

**GENOME371**

**PRACTICE EXAM 2**

**KEY**

**A2003**

**1A)** Paternal: A, D, E

Maternal: B, C, F

**1B)** E

**1C)** Son 1: recombinant

Son 2: parental

Son 3: recombinant

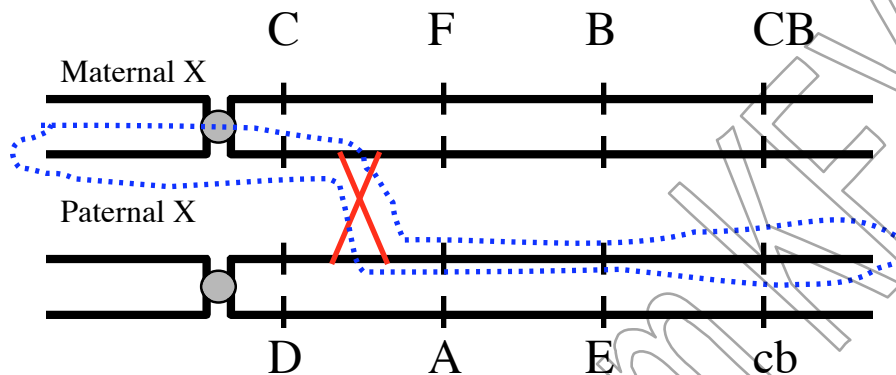
**1D)** A = F

D = C

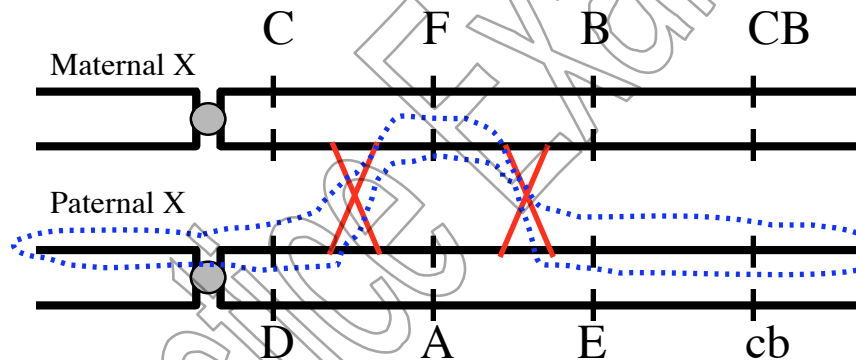
E = B

**1E/F)** Note: several answers are possible for the gene order. Only one is shown. E must be located near the recessive colorblindness allele, cb, on the paternal chromosome. The problem states that the colorblindness locus lies near the end of the chromosome. B must be located near the dominant Cb allele on the maternal X; the maternal X must also have C and F. On the paternal chromosome, D must be allelic to (located opposite to) C and A must be allelic to F.

Son 1:



Son 3:



**2)** **Ligase, A:** Both recBCD and ruvC are endonucleases which make cuts in the DNA. In this mutant ligase failed to repair these breaks and didn't reattach the DNA backbone. As a result, fragments of DNA are left behind on the metaphase plate during anaphase.

**Cohesin, C:** Cohesin protein holds sister chromatids together. It must be bound to the sister chromatids for the Synaptonemal Complex to form and for recombination enzymes to initiate crossing-over. Without crossovers to create a force that opposes microtubules, homologous chromosomes can be pulled to either pole, resulting in non-disjunction, shown in C.

**Resolvase, B:** Resolvase (ruvC) cleaves the heteroduplex DNA generated during crossing-over, thereby releasing non-sister chromatids and allowing the homologues to move to opposite poles of the spindle. Loss of function would yield attached chromosomes that fail to separate in anaphase.

Alternate answer, A: If resolvase were mutant, non-sister chromatids would remain

covalently attached to each other. The microtubules could continue pulling on the homologues and detach them from each other by brute force, creating broken chromosomes.

**Synaptonemal Complex, C:** The Synaptonemal Complex must be formed correctly for recombination to occur. Without crossing-over, microtubules could pull the homologues randomly to one pole, leading to the non-disjunction event shown in C. Note: the spindle is normal but the chromosomes behave abnormally.

**recBCD Endonuclease, C:** The endonuclease complex RecBCD cuts the DNA to initiate recombination. Without this complex, crossing over would not occur and homologues would not line up at the metaphase plate properly. Microtubules could pull the homologues randomly to one pole, leading to the non-disjunction event shown in C.

Some students argued that a mutant endonuclease could cleave the DNA too often and create the fragments shown in A. That type of mutation is due to an overactive enzyme, resulting from a gain of function mutation. We specified loss of function mutations.

**3A) Dominant, Autosomal.**

$\underline{D}$  = white,  $d$  = red

White-eyed Male  $\underline{D}/d$  x Wild-type Female  $d/d$

**3B) White-eyed sons =  $X^R/Y$  ;  $D/d$  Morgan's white-eyed females =  $X^r/X^r$  ;  $d/d$**

**3C) Single red-eyed male =  $X^R/0$  ;  $d/d$**

**3D) Non-disjunction in Mom's meiosis, could be mei I or mei II (no X from Mom)**

**4A) Phenotype of F1 tells us that African and National are dominant to Stars and Stripes.**

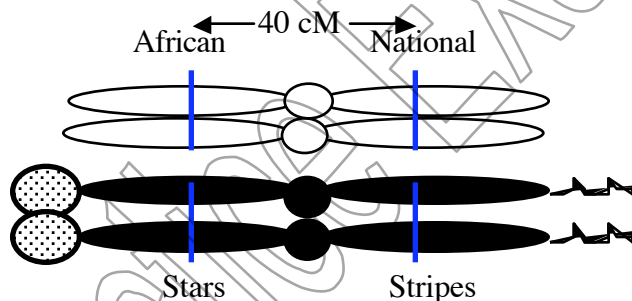
Presence of four categories of testcross progeny tells us that two genes determine the pattern.

The testcross progeny do not appear in a 1:1:1:1 ratio. Thus, these genes are linked.  $400 \text{ recombinants} / 1000 \text{ total} \times 100 = 40 \text{ cM}$ .

**4B) Karyotype  $F_1$  female: C**  
**Karyotype  $F_1$  male: E**

**4C) D**

**4D)**



**5A) Class A = tetratype (1:1:1:1, one of each, see below)**

**Class B = Parental type (2 SUP3; sup11 and 2 sup3; SUP11)**

**Class C = Non-parental type (2 SUP3; SUP11 [dead] and 2 sup3; sup11 [large])**

**5B) large colony = sup3; sup11**

**small colonies = SUP3; sup11 and sup3; SUP11**

**dead colony = SUP3; SUP11**

**5C) The two tRNA suppressors disrupt many proteins by inserting amino acids into the proteins when termination should occur at stop codons. The diploid barely survives, since twice as much message and the wild-type tRNA out-compete the suppressors. In a haploid, the suppressors win out and too many proteins are disrupted by the added amino acids.**