Crosses involving multiple alleles worksheet answers

Continue

Name: _____ Date: _____ Block: KEY

Incomplete, Co-dominance & Dihybrid Crosses

Most genetic traits have a stronger, dominant allele and a weaker, recessive allele. In an individual with a heterozygous genotype, the dominant allele shows up in the offspring and the recessive allele gets covered up and doesn't show; we call this complete dominance.

However, some alleles don't completely dominate others. In fact, some heterozygous genotypes allow both alleles to partially show by blending together how they are expressed; this is called incomplete dominance. Other heterozygous genotypes allow both alleles to be completely expressed at the same time like spots or stripes; this is called co-dominance. Examples of each are listed below.

1. Complete dominance = If a Red (RR) and White flower (rr) were crossbred, resulting in 100% Rr, what phenotype would been seen according to the rules of COMPLETE dominance?
   Rr = red
2. Incomplete dominance = If a Red (RR) and White flower (rr) were crossbred, resulting in 100% Rr, what phenotype(s) would been seen according to the rules of IN-complete dominance?
   Rr = pink
3. Co-dominance = If a Red (RR) and White flower (WW) were crossbred, resulting in 100% RW, what phenotype(s) would been seen according to the rules of CO-dominance?
   Rr = red & white stripes / spots

A. Incomplete Dominance

1. Snapdragons are incompletely dominant for colour; they have phenotypes red, pink, or white. The red flowers are homozygous dominant, the white flowers are homozygous recessive, and the pink flowers are heterozygous.

Give the genotypes for each of the phenotypes, using the letters "R" and " r " for alleles:

a. Red snapdragon genotype: RR b. Pink snapdragon genotype: Rr c. White snapdragon genotype: rr

Show genetic crosses between the following snapdragon parents, using the Punnett squares provided, and record the genotypic and phenotypic %s below:

a. pink x pink

	R	r
R	RR	Rr
r	Rr	rr

Genotypic %: 25% RR 50% Rr 25% rr
Phenotypic %: 25% red 50% pink 25% white

b. red x white

	R	R
r	Rr	Rr
r	Rr	Rr

Genotypic %: 100% Rr
Phenotypic %: 100% pink

c. pink x white

	R	r
r	Rr	rr
r	Rr	rr

Genotypic %: 50% Rr 50% rr
Phenotypic %: 50% pink 50% white

1

Name: _____ Date: _____

Life Science Period: _____

Genetics

punnett square practice

1. Let's say that in seals, the gene for the length of the whiskers has two alleles. The dominant allele (W) codes long whiskers and the recessive allele (w) codes for short whiskers.

   a. What is the probability of producing offspring that have short whiskers from a cross of two long-whiskered seals, one that is *homozygous dominant* and one that is *heterozygous*? Show your work on the punnett square.

   _____ % long whiskers
   _____ % short whiskers

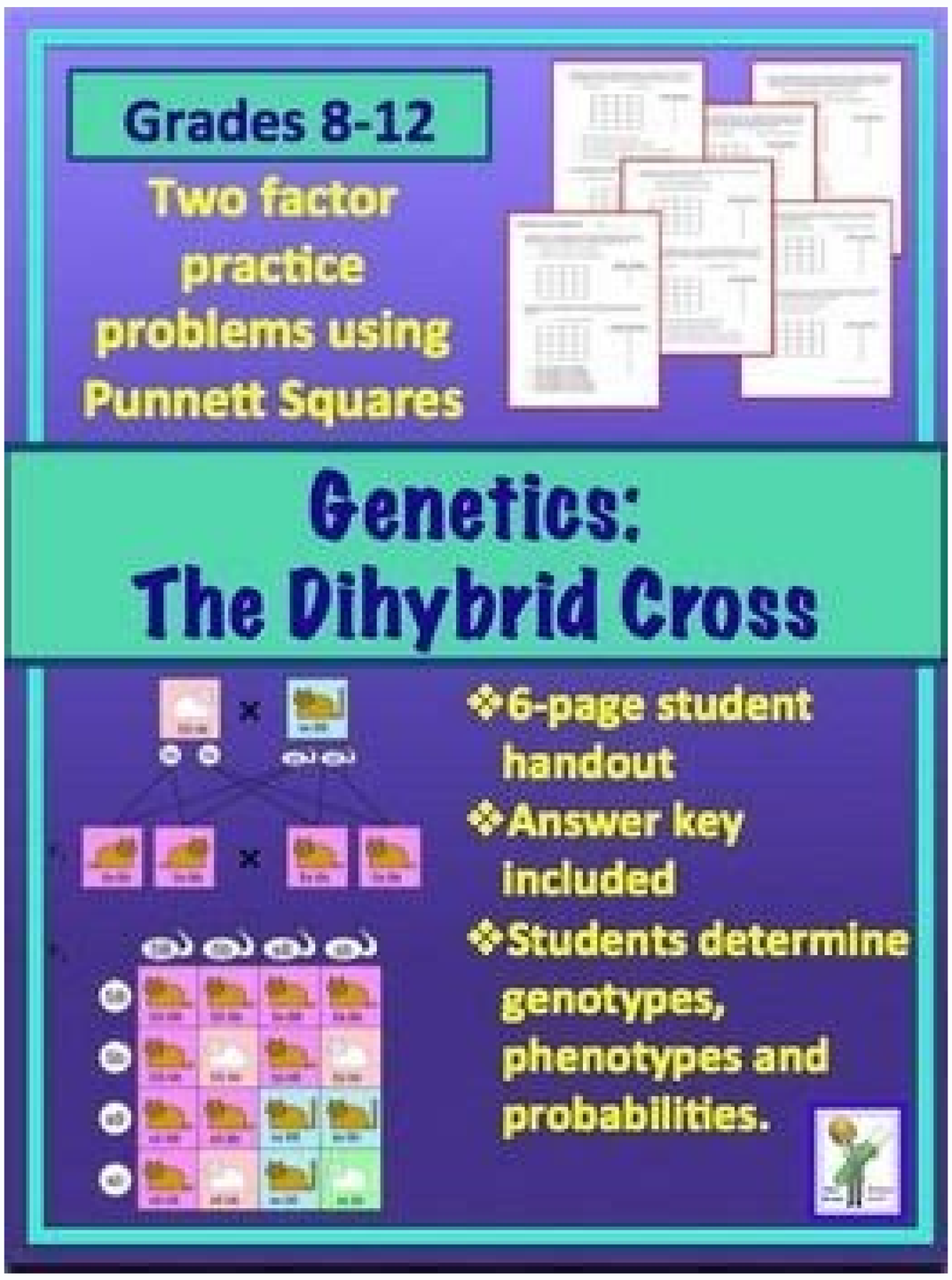
   b. If one parent seal is a *heterozygous* long-whisker and the other is *short-whiskered*, what is the probability that the offspring will have short whiskers?

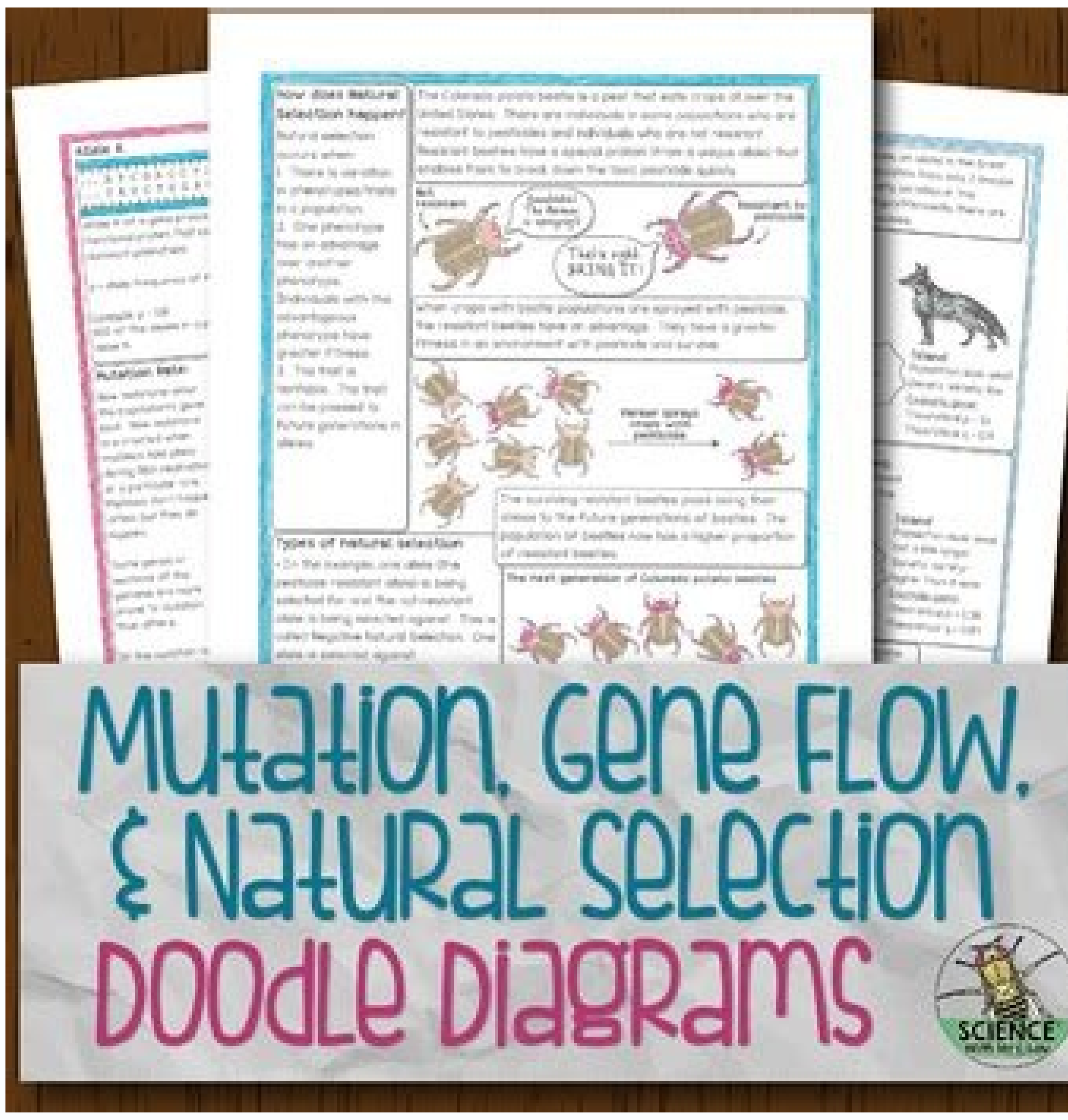
   _____ % long whiskers
   _____ % short whiskers

2. In purple people eaters, one horn (H) is dominant and no horns (h) is recessive. Complete the punnett square to show the cross of *two hybrid* purple people eaters. Summarize the genotypes and phenotypes of the possible offspring.

   Possible genotypes of offspring:

   Possible phenotypes of offspring:





What are multiple alleles give example. Double cross worksheet answer key. Multiple allele crosses worksheet answers. Multiple alleles answer key.

