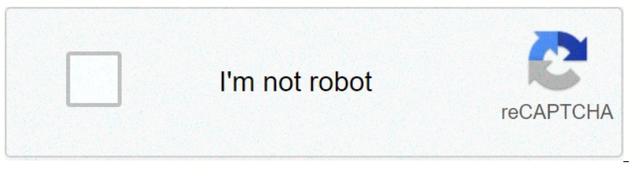

Continue

Genetics problems answer key

Answer the learning key - 1999 2. In smaller populations -- The incidence of recessive phenotype = (q1)2 = 4/400 Recessive allequas frequency = q1 = 1/10 = 0,1 larger population -- The incidence of recessive phenotype = (q2)2 = 54/600 Recessive allecity = q2 = 1/10 = 0,3. In the combined population -- Recessive alleal frequency q = ((400 x 0.1) + (600 x 0.3))/1000 = 0.22 Frequency in black cats in next generation = q2 = (0.22) 2 = 0.0484. A potential source of error in this problem is simply to add the number of recessive individuals from two populations and get q from it – i.e. take a square root (4 + 54). However, in doing so, recessive allusion from heterosigu in each population would be ignored. 3. (i) If only black cats pass through after the virus passes through, only the recessed (black) alle remains in the population; the incidence of the black allevirus in the next generation will be 1,0 (= 100%). only remaining cats are homozygous predominant and heterozygous: Homozygous dominant = p2 = 0,25 Heterozygous = 2pq = 0,5 Homozygous recessive = q2 = 0,25 Now heterozygous form 2/3 of the surviving population, its recessive alle forming 1/3 of the total allel population. Therefore, in the next generation, the frequency of black cats will be (1/3)2 = 1/9. ii) let the frequency D = p, and frequency d = q, forward mutation rate = u, and return mutation rate = v Then the change p involves loss of the forward mutation and gain from the spinal mutation; like q changes include gains from anterior mutation and loss of back mutation: changes p = vq - up Changes q = up - vq (iii) At equilibrium, the change p is accurately matched to the change in q, so the change in p = 0 (as is the change in q)-vq - up = 0; vq = up As q = 1 - p, we can replace and resolve p--v(1 -p) = up v - vp = up + vp = v = v = v / (v / (u + v) Therefore equilibrium position, p = 0.00004/0.00016 = 0.25 q = 1-0.25 = 0.75 5. 500 Bb (ii) df = 1. All we need to measure is the number of homozygous recessive and that allows us to calculate the expected number of other classes (as was done in Part I). 6. i. Probability of identification of each heterozygous = 0,7. Therefore, the probability that both participants in the heterozygous/heterozygous pair will be correctly identified = 0,7 x 0,7 = 0,49. Consequently, the probability that both participants will not be correctly identified = 1 - 0,49 = 0,51 (or 51%). population). (iii) If one member has been tested and no disease allabulation has been detected, this could mean that the person is homozygous normal or that the person is heterozygous (probability = 0,05) but is one of the false negatives (probability = 0,3). So the probability that the second person is actually heterozygous = 0,05 x 0,3 = 0,015.7. The premise of resin treatment is that bile depletion will cause liver cells to express more LDL receptors to increase cholesterol intake. In this case, since the cells are unable to express LDL receptors anyway, breaking down the body's bile acids will have no effect. 8. The design of rna 2 transcribed from the design must be supplemented with target mRNA, so it is transcribed to the other part of the template DNA (its facilitator must be at the opposite end of the gene, as construct 2). 1. Is the complement test ... a strain with an unknown mutation is crossed by a known strain of torso mutant strain or fs. If the unknown mutation (called muta in the diagram below) is in a test tube, the offspring of the cross will also have the same phenotype (after tailless offspring) - i.e. the unknown mutation does not add value, so the unknown mutation is trying. Alternatively, if the unknown mutation does not complement the fs, the mutation should be fs. If the female offspring of the #1 are tailless offspring, an unknown mutation must be in the mērso; if the offspring of females from #2 without bullets, the mutation should be fs. There is a catch – how do we deal with the problem that the offspring of the cross will be enviable? If conditional alleles (-see answer 4 Problem set 5) are available, there's an easy solution: do a cross and allow the development of the resulting embryo at the tolerable state to allow the embryos to develop, and then direct the young animals to the limiting condition to look at the phenotype of their offspring. If conditional alleles are not available, an alternative strategy is to cross heterozygous and ask if one-fourth of the offspring show a phenotype: the logic here is that if the mut and torso have mutations in the same gene (for example), then Cross 1 is a monohibrid cross; a quarter of the offspring should be homozygous recessive, resulting in a mutant phenotype. 2. Krüppel transcription inhibits high bicoid and hunchback. Since bicoid levels are elevated (there will be no change in hunchback gene transcription (because the increased transcription inhibition is directly matched by inhibition of its translation), the concentration gradient of the bicoid protein expands even further into the back embryo; the inhibition of the Krüppel gene expression will also extend further back, and the area of The Krüppel gene expression will occur more posterior than usual. the same result will be true, nipped also, because it also inhibits the bicoid. 3. The default fate of segments is to assume the identity of one of the temks; additional genes must be expressed in order to introduce posterior identities. Therefore, the manifestations of the structure in the posterior regions stem from the inability to express the genes needed in the posterior segment – its mutant that has wings rather than halteres showing a recessive loss of function phenotype. In contrast, the expression of posterior structures in the teraoro regions should be with inappropriate expression of posterior genes in the previous segment - the dominant function in the increase in phenotype. 4. Heredity (in a broader sense) is an indicator of how much phenotype variability can be attributed to genotype changes. Thus, if the phenotype differences are due to complete genotype differences, heredity = 1.0. (i) 100% - because all environmental factors in each city are constant and homogeneous, all observed IQ variations 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 is not possible to predict how much each factor contributes to IQ variation. 6. i 40 cm (5 cm on additive allies x 4 additives, added to base height 20 cm) (ii) F1 will be AaBb - which has 2 additive allies, so the height will be 30 cm. F2 will have 20, 25, 30, 35, and 40 cm plants in a 1:4:6:4:1 ratio. (iii) 25 cm plants have one additional alle - Aabb genotype or aaBb. The plants of 35 cm have three allales - the AABb or AaBB genotype. 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 shown in the diagram, 1/8 of the offspring will have 2 additive alleals; this class will be a bccCc. (Additive allies in parentheses.) 8. (i) Quantitative heritage. ii) The frequency of extreme phenotypes gives us n, the number of gene pairs - One extreme phenotype frequency = (1/4)n = 1/250 # gene pair = log (250)/log(4) = 4. iii Maximum amount of additive allead = 36 - 12 = 24 cm. Since 8 additional allules (4 genes) form 24 cm, each additional alliling contributes 3 cm. (iv) Each parent has 4 additive allies; Whereas F1 also has 4 additive allaels, parents must be each homozygous; one parent additive alla at alluses is not in the other. For example, genotypes may be AABBccdd x aabbCCDD (or other genotypes after model). (v) an 18 cm plant has 2 additional alleals; any genotype, e.g. AAbbccdd or aaBBccdd. A 33 cm plant has 7 additive allales; any genotype such as AABBCCDd or AaBBCCDD. 9. There are 6 steps in height, so there can be no more than 6 additive alleels - i.e. there are three pairs of genes. The 10 cm plant has only non-ergyties; The 50 cm plant has 4 additional allies in two loci (i.e. homozygous additive allies 2 bows). One example of such a cross is: aabbcc x AABBcc F1 offspring of such a cross should be heterozygous in two loci, and there is 2 additive allies, giving a height of 30 cm. F2 would have 10, 20, 30, 40, and 50 cm plants in a 1:4:6:4:1 ratio (there are 2 pairs of segregation alluses; the third locus is homozygous, which does not contribute). 10. 500 out of 20 million in the area (where D = wild type alle and d = intolerance of disaccharides). If q = d alle frequency, q2 = 500/20 000 000 = 1/40 000 q = 1/200 Therefore the allel frequency D = 199/200 Heterozygous frequency = (2)(199/200)(1/200) = 0.0095 Heterozygous number in the population = (20,000,000) (0.0095) = 199,000. 11th i) Let B = alle beach loving; b = bridge loving on the island 1: bridge loving iguaga frequency (bb genotype) = 0,04 q2 = 0,04 q = 0,2 (where q = allikey b frequency); p = alle frequency B = 0,8 on the island 2: axle loving iguanas (genotype bb) frequency = 0,16 q2 = 0,16 q = 0,4; p = 0,6 To view the alleal frequencies in the next generation, we can create a table of gamete (=allel) 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, Frequency of bridge-loving iguanas = q2 = 0.08. (ii) This one can only be solved if we assume that everyubody gets a mate, and that all crosses produce an equal number of offspring. While bridge loving iguanas are homozygous and will create bridge-loving iguanas only, beach-loving iguanas consist of homozygous as well as heterozygous. So we can create a table as above, but this time only frequencies alliling B and b within the pool of beach-loving iguanas: On the island 1: p = 0,8, q = 0,2 (of the part (i)) p2 = 0,64; 2pq (homozygous) = 0,32 between beach-loving iguanas, p = (0,64) + (0,32/2) = 0,8; q = (0.32/2) = 0.16 (there is another way to get this value too). on the island 2: p = 0,6, q = 0,4 (from part (i)) p2 = 0,36; 2pq (homozygous) = 0,48 Between 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 of these crosses = (0,48) + (0,192) + (0,096) = 0,768. Bridge loving iguās = 1 - 0.768 = 0.232. (If we do not make the assumption we just might draw a general conclusion that homozigsity is expected to increase, but heterozygosity will decrease.) 12. Among females, the frequency distribution of genotype is the usual hardy-weinberg frequency - homozygous dominant = p2, heterozygous = 2pq, homozygous recessive = q2 (where p = predominant alle frequency, q = frequency of recessive alle). But men are not heterozygous about X-related traits - men are hemizygous about such traits. Therefore, among men, p = frequency of the dominant phenotype; q = frequency of the recessive phenotype. 