromosome. An **Hfr** is isolated from this strain that can transfer **ProB**$^+$ within 10 minutes of mating to a **ProB**$^-$ recipient. Draw the origin of transfer on the **F'** in the diagram above, showing the correct orientation for the direction of transfer.

(b 8 pts.) In the space below, draw a diagram showing the organization of the **Lac** genes in the Hfr isolated in part **a**. You only need to show the portion of the chromosome flanking the **Lac** operon, but be sure to show all copies of the **Lac** operon genes and the integrated **F** factor including the orientation of the origin of transfer.



(c 5 pts.) Will the **Hfr** isolated in part **a** be able to use lactose as a carbon source? Explain why or why not.

No. The regulatory sites (the promoter) is separated from LacY. Without expression of the permease, the cell cannot import lactose, and will not be able to use lactose as a carbon source.

Name: _____

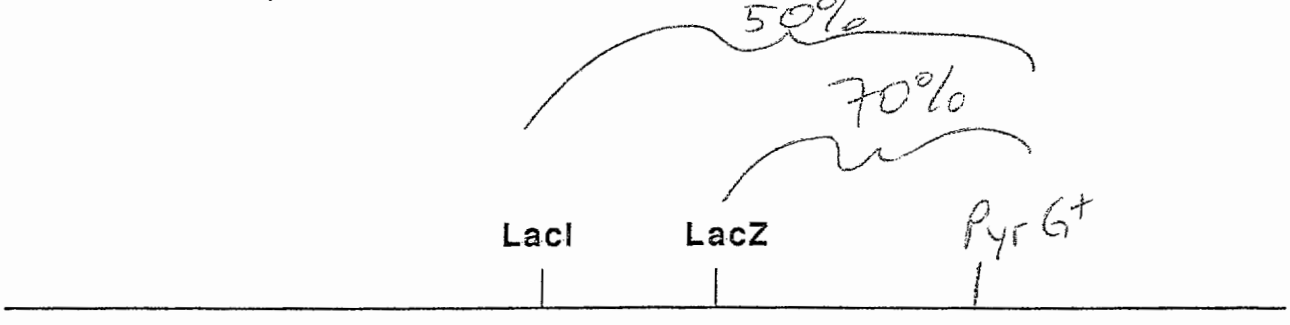
(d 5 pts.) The **Hfr** isolated in part **a** is mated to a strain that has a recessive mutation causing constitutive expression of the **Lac** operon. From this mating you wish to isolate recombinants that show normal **Lac** regulation. Would you expect such recombinants to arise early or late after mating is initiated? Explain briefly.

these recombinants will arise LATE because LacI:O will be transferred very late after mating.

5. (10 pts.) The **PyrG** gene is found to lie about one minute away from the **Lac** operon on the *E. coli* chromosome. You grow P1 phage on a **PyrG$^+$ LacZ$^-$** strain and then use the resulting phage to infect a **PyrG$^-$ LacI$^-$** strain, selecting for **PyrG$^+$**, Among 100 **PyrG$^+$** transductants, 5 show normal regulation of ß-galactosidase, 25 show constitutive expression of ß-galactosidase, and 70 show uninducible expression of ß-galactosidase.

For the reciprocal cross, you grow phage P1 on a **PyrG$^+$ LacI$^-$** strain and then infect a **PyrG$^-$ LacZ$^-$** strain, selecting for **PyrG$^+$**, Among 100 **PyrG$^+$** transductants, 20 show normal regulation of ß-galactosidase, 50 show constitutive expression of ß-galactosidase, and 30 show uninducible expression of ß-galactosidase.

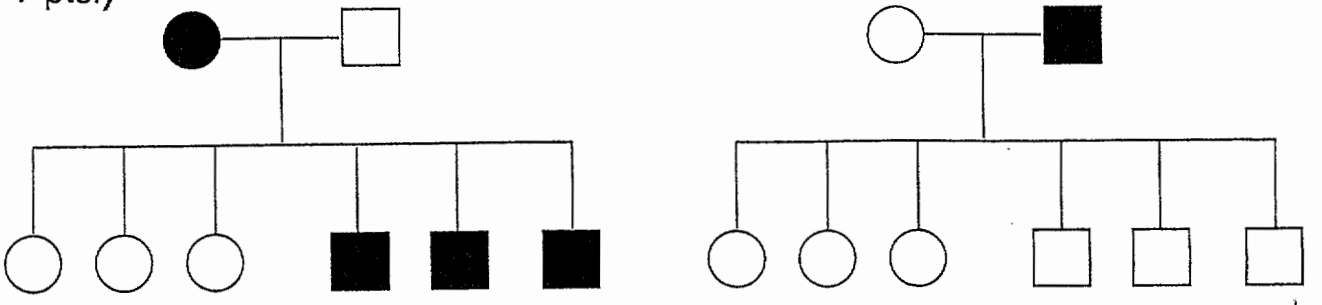
On the diagram below show where the **PyrG** gene maps relative to **LacZ** and **LacI**, giving the two-factor map distances that can be derived from this data.