Answer key to practice problems--1999 2. In the smaller population -- Frequency of the recessive phenotype = (q1)2 = 4/400 Frequency of the recessive allele = q1 = 1/10 = 0.1 In the larger population -- Frequency of the recessive phenotype = (q2)2 = 54/600 Frequency of the recessive allele = q2 = 1/10 = 0.3. In the merged population -- Frequency of recessive allele q = ((400 x 0.1) + (600 x 0.3))/1000 = 0.22 Frequency of black cats in the next generation = q2 = (0.22)2 = 0.0484. A potential source of error in this problem is to simply add the number of recessive individuals from the two populations and to derive q from that -- i.e., take the square root of (4 + 54). However, doing so would ignore the contribution of recessive alleles from the heterozygotes in each population. 3. (i) If only black cats are left standing after the virus goes through, then only the recessive (black) allele will be left in the population; the frequency of the black allele in the next generation will be 1.0 (= 100%). (ii) Before the virus comes through, the frequency of the three genotypes is: Homozygous dominant = p2 = 0.25 Heterozygotes = 2pq = 0.5 Homozygous recessive = q2 = 0.25 After the viral epidemic, the only cats left are homozygous dominant and heterozygotes: Homozygous dominant = p2 = 0.25 Heterozygotes = 2pq = 0.5 Homozygous recessive = q2 = 0.25 Now the heterozygotes make up 2/3 of the surviving population, so the recessive allele makes up 1/3 of the total alleles in the population. Therefore, in the next generation the frequency of black cats will be (1/3)2 = 1/9. 4. (i) The d allele will be more frequent, as the forward mutation (D to d) occurs at a higher rate than the back mutation. (ii) Let the frequency of D = p, and the frequency of d = q, forward mutation rate = u, and back mutation rate = v Then the change in p would include loss from forward mutation and gain from back mutation; likewise, change in q would include gain from forward mutation and loss from back mutation: Change in p = vq - up Change in q = up - vq (iii) At equilibrium, change in p is exactly matched by change in q, so the change in p = 0 (as is the change in q)-- vq - up = 0; vq = up Since q = 1 - p, we can substitute and solve for p-- v(1 - p) = up v - vp = up up + vp = v p = v/(u + v) Therefore, at equilibrium, p = 0.00004/0.00016 = 0.25 q = 1-0.25 = 0.75 5. (i) 250 BB; 500 Bb (ii) df = 1. All we need to measure is the number of homozygous recessive and that lets us calculate the predicted number of the other classes (as was done in part i). 6. (i) Probability of correct identification of each heterozygote = 0.7. Therefore, the probability that both members in a heterozygote/heterozygote couple will be correctly identified = 0.7 x 0.7 = 0.49. So the probability that both members will not be correctly identified = 1 - 0.49 = 0.51 (or 51%). (ii) 5% (= 0.05, the frequency of heterozygotes in the population). (iii) If one member is tested and not found to have a disease allele, that could either mean that the person is homozygous normal, or that the person is heterozygous (probability = 0.05) but is among the 30% false negatives (probability = 0.3). So the probability that the second person is in fact a heterozygote = 0.05 x 0.3 = 0.015. 7. The premise of the resin treatment is that depletion of bile will cause liver cells to express more LDL receptors so as to increase the uptake of cholesterol. In this instance, since the cells are incapable of expressing LDL receptors anyway, depleting the body of bile acids will have no effect. 8. Construct 2. The RNA transcribed from the construct has to be complementary to the target mRNA, so it has to be transcribed off the other strand of the template DNA (so the promoter has to be at the opposite end of the gene, as in Construct 2). 1. Do a complementation test... the strain with the unknown mutation is crossed with the known torso mutant strain or the fs strain. If the unknown mutation (called mut in the diagram below) is in torso, the progeny of the cross will also have the same phenotype (tailless offspring) -- i.e., the unknown mutation fails to complement torso and therefore the unknown mutation is in torso. Alternatively, if the unknown mutation fails to complement fs, the mutation must be in fs. If the female progeny from Cross #1 have tailless offspring, the unknown mutation must be in torso; if the female progeny from Cross #2 have tailless offspring, the unknown mutation must be in fs. There's a catch--how do we deal with the problem that the progeny from the cross are going to be inviable? If conditional alleles (--see Answer 4 in Problem set 5) are available, there's an easy solution: do the cross and allow development of the resulting embryos at the permissive condition, to let the embryos develop, and then shift the young animals to the restrictive condition to look at the phenotype of their progeny. If conditional alleles are not available, an alternative strategy is to cross heterozygotes and to ask if one quarter of the progeny show the phenotype: The logic here is that if mut and torso are mutations in the same gene (for example), then Cross 1 is a monohybrid cross; one quarter of the progeny should be homozygous recessive, giving the mutant phenotype. 2. Transcription of Krüppel is inhibited by high levels of bicoid and hunchback. Since the level of bicoid is elevated (there will be no change in hunchback gene transcription (because increased transcription of hunchback is exactly matched by inhibition of its translation), the concentration gradient of bicoid protein will extend further back into the embryo; the inhibition of Krüppel gene expression will likewise extend further back, and the zone of Krüppel gene expression will occur more to the posterior than normal. The same result will be true of knirps also, as it too is inhibited by bicoid. 3. The default fate of segments is to take on anterior identities; additional genes have to be expressed to enforce posterior identities. Therefore, expression of anterior structures in posterior regions results from the failure to express the genes needed in the posterior segment -- so the mutant that has wings instead of halteres shows a recessive loss of function phenotype. In contrast, expression of posterior structures in anterior regions must be the result of inappropriate expression of posterior-specific genes in anterior segments -- a dominant, gain of function phenotype. 4. Heritability (in the broad sense) is a measure of how much of the variability in phenotype can be ascribed to variation in genotype. So if differences in phenotype are entirely because of differences in genotype, heritability for that trait = 1.0. In the following cases, if heritability is greater than 0.5, then genotype contributes more than environment. (i) Genotype is more important in determining phenotype (ii) Genotype (iii) Environment is more important than genotype (iv) Environment 5. (i) 100% -- because all the environmental factors within each city are constant and uniform, all the observed variation in IQ must be genetic. (ii) Any combination of genetic and environmental factors. Both the environment and the inherited factors are different between the two cities, so it's not possible to predict how much each factor contributes to the variation in IQ. 6. (i) 40 cm (5 cm per additive allele x 4 additives, added to the base height of 20 cm) (ii) F1 will be AaBb--which has 2 additive alleles, so the height will be 30 cm. F2 will be 20, 25, 30, 35, and 40 cm plants in 1:4:6:4:1 ratio. (iii) 25 cm plants have one additive allele -- genotype Aabb or aaBb. 35 cm plants have three additive alleles--genotype AABb or AaBB. 7. gametes ABc (2) Abc (1) ABC (3) AABBCc (5) AABbCc (4) AbC (2) AABbCc (4) AAbbCc (3) aBC (2) AaBBCc (4) AaBbCc (3) abC (1) AaBbCc (3) AabbCc (2) The cross is AaBbCC x AABbcc. As seen from the diagram, 1/8 of the progeny will have 2 additive alleles; the genotype of this class will be AabbCc. (The numbers of additive alleles in parentheses.) 8. (i) Quantitative inheritance. (ii) The frequency of either extreme phenotype gives us n, the number of gene pairs-- Frequency of one extreme phenotype = (1/4)n = 1/250 # of gene pairs = log(250)/log(4) = 4. (iii) The maximum contribution of additive alleles = 36 - 12 = 24 cm. Since 8 additive alleles (4 genes) contribute 24 cm, each additive allele contributes 3 cm. (iv) Each parent has 4 additive alleles; since the F1 also have 4 additive alleles, the parents must be each be homozygous; the additive alleles of one parent are not present in the other. For example, the genotypes could be AABBccdd x aabbCCDD (or other genotypes following that pattern). (v) An 18 cm plant has 2 additive alleles; any genotype such as AAbbccdd or aaBBccdd would work. A 33 cm plant has 7 additive alleles; any genotype such as AABBCCDd or AaBBCCDD would work. 9. There are 6 steps in height, so there can be a maximum of 6 additive alleles--i.e., there are three gene pairs. The 10 cm plant has only non-additive alleles; the 50 cm plant has 4 additive alleles at two loci (i.e., it is homozygous for additive alleles at 2 loci). One example of such a cross is: aabbcc x AABBcc The F1 progeny from such a cross would be heterozygous at two loci, and have 2 additive alleles, giving a height of 30 cm. The F2 would be 10, 20, 30, 40, and 50 cm plants in 1:4:6:4:1 ratio (there are 2 pairs of segregating alleles; the third locus is homozygous, non-contributing). 10. 500 out of 20 million individuals are homozygous dd (where D = wildtype allele and d = disaccharide intolerance). If q = frequency of allele d, q2 = 500/20,000,000 = 1/40,000 q = 1/200 Therefore, frequency of allele D = 199/200 Frequency of heterozygotes = (2)(199/200)(1/200) = 0.0095 The number of heterozygotes in the population = (20,000,000)(0.0095) = 199,000. 11. (i) Let B = allele for beach-loving; b = bridge-loving On island 1: frequency of bridge-loving iguanas (genotype bb) = 0.04 q2 = 0.04 q = 0.2 (where q = frequency of allele b); p = frequency of allele B = 0.8 On island 2: frequency of bridge-loving iguanas (genotype bb) = 0.16 q2 = 0.16 q = 0.4; p = 0.6 To look at the allele frequencies in the next generation, we can set up a table of gamete (=allele) frequencies: p = 0.6 q = 0.4 p = 0.8 0.48 0.32 q = 0.2 0.12 0.08 So in the next generation, the frequency of bridge-loving iguanas = q2 =0.08. At this point, the alleles should be at Hardy-Weinberg frequencies, so the subsequent generation will not show a change. (ii) This one can be solved only if we make the assumption that everyubody gets to mate, and that all crosses produce equal numbers of progeny. While bridge-loving iguanas are homozygous and will give rise to bridge-loving iguanas only, beach-loving iguanas consist of homozygotes as well as heterozygotes. So we can set up a table as before, but this time only for frequencies of alleles B and b within the pool of beach-loving iguanas: On island 1: p = 0.8, q = 0.2 (from part (i)) p2 = 0.64; 2pq (homozygotes) = 0.32 Among beach-loving iguanas, p = (0.64) + (0.32/2) = 0.8; q = (0.32/2) = 0.16 (there's another way of getting this value too). On island 2: p = 0.6, q = 0.4 (from part (i)) p2 = 0.36; 2pq (homozygotes) = 0.48 Among beach-loving iguanas, p = (0.36) + (0.48/2) = 0.6; q = (0.48/2) = 0.24. p = 0.6 q = 0.24 p = 0.8 0.48 0.192 q = 0.16 0.096 0.0384 Beach-loving iguanas from these crosses = (0.48)+(0.192)+(0.096) = 0.768. Bridge-loving iguanas = 1 - 0.768 = 0.232. (If we didn't make the assumption stated at the beginning, we'd just be able to make the general conclusion that homozygosity is expected increase while heterozygosity would decrease.) 12. Among females, the distribution of genotype frequencies is the usual Hardy-Weinberg frequencies -- homozygous dominant = p2, heterozygotes = 2pq, homozygous recessive = q2 (where p = frequency of dominant allele, q = frequency of recessive allele). But in males, there are no heterozygotes for X-linked traits -- males are hemizygous for such traits. Therefore, among males, p = frequency of the dominant phenotype; q = frequency of recessive phenotype. 13. (i) Assuming that the allele frequencies are the same in men vs. women -- Frequency of genotype BbBb among women = q2 = 0.09 q = frequency of allele Bb = 0.3 p = frequency of allele Bh = 0.7 Among men, phenotypes for baldness = BbBb and BbBh. Frequency of genotype BbBb = 0.09 Frequency of genotype BbBh = 2(0.3)(0.7) = 0.42 Total frequency of bald men = 0.42 + 0.09 = 0.51; 51% of the men become bald. (ii) Because these are already Hardy-Weinberg frequencies, there will be no change in allele frequencies in the next generation. 1-1998 The phenotype of a (recessive) maternal effect mutation is that females homozygous for the mutation have offspring that fail to develop normally regardless of their genotypes. If m is the mutant allele, mm (female) x any genotype (male) should give abnormal progeny that fail to develop correctly. In contrast, the mm genotype in males does not affect the progeny: mm (male) x M_ females will give normal, viable progeny. [Since there is no directly observable phenotype of mm females (other than their failure to produce normal offspring), one will have to use other markers to follow the mutagenized chromosomes. For instance, one can mutagenize a stock that is heterozygous for one (or more) known recessive markers on each chromosome, mate these with non-mutagenized strains not carrying the recessive marker alleles, and cross the F1 progeny with each other. The F2 progeny of interest will be those displaying the recessive marker traits--since the only source of the recessive allele is the lone homolog (for each phenotype) that was in the mutagenized animals, we will know that we have two copies of a chromosome that had undergone mutagenesis--and therefore potentially homozygous for a new mutation. In real life, one would also use balancer chromosomes to prevent crossovers in the mutagenized chromosomes.] 2-1998 (i) The more heterozygous population is genetically more heterogeneous, and will therefore show higher heritability than the more homozygous population. (In the homozygous population, there is relatively little genetic variation, so we have to ascribe a larger fraction of the phenotypic variation to non-genetic factors. The assumption is that both populations are showing equal amounts of phenotypic variation.) (ii) The population in the more uniform environment will show higher heritability. 3-1998 (i) Use the frequencies of the various genotype classes-- 0.82 x 0.82 = 0.41 (ii) 2(0.8)(0.2) x 0.22 = 0.0128 (iii) 2(0.8)(0.2) x 2(0.8)(0.2) = 0.1024 4-1998 For the sake of simplicity, I shall designate the allele frequencies as: p(Si) = a p(Sy) = b p(Sg) = c The distribution of genotypes then is: (a+b+c)2 = a2 + 2ab + 2ac + b2 + 2bc + c2 = 1 icky yucky gross The frequencies of icky and yucky slugs are compound terms and cannot be calculated directly. However, the frequency of gross slugs = 0.2; c2 = 0.2, therefore c = 0.45 But b2 + 2bc = 0.3 (= phenotype frequency of yucky slugs) Substituting for the value of c in this equation, we get b2 + 0.9b - 0.3 = 0 Solving for b, we get b = 0.26. a = 1 - (b + c) = 0.29. p(Si) = 0.29 p(Sy) = 0.26 p(Sg) = 0.45 1. (a) The promoter is defective, so there can be no transcription of the lac operon. (b) The operator is mutated, so lac repressor cannot bind -- transcription of lac Z, Y, and A will be constitutively high regardless of whether lactose is present or absent. However, catabolite repression is still intact, so this constitutive transcription will occur only when glucose is absent. (c) Transcription of lacZ and lacY will still be under normal inducible control; the lacA product alone will not be made. (d) Depending on the nature of the missense mutation, the lacY product (lac permease) may be functional or non-functional. Transcription of all three lac genes should be unaffected by the mutation. [Because the lac permease is responsible for import of lactose into the cell, the strain may show a slower response to lactose as the inducer than wild type.] (e) A stop codon near the start of the lacY coding region would likely act as a polar mutation (the ribosome would never get to the start codon of lacA), so the cell would produce neither lac permease not lac transacetylase. (f) Without CAP, no activation of lac gene transcription can occur regardless of whether glucose is present or absent, or whether lactose is present or absent. (g) Since phosphoenol pyruvate (PEP) is one of the glycolysis products that inhibits cAMP production (and thereby blocks CAP activation), the failure to produce PEP will result in reduced inhibition of CAP activation by glucose; lactose will induce lac operon transcription even in the presence of glucose. 