Fall 2001 Biology 111 Exam #2.5 - Molecular Genetics Half Exam

There is no time limit on this test, though I have tried to design one that you should be able to complete within 1.5 hours, except for typing. You are not allowed to use your notes, old tests, any electronic sources, any books, nor are you allowed to discuss the test with anyone until all exams are turned in by class on Monday October 29. **EXAMS ARE DUE AT CLASS TIME ON MONDAY OCTOBER 29**. You may use a calculator and/or ruler. The **answers to the questions must be typed on a separate sheet of paper** unless the question specifically says to write the answer in the space provided. If you do not write your answers on the appropriate pages, I may not find them unless you have indicated where the answers are. There are 3 pages to this exam, including this cover sheet.

- 3 pts if you do not follow this direction.
Please do not write or type your name on any page other than this cover page. Staple all your pages (INCLUDING THE TEST PAGES) together when finished with the exam.

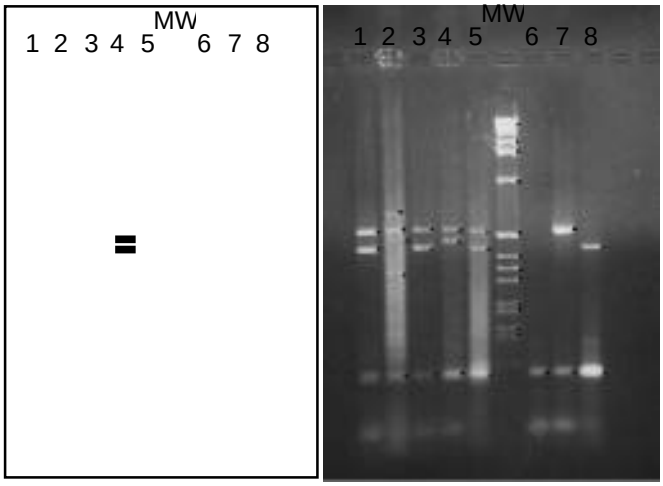
Name (please print):

Write out the full pledge and sign:

How long did this exam take you to complete (excluding typing)?

10 pts.

1) To the far right is a gel where 8 chromosomes have been isolated from one person using FACS. Each chromosome was cut with a restriction enzyme and electrophoresed from top to bottom. The gel was then Southern blotted and probed with a sequence called "DCnU". Shown to the left of the gel is an autoradiograph of the blot.



a) Interpret this Southern blot.

The marker sequence is on chromosome #4.

b) If you knew the DCnU marker sequence were linked to a disease gene by 3.7 map units, what else can you conclude about this figure?

The disease gene is also located on chromosome #4. It appears that the person may be a heterozygote, based on the RFLP for this chromosome.

10 pts.

2) A few more rare recessive traits have been discovered lately. They are: elbows backwards (hinges the other way); upside down (toe prints on top and nails on the bottom); navel in back (a belly button in back and none in front).

A couple commit to each other and the male is forwards, upside down and navel in back and his lovely partner is backwards, right side up and navel in front.

They produce a lot of progeny and the outcome is as follows:

elbow	toes	navel
forwards	upside down	navel in back = 130
forwards	right side up	navel in back = 17
forwards	upside down	navel in front = 14
forwards	right side up	navel in front = 1
backwards	right side up	navel in front = 134
backwards	upside down	navel in front = 16
backwards	right side up	navel in back = 13
backwards	upside down	navel in back = 0

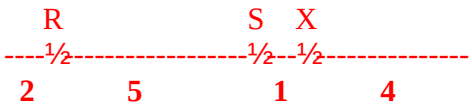
a) Draw a map to indicate the location and distances of these loci.

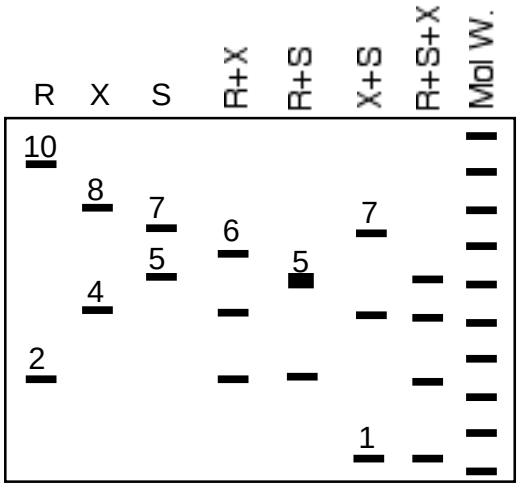
←----------18.5 map units----------→
T----------E----------N
10.5 map units 8.6 map units

b) Indicate which alleles were on each of the parental chromosomes.
E = wt elbow; T = wt toes; N = wt navel
male E t n on one chromosome and e t n on the other.

female e T N on one chromosome and e t n on the other.

10 pts.
3) Generate a restriction map using the data on the following page. Be sure the label the distances between, and identity for, each restriction site.





7 pts.
4) Explain how Francis Collins and his team were able to generate a CF cDNA probe. Begin with a human being and end your answer the a radioactive probe. You can make this a list of steps if you find it easier.

- Tissue was taken from a wt person.
- mRNA was isolated from these cells.
- cDNA was produced by incubating mRNA with reverse transcriptase, dNTPs (radioactive), and oligodT primer.

4 pts.
5) What was the cause of CF if patients with ΔF508 make functional CFTR proteins that can transport Cl^- ions as well as homozygous wild-type people can?

The functional protein was trapped in the ER and was unable to reach the plasma membrane.

6 pts.

6) What is the genetic cause of HD? Does it always occur around 40 years after birth? Explain your answer.

- A trinucleotide repeat expansion that produced more glutamate amino acids in a row. This leads to a tighter than normal binding of Huntingtin to HAP1.
- No, HD can begin earlier if the number of trinucleotide repeats is larger. The larger the repeat, the earlier the onset.

3 pts.

7) Is APOE-4 ***THE*** genetic cause for Alzheimer's disease? Explain your answer.

No, APO-E4 is not the only genetic cause for Alzheimer's disease. Homozygotes are not 100% certain to get the disease. It appears that Alzheimer's is a polygenic disease.