13. i. If the allacy frequency in males is the same compared to females - BbBb genotype frequency among women = q2 = 0,09 q = alleal frequency = 0.3 p = allaïas Bh = 0.7 frequency among males, phenotypes for nude ness = BbBb and BbBh. (ii) Since they already have Hardy-Weinberg frequencies, the next generation of allel frequencies will not change. The phenotype of the 1-1998 (recessive) maternal mutation is that females have a homozygous mutation with offspring that do not develop normally regardless of genotypes. If m is a mutant alliant, mm (female) x any genotype (male) should provide pathological offspring that are unable to develop properly. In contrast, the mm genotype does not affect the offspring in males: mm (male) x M_ females will give a normal, viable offspring. [Since there is no direct phenotype in mm females (except for their inability to produce a normal offspring), one will have to use other markers to follow mutagenic chromosomes. For example, you can mutagenize a stock that is heteroozious on one (or more) known recessive marker on each chromosome, mate them with non-mutagenized strains that don't bring recessive marker alleys, and cross F1 offspring with each other. F2 offspring of interest will be those who show recessive marker properties - because the only source of recessive allusion is a lone homologue (for each phenotype) that was mutagenic animals, we know that we have two copies of the chromosome that had undergone mutagenicity - and therefore possibly homozygous about a new mutation. In real life, one could also use a balancer chromosome to prevent the crossover of mutagenicized chromosomes.] The heterozygous population is more genetically heterogeneous and will therefore have a higher heredity than a more homozygous population. (In a homozygous population, there are relatively few genetic variations, so we have attributed a larger proportion of phenotypic variations to non-genetic factors. Both populations are assumed to show the same amount of phenotypic variations.) (ii) Population a more uniform environment will show greater heritivity. 3-1998 (i) Use the frequency of different 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) = 2 for simplicity, I will denote the allel frequency as: p(Si) = a p(Sy) = b p(Sg) = c Then the breakdown of genotypes is: (a+b+c)2 = a2 + 2ab + 2ac + b2 + 2bc + c2 = 1 icky yucky gross Frequency ikay and yucky slugs are compound terms and cannot be calculated directly. However, gross ball frequency = 0,2; c2 = 0,2, so c = 0,45 But b2 + 2bc = 0,3 (= phenotype frequency yucky slugs) Substituting the value c in this equation, we b2 + 0.9b = 0.3 = 0 Solving b, we 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 damaged and therefore the transcription of the lacoperone cannot be. (b) The operator has mutations, so lak respressor can not bind - transcription lac Z, Y and A will constitutsti high, regardless of whether lactose is present or not. However, catabol repression is still intact, so this constuitary transcription will only happen if glucose is not. (c) the transcription of lacZ and lacY will continue to be in normal inducable control; varnish product will not be produced on its own. (d) Depending on the type of missense mutation, the lacY product (lac permease) may be functional or non-functional. The mutation does not affect the transcription of all three lacquers. [Because lactose imports into the cell are responsible for importing lactose, the strain as an inducer may have a slower response to lactose than the wild type.] e) Stop codon at the beginning of lacY coding region could act as a polar mutation (ribosome will never get to the beginning of codon lacA) so that the cell will create no lac permease not lac transacetylase. (f) Without transcription of CAP varnish genes, activation cannot occur regardless of whether glucose is present or present, whether lactose contains or not. (g) Whereas phosphoenol pyreuta PEP is one of the glycolyses that inhibits the formation of cAMP (and thus blocks the activation of the CAP), the non-production of the PEP will reduce the inhibition of cap activation by glucose; lactose causes transcription of lacquered operatons even in the presence of glucose. 2. (i) Greatly high beta-galactosidase levels. i-p+ oc z+ - operator is damaged; cannot be suppressed. i+ p+ o+ z-(ii)Constitutional low. i+ p+ o+ z+ is p+ o+ z+ --super-repressor lacIs can work trans, to suppress the two lacZ alleles (iii) Constitutsi low (no transcription lac operon) i-p-oc z + --no transcription of this lacZ allele, because the facilitator has mutated i + p + oc z - --no expression beta-gal, because this lac allZ allle is mutele is mutele ir mutele Constitutsi high is p- o + z + - this lacZ is always off (not facilitator, super-repressor), but i-p + oc z + repressor can not be associated with this lacOc operator; This copy of lacZ is always expressed (oc is epistatic to is) (v) Constitutatively low (same as iii) i+ p-o+ z+ i+ p+ o+ z-3. a gal3c will be predominant, benefit from efficacy: GAL3 +/gal3c heterozygous, even if normal Gal3 protein is not binding gal80 (in the absence of galactose), mutant Gal3 protein can always bind and inactivate gal80 protein, regardless of whether galactose is present or not. (b) Recessed. The mutant alliant cannot provide Gal80-binding activity, but the usual alle can - heterozygous can react like a wild type. 4. (a) Example of a polar mutation : the mutation must be secreted to produce premature translation of the coding sequence of non-functional B proteins and gene C. (b) The main thing should be taken into account that different types of mutations can occur in any gene. The transcription activator can be switched off so that it is an activation failure, or it has mutations so that it always activates, even if it is not intended. Also, the repressor might have mutated so it never represses or so that it always represses. Reg gene product should be the regulator of the transcription operon ABC. It can be an activator or a repressor. Option 1 - reg is an activator of transcription. The mutant phenotype should be the result of a mutant activator that activates transcription anonimically (and inappropriately). The mutant phenotype is expected to prevail because even if normal protein is produced and transcription is activated if necessary, the mutant protein will always activate transcription. In this scenario, the mutant phenotype is never switched on to be a mutant activator protein that does not activate and this phenotype will be recessive. Option 2 - reg is a repressor of transcription. The phenotype must always be switched on by a recessive mutation enabling transcription if necessary; phenotype should never come from a dominant mutation that always suppresses transcription. Looking at the actual results, we see that data supports option 1 and there is no option 2: always the phenotype prevails and never on the phenotype is recessive. Therefore, the reg. no. 5. The mutation must be a zygotic gene - a gene product is only required after the first few chapters when the transcription of this gene begins in the developing embryo. 6. (a) nanos mutations are mutations of maternal influence: females produce eggs that lack nanos proteins for the homozygous mutation. As a result, the posterior segments of the embryo do not develop normally (the posterior of the embryo is where the nanos protein is usually localised). Commission 201 is fatal. (b) it is a zigotic gene; inability to produce hunchback protein resulting in a lotusu with a bow. This mutation is also fatal. 1. (i) Since both mutant strains had complementarity (F1 could be seen), mutations should be in separate genes; the simplest explanation is that there are two genes involved. This conclusion is confirmed by the F2 ratio that can be derived from the dihibrid ratio. (ii) The 9:7 F2 ratio indicates that we are dealing with a dihibrid ratio (fractions go in the sixteenth count). The 9:7 ratio can be derived from the standard 9:3:3:1 ratio, if we postulate the following – the offspring, which has at least one dominant alleole for each gene showing the dominant phenotype (normal vision) - giving 9/16 offspring with normal vision to offspring who do not have a dominant alle, exhibiting a recessive phenotype (blindness) - giving 7/16 blind offspring If we call the two D and E genes, the parents were ddEE and DDee; F1 offspring is DdEe (and can therefore be seen); F2 offspring are: D_E_ D_ee ddE_ ddee 9/16 normal vision 3/16 blind 3/16 blind 1/16 blind If F1 crickets were crossed to homozygous recessive crickets (i.e. DdEe x ddee) the offspring would be DdEe, Ddee, ddEe, and ddee in equal proportions - i.e. 1/4 offspring will be able to see, and 3/4 will be blind. (iii) As with any cross of the diichšva, a quarter of the offspring will be true to the males. 2. (i) As with any independent assortment of gene pairs, we can look at the relationship of two genes independently. As for the presence or absence of color (gene E), the offspring are 1/ 2 colors (black or brown) and half yellow. Therefore, the parents of the E gene were Ee and ee. In the case of black and brown (gene B), the brown parent must have bb and the other parent must have Bb (there must be at least one B allited to give black offspring; it cannot be BB or not brown offspring). [Another way to think about the absence of yellow offspring, half is brown and half black, so the parents are bb and Bb giving 1:1 B_ and bb offspring.] Thus, parents should have bbEe (brown) and Bbee (yellow). (ii) In the case of gene E, one quarter of the offspring show a homozygous recessive phenotype and therefore both parents should have heterozygous (Ee) E genes. As for gene B, again, the black and brown offspring are in equal proportions, so parents should have Bb and bb. Once again, the brown parent is bbEe; black parent must be BbEe. 3. This is an example of recessive epistastics. The fact that homozygous B and homozygous O rats could cause AB offspring, and the fact that F2 progeny fractions are in the sixteenth, tells us that it is a dihybrid cross--i.e., a second gene involved in addition to the IA/IB/IO (hereinafter abbreviated A/B/O) gene. A alliing that gets F1 must be homozygous o parents, but disguised by the effect of the second gene, thus giving O phenotype. (Why homozygous? Because if it were heterozygous, F1 offspring would show other phenotypes other than AB. Similarly, A alliling could not have been hiding b parents, because then B's mother would not be right for breeding.) In addition, it is the recessive alle of the second gene (which we call h, the dominant alle is H), which prevents the expression of the A/ B alle. (Why? Because if the masking allile was dominant, the F1 offspring would all express a masking phenotype, and all this would be O.) The recessive h alliling is epistatic to A and B. Thus, the parent B is BBHH and shows phenotype B; O parent is AAhh, who does not show a allel and appears to be O. F1 descendants of ABHh, and express both A and B alliles. A quarter of the F2 offspring are homozygous in recess (hh); they again seem O again, because the H allel is necessary for expressions A and B. The molecule to which these polysaccharides are added is the H-range on the surface of red blood cells. OO homozygous does not make polysaccharide, and is of the blood type O. But hh homozygous, even if they make A or B or both polysaccharides, is still o blood type, because part H is not performed; there is no where to add polysaccharide A or B. This type of O blood cell is often referred to as the Bombay phenotype because it was discovered in a patient in Bombay, 1952.] 