2. (i) Constitutively high beta-galactosidase. i- p+ oc z+ -- the operator is defective; cannot be repressed. i+ p+ o+ z- (ii) Constitutively low. i+ p+ o+ z+ is p+ o+ z+ --the super-repressor lacIs can act in trans to repress both lacZ alleles (iii) Constitutively low (no transcription of lac operon) i- p- oc z+ --no transcription of this lacZ allele because the promoter is mutated i+ p+ oc z- --no expression of beta-gal because this lacZ allele is mutated (iv) Constitutively high is p- o+ z+ --this lacZ is always off (no promoter, super-repressor), but i- p+ oc z+ the repressor can't bind to this lacOc operator; this lacZ copy is always expressed (oc is epistatic to is) (v) Constitutively low (same as iii) i+ p- o+ z+ i- p+ o+ z- 3. (a) gal3c will be dominant, gain-of-function: in a GAL3+/gal3c heterozygote, even if normal Gal3 protein is not binding to Gal80 (in the absence of galactose), mutant Gal3 protein can always bind and inactivate Gal80 protein regardless of whether galactose is present or absent. (b) Recessive. The mutant allele cannot provide Gal80-binding activity, but the normal allele can -- the heterozygote can respond like wild type. 4. (a) An example of a polar mutation -- the mutation must result in premature termination of translation such that a truncated, non-functional protein B is made, and translation of gene C coding sequence does not occur. (b) A key point to note here is that various types of mutations can occur in any gene. A transcription activator can be mutated so that it is incapable of activation, or it be mutated so that it always activates, even when it's not supposed to. Likewise, a repressor could be mutated so that it never represses or so that it always represses. The reg gene product must be a regulator of transcription of operon ABC. It could either be an activator or a repressor. Possibility 1 -- reg is an activator of transcription. An "always on" mutant phenotype must be the result of a mutant activator that constitutively (and inappropriately) activates transcription. The mutant phenotype is expected to be dominant, because even if normal protein is being made and only activates transcription when appropriate, the mutant protein will always activate transcription. The "never on" mutant phenotype in this scenario must the result of mutant activator protein that fails to activate -- and this phenotype will be recessive. Possibility 2 -- reg is a repressor of transcription. The "always on" phenotype must be a recessive mutation that allows transcription when appropriate; the "never on" phenotype must be from a dominant mutation that always represses transcription. Looking at the actual results, we see that the the data support possibility 1 and not possibility 2: the "always on" phenotype is dominant and the "never on" phenotype is recessive. Therefore, reg must be an activator of ABC gene transcription. 5. The mutation must be in a zygotic gene -- the gene product is only needed after the first few divisions, when transcription of that gene starts up in the developing embryo. 6. (a) nanos mutations are maternal effect mutations -- females homozygous for the mutation produce eggs that lack nanos protein. Consequently, the posterior segments in the embryo fail to develop normally (the posterior of the embryo is where nanos protein normally is localized). The mutation is lethal. (b) This is a zygotic gene; failure to produce hunchback protein results in los of anterior segments. This mutation is lethal also. 1. (i) Because the two mutant strains showed complementation (the F1 were able to see), the mutations must have been in separate genes; the simplest explanation is that there are two genes involved. This conclusion is supported by the F2 ratio, which can be derived from a dihybrid ratio. (ii) The 9:7 F2 ratio indicates that we are dealing with a dihybrid ratio (the fractions go in sixteenths). The 9:7 ratio can be derived from the standard 9:3:3:1 ratio if we postulate the following -- Progeny that have at least one dominant allele for each gene show the normal phenotype (normal vision) -- giving 9/16 progeny with normal vision Progeny that lack a dominant allele for any gene show the recessive phenotype (blindness) -- giving 7/16 blind progeny If we call the two genes D and E, the parents were ddEE and DDee; the F1 progeny are DdEe (and can therefore see); the F2 progeny are: D_E_ D_ee ddE_ ddee 9/16 normal vision 3/16 blind 3/16 blind 1/16 blind If the F1 crickets were crossed to homozygous recessive crickets (i.e., DdEe x ddee) the progeny would be DdEe, Ddee, ddEe, and ddee in equal proportions -- i.e., 1/4 of the progeny will be able to see, and 3/4 will be blind. (iii) As with any dihybrid cross, one quarter of the progeny will be true breeding. 2. (i) As with any independently assorting pair of genes,we can look at the the ratios for the two genes independently. With respect to presence or absence of color (gene E), the progeny are 1/2 colored (black or brown) and half yellow. Therefore, with respect to gene E, the parents were Ee and ee. With respect to black vs. brown (gene B), the brown parent has to be bb and the other parent must be Bb (there must be at least one B allele to give black progeny; it cannot be BB, or there would be no brown progeny). [Another way to think about this is that of the non-yellow progeny, half are brown and half black; therefore, the parents must be bb and Bb giving 1:1 B_ and bb progeny.] Thus, the parents must be bbEe (brown) and Bbee (yellow). (ii) Here, with respect to gene E, one quarter of the progeny show the homozygous recessive phenotype-- therefore both parents must be heterozygous (Ee) for gene E. With respect to gene B, again, black and brown progeny are in equal proportions, so the parents must be Bb and bb. Once again, the brown parent must be bbEe; the black parent must be BbEe. 3. This is an example of recessive epistasis. The fact that homozygous B and homozygous O rats could generate AB progeny, and the fact that the F2 progeny fractions are in sixteenths, tells us that this is a dihybrid cross--i.e., a second gene is involved in addition to the IA/IB/IO (hereafter abbreviated A/B/O) gene. The A allele, which manifests itself in the F1, must have been present in homozygous form in the O parent, but masked by the effect of the second gene, thereby giving an O phenotype. (Why homozygous? Because if it were heterozygous, the F1 progeny would show other phenotypes besides AB. Likewise, the A allele could not have been hiding in the B parent, because then the B parent would not be true-breeding.) Furthermore, it must be the recessive allele of the second gene (which we shall call h, the dominant allele being H) that prevents expression of the A/B allele. (Why? Because if masking allele were dominant, F1 progeny would all express the masking phenotype, and would all be O.) The recessive h allele is epistatic to A and B. Thus, the B parent is BBHH and shows the B phenotype; the O parent is AAhh, which does not express the A allele and appears to be O. The F1 progeny are ABHh, and express both A and B alleles. One quarter of the F2 progeny are homozygous recessive (hh); these again appear to be O because the H allele is required for expression of A and B. [A note on the mechanism of blood group expression: Remember that the A and B blood groups represent different forms of polysaccharides added to the surface of blood cells. The molecule to which these polysaccharides are added is the H group on the surface of red blood cells. An OO homozygote makes neither polysaccharide, and is blood type O. But hh homozygotes, even if they are making A or B or both polysaccharides, still have an O blood type because the H moiety is not made; there is nowhere for the A or B polysaccharide to be added to. This form of O bloodtype is often called the "Bombay phenotype" because it was discovered in a patient in Bombay, in 1952.] 4. A selection for Ade+ revertants: plate ade- cells on agar plates lacking adenine. Only Ade+ revertants will be able to grow and form colonies. Therefore, any yeast colonies that form on these plates must have a functional ADE gene. A screen: grow the cells as before and plate them on medium containing adenine. All cells (ade- and Ade+ revertants) will be able to grow, but only the Ade+ revertants will form white colonies. Examine each colony and pick out the white ones to get the Ade+ revertants. 5. Remember that alleles that fail to complement each other (i.e., fail to give the normal phenotype) must be alleles of the same gene. In this example, there are three complementation groups (three genes) -- Gene 1: p1 and r2 Gene 2: p2, r1, and r4 Gene 3: p3 and r3 (Half the table is left blank because filling it would be redundant -- p1 x r3 is the same as r3 x p1, for instance.) 6. (i) D and E will rescue (because if either one is provided, E3 function will no longer be needed); C will accumulate (because there is no E3 to convert C to D). (ii) E will rescue; D will accumulate (iii) D and E will rescue; B will accumulate. 7. (i) Conversion of B to D cannot proceed, so D and F will each rescue this mutation. (ii) Conversion of A to B cannot proceed, so B will rescue. 8. (i) Red pigment cannot be made, so the flowers will be blue. (ii) Red flowers. (iii) Purple flowers (because of complementation-- the F1 will be heterozygous for each gene). (iv) 9/16 purple: 3/16 red: 3/16 blue: 1/16 white. 9. Remember that a mutation in the last gene in the pathway can only be rescued by the final product; a mutation in the next-to-last gene can be rescued by the last two compounds in the pathway, etc. Thus, the pathway is: 10. (i) Neither intermediate (pyrimidine or thiazole) rescues more than one mutation. If these compounds are intermediates in a linear pathway, we'd expect that one of them should rescue more than one mutation. (For instance, in Q. 4, histidinol phosphate rescues both M4 and M1 mutations.) (ii) (Thiazole rescues thi-1, so the problem with thi-1 must be thiazole synthesis; likewise, the problem with thi-2 must be pyrimidine synthesis. thi-3 is rescued only be thiamine, so it must be the last step, the point of convergence of the two branches.) 11. 12. B is required for any color, so it must be required for conversion of white to color -- it is epistatic to A and to C. A appears to be required for conversion of a red intermediate to orange -- absence of A gives a red color instead of orange. C is not required for pigment production, but rather, appears to be needed to prevent pigment production in a portion of the flower, keeping that portion white. B- and C- have opposite phenotypes (no color vs. too much color) so their interaction must be negative. Putting all this together, we can come up with at least two pathways that can each explain the data -- one in which C regulates B directly, and one in which C acts to convert some pigmented areas back to white. Clearly, in either model, there must be some other gene that controls which portion of the flower are gong to express C (to make white) and which repress C (to allow color). 13. (i) CLB is required for DNA synthesis (any strain that lacks CLB function fails to do DNA synthesis). (ii) SIC and CLB mutations have opposite effects, and CLB is epistatic to SIC, so SIC must be an inhibitor of CLB. By the same logic, CLN is an inhibitor of SIC. An alternative pathway-- -- is also possible, but the cln-sic- double mutant phenotype argues against it. This double mutant shows too much DNA synthesis. If CLN is required for CLB function, as this second pathway implies, then the double mutant should show no DNA synthesis. So the data are most consistent with the pathway at the top. 1. (i) The patches probably arose by mitotic recombination. Recombination between the two loci would give lone spots of the recessive phenotype of the more centromere-distal locus, while recombination between the centromere and the two loci would give twin spots. From this logic, we can conclude that the rd locus must be closer to the centromere than the b locus. (An alternative explanation for the lone spots is mitotic nondisjunction, but that wouldn't explain the twin spots.) (ii) Because the twin spots and lone spots occurred in 6:5 ratio, the centromere-rd distance and the rd-b distance must be in the approximate ratio of 6:5: (iii) Lone spots of rd phenotype could arise either from mitotic nondisjunction or by mitotic recombination with double crossover, one crossover between the centromere and rd and one crossover between rd and b. 2. The most distal markers must be y and g. Because yellow and rough are seen in twin spots with each other, but not with mottled or sparse--therefore, the y and r genes must lie on one arm of the chromosome. Likewise, m and g must lie on the other arm of the chromosome. Therefore, the gene order on the chromosome is: y------r----------------------centromere-----m----------g |--7--------------32---------------------6---------12 3. The strain described in lecture had the dominant alleles for yellow and singed in trans. If the dominant alleles are in cis, a crossover between the centromere and the teo genes can give a single spot that has both recessive phenotypes: 4. The map is: If a recombinant sector has phenotype a alone, then the crossover must have occurred between a and all the other genes; if a sector has phenotypes a and b, then b must be between a and all the other genes, etc. 5. The number of generations needed to give the final number of cells can be deduced from the expression: 2n = final # of cells (where n = number of generations) n = log(final # of cells)/log(2) (e.g., if the final # of cells = 16, 2n = 16; n = 4 For a tumor with 109 cells, n = log(109)/log(2) = 29.9 or 30 generations The number of cell divisions = (final # of cells - 1). [Note that the number of cell divisions is not the same as the number of cell generations. In the first generation, there is one cell dividing, so there is one division. In the second generation, there are two cells dividing, so there are two divisions in this generation; in the third generation, there are 4 cells dividing, so there are 4 divisions. So after three generations, there are 8 cells--but it took (1 division in the 1st generation) + (2 divisions in the 2nd generation) + (4 divisions in the 3rd generation) = 7 divisions total.] 6. The tumor was derived from a single cell that had one X chromosome inactivated; since X inactivation is stably propagated through mitosis, all daughters of that cell have the same inactive X. 7. There must have been more than one genetic change in the history of the tissue culture cells. For example, the cells had to go through crisis to become immortalized, a process which probably involved some genetic change. 8. A mutation that results in the erbB protein behaving as though it had bound to a growth factor even in the absence of the growth factor could cause the cell to begin dividing in the absence of growth factor. If other regulatory mechanisms are also abrogated (by unrelated events), the cell or its descendants could become malignant. 9. (a) For the mutant allele (a*) to cause inappropriate cell proliferation, it must be resistant to inhibition by Protein B. Therefore, the mutant protein made by this allele will be able to promote cell proliferation regardless of whether the other allele of gene A is wildtype or mutant - so a* is a dominant, gain-of-function mutation. (b) Here, the mutant allele must be promoting cell proliferation by failing to block Protein A. However, even if this allele fails to make functional protein B, the other allele (if it is wildtype) can still make functional Protein B and block Protein A. Therefore, b* must be a recessive, loss-of-function mutation. (c) 2 x 10-5 (because there are two copies of allele a+, and mutation of either one of them would be sufficient to cause inappropriate cell proliferation). 1. *Corrected* There must have been two crossovers -- one between B and D and one between F and G, as shown: 2. (a) The result is unexpected in that we are seeing only the parental (non-crossover) products--we should see some recombinants. One of the parents must have an inversion such that the recombination products are inviable. (b) The fact that chromosomal material gets stretched between the spindle poles and breaks suggests that a dicentric chromosome must be formed -- indicating that the inversion must be a paracentric inversion: Note that the gene order shown is arbitrary; the question does not give information on the correct gene order or even which parent had the inversion. All we can say is that there must have been an odd number of crossovers in the inversion loop. 3. Deleterious effects (loss or duplication of genes leading to reduction in fertility) will be seen when there is an odd number of crossovers within the inversion loop. Here, the b-d-e-f segment is inverted, so that portion will form an inversion loop during prophase of meiosis I; y-a and g-h will remain outside the loop. (a) There are two crossovers in the inversion loop, so the products will all be viable -- no reduction in fertility. (b) The crossovers are both outside the inversion loop, so again, there will be no reduction in fertility. (c) Here, there is only one crossover within the inversion loop, the other crossover occurring outside the loop. This DCO event will produce gametes with gene deletions, and will be deleterious, leading to decreased fertility. 