Question 6 omitted **Name:** _____

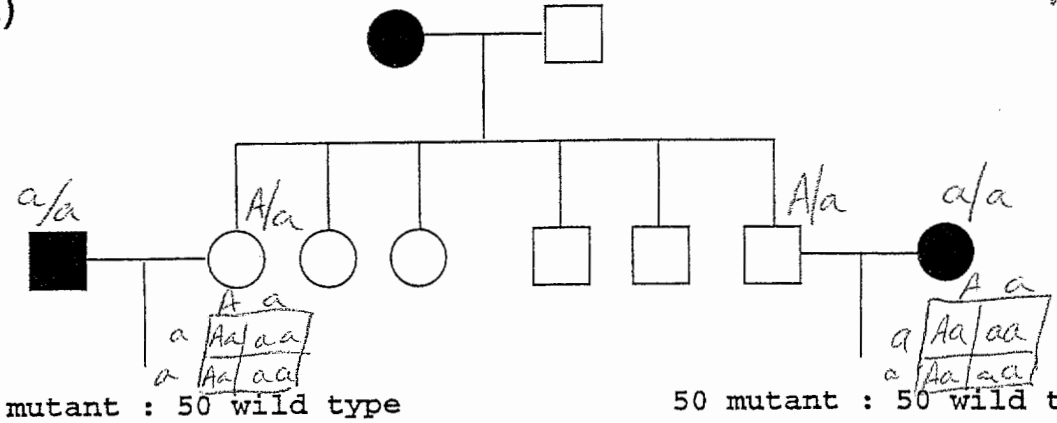
7. The following four crosses involve mice from either true-breeding mutant strains or true-breeding wild-type strains. In each case mice exhibiting the mutant traits are indicated by solid symbols. Square symbols designate males and circles designate females. In each case describe the mode of inheritance that best explains the data and give your reasoning.

(a 7 pts.)



X linked Recessive only trait
Affected mother ~~only~~ passes on to sons
Affected father mating w/ unaffected mother results in ♀ carriers & Normal males

(b 7 pts.)



50 mutant : 50 wild type

50 mutant : 50 wild type

Autosomal Recessive

Parts c+d omitted

Question 8 omitted

Name: _____

9. **(a** 9 pts.) Consider a codominant blood antigen where individuals homozygous for one allele express only antigen M, individuals homozygous for the other antigen express only antigen N, and heterozygous individuals express both N and M. In a study of three populations you determine the genotype frequencies of individuals that express only M or only N. Based on this information fill in the table below, giving the N and M allele frequencies and stating whether the population is in Hardy-Weinberg equilibrium.

Population	Frequency expressing M only	Frequency expressing N only	MN = 1 − Monly − Nonly	Allele frequencies M	Allele frequencies N	H-W equilibrium?
1	0.25	0.25	0.5	0.5	0.5	Yes
2	0.36	0.16	0.48	0.6	0.4	yes
3	0.01	0.64	0.35	0.185	0.815	No

population is in HW Eq if MM genotype freq = (M allele freq)^2 & MN genotype freq = $2 \cdot M_{\text{allele freq}} \cdot N_{\text{allele freq}}$

(M) allele freq = (Monly) + $\frac{1}{2}$(MN)

(N) allele freq = (Nonly) + $\frac{1}{2}$(MN)

(b 8 pts.) Consider a deleterious allele in a human population which has a selective disadvantage S = 1 in the homozygote and a selective disadvantage S = 0.1 in the heterozygote. The mutation rate $\mu = 10^{-5}$ for this allele. In a balance between new mutation and selection, what will the steady state allele frequency be? (Your answer can be an estimate accurate to ± 10%).

the allele is present only in heterozygotes & has a selective disadvantage in heterozygotes (dominant)

$q = \mu / s_{net} = 10^{-5} / 0.1 = \boxed{10^{-4}}$

$\Delta q_{mut} = \Delta q_{sel}$ $\quad p \approx 1$

$\mu = s_{hom} q^2 + \frac{1}{2} \cdot 2pq \cdot s_{het}$

$10^{-5} = q^2 + 0.1q \Rightarrow q^2 \approx 0 \Rightarrow 10^{-5} = 0.1q$

(c 8 pts.) If a recessive disorder occurs at a frequency of 10^{-3} in the offspring of first cousins in a population, what is the probability that a brother-sister mating from the same population would produce a child with the disorder?

F(1st cousin mating) = $\frac{1}{16}$

F(brother-sister mating) = $\frac{1}{4}$

f(affected child) = (F)(allele freq)

$10^{-3} = \frac{1}{16} \cdot q$ for first cousin matings $\Rightarrow q = 0.016$

f(affected child from brother-sister mating) = $\frac{1}{4} \cdot 0.016 = \boxed{4 \times 10^{-3}}$