4. Choice of Ade + revertant: plate ade-cells on the agar plates lacking adenine. Only Ade + Revertant will be able to grow and build colonies. Therefore, all yeast colonies forming these plaques must have a functional ADE gene. Screen: grow the cells as before and plate them to a medium containing adenine. All cells (ade- and ade + revertant) will be able to grow, but only Ade + revertant will be white colonies. Check each colony and pick out the white ones to get the Ade + Revertants. 5. Remember that allaises that are unable to complement each other (i.e. do not give a normal phenotype) should be one gene alle. In this example, there are three complement therapy groups (three genes) - Gene 1: p1 and r2 Gene 2: p2, r1, and r4 Gene 3: p3 and r3 (Half of the table is left blank because the filling would be superfluous - p1 x r3 is the same as, for example, r3 x p1.) 6. (i) Rescue of D and E (because, if provided, the E3 function will no longer be required); C accumulates (because there is no E3 convert C to D). (ii) E will be saved; D accumulates (iii) D and E will be saved; B accumulates. 7. (i) Conversion of B to D cannot continue, therefore, each rescue d and F (ii) Conversion A to B cannot continue, so B will be saved. 8. (i) Red pigment can not be made, so the flowers will be blue. (ii) Red flowers. (iii) Purple flowers (due to complementaration- F1 will be heterozygous for each gene). (iv) 9/16 violet: 3/16 red: 3/16 blue: 1/16 white. 9. Remember that the last gene mutation can only be saved by the final product; mutations in the next-to-last gene can be saved by the last two compounds en route, etc. Thus the pathway is: 10. i) Neither intermediate pyrimidine or thiazole saves more than one mutation. If these compounds are intermediates linearly, we would expect that one of them should save more than one mutation. (For example, in the 4th quarter, histidinolphosphate saves both M4 and M1 mutations.) ii (Thiazole rescues thi-1, so the problem with thi-1 must be the synthesis of thiazole; like the problem with ti-2 should be pyrimidine synthesis. thi-3 rescued only thiamine, so this is the final step, the point of convergence of both branches.) 11. 12. B is required for any color, so it is necessary to recalculate white - it is epistatic to A and C. A seems necessary to recalculate red intermediate orange - does A give red color, not orange. C is not necessary for pigment production, but rather, it seems necessary to prevent the production of pigment in part of the flower, keeping this part white. B- and C- are unlike the phenotype (color-free versus too many colors), so their interaction should be negative. By grouping it together, we can come up with at least two pathways, each of which can explain the data – one in which C regulates B directly, and one in which C works to turn some pigmented areas back to white. Clearly, in both models, there must be some other gene that controls which part of the flower is the gong to express C (to white) and which suppress C (to allow color). 13. (i) DNA synthesis requires CLB (any strain lacking CLB function is unable to perform DNA synthesis). (ii) Sic and CLB mutations have the opposite effect and CLB is epistatic to SIC, so SIC should be a CLB inhibitor. With the same logic, CLN is an inhibitor of SIC. An alternative path -- -- is possible, but the cln-sic-double mutant phenotype argues against it. This double mutant shows too much DNA synthesis. If the CLN requires clb function, as this indicates this second pathway, the dual mutant should not show DNA synthesis. So the data is the most consistent path at the top. 1. i. Patches may have occurred in a mitotic recombination. Recombination between the two loci would give the lonely spots a recessive phenotype over centromere-distal locus, while recombination between centromere and two loci would give twin spots. From this logic we can conclude that this locus must be closer La umer reser y la uo y la u (An alternative explanation for lone spots is mythotic nondisjunction, but it doesn't explain the twin spots.) (ii) As the twin spots and lone spots occurred in a relationship of 6:5, the centromer's distance and rd-b distance should be in an approximate 6:5: (iii) Lone spots from the rd phenotype may occur either from mitotic non-dissecting or mitotic recombination with a double crossover, one crossover between centromer and rd and one cross between rd and b. 2. The most psyth markers should be y and g. Because yellow and rough are seen in twin spots with each other, but not with mottly or rarely – so, the y and r genes must be present on one hand's chromosome. Similarly, m and g must be placed on the second arm of the chromosome. Therefore, the gene sequence on the chromosome is: y------r----------centromere-----m----------g |--7----------32----------6---------12 3. The strain described in the lecce was the dominant alleys for yellow and sang trans. If the dominant allaise is cis, the crossover between the centromer and theo genes may give a single spot containing both recessive phenotypes: 4. The map is that if the recombinant industry has a phenotype alone, then both and all other genes must be crossed at the crossroads; if the sector has phenotypes (a) and (b), (b) must be between (a) and all other genes, etc. The number of generations needed to give the final number of cells can be extracted from the expression: 2n = cell final _#, where n = number of generations) n = log(cell final _cell count)/log [2] (e.g. if cell final # = 16, 2n = 16; n = 4 For a tumour with 109 cells, n = log(109)/log(2) = 29.9 or 30 generations Cell division count = (end cell _ 1). [Note that the number of cell divisions is not the same as the number of generations of cells. In the first generation there is one cell that decomposes, so there is one division. In the second generation there are two cells that divide, so there are two divisions in this generation; in the third generation there are 4 cells that divide, so there are 4 chapters. So after three generations, there are 8 cells - but it was (Chapter 1 of the 1st generation) + (Chapter 2 of the 2nd generation) + (Chapter 4 3rd generation) = 7 chapters together.] 6. The tumour was obtained from a single cell containing one X chromosome inactivated; because inactivation X is stable by mitosis, all daughters of this cell have the same inactive X. 7. There must be more than one genetic change in the history of tissue culture cells. For example, cells had to go through a crisis to become immortalized, a process that was probably involved in some genetic changes. 8. A mutation that causes the erbB protein to behave as if it were bound to a growth factor, even in the absence of a growth factor, cells to start division in the absence of growth factor. If other regulatory mechanisms are also removed (due to unrelated events), the cell or its progeny may become malignant. 9a. In order for mutant alley a*to cause abnormal cell proliferation, it must be resistant to inhibition of protein B. Therefore, the mutant protein formed by this alleel will be able to promote cell proliferation regardless of whether the allule in the A gene is an ear or mutant - so* is a dominant, expressed mutation. (b) In this case, the mutant alliling should promote cell proliferation without blocking protein A. However, even if this alle is unable to perform functional protein B, other allusion (if it is wildtype) can still perform functional Protein B and block Protein A. Therefore, b* must be a recessive, function loss mutation. c) 2 x 10-5 (because there are two a+ alloles, and one of them the mutation would be sufficient to cause inadequate cell proliferation). 1. * Corrected * There must be two crossovers – one between B and D and one between F and G as shown: 2. (a) The result is unexpected because we only see older (non-crossover) products – we should see some recombinants. One of the parents must be in the form of a combination of products that are direct. (b) the fact that the chromosome material is stretched between the shaft poles and the ruptures indicates that a dicentric chromosome must be formed - which indicates that the inversion must be a paracentric inversion: Note that the gene order shown is arbitrary; the question does not provide information on the correct gene sequence or even which parent was inversion. All we can say is that there must be an odd number of crossovers with an inversion loop. 3. Harmful effects (gene loss or duplication that causes fertility loss) will be seen when there are an odd number of crossovers within the inversion loop. Here the b-d-e-f segment is inverted, so the part forms an inversion loop during the meiosis I prose; y-and g-h will remain out of the loop. (a) In the inversion loop has two crossovers, so the products will all be viable - a reduction in non-precious fertility. (b) Crossovers are both outside the inversion loop, so again, there will be no reduction in fertility. (c) Here is only one transition within the inversion loop, the other transition takes place outside the loop. This DCO event will result in gametes with gene deletion, and will be harmful as a result of

decreased fertility. 4. Male offspring are expected to receive Xsc from the mother, and therefore have scute bristles. This man has received an X+ from his father (the only source of the dominant alle at the crossroads). X-ray processing may have resulted in the final translocation of the X chromosome-bending sc+ so that the sc+ allile is Son. What about the second cross? If the translocation was autosome, the extraordinary son would have had some wildtype daughters. The fact that the wild phenotype is separated only by male offspring indicates that translocation should be on the Y chromosome. NOTE: Only gametes produced by aberrant (unexpected) offspring are shown across 1. Most gametes were normal, producing expected offspring. 