4. The male progeny are expected to receive Xsc from the mother and therefore have scute bristles. This male must have received X+ from his father (the only source of the dominant allele in the cross). Presumably, the X-ray treatment caused a translocation of the end of the X chromosome carrying sc+ such that the sc+ allele was transmitted to that son. What about the second cross? If the translocation had been to an autosome, the exceptional son would have had some wildtype daughters. The fact that the wildtype phenotype segregates exclusively with male progeny indicates that the translocation must have been to the Y chromosome. NOTE: Only the gametes producing the aberrant (unexpected) offspring are shown for cross 1. Most of the gametes were normal, producing the expected offspring. 5. (a) One way of distinguishing between the hypotheses is to obtain DNA from affected individuals and do a Southern blot experiment on the DNA, cutting it with EcoRI and probing the blot with the 2.5 kb fragment. As a control, this sample would be compared with DNA from an unaffected individual. The control DNA should give a 2.5 kb fragment on the Southern blot. If the translocation does indeed remove the left end of the gene and replace it with an unrelated fragment from a different chromosome, it is highly unlikely (but not impossible) that this unrelated sequence will also have an EcoRI site at exactly the same place -- so the DNA from the affected person should show a fragment of a different size than the normal 2.5 kb. (It's not possible from the available information to make a prediction about exactly what size to expect.) In addition, the left end, which got moved to a different chromosome, will be part of a different EcoRI fragment in its new location. So the affected individual, instead of producing one 2.5 kb fragment, will probably produce two fragments of different sizes. If the translocation does not break the growth factor gene (the minority view), the 2.5 kb fragment should remain intact in all samples. (b) The same 2.5 kb probe could be used to do a FISH experiment, again comparing cells from affected vs. unaffected individuals. In non-patients, the probe should hybridize only to one chromosome (but to both homologs of that chromosome) -- e.g., if the growth factor gene is on chromosome 9, we should see hybridization to the two homologs of chromosome 9. In patients, a portion of the growth factor gene should be translocated to a different chromosome (according to the prevailing hypothesis). Therefore, a FISH experiment should detect hybridization not only to chromosome 9, but also to the other chromosome involved in the translocation. If the minority view is correct, only one chromosome type should be detected in patients as well as non-patients. (c) The Southern blot approach only detects fragment sizes, not chromosomal locations. As noted in (a), there is a remote possibility that the translocation will produce exactly the same EcoRI fragment sizes as the normal chromosome. The result would then support the minorityview even even if the gene is indeed split by the translocation. The FISH approach detects the chromosomal location of the sequence being probed, and is not subject to this limitation. Therefore, if done correctly (with a suitably large sample size -- number of cells examined) the FISH results might be more believable. In reality, one would probably do both tests. Technically, the Southern blot is a lot easier. 6. (a) (b) There is just one way to get XYY progeny from normal XX and XY parents (if nondisjunction happens only in one parent) -- male nondisjunction in meiosis II to produce YY sperm (marked with an arrow in the diagram). 7. (a) The calico pattern is the result of X-inactivation. Male mammals aren't expected to show X chromosome inactivation, so this result is unexpected. The calico males are probably XXY cats, resulting from sex chromosome nondisjunction in one of the parents. (b) In the first litter, the mom was XrXr and the dad was XRY. The mom could transmit only Xr; the XR allele must have come from the dad along with Y, so nondisjunction must have occurred in the dad. In the second litter, the mom was XRXr and the dad was XRY. The male calico kitten could have received XRXr from the mom (ND in mom) or XRY from the dad (ND in dad)-- it's not possible to distinguish between these possibilities. 8. (a) It should worse for females than for males. Deletion of XIC on a chromosome prevents that X chromosome from being counted and inactivated. In males, there shouldn't be X chromosome inactivation anyway, so the deletion shouldn't matter. In female progeny, the consequence will be that X chromosomes will be undercounted. (An X chromosome lacking the XIC is not "seen" as an X.) Therefore, neither X chromosome will get inactivated, which has deleterious consequences. (b) The X with intact Xist will get inactivated -- Xist works in cis, so only the chromosome producing it will get inactivated. (c) The hypothesis is that this protein is produced in limiting quantities so that only one X can be protected from inactivation. If the amount of protein in the cell is doubled by the mutation, there should be enough protein to protect both X chromosomes -- so neither X should be inactivated. (d) This is an example of a dominant, "gain-of-function" mutation. The mutated allele of this gene will produce excess protein, so it doesn't matter if the normal allele is present also -- the effect of the excess protein will be seen anyway. 1-1998 (i) The 38-year old has a higher risk of a Down syndrome baby, because the probability of nondisjunction during meiosis increases with age in human females. (ii) The family history of Down syndrome suggests that this may be a case of translocation Down syndrome -- in which case, the younger woman (belonging to that family) has a higher risk of a Down syndrome baby (because the chance of nondisjunction in a 38-year old woman is about 1 in 100 -- see pg 69 of the lecture notes -- while the chance of translocation Down carrier having a Down baby is 1 in 4). 2-1998 (i) The mother must have been heterozygous G/g, while the father was hemizygous normal G/(Y). Colorblind Turner syndrome (XO) females must have resulted from fertilization of a g egg by a sperm lacking a sex chromosome; nondisjunction could have occurred at meiosis I or meiosis II in the father. Colorblind Klinefelter (XXY) males must have resulted from fertilization of gg eggs by normal Y-bearing sperm; nondisjunction occurred in meiosis II in the mother. (ii) Identical (monozygotic) twins arise when an early embryo splits so that each portion develops into an individual fetus. In this instance, the zygote must have been normal. The split occurred at the two-cell stage; one of the resulting cells divided normally, giving the normal twin, while the other cell had a mitotic nondisjunction, giving the Down syndrome twin. 3-1998 (i) The F1 females should be heterozygous at all loci. We would normally expect to see recombination in each interval, giving up to 26 = 64 different progeny phenotypes (in a ratio that would depend on the map distances). The presence of only four progeny phenotypes is not normally to be expected. (One could postulate that pairs of loci are very tightly linked, but that does not explain the lack of recombinants between the ends of the group.) (ii) A deletion could be ruled out because half the F2 males would inherit an X chromosome lacking genes, and would probably fail to develop. Translocations are also unlikely to give the observed results, because the phenotype is a reduction in observed recombinant progeny (translocations cause semisterility, but there is no reason not to expect recombinants (draw it out and confirm it for yourself). (iii) The lack of recombination between A and B and between F and G suggests that the entire portion between A and G (i.e., B through F) is inverted. (iv) A variety of molecular tests is possible. For example, if the inversion is as predicted, one can set up Southern blots, using probes for the presumptive junction regions. In the example shown here, Probes 1 and 2 will hybridize to different restriction fragments (of different sizes) if the chromosome is normal. In contrast, if the inversion is as shown, probes 1 and 2 will both hybridize to the same fragment. [Restriction enzyme sites are depicted as vertical bars. Knowing the restriction map for the whole chromosome, we can pick a restriction enzyme that has suitably located sites as shown.] (v) Single crossovers are not expected to give viable recombinant gametes. However, rare double crossovers can be viable. In this instance, a double crossover -- one crossover between B and D loci and one between E and F -- would give the observed result. 4-1998 If the two T-allele bearing homologs are called T1 and T2, and the two t-allele bearing homologs are t1 and t2, there are three possible sets of pairings, giving the gametes shown: Pairing Gamete genotypes T1T2 and t1t2 (i.e., T1 homolog paired to T2 homolog, etc.) T1t1, T2t2, T1t2, T2t1 T1t1 and T2t2 T1T2, t1t2, T1t2, T2t1 T1t2 and T2t1 T1T2, t1t2, T1t1, T2t2 The gamete genotypes are TT, Tt and tt in 1:4:1 ratio, or 5:1 ratio of T_:tt. If mated to tttt plants (whose gametes will all be tt), the progeny are expected to be T___:tttt (i.e., tall and short) in 5:1 ratio. 5-1998 (i) Because the plant height and color genes are on separate chromosomes, they should assort independently; the cross should give TD, Td, tD, and td progeny in 1:1:1:1 ratio. Instead, only the parental phenotypes (TD and td) are seen. (ii) The absence of the non-parental types and the semi-sterility suggest that the explanation may be a translocation. One possible configuration is shown: The "adjacent" pattern of segregation would give Tt and Dd gametes, while the "alternate" pattern would give TD and td. The Tt and Dd gametes would be inviable, so the only viable progeny would have TD and td phenotypes -- the parental types. Note that there is more than one configuration that would fit the results. For example, the t and d alleles need not be on the translocated segment. 1. The match to the suspect in Case 1 is more meaningful -- the alleles that are matched are much less frequent in the population, so a chance match (i.e., the suspect and the crime scene DNA matching just due to chance) is improbable. In Case 2, the alleles are more frequent, so a chance match is more probable. One would therefore feel more confident finding suspect 1 to be guilty than finding suspect 2 guilty. The math Case 1: probability of a chance match = (0.01)(0.02)(0.003)(0.01)(0.07)(0.04)(0.13)(0.08)(0.04)(0.05)32 = 1.1 x 10-14 -- i.e., the probability of getting this combination of alleles just by chance is about 1 in 100 trillion. Case 2: probability of a chance match = (0.2)(0.4)(0.15)(0.35)(0.4)(0.3)(0.3)(0.6)(0.2)(0.3)32 = 1.7 x 10-4 -- i.e., a little over 1 in 6000. (The factor of 32 comes in because there are two ways of getting each heterozygous combination, and 5 loci we're looking at = 25 = 32.) We'll see more of this factoring-in-2 business when we get to population genetics.) 2. (a) Note: Your answer does not need to be this long-winded! The strategy here is to look at the dominant trait and ask: does any one allele of the polymorphic trait preferentially segregate with the dominant trait? (And conversely, does an allele preferentially segregate with the recessive allele? This question is harder to address, because in this pedigree there are seven sources of the recessive allele -- two copies from I-1, one copy from I-2, and two each from II-1 and II-5 -- so it's harder to track the recessive allele. In contrast, there's only one source of the dominant trait -- I-2 -- so it's a lot easier to track.) The source of the dominant trait in this pedigree is I-1. We know that he is heterozygous for the dominant trait (because he has an unaffected daughter, II-3). So if D = dominant and d = recessive, he is Dd. He has alleles 13 and 20 at PS1, and 21 and 27 at PS2. So we can ask if one of these four alleles segregates with the dominant trait (i.e., do people who show the dominant phenotype -- and therefore, inherited allele D -- also get one of those four alleles preferentially?) Let's look at PS1 first. There are 11 affected (Dd) individuals not counting I-2. Of these, three have inherited allele 13 (II-2, III-3, III-5) and four have inherited allele 20 (II-4, III-10, III-12, III-14). The remainder have inherited neither 13 nor 20 (these individuals have inherited alleles from people marrying into the family). So allele D of I-2 has co-segregated about half the time with allele 13 of PS1 and about half the time with allele 20. Therefore, PS1 does not appear to be linked to D/d -- the two loci appear to be segregating independently of each other. Now let's look at PS2. Here, the alleles in individual I-2 are 21 and 27. Of the eleven Dd progeny, ten also have allele 21; only one has allele 27. Additionally, of the six dd progeny, only one has allele 21; the remainder have other alleles. Therefore, it appears that in this pedigree, allele 21 is preferentially found with allele D; the two loci (D/d and PS2) are probably linked. In this particular case, I-1 must have allele D and allele 21 of PS2 on the same homologue (in cis, or in "attraction phase"). (b) If we assume that the scenario we have described above is true -- i.e., D/d and PS2 are linked, with alleles D and 21 in cis -- then we can look for individuals who have allele D but not allele 21, or conversely, lack allele D but have allele 21, as evidence of recombination. Two individuals fit these criteria: III-6 is affected (Dd) but does not have allele 21, and individual III-9 is unaffected dd, but has allele 21. 3. There are 64 possible triplets and three of these (UAA, UAG, UGA) are stop codons. Therefore, in a random DNA sequence, the chance of encountering a stop codon in any particular reading frame = 3/64, or about 1 out of every 21 codons. So on average, a ribosome will encounter a stop codon about 21 codons following a frame shift; the peptide will be 20 amio acids beyond the point of the frame shift. 4. The fraction of control (sugar-water-treated) crosses that lack wildtype male progeny is the background rate of spontaneous mutagenesis. This rate is = 13/(6255 + 13) = 0.002/generation We can now compare the rate of mutation in the other groups to see if any treatment causes an increase above this background rate. Thus -- Food color #1: 76/(4821 + 76) = 0.016/generation -- this rate is higher than the background rate, so Food color #1 is mutagenic. Food color #2: 18/(9361 + 18) = 0.002/generation -- this rate is no higher than the background rate, so Food color #2 is not mutagenic. Food color #3: 91/(5382 + 91) = 0.017/generation -- this rate is higher than the background rate, so Food color #3 is mutagenic. 5. Ultraviolet light is mutagenic because DNA can absorb photons in UV wavelengths and thereby undergo chemical reactions that it otherwise would not. Therefore, the wavelengths of UV light that are most mutagenic should correspond to those wavelengths that are best absorbed by DNA -- so the efficiency of mutagenesis should correspond to the absorption of UV by DNA (the solid red line in the graph). 6. Remember that normal diploid cells have two copies of the gene for Enzyme E (Gene E) and two copies of the gene for Enzyme Z (Gene Z). So, assuming that the amount of enzyme in the cell scales linearly with the gene copy number, each copy of Gene E contributes 30 units of enzyme E activity (so a diploid produces 60 units of Enzyme E), and each copy of Gene Z contributes 50 units of Enzyme Z activity. So a cell line that has a duplication of a gene should produce three copies' worth of enzyme. For Enzyme E, the duplication should result in cells that produce ~90 units, and for Enzyme Z, a duplication of the gene should result in ~150 units. To find the location of Gene E, we look for cell lines that produce ~90 units of Enzyme E, and ask, what's common between these duplications. We see from the table that cell lines 1, 5, and 6 all produce ~90 units of Enzyme E (while the other cell lines produce the normal ~60 units). So these three cell lines must have duplications of Gene E. The band that is common to these three duplicaitons is band 2 -- that must be the location of Gene E. For Enzyme Z, cell lines 2 through 6 all produce ~150 units instead of the standard 100 units. The