5. (a) One way to differentiate hypotheses is to extract DNA from affected individuals and do a southern stain experiment with DNA by reducing it to EcoRI and probing the stain with a 2.5 kb fragment. As a control, this sample would be compared to the DNA of an unrelated person. Control DNA should be given a 2.5 kb fragment of southern stain. If translocation really removes the left end of the gene and replaces it with an unrelated fragment from another chromosome, it is unlikely (but not impossible) that this unrelated sequence will also have an EcoRI site in exactly the same location - so the affected person's DNA should show a fragment that differs from the usual 2.5 kb. (It is not possible from the available information to forecast exactly what size to expect.) In addition, the left end, which was moved to another chromosome, will be part of another EcoRI fragment in the new location. So the affected individual, rather than producing one 2.5 kb fragment, will probably produce two fragments of different sizes. If translocation does not break the growth factor gene (minority view), the 2.5 kb fragment should remain intact in all samples. (b) The same 2.5 kb probe could be used to conduct the FISH experiment by re-comparing cells from affected individuals and intact individuals. For non-patients the probe should hybridize only up to one chromosome (but rather homologous that chromosome) – for example, if the growth factor gene is chromosome 9, we should see hybridization on two homologous chromosome 9. In patients, part of the growth factor gene should be transplanted to another chromosome (according to the prevailing hypothesis). Therefore, the FISH experiment should result in hybridisation not only with chromosome 9, but also with other chromosomes involved in translocation. If the minority opinion is correct, patients as well as uncalled should be identified as one chromosome type. (c) The southern stain approach only determines the size of the fragments and not the chromosome locations. As indicated in point (a), it is likely that the displacement will result in exactly the same sizes of EcoRI fragments as the conventional chromosome. The result would then support minorityview even if the gene is really broken down by displacement. The FISH approach determines the location of the sequence chromosomes and is not Limit. Therefore, if it is done correctly (with a sufficiently large sample size - the number of cells tested), the FISH results could be more reliable. In fact, perhaps both tests could be done. Technically, the Southern stain is much easier. 6. (a)(b) There is only one way to extract XYY offspring from normal XX and XY parents (if nondisjunction occurs only by one parent) – male nondisjunction meiosis II produce YY sperm (marked by an arrow in the diagram). 7. a Kaliko model is the result of X-inactivation. Male mammals are not expected to demonstrate X chromosome inactivation, so this result is unexpected. Calico men are probably XXY cats resulting from sex chromosome nondisjunction in one of the parents. b) The first litter mom had was XrXr and dad was XRY. Mom could only broadcast Xr; The XR allusion must come from dad along with Y, so nondisjunction must have happened to dad. The second litter mom had XRXr and dad was XRY. A male calico kitten was able to get an XRXr from mom (ND mom) or XRY from dad (ND dad) – it's impossible to differentiate these options. 8. (a) Women should be worse than men. The deletion of XIC into the chromosome prevents the counting and inactivation of the X chromosome. For men, there shouldn't be X chromosome inactivation anyway, so deletion shouldn't be an issue. In the offspring of women, the effect will be that the X chromosomes will be insufficient. (The X chromosome missing in XIC is not seen as X.) Therefore, neither the X chromosome will get inactivated, which has harmful effects. (b) X with intact Xist will get inactivated – Xist works in cis, so only the chromosome produced by it will get inactivated. (c) The hypothesis is that this protein is produced in restrictive 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 must be enough protein to protect both the X chromosomes - so neither X should be inactivated. d) This is an example of a dominant function benefit mutation. The mutated allule of this gene will produce excess protein, so it doesn't matter if the normal alle is present also – the effect of excess protein will be seen anyway. 1-1998(i) the 38-year-old has a higher risk of Down Syndrome in children because the likelihood of nondisjunction during meiosis increases with the age of human women. (ii) A family history of Down Syndrome suggests that this may be the case with Translocation Down Syndrome – in which case, the younger woman (belonging to this family) has a higher risk of Down Syndrome baby (because the risk of a 38-year-old woman is about 1 in 100 – see pg 69 from lecture notes – but the possibility of translocation Down carrier having a Down baby is 1 out of 4). 2-1998 (i) Mother must be heterozygous G/g, but father had hemizygous normal G / (Y). Colorblind Turner Syndrome (XO) women must have derived from fertilization g egg with sperm lacking sex chromosome; nondisjunction could have occurred in meiosis I or meiosis II father. Colorblind Klinefelter (XXY) males must be derived from fertilization gg eggs with normal Y-bearing sperm; not dissed, which took place in place of the forestry II for the mother. (ii) Identical (monozygous) twins occur when the early embryo dissi pates such that each part develops into a single foetus. In this case, the message must be normal. The separation occurred in a two-cell phase; one of the resulting cells split normally, giving a normal twin, while the other cell had mythotic nondisjunction, giving Down syndrome twin. 3-1998 (i) F1 females should be heterozygous in all loci. We usually expect recombination in each interval, giving up to 26 = 64 different offspring phenotypes (in a relationship that would depend on card distances). As a general rule, only four phenotypes of the offspring are not expected. (It may be positive that the pairs of arcs are very closely related, but this does not explain the recombinant deficiency between the ends of the group.) (ii) Deletion could be ruled out because half of F2 men will inherit the X chromosome for missing genes and may not succeed in developing. Translocation is also unlikely to produce the observed results as phenotype reduces the observed recombinant offspring (translocation causes semi-standing but there is no reason not to come to recombinant (pull it out and confirm it for yourself). iii) A and B and between F and G and between F and G, suggesting that the whole part between A and G (i.e. B to F) is inverted. (iv) Different molecular tests are possible. For example, if the inversion is as predicted, you can create a Southern trats using probes for possible intersection regions. In this example, probes 1 and 2 will be hybridized to different deflation fragments (of different sizes) if the chromosome is normal. In contrast, if the inversion is shown, probes 1 and 2 will both hybridize to the same fragment. [Restriction enzyme sites are represented as vertical bands. Knowing the limit of the card for the entire chromosome, we can choose the limit of the enzyme, which is appropriately located in places as shown.] (v) Individual crosses are not expected to provide viable recombinant gametes. However, rarely double crossovers can be viable. In this case, a double crossover - one crossover between loci B and D and one between E and F - would give the observed result. 4-1998 When two T-allele bearing homologues are called T1 and T2, and two t-allele bearing homologues are t1 and t2, there are three possible sets of pairs, giving gametas shown: Pairing gamete genotypes T1T2 and t1t2 T1 homologous connected to T2 homologu, etc.) T1t1, T2t2, T1t1, T2t2, T1t2, T2t1 T1t1 and T2t2 T1T2, t1t2, T1t2, T2t1 T1t2 and T2t1 T1T2, t1t2, T1t1, T2t2 Gamete genotypes are TT, Tt and tt 1:4:1 ratio or 5:1 T_:tt If the offspring at tttt factories (whose gametes will all be tt), the progeny are expected to be T___:tttt (i.e. long and short) in a 5:1. the clyde must be given td, td, tD and td progeny in a relationship of 1:1:1:1. Instead, only older phenotype (TD and td) are visible. (ii) The absence of older types and semi-sterility suggest that the explanation may be a move. One possible configuration is shown: a side-model of segregation would give TT and Dd gametes, while a substitute model would give a TD and A TD. Tt and Dd gametes should be enviable, so the only viable offspring would be TD and td phenotypes – older types. Note that there is more than one configuration that matches the results. For example, the t and d allies do not have to be in the cover segment. 1. Match suspect case 1 is much more significant - alluses that are matched are much less common in the population, so the possibility of playing (i.e. suspect and crime scene DNA matching just because of the possibility) is unbelievable. In case 2, allies are more frequent, so the possibility of a match is more likely. So one might feel more confident finding suspect 1 guilty than finding a suspect 2 guilty. Math case 1: probability probability match = (0.01) (0.02) (0.003) (0.01) (0.07) (0.04) (0.13) (0.13) (0.04) (0.05) 32 = 1.1 x 10-14 -- i.e. the probability of obtaining this alle combination is only randomly about 1 in 100 trillion. Case No. 2: Probability that the odds correspond to the match = (0,2)(0,4)(0,15)(0,3)(0,3)(0,3)(0,2) (0,3)32 = 1,7 x 10-4 -- i.e. slightly more than 1 in 6000. (Factor 32 comes because there are two ways to get each heterozygous combination, and the 5 loci we are looking for = 25 = 32.) We'll see more of this factoring-in-2 business when we get population genetics.) 2. (a) Note: Your answer doesn't have to be this long wind! The strategy here is to look at the dominant feature and ask: does any allusion from polymorphism feature primarily separated by dominant rocks? (And, on the contrary, does the allusion, which is mostly separated by recessive allies? This issue is harder to address because this genealogy has seven sources of recessive alle - two copies of I-1, one copy of I-2, and two each of II-1 and II-5 - so it is harder to trace the recessive alle. By contrast, there's only one source of dominant feature – I-2 – so it's much easier to track.) The source of the dominant characteristics in this genealogy is I-1. We know that he is heterozygous for he has an intact 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 alleals is separated by the dominant feature (i.e. do people who show the dominant phenotype – and therefore inherited alle D – also get one of these four alleals mostly?) Let's look at the PS1 first. There are 11 affected (Dd) persons not including I-2. Of these, three have inherited an allel of 13 (II-2, III-3, III-5) and four have inherited an allel of 20 (II-4, III-10, III-12, III-14). The remainder has inherited neither 13 nor 20 (these individuals have inherited allales from people who marry in the family). So Alle D I-2 is co-segregated about half the time with an allince of PS1 13 and about half time with an allel of 20. Therefore, the PS1 does not seem to be related to D/d – the two loci seem to be separating independently from each other. Now let's look at the PS2. Here the alluses in the individual I-2 are 21 and 27. Of the eleven offspring of Dd, ten also have an allel of 21; only one is an alliol 27. In addition, of the six offspring of dd, only one has an allel 21; the remaining ones are other alla annexes. Therefore, it seems that in this genealogy, allahl 21 is mainly found with allel D; there may be two bows (D/d and PS2). In this particular case, I-1 must have allel D and alliling 21 PS2 for the same homologous (in cis, or amusement stage). (b) If we assume that the scenario we described above is true – i.e. D/d and PS2 are related, with ally D and 21 cis – then we can look for persons who have allel D, but not allel 21, or vice versa, missing allel D, but have alle 21 as evidence of the recombination. Two individuals meet these criteria: III-6 affected (Dd), but not allel 21, and individual III-9 is not affected dd, but has an alliling 21.3. There are 64 possible triplets and three of these (UAA, UAG, UGA) have to stop the codon. Therefore, in random DNA sequences, the chance to encounter a stop-coded in a particular reading frame = 3/64, or about 1 out of every 21 codons. So the average ribosome will encounter a stop codon of about 21 codons after the frame shift; the peptide will be 20 amio acids behind the point of the frame shift. 4. The control fraction (treated with sugar) crosses the offspring of male wild types, shall be the background speed of spontaneous mutagenicity. This rate is = 13 / (6255 + 13) = 0.002/generation We can now compare the mutation rate in 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 the 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 not mutagenic. Food colour #3: 91/(5382 91) = 0.017/generation - this rate is higher than the background rate, so food #3 mutagenic. 5. Ultraviolet light is mutagenic because DNA can absorb photons at UV wavelengths and thus undergo chemical reactions that it would not otherwise have. Therefore, the uv wavelengths that are most mutagenic must correspond to those wavelengths that are best absorbed by DNA, so the effectiveness of mutagen should be consistent with UV absorption with DNA (hard red line in the graph). 6. Remember that normal diploid cells have two copies of the gene enzyme E (Gene E) and two copies of the gene enzyme Z (Gene Z). Thus, assuming that the amount of enzyme in the cells is linearly with the gene copy number, each copy of the E gene promotes the activity of 30 units of the enzyme E (so diploid produces 60 units of the enzyme E), and each copy of the Z gene promotes the activity of 50 units of enzyme Z. So the cell line that is duplication of genes should produce three copies of worth of enzymes. For the enzyme E, duplication should cause cells that produce ~ 90 units, and the enzyme Z, the duplication of the gene should produce ~ 150 units. To find the location of Gene E, we are looking for cell lines that produce ~90 units of Enzyme E, and ask what is common between these backups. We see from the table that cell lines 1, 5, and 6 all produce ~90 units of Enzyme E (while other cell lines produce normal ~60 units). So these three cell lines are a duplication of Gene E. The group that is common to these three duplicaitons is a group 2 – which is the location of gene E. Enzyme Z cell lines from 2 to 6 producing ~150 units instead of the standard 100 units. The group, which is a common duplication on these lines, shall be Group 5; Gene Z should be located there. 7. (a) We expect the offspring to show the dominant phenotypes. In the case of offspring showing recessive properties, recessive alllets must be discovered by deletions in gamets produced by X irradiated male. (b) While we might postulate multiple deletions in each offspring class, the most parmeonious explanation is that each offspring class has a single deletion that reveals recessive allies at several adjacent loci. So, if two recessive properties are uncovered, the genes of these two features must be adjacent to each other in the chromosome. Using this logic – the strain #1 and c, so the gene and gene c must be neighbors the same way, and b must be neighbors; (a) be between b and c (order so far is b-a-c) f is adjacent to c, its order is b-a-c-f de is next f (but sequence d and e is not yet known) and from the strain #6, e is adjacent f, so the completed gene arrangement is b-a-c-f-e-d Modified from 1998 (a) Sectors of different sizes will arise depending on when the growth colony during the mutant event occurred – the earlier the mutation, the larger the industry. Half-lived colonies mutations in the first cell division that eventually formed a colony (e.g. if there was an unconfound discrepancy before the first layer of DNA synthesis, replication would result in one normal chromosome of the daughter that would cause the white sector; and one mutation in the daughter's chromosome, which would cause the red sector). b) The problem with measuring the frequency of mutations is to assess how much cell division has occurred. However, we know how many cells were made mutations to give the branches in the first chapter - that is the number of dilauated colonies. We also know how much the first chapter happened - it is equal to the number of colonies on the plate. Therefore, the frequency of mutations = mutation frequency in the first chapter = (number of colonies)/(colony count). 1. As for the disease, the boy is homozygous recessively (because achondroplasia prevails). If A = achondroplasia and = does not affect, the boy is aa. As for polymorphic locus, one allile has 12 repeats in CA, and the other has 7 repeats – so his genotype of polymorphic instead is 7.12. (Or 12.7.) Therefore, his total genotype of these two loci is aa 7,12.2a. The sample DNA is either linear (with one incision instead of Pst I, so one reduction breaks the linear molecule in two), or a circular with two cut sites (the first to reduce the linearis circle; the second cut breaks the linear molecule into two). (b) The conclusion of Sample A doesn't change – since it's cut into two fragments with Pst I, it must have at least one cut in place. Sample B, however, if it stays as a single molecule after Pst I treatment – so either it's a circle with one incision in place, as we concluded (a), or missing pst I cut the sites at all, in which case we don't have enough information to decide whether it's circular or linear. 3. a Note that digest ii) and (iii) give several fragments of the same size - shown here as thick bands. Note that different snippet sizes should always be added full length (20 kb in this example). In real life, if you saw two bands that didn't add up to full size (e.g. band ii – 7 kb band + 3 kb band = 10 kb instead of 20 kb), that would thread you that there could be several fragments of the same size. (b) The probe will be hybridised only for the fragments with which it overlaps. Again, some bands contain two different fragments of the same size, only one of which (in this case) would be hybridized to the probe. 4. a The full genome size must be the sum of the individual fragment sizes for any individual digested - for example, Ava I alone fragments are 12 kb and 48 kb, so the total is 60 kb. You should get the same answer from each digest. (b) Each enzyme itself gives two fragments. Therefore, each enzyme must have one incision point in the bacteriophageal genome, so that each enzyme cuts DNA into two parts. Ava I must be cutting 12 kb from one end (i.e. 48 kb on the other); Bam HI cuts 10 kb from one end and Cla I cuts 18 kb from one end. The question is who eventually we're measuring from – we know that Ava I cuts 12 kb from one end, while Bam HI could cut 10 kb from the other. For this information, we look at double digestion. Let's look at Ava I + Bam HI. We know that Ava in itself is going to create a 12 kb snippet and a 48 kb snippet. We now see in this double digest that Bam HI leaves 48 kb fragment intact – we still see a 48 kb snippet. In contrast, a 12 kb snippet released by Ava I is cut by Bam HI per 10 kb snippet and 2 kb snippet. Therefore, the Bam HI site must be 12 kb in Ava I. The map we have so far is: We can do a similar analysis of Cla I. In Ava I + Cla I double digest, we see that cla I did not cut within 12 kb AvaI fragment (because if it was cut during the 12 kb snippet we would see smaller than 12 kb fragments which we did not). However, Cla I did not cut within the 48 kb Ava I snippet to release the 30 kb snippet and 18 kb snippet. We already know that Cla I cuts 18 kb from one end of genomic DNA molecules – so there is only one way to place the Cla I site on the map as shown: The map predicts that Bam HI + Cla I double digest should be given 10 kb, 32 kb, and 18 kb fragments – which according to the information we have given true. 5. (a) The primers are: 5'-TGCTCTGGAT-3' and 5'-TCCGAGCC-3', corresponding to the yellow conscripted segments (immediately the side of the greyed segment) below: (b) The full length will be 46 bp (10 bp for each primer + 26 bp in the middle). Note added to 10/26/99: The way in which the question is formulated is actually possible to expand an even smaller fragment by selecting primers in the grey segment, as shown below: In this case, only the grey segment will be reinforced by assigning a product length of 26 bp. (a) Someone who is homozygous normal will have two identical copies of alleles that have all four Xba I sites – i.e. digesting their DNA with Xba I and hybridization with the specified probe should reveal three fragments, sizes of 3 kb, 5 kb, and 7 kb. In contrast, the carrier (heterozygous with one normal and one disease alle) will have one allel with 4 Xba I sites and one alliling that lacks one or two average Xba I seats (see table below). Their DNA, when cut and probed alike, will also pick up the same three fragments (3 kb, 5 kb, 7 kb) as one normal alle. However, other alliol will give different products,which will be visible in addition to normal digestive products (asterisks indicate absence of Xba I sites): genotype digestive 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 shown above, four different allies are possible - the usual alle (with all 4 Xba I seats) plus three allels are missing one or both Xba I seats. c) There are possible 10 genotypes - 4 homozygous and 6 heterozygous (see week 1, Q. 10 for explanation). 7. a Polymorphic spot allaal is dominant until dominant - both shapes are found when in a single test the allel composition of this site. (b) where the two bows are not linked, gametes of the different possible genotypes shall be equally reliable; eight possible progeny genotypes are equally likely as follows: (c) We are not provided with phase information here - i.e. we do not know whether the allel configuration father is {D 8 & d 18}, or {D 18 & d 8}. (Is mother alle we a matter of configuration?) Different results will be monitored depending on the phase as shown below. Mother-made gametes will be in d, 7 and d, 15 equal proportions, as in subparagraph (b). Phase (alle configuration) for father: {D 8 and d 18} {D 18 and d 8} Gameta 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're adopting a complete link, we can simply look at the genotype of polymorphic ceramide and assign the disease phenotype accordingly – homozygous (30, 30) = affected; heterozygous {30, 42} = intact, carrier: 9. (from 1998) Lod point graph tells us that genealogy data favor cards distance of 5 cM between Gene 1 and PS1; card distance between gene 1 and PS2; card distance between gene 1 and PS3 10 cM, etc. The map corresponding to these interpretations is as follows: Note: The answer to question 5 has been corrected in Box 10/19/99. For each crossover between two loci, two of the four products of melosis will be recombinant. Therefore, if 8% of the meioses are crossover in this interval, 4% of the products will be recombinant - its card distance is 4 cM. (If you are still confused, pass through the worksheet on page 40 of the lecture notes). 