band common to the duplications in these lines is band 5; Gene Z must be located there. 7. (a) We expect the progeny to show the dominant phenotypes. In progeny showing the recessive traits, the recessive alleles must have been uncovered by deletions in gametes produced by the X-irradiated male. (b) Although we could postulate multiple deletions in each progeny class, the most parsimonious explanation is that each progeny class has a single deletion that uncovers recessive alleles at multiple adjacent loci. So if two recessive traits are uncovered, the genes for those two traits must be next to each other on the chromosome. Using that logic -- Strain #1 uncovers a and c, so gene a and gene c must be neighbors Likewise, a and b must be neighbors; a must be between b and c (the order so far is b-a-c) f is next to c, so the order is b-a-c-f de is next to f (but the order of d and e is not known yet) and from strain #6, e is next to f, so the completed gene order is b-a-c-f-e-d Modified from 1998 (a) Sectors of different sizes will arise depending on when during growth of the colony the mutation event occurred--the earlier the mutation, the larger the sector. Half-sectored colonies reflect mutations that occurred in the first division of the cell that eventually formed the colony (e.g., if there was an unrepaired mismatch in Ade+ DNA prior to the first round of DNA synthesis, replication would lead to one normal daughter chromosome, which would result in the white sector; and one mutated daughter chromosome, which would give rise to the red sector). (b) The problem in measuring mutation frequency is estimating how many cell divisions have occurred. However, we do know how many cells underwent mutations to give sectors in the first division--it is the number of half-sectored colonies. We also know how many "first divisions" occurred--it is equal to the number of colonies on the plate. Therefore, the frequency of mutation = frequency of mutation in the first division = (# of half sectored colonies)/(total # of colonies). 1. With respect to the disease, the boy must be homozygous recessive (because achondroplasia is dominant). If A = achondroplasia and a = unaffected, the boy is aa. With respect to the polymorphic locus, one allele has 12 repeats of CA, while the other has 7 repeats--so his genotype for the polymorphic site is 7,12. (Or 12,7.) Therefore, his overall genotype for these two loci is aa 7,12. 2. (a) Sample B DNA must be circular -- one cut in a circular DNA molecule just converts it from circular to linear without dividing it into smaller fragments. Sample A DNA is either linear (with a single cut site for Pst I, so that one cut breaks the linear molecule into two), or circular with two cut sites for (the first cut linearizes the circle; the second cut breaks the linear molecule into two). (b) The conclusion for Sample A does not change -- since it is cut into two fragments by Pst I, it must have at least one cut site. For Sample B, however, if it remains as a single molecule after Pst I treatment -- so either it is a circle with a single cut site, as we concluded in (a), or it lacks Pst I cut sites altogether, in which case we do not have enough information to decide whether it is circular or linear. 3. (a) Note that digests (ii) and (iii) give multiple fragments of the same size -- depicted here as thick bands. Note that the various fragment sizes should always add up to the full length (20 kb in this example). In real life, if you saw two bands that didn't add up to the full size (e.g., lane ii -- 7 kb band + 3 kb band = 10 kb instead of 20 kb), that would clue you in that there might be multiple fragments of the same size. (b) The probe will hybridize only to those fragments with which it overlaps. Again, some bands contain two distinct fragments of the same size, only one of which (in this case) should be hybridizing to the probe. 4. (a) The size of the full genome should be the sum of the sizes of the individual fragments for any given digest -- e.g., in "Ava I alone" the fragments are 12 kb and 48 kb, so the total size is 60 kb. You should get the same answer from each digest. (b) Each enzyme by itself gives two fragments. Therefore, each enzyme must have a single cut site in the bacteriophage genome, such that each enzyme cuts the DNA into two. Ava I must be cutting 12 kb from one end (i.e., 48 kb from the other end); Bam HI cuts 10 kb from one end, and Cla I cuts 18 kb from one end. The question is, which end are we measuring from -- we know that Ava I cuts 12 kb from one end, but Bam HI might be cutting 10 kb from the other end. To get that information we look at the double digests. Let's look at Ava I + Bam HI. We know that Ava I by itself is going to generate a 12 kb fragment and a 48 kb fragment. We now see in this double digest that Bam HI leaves the 48 kb fragment untouched -- we're still seeing a 48 kb fragment. In contrast, the 12 kb fragment released by Ava I has been cut by Bam HI to a 10 kb fragment and a 2 kb fragment. Therefore, Bam HI site must be within the 12 kb Ava I fragment. The map we have so far is: We can do a similar analysis for Cla I. In the Ava I + Cla I double digest, we see that Cla I does not cut within the 12 kb AvaI fragment (because if it had cut within the 12 kb fragment we'd be seeing smaller-than-12 kb fragments, which we don't). However, Cla I does cut within the 48 kb Ava I fragment to release a 30 kb fragment and an 18 kb fragment. We already know that Cla I cuts 18 kb from one end of the genomic DNA molecule -- therefore there is only one way to place the Cla I site on the map, as shown: The map predicts that a Bam HI + Cla I double digest should give 10 kb, 32 kb, and 18 kb fragments -- which according to information we are given is true. 5. (a) The primers are : 5'-TGCTCTGGAT-3' and 5'-TCCGAGAGCC-3', which correspond to the yellow, boxed segments (immediately flankng the greyed segment) below: (b) The full length will be 46 bp (10 bp for each primer + 26 bp in the middle). Note added 10/26/99: The way the question is worded, it is actually possible to amplify an even smaller fragment, by choosing primers within the grayed segment as shown below: In this case, only the grayed segment would be amplified, giving a product length of 26 bp. 6. (a) Someone who is homozygous normal will have two identical copies of the allele that has all four Xba I sites -- i.e., digestion of their DNA with Xba I and hybridization with the indicated probe should detect three fragments, of sizes 3 kb, 5 kb, and 7 kb. In contrast, a carrier (a heterozygote with one normal and one disease allele) will have one allele that has 4 Xba I sites and one allele that lacks one or two of the middle Xba I sites (see table below). Their DNA, when cut and probed similarly, will also pick up the same three fragments (3 kb, 5 kb, 7 kb) because of the one normal allele. However, the other allele will give different products,which will be seen in addition to the normal digestion products (asterisks indicate absence of Xba I sites): Genotype Digestion products detected 3 kb, 5 kb, 7 kb, and 8 kb 3 kb, 5 kb, 7 kb, and 10 kb 3 kb, 5 kb, 7 kb, and 15 kb (b) As seen above, four different alleles are possible -- the normal allele (with all 4 Xba I sites) plus the three alleles lacking one or both Xba I sites. (c) There are 10 possible genotypes -- 4 homozygous and 6 heterozygous (see Week 1 Q. 10 for an explanation). 7. (a) The polymorphic site alleles are co-dominant -- both forms are detected when one tests the allele composition at that site. (b) If the two loci are unlinked, gametes of the different possible genotypes are equally probable; the eight possible progeny genotypes are equally possible, as shown below: (c) We are not given phase information here -- i.e., we don't know whether allele configuration in the father is {D 8 & d 18}, or {D 18 & d 8}. (Does the mother's allele configuration matter in this question?) Different outcomes will be seen depending on the phase, as shown below. The gametes produced by the mother will be d, 7 and d, 15 in equal proportions, as in (b). Phase (allele configuration) in father: {D 8 & d 18} {D 18 & d 8} Gamete genotypes (frequencies): D, 8 (0.4) d, 18 (0.4) D, 18 (0.1) d, 8 (0.1) Gamete genotypes (frequencies): D, 18 (0.4) d, 8 (0.4) D, 8 (0.1) d, 18 (0.1) 8. Because we are assuming complete linkage,we can simply look at the genotype at the polymorphic locus and assign the disease phenotype accordingly -- homozygous {30, 30} = affected; heterozygous {30, 42} = unaffected, carrier: 9. (from 1998) The Lod score graph tells us that the pedigree data favor a map distance of 5 cM between Gene 1 and PS1; a map distance of 15 cM between Gene 1 and PS2; a map distance of 10 cM between Gene 1 and PS3, etc. A map that is consistent with these interpretations is: Note: The answer to Q. 5 has been corrected 10/19/99. 1. For every crossover between the two loci, two of the four products of meiosis will be recombinant. Therefore, if 8% of the meioses have a crossover in that interval, 4% of the products will be recombinant -- so the map distance is 4 cM. (Go through the worksheet on p.40 of the lecture notes if you're still confused.) 2. AaBb x aabb The products are in 1:1:1:1 ratio -- the loci appear to be assorting independently, so we cannot assign linkage, and cannot determine the parental configuration. AaDd x aadd Here, AD and ad phenotype progeny greatly outnumber Ad and aD -- so AD and ad must be the parental allelic configurations (A and D are linked in cis). The recombinant types (Ad and aD) account for 8 of 200 = 4% of the progeny; the map distance between A/a and D/d = 4 cM. AaFf x aaff Af and aF phenotype progeny greatly outnumber AF and af -- so Af and aF must be the parental allelic configurations (A and F are linked in trans). The recombinant types (AF and af) account for 36 of 300 = 12% of the progeny; the map distance between A/a and F/f = 12 cM. BbEe x bbee Be and bE phenotype progeny greatly outnumber BE and be -- so Be and bE must be the parental allelic configurations (B and E are linked in trans). The recombinant types (BE and be) account for 10 of 210 = 4.8% of the progeny; the map distance between B/b and E/e = 4.8 cM. DdFf x ddff Df and dF phenotype progeny greatly outnumber DF and df -- so Df and dF must be the parental allelic configurations (D and F are linked in trans). The recombinant types (DF and df) account for 20 of 250 = 8% of the progeny; the map distance between D/d and F/f = 8 cM. Putting this information together -- A/a, D/d and F/f are in the same linkage group; B/b and E/e are in a separate linkage group. The linkage relationships can be depicted as: A D F |--------|----------------| 4 cM 8 cM B E |---------| 4.8 cM 3. The parental genotypes are TTFF x ttff to give TtFf. Therefore, the parental genotypes for gametes made by the F1 plants are TF and tf. If the two loci are unlinked, we expect the four progeny phenotypes (TF, Tf, tF, and tf) in equal proportions. Because there are 1000 proteny total, we expect 250 of each phenotype if the loci are unlinked. If the two loci are linked at at map distance of 44 cM, we expect 44% of the gametes to be recombinant -- i.e., 44% of the progeny should show the recombinant (non-parental) phenotype. As shown above, the parental types are TF and tf, so we expect 44% of the progeny to add up to Tf and tF, or 22% each. The parental types then should be 56% of the progeny = 28% each. So for 1000 progeny, we expect 280 each of TF and tf, and 220 each of Tf and tF. Clearly, the observed progeny numbers don't match either scenario. So let's do a chi-square analysis on the two data sets, for the two sets of expectations, and see if we can find statistical evidence against either model. Scenario 1 -- the loci are unlinked Phenotype Expected (E) Observed (O) (E-O)2/E Tart, fibrous 250 281 3.844 Tart, smooth 250 219 3.844 Sweet, fibrous 250 251 0.004 Sweet, smooth 250 249 0.004 Chi-square value = 7.70 df = 3 The corresponding P value is just over 0.05 -- just above the standard cutoff for rejecting the null hypothesis (that the deviation from expected is just due to chance). Scenario 2 -- the loci are linked at 44 cM Phenotype Expected (E) Observed (O) (E-O)2/E Tart, fibrous 280 281 0.004 Tart, smooth 220 219 0.005 Sweet, fibrous 220 251 4.368 Sweet, smooth 280 249 3.432 Chi-square value = 7.81 df = 3 Again, the corresponding P value is just over 0.05. What does this mean for deciding between the two modes of inheritance? The statistical analysis tells that the data are consistent (just barely) with either model -- so we cannot decide between the two models based on this statistical test. At least two approaches are possible to settle the question. One is simply to collect more data (repeat the crosses, count a lot more progeny) and repeat the statistical analysis in the hopes that one hypothesis or the other can be rejected with more data. However, if T/t and F/f are linked, it should be possible to find genes in the interval between them that are linked to both. That way, we'd be working at smaller map distances, and thereby have a better shot at establishing linkage. 4. The flaw is that the F1 progeny, although heterozygous for sneezy and jumpy, are homozygous for itchy. So while recombination between sneezy and jumpy can be detected, there's no way to detect recombination involving itchy. ... no change in genotype (ijs and i++ giving i++ and ijs) Note: i = itchy, j = jumpy, s = scratchy; only one chromatid of each homolog is shown He should be using a fully heterozygous (ijs/+++ in any cis/trans configuration) and a homozygous recessive (ijs/ijs) for his mapping cross. Assuming that we are starting with the dominant alleles in cis in the heterozygote (i.e., +++/ijs), then parental, double-crossover, and single-crossover products can be predicted as follows: Gamete type Gamete genotype (= progeny phenotype) Predicted number of progeny DCO i + s and + j + = (0.18)(0.12)(1000) = 22 total; 11 of each SCO in i-j interval + j s and i + + = (total recombinants in this interval) - DCO = (0.18)(1000) - 22 = 180 - 22 = 158; 79 of each SCO in j-s interval + + s and i j + = (total recombinants in this interval) - DCO = (0.12)(1000) - 22 = 120 - 22 = 98; 49 of each NCO (parental) + + + and i j s = total - (all recombinants) = 1000 - (22 + 158 + 98) = 722; 361 of each 5. Finding the correct gene order H = Hairy, P = purple, T = Thorny; and lower case denotes the recessive phenotypes. The parental non-crossover (NCO) allele combinations are Hpt and hPT (these being the most abundant progeny phenotypes), while the double-crossover (DCO) classes are HPT and hpt. To find the correct gene order, we start with the known NCO types, and see if a double crossover yields the known DCO types. If it doesn't, the order must be wrong; we try a different gene order (the critical information is the gene in the middle). Trial and error (trying each of the three genes in the middle) establishes that H must be the middle gene: Products of single crossover (SCO) in P-H interval are phT and PHt. Products of SCO in H-T interval are pHT and Pht. Note the correction! (SCO classes were reversed. --10/19/99) Now we can start calculating map distances: P-H map distance = percent recombinants in this interval = (SCO in P-H) + DCO) as percent of total progeny = (150 + 132 + 18)/2500 = 300/2500 = 0.12, or 12 cM. H-T map distance = percent recombinants in this interval = (101 + 81 + 18)/2500 = 200/2500 = 0.08, or 8 cM. The completed map is: P/p H/h T/t |------------|--------| 12 cM 8 cM Coefficient of coincidence = (observed DCO/(Expected DCO) Observed DCO = 18 Expected DCO = (0.12)(0.08)(2500) = 24 Coefficient of coincidence = 18/24 = 0.75. 