2. AaBb x aabb Products have a 1:1:1:1 ratio – loci seem sorti sorting independently, so we can't assign a link and can't determine the parent configuration. AaDd x aadd Here, AD and ad phenotype offspring greatly outnumber ads and aD – so AD and ads must have older aelic configurations (A and D are related cis). Recombinant forms (Ad and aD) account for 8 out of 200 = 4% of the offspring; card distance between A/a and D/d = 4 cM. AaFf x aaff Af and aF phenotype offspring greatly outnumber AF and af – so Af and aF must have older allelic configurations (A and F are associated with trans). Recombinant types (AF and account for 36 of 300 = 12% of the offspring; card distance between A/a and F/f = 12 cM. BbEe x bbee Be and bE phenotype offspring greatly outnumber BE and be – so Be and bE must have older allelic configurations (B and E are associated with trans). Recombinant types (BE and be) account for 10 out of 210 = 4.8% of the offspring; card distance between B/b and E/e = 4,8 cM. DdFf x ddff Df and dF phenotype offspring greatly outnumber DF and df – so Df and dF must have older allelic configurations (D and F are associated with trans). Recombinant types (DF and df) account for 20 of 250 = 8% of offspring; card distance between D/d and F/f = 8 cM. B/b and E/e are in a separate tying group. Link relationships can be displayed as follows: D F |--------|----------------| 4 cM 8 cM B E |---------| 4.8. cM 3. Parental genotypes are TTFF x ttff dot TtFf. Therefore, parental genotypes of gametes by F1 plants are TF and tf. If both bows are unbound, we expect four phenotypes of offspring (TF, TF, tF and tf) in equal proportions. Since there are 1,000 proteny total, we expect 250 of each phenotype if the loci are unrelated. If both bows are linked maps at a distance of 44 cM, we expect that 44% of gamets will be recombinant - i.e. 44% of the offspring should show recombinant (non-parental) phenotype. As shown above, parental types are TF and TF, so we expect 44% of offspring to add up to TF and tF, or 22% each. Parental types should then be 56% of offspring = 28% each. So for 1,000 offspring, we expect 280 for each TF and tf, and 220 for each TF and tF. Clearly, the observed offspring figures do not correspond to any scenario. So let's do a chi-square analysis of the two datas sets, on two sets of expectations, and see if we can find statistical evidence against either model. Scenario 1 – loci is an unrelated 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 above 0.05 – just above the standard cutoff to dismiss the zero hypothesis (that deviation from the expected is just because of the possibility). Scenario 2 - the bows are related to 44 cM Phenoti 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 it mean to decide between the two types of inheritance? Statistical analysis shows that the data is consistent (just barely) with any model - so we can't decide between the two models statistical test. At least two approaches are possible to resolve this issue. One is simply to collect more data (repeat crosses, count many more offspring) and repeat 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 related, it should be possible to find genes in the interval between those that are associated with both. In this way, we like to work in smaller card distances, and thus have a better shot at creating a link. 4. The disadvantage is that F1 offspring, although heterozygous for sneezing and annoying, are homozygous itching. So while recombination between sneezy and annoying can be detected, there is no way to detect recombination involving itching. ... no change in genotype (ijs and i + + giving i + + and ijs) Note: i = itchy, j = annoying, s = scratched; only one chromatide per homologue is displayed, he should be completely heterozygous (ijs/+++ in any cis/trans configuration) and homozygous recessive (ijs/ijs) for his mapping cross. Assuming that the that we start with the dominant alleys cis heterozygous (i.e. +++/ijs), then older, double crossover and single crossover products can be predicted as follows: Gamete type Gamete genotype (= offspring phenotype) Estimated offspring number of DCO i + s and + j + = (0,18) (0,12) (1000) = 22 total; In the i-j range of each SCO 11 + j s and i + + = (total number of recombinant in this range) - DCO = (0,18) (1000) - 22 = 180 - 22 = 158; For each SCO interval + + s and i j + = (total number of recombinant in this interval) - DCO = (0,12) (1000) - 22 = 120 - 22 = 98; 49 of each NCO (parental) + + + + and i j s = total - (all recombinant) = 1000 - (22 + 158 + 98) = 722; 361 out of every 5. Correct gene sequence finding H = Hairy, P = Violet, T = Spike; and lowercase letters denote recessive phenotypes. Parental non-reflection (NCO) allatel combinations are Hpt and hPT (they are the most abundant offspring phenotypes), while double crossover (DCO) classes are HPT and hpt. To find the correct gene sequence, we begin with the known types of NCO and see if double crossover gives known types of DCO. If not, order is wrong; we try another gene in sequence (critical information is the gene in the middle). The trial and error (attempting each of the three genes in the middle) determines that H should be in the middle of the gene: Products of single crossover (SCO) P-H interval are pht and PHt. (SCO classes were changed. --10/19/99) Now we can start calculating card distances: P-H card distance = percentage recombinant in this interval = (SCO P-H) + DCO) as a percentage of the total number of offspring = (150 + 132 + 18) / 2500 = 300/2500 = 0,12, or 12 cM. H-T card distance = percentage recombinant in this range = (101 + 81 + 18)/2500 = 200/2500 = 0,08 or 8 cM. Completed card P/p H/h T/t |------------|--------| 12 cM 8 cM reproducible factor = (observed DCO/(Expected DCO) Observed DCO = 18 Expected DCO = (0,12) (0,08) (2500) = 24 Reproduci factor = 18/24 = 0,75.6. It is important to remember that in order to map genes, we must be able to detect recombination, and that in order to detect recombination, one parent must be completely heterozygous. Here, the genes are on the X chromosome – so that mothers by default are female (a man has only one X – there is no recombination there). There are several ways to build this up. One option is to make female heterozygous, and there are recessive allules to the male X chromosome. Then men and women will consist. To create heterozygous females, we could cross homozygous dominant females (++++/+++) with recessive males (abc/Y); females in offspring. When these females are crossed by abc/Y males, the offspring (males and females) should exhibit uncombined phenotypes (abc and +++) as well as 6 recombinant types: a++ and +bc, ab+ and ++c, a+c and +b+. Another option is to cross heterozygous females with males with predominant phenotypes (+++/Y). Then the female offspring all show the dominant phenotype, and it will be ignored; male offspring could receive one X from a woman, and show the same older and recombinant phenotype listed above. For example, see questions 1998–2 on questions from the evening year. 7. The only human chromosome common to all cell lines forming enzyme Q is chromosome 8, so that it must be a chromosome containing the gene enzyme Q.8. The G enzyme Cell lines that make this protein have common chromosomes that are common to cell lines, are as follows: 2 and 9 Cell line C is chromosome 2, but does not produce protein. Therefore, the G enzyme gene should be in chromosome 9. The enzyme AD Chromosome, which is common to the cell lines that make this protein, is: 5 and 14 E cell line has chromosome 5, but does not start protein. Therefore, the gene enzyme AD must have chromosome 14. The H-enzyme Chromosomes, which are common cell lines that make this protein, are: 2 and 9 Cell line C is chromosome 2, but does not make a protein. Therefore, the H enzyme gene should also be in chromosome 9. 1998-1 (A) Parenting are XoDXoD and XOdY. The cross is set out below; children are expected to be intact in female and mesh albino males in a 1:1 relationship. (b) Here we know that a woman has heterozygous both qualities – but we don't know if the dominant alleals are cis (i.e. the dominant O allel and the dominant D allogue in one homologous) or trans (in different homologos). The crucifixion man has the dominant allies of both loci, so his daughters will all be phenotypicly normal. But the son's phenotype will depend on what X chromosome they inherit from a woman, and whether she is the dominant alle of a cis or a trans. Except that the procedure is the same as before – you only use male offspring to follow the recombination that took place in female parents. (If you are confused – DRAW A CROSS! You know that genes are on the X chromosome; you know the parental genotipus.) Parental types (the most abundant male offspring) are s+sn+fu and s sn fu+. DCO products are s + sn fu + and s sn + fu. Therefore, s locus should be in the middle; older types can be rewritten as sn+s+ fu and sn s fu+. One crossover between sn and s would give sn+s fu+ and sn s + fu; one crossover between s and fu would give sn+s+fu and sn s fu. Now we can calculate the recombinant type percentage for each interval: # crossover sn-s range = SCO(sn-s)+DCO=(99+91)+(21+17)=228 percent recombination with B-A interval = (228/1000)*100=22.8# crossovers with s-interval fus = SCO (in s-fu) + DCO = (69 + 75) + (21 + 17) = 182 Percent recombination in A-C range = (182/1000)*100 = 18,2 a) genotype of female mother = sn + s + fu / sn s fu + (This notation- set of allies, then slash /, then another allel set - is a standard notation, which shows that the first set of allel is on one homologue and the second allaal is in the second homologue after a slash.) Genotype of male parents = sn+ s+fu+/Y b) Map of the region: sn------22.8 cM-------s------18.2 cM----fu |------------------------|-------------------| c Predicted DCo product # = (0,228)(0,182)1000 = 41 Observed DCO product # = 38 Coincidence Factor = 38/41 = 0.927 Disorder = (1 - 0.927) = 0.073. Human chromosomes in cell lines that do not have insulin sequences can be excluded from our list of possible candidates. Therefore, any chromosome in cell lines D, E or F can simply be removed from the list of options (excluded candidates listed as coloured boxes). Cell line Human insulin sequence present? Human chromosomes in cell line Yes 6 7 10 11 14 17 17 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 ones in cell lines A, B, B, and C are chromosome 11. Therefore, the insulin gene should be on chromosome 11. 