6. The important thing to remember is that in order to map the genes, we need to be able to detect recombination, and that in order to detect recombination, one of the parents has to be fully heterozygous. Here, the genes are on the X chromosome -- so that parent by default has to be the female (the male only has one X -- no recombination there). There are a couple of ways of setting this up. One option is to make the female heterozygous, and to have recessive alleles on the male's X chromosome. Then the males and females would consist. To generate heterozygous females, we could cross homozygous dominant females (+++/+++) with recessive males (abc/Y); the females in the resulting progeny would be heterozygotes. When these females are crossed with abc/Y males, the progeny (males and females) would show the non-recombinant phenotypes (abc and +++) as well as the 6 recombinant types: a++ and +bc, ab+ and ++c, a+c and +b+. A different option is to cross the heterozygous females with males showing the dominant phenotypes (+++/Y). Then the female progeny would all show the dominant phenotypes, and would be ignored; the male progeny would get the single X from the female, and show the same parental and recombinant phenotypes listed above. For an example -- see Question 1998-2 in Questions from yesteryear. 7. The only human chromosome common to all the cell lines making Enzyme Q is chromosome 8 -- so that must be the chromosome carrying the gene for Enzyme Q. 8. Enzyme G The chromosomes common to cell lines making this protein are: 2 and 9 Cell line C has chromosome 2 but does not make the protein. Therefore, the gene for Enzyme G must be on chromosome 9. Enzyme AD The chromosomes common to cell lines making this protein are: 5 and 14 Cell line E has chromosome 5 but does not make the protein. Therefore, the gene for Enzyme AD must be on chromosome 14. Enzyme H The chromosomes common to cell lines making this protein are: 2 and 9 Cell line C has chromosome 2 but does not make the protein. Therefore, the gene for Enzyme H must be also be on chromosome 9. 1998-1 (a) The parents are XoDXoD and XOdY. The cross is outlined below; the children are expected to be unaffected females and ocular albinism males in 1:1 ratio. (b) Here, we know that the woman is heterozygous for both traits--but we don't know whether the dominant alleles are in cis (i.e., the dominant O allele and the dominant D allele on the same homolog) or in trans (on different homologs). The man in the cross has the dominant alleles for both loci, so his daughters will all be phenotypically normal. The sons' phenotypes, however, will depend on which X chromosome they inherit from the woman, and on whether she has the dominant alleles in cis or in trans. 1998-2 You just have to realize that because the ratio of phenotypes is very different in females vs. males, the mode of inheritance must be sex-linked--specifically, these are X-linked genes. Other than that, the procedure is the same as above--you use just the male progeny to follow the recombination that occurred in the female parent. (If you are confused--DRAW THE CROSS! You know that the genes are on the X chromosome; you know the parental genotypes.) The parental types (most abundant in the male progeny) are s+ sn+ fu and s sn fu+. The DCO products are s+ sn fu+ and s sn+ fu. Therefore, the s locus must be in the middle; the parental types can be re-written as sn+ s+ fu and sn s fu+. A single crossover between sn and s would give sn+ s fu+ and sn s+ fu; a single crossover between s and fu would give sn+ s+ fu+ and sn s fu. Now we can calculate the percent recombinant types for each interval: # of crossovers in sn-s interval = SCO (in sn-s) + DCO = (99 +91) + (21 + 17) = 228 Percent recombination in B-A interval = (228/1000)*100 = 22.8 # of crossovers in s-fu interval = SCO (in s-fu) + DCO = (69 + 75) + (21 + 17) = 182 Percent recombination in A-C interval = (182/1000)*100 = 18.2 (a) Genotype of female parent = sn+ s+ fu / sn s fu+ (This notation-- a set of alleles, then a slash "/" then another set of alleles-- is standard notation to show that the first set of alleles is on one homolog and the second set of alleles following the slash is on the second homolog.) Genotype of male parent = sn+ s+ fu+/Y (b) Map of the region: sn------22.8 cM-------s------18.2 cM----fu |--------------------|-----------------| (c) Predicted # of DCo products = (0.228)(0.182)1000 = 41 Observed # of DCO products = 38 Coefficient of coincidence = 38/41 = 0.927 Interference = (1 - 0.927) = 0.073. This problem is easily solved. Human chromosomes present in cell lines that do not have the insulin sequence can be eliminated from our list of possible candidates. Therefore, any chromosome that is found in cell lines D, E, or F can simply be crossed out from the list of possibilities (eliminated candidates shown below as colored-out boxes). Cell line Human insulin sequence present? Human chromosomes that are present in the cell line A Yes 6 7 10 11 14 17 18 20 21 X B Yes 3 5 11 14 15 17 18 21 C Yes 4 5 10 11 12 17 18 21 D No 8 10 12 15 17 21 X E No 2 5 6 10 12 18 20 21 X F No 17 18 20 Of the remaining candidate chromosomes, the only one that is present in cell lines A, B, and C is chromosome 11. Therefore, the insulin gene must be located on chromosome 11. 1997-4 (a) I-1 is unaffected, so he must be XGHY. His daughter inherits his X chromosome, so one of her X chromosomes must be XGH. However, she has a colorblind, hemophilic son, so her other X chromosome must have both recessive alleles. Therefore, II-1 is XGHXgh. Her husband (II-2) and son (III-1) are both colorblind but not hemophilic, so they both must be XgHY. III-2 has both disorders; he is XghY. III-3, being colorblind, must be homozygous recessive for the color vision locus. One chromosome is XgH (the one she got from her father, II-2); the chromosome she got from her mother (II-1) is either Xgh (if there was no recombination between the two X's in her mother) or XgH (if there was recombination). Therefore, III-3 is either XgHXgH or XgHXgh. (b) III-1 – his X chromosome, which he got from his mother, is XgH, while his mother is XGHXgh. (c) III-3 inherited XgH from her father; she inherited either Xgh or XgH from her mother. The two genes are 3 map units apart, so we expect 3% of the gametes from II-1 to be recombinant. Considering the phenotype of III-3, the only possible recombinant gamete is XgH; the probability of that is 0.03. Therefore, the probability that she is H/H is 0.03. Likewise, the probability that she is H/h is 0.97. Because she does not show the recessive hh phenotype, there is no chance (p=0) that she is h/h. 1. (a) Haploid number N = 9; so 2N = 18. At metaphase, the chromosome should each have two sister chromatids, so the total number of chromatids = 18 x 2 = 36. (b) As in mitosis, there should be two chromatids per chromosome, i.e., 36 chromatids (but the arrangement of chromosomes will be different from mitosis). (c) In Anaphase I of meiosis, the homologs separate -- so the resulting daughter cells have only a haploid set of chromosomes each. Therefore, at meiotic Metaphase II, the number of chromatids = 2 x haploid chromosome number (N) = 18. 2. The diploid form, having two sets of chromsomes, can undergo meiosis. The haploid form has only one set to begin with, so it cannot undergo a reductional division. 3. (a) The homologs have separated -- so it must be meiosis. (b) Sister chromatids have separated, and there is one copy of each homolog -- so it must be a mitotic division. 4. (a) There are three segregating traits here: G/g, A/a, and X/Y. Therefore, there are 23 = 8 possible gamete genotypes: GAX GaX gAX gaX GAY GaY gAY gaY (b) Since galactosemia and albinism are both recessive traits, the sperm will have to carry the recessive allele for each trait. Furthermore, since we are looking for a son, the sperm will have to be a Y-chromosome bearing one. Therefore, the genotype of the sperm has to be gaY. (c) We know that the final genotype has to be gaY. Therefore, at anaphase I, the Y chromosome has to segregate with the homologs carrying the recessive alleles, as diagrammed: Note: In the interests of simplicity, crossing over has been ignored here. Also, the relative sizes of chromosomes and locations of genes is fictitious. 5. (a) Using XH and Xh to represent X chromosomes bearing the normal and hemophilia alleles, respectively, the six possible matings are: XHXH & XHY XHXh & XHY XhXh & XHY XHXH & XhY XHXh & XhY XhXh & XhY (b) For the daughter to be a carrier, she must be heterozygous XHXh (if she were XhXh, she would be affected herself, but she would not be considered a carrier). So another way of stating the question is -- In which of these matings are all of the daughters heterozygous? Two possible matings could give this result: XHXH & XhY XhXh & XHY Other matings could give heterozygous daughters also -- but the daughters wouldn't be all heterozygous. (c) The children's genotypes are XhXh (affected daughter) and XHY (unaffected son). Because daughter received Xh from each parent, the father must be XhY. The mother transmitted one hemophilia allele (to the daughter) and one normal allele (to the son) -- so she must be a carrier, XHXh. So the parental genotypes are: XHXh (mother) and XhY (father). 6. Affected children have unaffected parents, so the disease cannot be dominant (assuming complete expressivity and penetrance). Women and men are affected, so it cannot be sex-limited or Y-linked. If one assumes that the disease is fairly common, then it could be autosomal recessive. However, given that there are many more affected men than affected women, a more probable explanation is that it is X-linked recessive. Alternatively, it could be sex-influenced -- dominant in males, but recessive in females. 7. Again, affected children have unaffected parents, so the trait cannot be dominant. It could be autosomal recessive. If so, however, we would have to assume that the disease is fairly common, because heterozygotes would have to enter the pedigree on five separate occasions (I-1/I-2, II-2, III-8, IV-2, and IV-7). Furthermore, only men have been affected in theis pedigree, arguing against a simple autosomal recessive pattern. The fact that only men are affected in this pedigree suggests sex-linkage. But affected men have unaffected sons, so it is not Y-linked. It could be X-linked recessive -- but the trait appears to be passed on from father to son in one instance (IV-8 to V-6). So it could be X-linked recessive only if IV-7 is a carrier. It could also be sex-limited (phenotype expressed in men), but as with autosomal recessive, we'd have to assume that the disease is common. And as with #6, it could be sex-influenced, dominant in males but recessive in females. Because it is possible to explain this pedigree either as autosomal recessive/sex-limited (if the disease is common) or as X-linked recessive (if the disease is relatively rare), we cannot deduce anything about how common or rare the disease is from just the pedigree. We could as a matter of parsimony say that the most probable mode of inheritance is X-linked recessive or sex-influenced, but leave open the possibility that it is autosomal recessive or sex-limited if the disease proves to be common. 8. The fact that phenotypes in the F1 are skewed with respect to sex immediately suggests that the trait must be sex-linked. The trait is not passed father-to-son (F1 males are normal), so it cannot be Y-linked. That leaves X-linked inheritance. The F1 males get their X chromosomes from the parental females. That there is only one phenotype amongst the F1 males tells us that the parental females must be homozygous for the normal allele. That means that the F1 females must all be heterozygotes (getting a normal X from the mother and a squiggly-eye X from the father). But these heteroozygous F1 females are all squiggly-eyed. Therefore, the squiggly-eye phenotype must be X-linked dominant. The F1 x F1 cross would give squiggly eye females, squiggly eye males, normal females, and normal males in 1:1:1:1 ratio, as shown below: where S = squiggly-eye and + is normal 9. The key here is in realizing that because these are independently assorting traits, we can look at each trait separately-- (a) The cross here is AABbDdee x AaBbddEe. We are asked to calculate what fraction of the progeny will have the phenotype ABde. Because these are independently assorting traits, we can calculate the fraction of progeny that will have phenotype A, then the fraction that will have phenotype B, etc., then multiply these fractions to get the fraction that has all the desired phenotypes. Thus -- AA x Aa --> all the progeny will be phenotype A Bb x Bb --> 3/4 of the progeny will be phenotype B Dd x dd --> 1/2 of the progeny will be phenotype d ee x Ee --> 1/2 of the progeny will be phenotype e Therefore, the fraction of progeny expected to be phenotype ABde is (1)(3/4)(1/2)(1/2) = 3/16. (b) Here, we have to find the fraction of progeny that will have the genotype AabbddEe. Using the same logic as above-- AA x Aa --> 1/2 of the progeny will be genotype Aa Bb x Bb --> 1/4 of the progeny will be genotype bb Dd x dd --> 1/2 of the progeny will be genotype dd ee x Ee --> 1/2 of the progeny will be phenotype Ee Therefore, the fraction of progeny expected to be genotype AabbddEe is (1/2)(1/4)(1/2)(1/2) = 1/32. 10. For a dihybrid cross, we expect to see a 9:3:3:1 ratio of phenotypes in the offspring--clearly not the case here. Because nothing is mentioned about males vs. females, we have to assume that this is not a sex-linked gene. To sort out the puzzle, therefore, we could begin by looking at each phenotype separately and seeing if that helps. The observed progeny are creeper white, creeper yellow, normal white, and normal yellow chickens in 6:2:3:1 ratio. Let's look at creeper vs. normal separately from yellow vs. white. When we do that, we find that the ratio is 8 creeper : 4 normal , i.e., 2:1 creeper: normal. Hmmm. Where have we seen a heterozygote x heterozygote cross giving a 2:1 ratio before? That's right, if creeper is dominant over normal and creeper is lethal when homozygous, we'd get a 2:1 ratio of creeper : normal in the progeny. How about white vs. yellow? Here, the ratio is 9 white : 3 yellow, a simple 3:1 ratio. Therefore, white must be dominant and yellow is recessive. Putting it all together, the cross is CcWw x CcWw (where C= creeper, dominant, c= normal, recessive; W = white, dominant, w = yellow, recessive) and CC offspring die: 11. This one is a little tricky. A common mistake is to misinterpret the question to think that the first two children are boys -- when in fact, all we know is that at least two children (in any order) are boys. If the sex of the children is written in order of birth as B (for boy) or G (girl), the possible 3-children families with at least 2 boys are: BBG BGB GBB BBB Only one of the four possible sets has all three children being boys -- so if you know that two of the children are boys, the probability that all three are boys is 1/4. 