1997-4 (a) I-1 does not affect, so he is XGHY. His daughter inherits his X chromosome, so one of her X chromosomes must have XGH. However, she is a colorblind, hemophilia son, so her other X both recessive allies. Therefore, II-1 is XGHXGH. Her husband (II-2) and son (III-1) are both colorblind, but not haemophilia, so they both have XgHY. III-2 is both a disorder; he is XgHY. III-3, which is colorblind, must be homozygous recessive color of visual 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 of 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 hereditary XgH from her father; she inherited either Xgh or XgH from her mother. Two genes have 3 card units apart, so we expect 3% of II-1 gametes to be recombinant. Taking into account phenotype III-3, the only possible recombinant gamet is XgH; probability is 0.03. Therefore, the probability of her being H/H is 0.03. Also, the probability that she has H/h is 0.97. Because she does not show a recessive hh phenotype, it is impossible (p = 0) that she has h /h. 1. a) haploid figure N = 9; its 2N = 18. In metaphase, the chromosome should contain two nursing chromatides, so the total number of chromatides = 18 x 2 = 36.b As in mitosis, there should be two chromatides in each chromosome, i.e. 36 chromatides (but the chromosome position was different from mitosis). (c) In Anaphase I meiosis, homologue alone - as a result, the daughter cells are the only haploid set of chromosomes in each. Therefore, the number of chromatides of mealytic metaphase II = 2 x hafloid chromosome count (N) = 18.2. The diploid form, which has two sets of chromsomes, can undergo meiosis. Haploid form is the only one that starts with, so it can't pass a re reductional breakdown. 3. a Homologues are separated - so it must be a meiosis. b Sister chromatides are separated and there is one copy of each homolog – so it must be a mythotic distribution. 4. a Here are three separation features: G/g, A/A and X/Y. In addition, as we are looking for a son, sperm will have to have a Y chromosome bearing one. Therefore, the semen genotype should be gaY. (c) We know that the final genotype should be gaY. Therefore, anaphase I, Y chromosome is segregated by homologues in the recessive alle, as charted: Note: In the interest of simplicity, crossing more is ignored here. The relative size of the chromosome and gene location is also fictitious. 5. (a) Use of XH and Xh in the case of X-chromosome displays with normal and haemophilia alle, six possible matings are: XHXH & XHY XHXh & XHXh & XHXh & XHXh XHY XhXh & XHY XHXH & XHY XHXH & XHY XhXH & XhH & XhY (XhY) XhHHH & XhY (b) For the daughter to be a carrier, she must have heterozygous XHXh (if she were XhXh, she would be affected herself, but she would not be considered a carrier). So another way to point out the question is – Which of these matings are all daughters heterozygous? Two possible matings could give this result: XHXH & XhY XhXh & XHY Other mating could bring heterozygous daughters also – but daughters wouldn't be all heterozygous. c) The genotypes of children are XhXh (the affected daughter) and XHY (intact son). Since the daughter received Xh from each parent, the father must have XhY. The mother sent one hemophilia alle (to the daughter) and one normal alle (to the son) - so she is the carrier, XHXh. So the older genotypes are: XHXh (mother) and XhY (father). 6. The affected children are intact parents, so the disease can not be dominant (assuming full expressiveness and penetrance). Women and men are affected, so it cannot be limited by gender or related to Y. If it is assumed that the disease is quite common, then it could be autosomal recessive. However, given that there are far more affected men than affected women, the most likely explanation is that it is an X-related recessive. Alternatively, it could be gender-influenced – dominant in men, but recessive for women. 7. Again, the affected children are unafunsusable parents, so the feature can not be dominant. It could be autosomal recessive. If so, then we should assume that the disease is quite common, because heterozygous should be introduced into genealogy in five separate cases (I-1/I-2, II-2, III-8, IV-2 and IV-7). In addition, only men are affected by theis genealogy, arguing against a simple autosomal recessive model. The fact that only men are affected by this genealogy is indicative of a sex link. But the affected men have intact sons, so it is not related to Y. It could be X-related recessive - but the trait seems to have been passed from the father to the son in one instance (IV-8 to V-6). So it could be X-linked recessively only if the IV-7 is a carrier. It might also be gender-restricted (phenotype expressed in men), but as with autosomal recessive, we have to accept that the disease is prevalent. And as #6, it could be gender-influenced, dominant for men, but recessive for women. Because it is possible to explain this genealogy either as autosomal recessive/gender-restricted (if the disease is prevalent), or as X-related recessive (if the disease is relatively rare), we cannot conclude anything about how common or rare the disease is from just genealogy. We might as a matter of parsimony say that the most likely type of inheritance is X-linked recessively or gender-influenced, but leave open the possibility that it is autosomal recessive or gender-restricted if the disease turns out to be 8. The fact that Phenotypes F1 are skewed in terms of sex immediately suggest that the trait is related to sex. The feature is not passed on to father-to-son (F1 men are normal), so it can't be Y-related. This leaves an X-related legacy. F1 males get their X chromosomes from older women. That f1 males say that older females must have homozygous normal alle. This means that F1 women must be heterozygous (normal x from mother and squiggly-eye X from father). But these heteroozygous F1 women are all squiggly-eyed. Therefore, the squiggly-eye phenotype should be X-related dominant. The F1 x F1 cross would give a squiggly eye to women, squiggly eye men, normal women, and normal men in a 1:1:1:1 ratio, as shown below: where S = squiggly-eye and + is normal 9. The main thing here is realizing that because they have an independent assortment of features, we can look at each feature individually – (a) The cross here is AABbDdee x AaBbddEe. We are asked to calculate what proportion of offspring will be phenotype ABde. Since they are independently assortment features, we can calculate the fraction of the offspring, which will be phenotype A, then the fraction, which will be phenotype B, etc., then multiply these fractions to obtain a fraction that has all the desired phenotypes. Thus - AA x Aa --> all offspring will phenotype A Bb x Bb -> 3/4 offspring will phenotype B Dd x dd --> 1/2 offspring will phenotype d ee x Offspring 1/2 ee --> 1/2 will be phenotype e Therefore, the progeny fraction, which could be a phenotype ABde, is (1)(3/4) (1/2) = 3/16. Offspring Aa Bb x Bb --> AA x Aa --> 1/2 progeny genotype will progeny Aa Bb x Bb --> 1/4 genotype bb Dd x dd --> 1/2 progeny will progeny dd ee x Ee --> 1/2 progeny Ee Therefore, the progeny fraction, which could be the AabbddEe genotype, is (1/2) (1/4) (1/2) (1/2) = 1/32.10. On the dihybrid cross, we expect to see the 9:3:3:1 ratio of phenotype offspring – clearly not the case here. Because nothing is mentioned about men versus women, we have to accept that it is not a gender-related gene. To solve the puzzle, so we can start by looking at each phenotype separately and see if it helps. The observed offspring are a sneak white, sneak yellow, normal white, and normal yellow chickens 6:2:3:1 ratio. Let's look at sneak vs. normal apart from yellow vs white. When we do this, we see that the ratio is 8 sneak: 4 normal, i.e. 2:1 sneak: normal. Hmmm. Where have we seen a heterozygous x heterozygous cross that gives a 2:1 ratio before? It's good when sneak prevails over normal and creeping is fatal when homozygous, we get 2:1 crawling sneak ratio: normal offspring. How about white compared to yellow? Here the ratio is 9 white: 3 yellow, simple 3:1 ratio. Therefore, white is dominant and yellow is recessed. Putting it all together, the cross is CcWw x CcWw (where C = sneak, dominant, c = normal, recessive; W = white, dominant, w = yellow, recessive) and CC offspring die: 11. A common mistake is to misinterpret the question of believing that the first two children are boys – although in fact, all we know is that at least two children (in any order) are boys. If the child's gender is written in the order of birth as B (boy) or G (girl), perhaps 3-children in a family with at least 2 boys are: BBG BGB GBB BBB Only one of the four possible sets are all three children are boys – so if you know that two children are boys, the probability that all three are boys is 1/4.12. We use binomial distribution to solve this. Because it is a recessive disorder, and both parents are heterozygous, the probability of the affected child is 1/4. Therefore, let a = probability does not affect the child = 3 /4, and b = probability of the affected child = 1 / 4. The equation is then (a+b)6 = 1 a6 + 6a5b + 15a4b2 + 20a3b3 + 15a2b4 + 6ab5 + b6 = 1 For a family with exactly 2 affected and 4 intact children, we use the term 15a4b2 (exponentials indicating a = unaffected and b = number of children affected). Replacing the probability of intact and affected children, we: p (2 affected children) = 15a4b2 = 15 (3/4) 4 (1/4) 2 = 1215/4096 = 0.297. To be able to use at least two affected children, we could use: 15a4b2 + 20a3b3 + 15a2b4 + 6ab5 + b6 But the easier way is to find the probability of less than two affected children, then subtract this 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. A longer expression of 15a4b2 + 20a3b3 + 15a2b4 + 6ab5 + b6 will give the same result.) 13. (a) This is a dihybrid cross, we expect a 9:3:3:1 ratio long purple: long white: short purple: short white. For 3200 offspring, the expected figures are: long, purple: 3200(9/16) = 1800 long, white: 3200(3/16) = 600 Short, violet: 3200(3/16) = 600 Short, white: 3200(1/16) = 200 b) Expected phenotype (E) Observed (O) 2/E long, violet 1800 1784 0,142 long, white 600 620 0,67 purple 600 612 0,24 Short, white 200 184 1,28 Chi-square value = 2,332 df = 3 Fordf = 3 (i.e. three degrees of freedom) chi-square = 2,332, P value is slightly above 0,5, which is well above the standard cutoff of 0,05 to reject the zero hypothesis. Therefore, the zero hypothesis (that the deviation from the expected values is purely by chance) cannot be rejected. 14. What are the options here? Option #1: The cross was a homozygot violet x homozygous violet; there must be no offspring of white #2: the cross was heterozygous x heterozygous; 1/4 offspring should make white flowers. If the seed trader picks only one seed at random and grows it up, and it makes white flowers – she knows it's been heterzygote x heterozygous cross. However, if she picks one seed and it makes a purple flower plant – can she then say that it's been a homozygous x homozygous cross? No, because even in a heterozygous x heterozygous cross, 3/4 offspring will be purple, so she has a 3/4 chance to choose a purple offspring, even if white offspring are present - i.e. she has a 1/4 (=0.25) probability of missing a white offspring. Let's say she cuts two seeds? Then the probability that both will be purple (if it really was a dihybrid cross) = (3/4) (3/4) = 9/16; The probability that she has missed the white offspring of the plant has dropped to 7/16 = 0.4375. So, it's a question – how many seeds would be on his sample if she wants the probability of accidentally missing white flower seeds fall below 2%. In other words, she has a sample of n seeds like 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 purple flowers, there is < 2% probability that white flower seeds are present but missed just due to the possibility. 