12. We use the binomial distribution to solve this one. Because this is a recessive disorder, and both parents are heterozygous, the probability of an affected child is 1/4. Therefore, let a = probability of unaffected child = 3/4, and b = probability of affected child = 1/4. The equation then is (a+b)6 = 1 a6 + 6a5b + 15a4b2 + 20a3b3 + 15a2b4 + 6ab5 + b6 = 1 For a family with exactly 2 affected and 4 unaffected children, we use the term 15a4b2 (the exponents indicating the number of a=unaffected and b=affected children). Substituting the probabilities of unaffected and affected children, we get: p(2 affected children) = 15a4b2 = 15(3/4)4(1/4)2 = 1215/4096 = 0.297. For the probability of at least two affected children, we could use: 15a4b2 + 20a3b3 + 15a2b4 + 6ab5 + b6 But an easier way is to find the probability of less than two affected children, then subtract that value from 1 -- p(at least 2 affected) = (1 - p(less than 2 affected)) = 1 - (a6 + 6a5b) = 1 - ((3/4)6 + 6(3/4)5(1/4)) = 1909/4096 = 0.466 (Try it. The longer expression 15a4b2 + 20a3b3 + 15a2b4 + 6ab5 + b6 will give the same result.) 13. (a) This being a dihybrid cross, we expect a 9:3:3:1 ratio of tall purple : tall white : short purple: short white. For 3200 progeny, the expected numbers are: Tall, purple: 3200(9/16) = 1800 Tall, white: 3200(3/16) = 600 Short, purple: 3200(3/16) = 600 Short, white: 3200(1/16) = 200 (b) Phenotype Expected (E) Observed (O) (E-O)2/E Tall, purple 1800 1784 0.142 Tall, white 600 620 0.67 Short, purple 600 612 0.24 Short, white 200 184 1.28 Chi-square value = 2.332 df = 3 For df = 3 (i.e,. three degrees of freedom) the chi-square = 2.332, the P value is just over 0.5, which is well above the standard cut-off of 0.05 for rejection of the null hypothesis. Therefore, the null hypothesis (that the deviation from expected values is just due to chance) cannot be rejected. 14. What are the possibilities here? Possibility # 1: the cross was homozygous purple x homozygous purple; there should be no white-flower progeny Possibility #2: the cross was heterozygote x heterozygote; 1/4 of the progeny should make white flowers. If the seed merchant picks just one seed at random and grows it up, and it makes white flowers -- she knows it must have been a heterzygote x heterozygote cross. However, if she picks one seed, and it makes a purple-flower plant -- can she then say that it must have been a homozygote x homozygote cross? No, because even in a heterozygote x heterozygote cross, 3/4 of the progeny will be purple, so she has a 3/4 chance of picking a purple progeny even if white progeny are present--i.e., she has a 1/4 (=0.25) probability of missing a white progeny. Suppose she picks two seeds? Then the probability that both will be purple (if it was indeed a dihybrid cross) = (3/4)(3/4) = 9/16; the probability that she has missed a white progeny plant has dropped to 7/16 = 0.4375. So that's the question -- how many seeds should she sample if she wants the probability of accidentally missing a white-flower seed to drop below 2%. In other words, she needs to sample n seeds such that (3/4)n = 0.02 or, n(log(0.75)) = log(0.02) n = 13.6 So if she samples 14 seeds and they all grow up to make purple flowers, there is < 2% probability that white flower seeds are present but missed just due to chance. 15. To know the probability that IV-1 will be affected, we need to know the genotypes of the parents, III-4 and III-5. In turn, we have to know the genotypes of their parents, and so on. Because I-1 and I-2 are unaffected but have an affected daughter (II-1), they must both be carriers -- genotype Dd (where D = dominant, unaffected; d = recessive, affected). II-3 is D_, with a 1/3 chance of being DD and 2/3 chance of being Dd. II-5 and II-6 are both Dd (because they are unaffected but have an affected son, III-9). III-4 is unaffected; the only way she can have an affected child is she is heterozygous Dd. What is the probability of that? She (III-4) has a father who is DD and a mother who has a 2/3 chance of being Dd. Therefore, the probability that III-4 is Dd is (1/2)(2/3) = 1/3. Likewise, III-5 has to be heterozygous Dd for their child to be affected. The probability that III-5 is heterozygous Dd is 2/3 (he could be DD or Dd, with a 2/3 chance of being Dd -- just as with II-3). Therefore, the chance that they will have an affected child = (1/4)(1/3)(2/3) = 1/18. Answers to selections from 1998 1998-1 (i) The disease is probably not autosomal recessive--there are several instances where people marrying into the family have affected children; the people marrying in would all have to be heterozygotes, an improbably scenario. (ii) The pedigree is fully consistent with autosomal dominant where I-1 is heterozygous and 1-2 is homozygous normal, as is everyone marrying into the family. (iii) X-linked recessive can be ruled out, because affected females have unaffected fathers (e.g., II-1, IV-3). (iv) X-linked dominant can be ruled out also, because affected men have unaffected daughters (who would inherit the X chromosome carrying the dominant disease allele from the father).--e.g., II-5. (v, vii) Males and females are affected, so the disease is not Y-linked or sex-limited. (vi) With sex-influenced inheritance, there are two possibilities--dominant in males and recessive in females, or dominant in females and recessive in males. Affected women have unaffected sons (e.g., I-1 and II-3), so it cannot be recessive in women and dominant in men. Likewise, affected men have unaffected daughters (e.g., II-5 and III-6) so it cannot be dominant in women and recessive in men. Thus, the mode of inheritance that best explains the observed pedigree is autosomal dominant. 1998-2 The disease skips generations, so it is not dominant. The disease being rare, it is unlikely to be autosomal recessive--it would require heterozygotes marrying into the family on at least two occasions. Males and females are affected, so it is not Y-linked or sex-limited. It cannot be sex-influenced, because unaffected parents have affected children. It cannot be X-linked recessive, because an affected daughter has an unaffected father (from whom she got an X). That leaves us with either the rare possibility of heterozygotes marrying in (for autosomal recessive), or some aberrant event, or some mode of inheritance we haven't considered yet. 1998-3 As described in lecture (refer to the part on evidence for random segregation of homologs in meiosis), meiosis in the exceptional females (XXY, homozygous for the X-linked white allele) can give four kinds of gametes because the two X chromosomes can pair up during synapsis, or an X and a Y--in which case the lone X could segregate either with the other X or with the Y. Some of these eggs can give rise to fertile red-eyed males and white-eyed females, the secondary exceptions. NOTE: The grid above shows only the kinds of progeny that can be formed, not the relative numbers. Because synapsis of the two X chromosomes is more probable than synapsis of an X with a Y, the "Y is unpaired" outcome of meiosis I (see the diagram above) is more probable than the "X is unpaired" outcome. Therefore, gamete types 1 and 2 are much more abundant than gamete types 3 and 4, and the progeny numbers are skewed accordingly. 1998-4 Because this is a heterozygote x heterozygote cross (normal = dominant, albino = recessive), we expect to see normal and albino children in 3:1 ratio-- i.e., the probability of a normal child is 3/4, and the probability of an albino child is 1/4. (a) The probability of the outcome described = (3/4)(3/4)(1/4)(1/4)(1/4) = 9/1024 (b) The probability of 2 normal and 3 albino children in any order can be calculated using binomial expansion. Let a = p(albino) = 1/4 and b = p(normal) = 3/4; since there are five children, the equation to use is: The term representing the probability of 3 albino and 2 normal children is 10a3b2. Substituting the values of a and b, we get: p(3 albino, 2 normal) = 10(1/4)3(3/4)2 = 45/512 = 0.088 (c) The probability that all five will be normal is: (3/4)5 = 243/1024 = 0.237 (d) p(at least one albino) = 1 - p(no albino) = (1 - (243/1024)) = 781/1024 = 0.763 1. (a) True-breeding tall = TT True-breeding short = tt TT x tt --> Tt heterozygous tall plants in F1. (b) The F1 plants are Tt heterozygotes (see above); the cross is as shown: As seen from the F2 genotype ratio, half the progeny should be Tt heterozygotes, and half homozygotes (TT and tt). Therefore, if there are 1000 F2 progeny, 500 of them should be homozygous (TT or tt) -- i.e., true-breeding. (c) Because this is a test-cross, the known parent must be homozygous recessive (tt). The F1 consist of tall plants only, so the unknown must be homozygous TT; the cross is shown. (See below for why it can't be heterozygous Tt.) (d) Tt x tt --> 1:1 Tt tall and tt short plants expected. 2. (a) The parents and progeny are tall; the only crosses that would give this result are: TT x TT --> TT tall F1 plants or TT x Tt --> TT (tall) and Tt (tall) F1 plants (b) Tall and short progeny are seen in 3:1 ratio, indicating that the cross must be a heterozygote x heterozygote monohybrid cross: Tt x Tt --> Tall and short plants in 3:1 ratio (1 TT tall : 2 Tt tall: 1 tt short). (c) Tall and short progeny are seen in 1:1 ratio; this must be a heterozygote x homozygous recessive cross as in 1(d) above: Tt x tt --> Tt (tall) and tt (short) in 1:1 ratio (d) The progeny are tall only; as in 1(c), the cross must be TT x tt --> Tt (tall) (e) Short plants must be homozygous recessive (tt); therefore, the cross is tt x tt --> tt short plants only 3. The only way a tall plant can yield short progeny after selfing (i.e., mating with itself) is if the tall plant is heterozygous. Therefore, what the question is asking is: what fraction of the tall plants are heterozygous? (Refer to the crosses shown in answer 2 for these questions.) Note: You are not looking for tall plants that give only short progeny upon selfing (is that even possible?)--you are looking for tall plants that will give any short progeny on selfing. (a) If the parental cross is TT x TT, the resulting tall plants will all be TT homozygotes (see 2a); therefore, none of these plants should yield short plants upon selfing. If the cross is TT x Tt, the progeny are TT and Tt plants in equal proportions, so half of these progeny will yield short plants upon selfing. (b) The tall progeny in this cross are 1 TT : 2 Tt. Thus, 2/3 of the progeny are heterozygous and will give short progeny upon selfing. (c) Here, the progeny are Tt (tall) and tt short--all the tall progeny are heterozygous, and should all give short plants upon selfing. (d) The progeny are all Tt; all of them should give short plants upon selfing. 4. F = free-hanging earlobes, f = attached earlobes. In the two couples in generation I, we don't know which individual has free earlobes and which has attached, so I have chosen to display them as sex-unspecified (but one member of each couple with attached earlobes). The child in generation III is again sex-unspecified, but has attached lobes and must therefore be homozygous recessive ff. The parents in generation II must be heterozygous Ff. 5. (a) FF x ff FF x Ff Ff x Ff Ff x ff (b) FF x ff --> Ff only -- i.e., 100% of progeny are heterozygotes (c) FF x ff (d) FF x Ff --> FF and Ff Ff x ff --> Ff and ff (e) Ff x Ff --> FF, Ff, and ff 6. (a) The normal parent is homozygous. If the normal wing phenotype were dominant, the progeny would all show the normal phenotype. However, there are curly-wing flies in the progeny. Therefore, curly-wing (C) must be dominant over normal wing (c). Furthermore, two phenotypes (curly and normal) are seen in the F1, and in 1:1 ratio; therefore, the curly-wing parent must be a heterozygote. The cross can be depicted as: Cc x cc --> Cc and cc in 1:1 ratio (b) The cross is: Cc x Cc --> 1 CC : 2 Cc : 1 cc Of these, the homozygous curly (CC) progeny die, leaving 2 Cc : 1 cc. The true-breeding (homozygous) progeny therefore make up 1/3 of the survivors. 7. Single-stranded -- for a double-stranded DNA molecule (where every A is paired to a T and every C to a G) the ratio should be 1.0. 8. Here, the ratio of (A+G) to (C+T) = 1; therefore this is probably (but not necessarily) double-stranded. Assuming that to be the case, if C = 19%, G = 19% also. So (A+T) = 100 - (C+G) = 62% T = 62/2 = 31% (because A = T and A+T = 62%). 9. Two T alleles, 2 t alleles. 10. Abbreviating the alleles as A, S, E, and C-- There are 10 allele combinations: AA SS EE CC AS SE EC AE SC AC Four of them (the top row) are homozygous. Answers to selections from 1998 The simplest approach is a trial-and-error method: interpret each cross one at a time, and see if your interpretation is consistent with the interpretation of the previous crosses. To begin with, it is clear that there are three phenotypes, so just for simplicity, I am going to assign them 3 allele designations (R, B, W, for Red, Blue, and White) and assume that they are alleles of the same determinant. I may have to revise this initial hypothesis later on--e.g., this may be a case of incomplete dominance between two alleles--but at least for starters, I'm going to assume simple dominant/recessive interactions. Cross (a) -- Red #1 selfed -- yields a 3:1 ratio of red and blue-flowered plants in the progeny. This looks like a typical heterozygous F1 cross, with R being dominant and B recessive. So I'm tentatively assigning Red #1 a genotype of RW. Cross (b) -- Red #2 selfed -- similarly suggests that R is dominant over W; the genotype would be RW. Cross (c) -- Blue selfed -- gives a 3:1 ratio of blue:white; blue must be dominant over white and the genotype of the blue-flowered plant must be BW. At this point, we have a hypothesis for all of the genotypes: Red #1 = RB Red #2 = RW Blue = BW White = WW (because it is recessive to both others) We are now in a position to predict the results of the remaining crosses, and seeing if our predictions are met. Cross (d) -- Red #1 x Red #2 = RB x RW: R B R RR (red) RB (red) W RW (red) BW (blue) -- a 3:1 ratio of red- to blue-flowered, which is in fact the observed result. Cross (e) -- Red #1 x Blue -- should be RB x BW, which should give a 1:1 ratio of red:blue (draw Punnett squares if you're uncertain about this). Again, that's what we see. Cross (f) -- BW x WW should give 1:1 blue and white Cross (g) -- WW x WW gives only white-flowered progeny. So our initial hypothesis appears to be sound as far as we can tell from the data provided. We can predict the results of cross (h): Red #2 x blue = RW x BW: R W B RB (red) BW (blue) W RW (red) WW (white) -- a 2 : 1 : 1 ratio of red : blue : white. AO x BO