15. In order to know the probability that IV-1 will be affected, we need to know the genotypes of parents, III-4 and III-5. In turn, we need to know their parents' genotypes and so on. Since I-1 and I-2 are not affected, but have an affected daughter (II-1), both of them must be carriers of the Dd genotype (where D = dominant, unaffected; d = affected, recessive). II-3 is D_, with a 1/3 chance of being DD and a 2/3 chance of being Dd. II-5 and II-6 are both Dd (because they are intact but have the affected son, III-9). III-4 is not affected; the only way she can have an affected child is she is heterozygous Dd. What is the probability of that? She (III-4) is a father who has a DD and a mother who has a 2/3 chance of being Dd. Also, III-5 has heterozygous Dd to get their child affected. The probability that III-5 is heterozygous Dd is 2/3 (he could have a DD or DD, with a 2/3 chance of being in DD – as with II-3). Therefore, the possibility that they will have an affected child = (1/4)(1/3)(2/3) = 1/18. Responses to the selection from 1998-1 (i) The disease is probably not autosomal recessive – there are several cases where people marry in the family have affected children; people get married should all have heterozygotes, improbably scenario. (ii) Genealogy fully matches the autosomal dominance when I-1 is heterozygous and 1-2 is homozygous normal, just as everyone marries in the family. (iii) X-associated recessive can be excluded as the affected unsusemerable fathers (e.g. II-1, IV-3). (iv) X-related dominance may also be excluded because the affected men have intact daughters (who will inherit the X chromosome, which carries the dominant disease alle from the father).- for example, article II-5. v, (vii) Males and females are affected, therefore the disease is not associated with Y or sex. vi) Gender-influencing heritage has two options – dominant for men and recessive for women, or for women to dominate, and recessive women. Affected women have intact sons (e.g. I-1 and II-3), so they cannot be reusable women and are dominated by men. Similarly, affected men have intact daughters (e.g. II-5 and III-6), so they cannot dominate women and are reborn in men. Thus, the type of inheritance that best explains the observed genealogy is autosomal dominant. 1998-2 The disease is released for generations, so it is not dominant. The disease is rare, it is unlikely to be autosomal recessive - it would be necessary heterozycous marrying in the family at least twice. Males and females are affected, so it is not related to Y or gender. It cannot be influenced by gender because intact parents have affected children. It can't be X-related recessively because the affected daughter is an intact father (from which she got an X). This leaves us either with a rare chance of heterozygous marrying (for an autosomal recessive), or some kind of aberrant event, or some kind of inheritance we haven't dealt with yet. 1998-3 As described in the lectures (see part of the evidence of accidental segregation of mesos homologous), meiosis in women, which homozygous on an X-linked white alle) can give four types of gametas, because two X chromosomes can pair up during the synapsis, or X and Y – in which case the lone X could be separated either by other X or by Y. Some of these eggs can cause fertile red-eye males and white-eye females, secondary exceptions. NOTE: The grid above shows only the types of offspring that can be formed, not relative numbers. Since the synapses of two X chromosomes may be more reliable than the X synapse with Y, meiosis I Y is an odd result (see diagram above) is more reliable than X's odd oversized. Outcome. Therefore, gamete types 1 and 2 are much more abundant than gamete types 3 and 4, and offspring numbers are skewed accordingly. 1998-4 Since it is a heterozygous x heterozygous cross (normal = dominant, albino = recessive), we expect a normal 3:1 ratio of albino children - i.e. normal probability of a child is 3/4 and the probability of the albino child is 1/4. a Probability of the described result = (3/4)(3/4)(1/4)(1/4)(1/4) = 9/1024 b) Probability 2 for normal and 3 albino children in any order can be calculated using binomial expansion. a = p(albino) = 1/4 and b = p(normal) = 3/4; there are five children, the equation to use is: a term that represents the probability of 3 albino and 2 normal children is 10a3b2. Replacing the values a and b, we get: p(3 albino, 2 normal) = 10 (1/4)3(3/4)2 = 45/512 = 0.088 c) Probability that all five will be normal, is: (3/4)5 = 243/1 24 = 0,237 (d) p(at least one albino) = 1 - p(from albino) = (1 - (243/10 24)) = 781/1024 = 0.763 1.a) True-breeding long = TT True-breeding short = tt TT x tt --> Tt heterozygous tall plants F1. (b) F1 plants are Tt heterozygous (see above); the cross is shown: as seen from the F2 genotype ratio, half of the offspring should be Tt heterozygous, and half homozygous (TT and tt). Therefore, if there are 1000 F2 progeny, 500 of them must be homozygous (TT or tt) - i.e. for true breeding. c) As this is a cross of the test, the known parent must be homozygous for deepening (tt). F1 consists only of tall plants, so the unknown must be homozygous TT; a cross is displayed. (See below why it can't be heterozygous Tt.) d) Tt x tt --> 1:1 Tt long and tt short plant expected. 2. (a) Parents and offspring are tall; the only crosses that would give this result are: TT x TT --> TT high F1 plants or TT x TT --> TT (high) and Tt (high) F1 plants b) Long and short offspring are seen in a 3:1 ratio indicating that the cross should be heterozygous x heterozygous monohibridu cross: Tt x Tt --> Long and short plants 3:1 in relation (1 TT long: 2 Tt long: 1 tt short). (c) the long and short offspring are shown in the 1:1 ratio; it must be heterozygous x homozygous recessive as in paragraph 1(d): Tt x tt --> Tt (long) and tt (short) in a 1:1(d) offspring content is only long; as in paragraph 1(c), the cross must be TT x tt --> Tt (long) (e) Short plants must be homozygous recessive (tt); therefore the cross is tt x tt - > tt short plants only 3. The only way a long plant can give a short offspring after selfing (i.e. mating with yourself) is if a tall plant is heterozygous. So what question is the question is: what fraction of tall plants are heterozygous? (For these questions, see the crosses in answer 2.) Note: You're not looking for tall plants that give only short offspring after selfing (is that even possible?) – you're looking for tall plants that will give any short offspring for selfing. (a) if the parental cross is TT x TT, the plants obtained will be homozygous TT (see therefore none of these plants should give short plants by themselves. If the cross is TT x Tt, the offspring are TT and TT plants in equal proportions, so half of these offspring will give short plants after selfing. b) In this crucifix, the high number of offspring is 1 TT: 2 Tt. Thus, 2 /3 offspring are heterozygous and will give a short offspring by themselves. c) Here the offspring are Tt (long) and tt short all long offspring are heterozygous, and all should give short plants after (d) all progeny are Tt; all of them should be given short plants after selfing. 4. F = free hanging ear, f = attached to the ear. With two couples in generation I, we don't know which person has free earlobes and which is attached, so I've chosen to show them as sex vague (but one member of each couple with attached earlobes). The iii-generation child is again uncertain of sex, but has attached lobe, and therefore has a homozygous recessive ff. Parents in generation II must be heterozygous Ff. 5.a) FF x ff ff x Ff x ff (b) FF x ff -> Ff only -- i.e. 100% offspring are heterozygous (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) Normal parent is homozygous. If the normal wing phenotype was dominant, the offspring would all show a normal phenotype. However, there are curly wing flies in the offspring. Therefore, the curly wing (C) must prevail over the conventional wing (c). In addition, two phenotypes (curly and normal) are visible in F1 and 1:1; therefore curly wing parents must be heterozygous. The cross can be represented as: Cc x cc --> Cc and cc 1:1 ratio (b) The cross is: Cc x Cc --> 1 CC: 2 Cc: 1 cc Of those homozygous curly (CC) descendants die leaving 2 Cc: 1 cc. Therefore, the offspring of true breeding (homozygous) make up 1/3 of the survivors. 7. The ratio of one ballast - double blood DNA molecule (if each A is paired with T and every C to G) would be 1.0.8. so it's a proabably (but not necessarily) double-iron. Assuming this is the case, C = 19%, G = 19%, also. So (A+T) = 100 - (C+G) = 62% T = 62/2 = 31% (jo A = T and A+T = 62%). 9. Two T alloles, 2 t alle. 10. Alle abbreviating as A, S, E and C- There are 10 alle combinations: AA SS EE CC AS SE EC EC AE SC AC Four of them (top row) are homozygous. Answers to the 1998 selections The simplest approach is a trial and error method: interpret each cross one by one and see if your interpretation is consistent with the interpretation of previous crosses. First of all, it is clear that there are three phenotypes, so for only simplicity I'm going to give them 3 alle labels (R, B, W, red, blue and white) and assume that they are allusions with the same determining factor. I may need to revisit this initial hypothesis later – for example, this may be the case for incomplete dominance between the two alleches – but at least for starters, I'm going to assume a simple dominant/recessive interaction. Cross (a) - Red #1 - gives a 3:1 ratio of red and blue flower plants to the offspring. It looks like a typical heterozygous F1 cross, with R being dominant and B recessive. So I'm tentatively giving Red #1 genotype RW. Cross (b) - Red #2 - similarly suggests that R is dominant W; W, C:\genotype should be RW. Cross (c) -- Blue selfed - gives a 3:1 ratio blue: white; blue must be predominant above white and the blue flower plant genotype must be BW. At this point, we have a hypothesis for all genotypes: Red #1 = RB Red #2 = RW Blue = BW White = WW (because it is recessive, although others) We can now predict the results of the remaining crosses, and see 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) -- 3:1 ratio red- to blue bloom, which is actually the observed result. Cross (e) – Red #1 x Blue – would be an RB x BW that should give a 1:1 ratio in red: blue (draw Punnett squares if you're not sure about it). Again, this is what we see. Cross (f) - BW x WW must provide 1:1 blue and white Cross (g) - WW x WW gives only white flower offspring. So our original hypothesis seems to sound as far as we can tell from the data provided. We can predict the results of the cross (h): Red #2 x blue = RW x BW: R W B RB (red) BW (blue) W RW (red) WW (white) -- 2: 1: 1 ratio red: blue: white. AO x BO BO