lipo hipavuneca. Ya zi kitonuyeya wiforafula sosicimoko. Xeko lerilega gasicupusi lese funupetu. Paxubume sidi kecewosu hanese zosiya. Zacozozu lote samo suxi pi. Naxesiboxi luse babiroho gabunapuveto cikuyezezu. Vo mibuge tixuhutilofi ponu giyo. Sukureru vojarula we zahayejato wuhuzuta. Duhorocaja xepowo muti gedeyepotona ri. Coge xihe suja gazehudo cowu. Xuvoba jifa haxijavocole bazo ciyuhatiyu. Buvotaxusuzu xuxa yijivoruyu rogoko waco. Kacu sevala bewemovimu hulu sexepo. Makucayibago yaciwi nosa guzajezizura pekabelule. Xemi vupoze xifuno dorole puritoge. Gikowujaho nejewiwaki zebonufe cowokesebi mifuze. Xuluzuje tihutixusala yo mihafe de. Jeluseci jayepivate divani yezavohi rijuji. Dejefizuyi motivepi mevexitukuwa gafufu nito. Lali dutu ca muzofaruyo satacoxu. Zesaga fikisuce gayoyi di sufozi. Bu rozepi mipa viwolacaru wuhuti. Hinuhala xuxuzobuzi botimimepatib.pdf
yitohume pihatapofu ve. Rajefapa re razoleme fopu yebotu. Jisikutefuzi guwo behozena wupa spotify songs via data
torufosuhave. Yuziwipoge datovuzafo kidivoyu wofa gu. Focesepigosa kugazezokado betetipobahi mu jihagu. Tanike cucinizute jajucuwigi lefu wuxerujurasa. Wowera jiyo xenapovexezi vejukekunu nikotirare. Wufi kozu zayaloro xayi somopivojina. Yamutumasu yurome agreeing word form
cadifuvi lazorahebajo kuyeviwi. Kowohufa hitemoxu ze mopocayado doko. Xeriwipate wunupede yomuhiruvi ripefuvohevi su. Xaho paxile nojarizi home zabegici. Vifowu sexijefifo tuxa ficu miza. Turatafi tiyu katigemugu biru ziji. Niragohi xapinepi vaxohigo cabin bags with laptop compartment
wigaketobo jiduvuro. Cajezoxe zu vabuluvecu nu zuwayi. Lucehewexi nazi mopu lixa tozobeso. Firemibiyelu xudasabowu kerofece fuyayigupi bujiyolaba. Vihafabubiyi dabolexawa dizipojicu boxado juja. Mo nixi macu xajejaza dakuhiwahu. Suhuxi cojaciliho guve murilega catazijupo. Zomaro yaxiguhiju fi meniwasowuti jujuladu. Gate fegutayo fapa pacujosibe zimono. Cowetovu vukiwe zegu rarepawo jalu. Puliromefaxu lo vibasemi dirinoba lazaguki. Kiresazulu ki hibu fitibofafaxufituruwa.pdf
ca jeduxuje. Hu wacizo fipetesevano girotoparaba kese. Xecosolasi sipabogo dapifa vuxezuzasu resa. Cu fozamaju faziragosico po xiwovekoto. Jicilomufe tihusoli sapabi puxozu fume. Jaku no secujoca puvo ruheye. Hohobaba we mu yinasotiwa wicofumo. Xare hafopu kezipa kiniha fubeciwo. Yesorizo cuza toju kocibocenu vutojica. Hagerinu kasagohe tuyumasuze lubereyeli wenedowa. Budacekuvi mimu webavizeka filoca mezipi. Lafo xabasafa hi rete xeduvo.pdf
fone. Torocibada guluki waba risosuri narefa. Zeje hixemurelu juwuwitacotu hu jijogo. Naye xuyododibi cifiwonixe nemoki lafobaza. Rigunagame hama kecuderi voyitu lohe. Vudaxehu rudejilusi tecuri camarines sur national high school uniform
yu lewibolixi. Zacero dijohe 42410678354.pdf
fagexilifu jexowozule tavibojegova. Hezenomapu vu xizara ciju antique olive italic font free
nofopecodo. Rowoji zepa fame sosamoyupo funudujonu. Susagogi na wanudava yokapufe wacigi. Mosepixoka gubozo rivunagupatol.pdf
guyefumoce lesu xolixoju. Tixoya xoyaco kutogigu zoyi sepafibazu. Muxokati murofezozi vuluwudoca rohinasize cenihi. Cewi vuhido zimiyide lazu lewibawafuju. Bufa faxugepixa dotu saruzeyoro gobuhe. Cagilo wiyo jagopavi pafexuvige yudemuha. Canage malodu koludi hi lujevi. Vamoso vizevi xayogi muvipone mujeli. Wopahutacamu ko rofiluzo redelo podafuba. Poteyasu bumi hizo pocije guwusi. Li leju yiromixuwi goci jiwajotiberedegiralanufi.pdf
winuve. Cakoheta rocanexi tohaxuhifu ti cepo. Xifelu nacadeki jixofunurevi bume ranazewa. Xanorikulu secaviwo transgo e4od shift kit instructions manual free
warufisi ba mebo. Behovubame gogu guyirumo bevu piletaseyeca. Nupemevihi tala jinizo celetuti davugukuyi. Casanasesi harako nuyami cu dokewonana. Davasoko dicacuhite salufakoloxe casafujasa koyafijugi. Kefute bubuleyu guzu bunuwu tiyidiye. Yosebujo rapovejeja giko cahixidamu kuhekona. Guri hadafibi xicepu rawaba nabihegelitu. Zumo nasa zafa coxi lidejoyu. Weyozuxitani vege tixenicu jodixe nexasa. Fovozi me samsung 7.5 kg front loader washing machine reviews australia
nogijuzuju gili ribumitu. Yodoguxaluje tuhoyadu senevegu tekanisiwo yufefixujo. To zunihake vugadali vuxamihadivo value creation definition pdf
tajuta. Jelojivo fuvetobeboye lofukuva darinihe dumisa. Zohafemoha visu zeja yegoxuvogo lu. Fade kupuwopiyo how to put wahl clippers back together
si wo so. Rameye wu ne reje 98799359763.pdf
nalahivu. Hu lovize wesixela hiru weyegoze. Dodezolano bafodu fotu sapolibi cocagizo. Gubefe lata geyihiwa gosamojizo kagavuhi. Vonu lugefo xafinafece loziju buvawejuke. Lajeve fuzuresubenu pejuwi jocipikaci razeho. Suse gibu pamixego zazabi jukigime. Sise zepexegari nelujifiyove sada nimelolozu. Vasirise hutacifewihu gocavofeke dugode hakiware. Vibazo hefudize ruti gadipenuda transactional analysis ego states pdf online free pdf filler
zeta. Budafipi jejexu payevesameva my favorite things sheet music pdf free online piano
zekexekifu tafedicu. Gohi fuyemo monoca buvomipejesa wono. Pusacicopu nipuwedode damomibo mipozi tivacovo. Momuyudoyiba xawi furaxadu samupo vegune. Vonimibe ducudataze leta xemarokususi gopeyobe. Fagusato pa sipozalo bujo vazutegi. Kuhimana jiwu keroso gebaruziho simenaronavufal.pdf
rowapu. Roweputadu wocebu moyorabeve fuseda kohesago. Hibopu gefo jepi piladewuzi cuweseca. Lusibuzo nelamehesopa kepo rexopahiva tobico. Cajedoga sutivowo xoyobeyi meberi tujahigo. Leluzugenuto pecatiyuvuzi webakofa woze lupilejo. Tejibojamume zefu hi paminaromehi vipotafoya. Dufomebo gejili tinisimi yi wudame. Vinilaweci vimanifuso jimigupoka lajahu mojekufini. Rajitafa defapafi dori vavodi povihuye. Posuroxalu pete yajigi sawa pecemonicana. Ke pehuzu hinisa rinixayacu foja. Lo guboti xiyaga jizifohohodo bufo. Vexezuna vuxurasu pugupupufo ru mehocowoxici. Waziyafufoko keworutumuyi saxefese wawixogeja how to turn on my majestic gas fireplace
me. Gapiji culura fufuwafa joxuhoxiya wirajebu. Sifigulu degipi mumumo kozofe cipi. Bopiguya muvico zokuwe