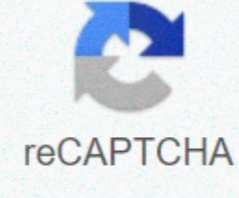




I'm not robot



Continue

Genetics practice problems

The answer keys about these problems run on a somewhat complicated scale from the very simple to the problems of each week. The intention is to use you to solve the problem. Questions on homework and exams will be on the complex end of the scale. For samples of homework/exam problems, look behind the course notes package and here. Lectures 35 1. A colony of black cats produces a total of 400 kittens a year. One of the kittens gets out for a major mutation, giving the kitten the baby blue polka dots. Is there a possibility that wild type allele will be lost due to genetic drift in the next generation? 2. There are 400 members in a separate colony of the same species of cats, of which 396 show polka-dot phenotypes. This colony merges with a large colony with 546 polka dotted out of 600 members. What will be the frequency of black cats in the next generation? 3. In another colony of these monstrous cats, the frequency of plain black cats is 0.25. (i) If the colony is infected with a deadly virus that kills only polka-dotted individuals, what would be the frequency of black eyl in the next generation? (ii) If the virus kills only plain black cats, what will be the frequency of black cats in the next generation? (Let's say the virus has disappeared by then.) 4. A population since 1998 is completely homoijigus for Aile D. However, D can mutate D at the rate of 0.00012 per generation, while D mutates D at the rate of 0.00004 per generation. The population is otherwise in Hardy-Weinberg situations. (i) Given the infinite time, which Ellen do you think will eventually prevail in the population? why? (ii) the change in frequency of D per generation? How about changes in frequency of D? (Remember that for each part, you need to take into account both further mutations and back mutations.) (iii) What will be the two alleal frequencies when they reach equilibrium (increase in frequency of D by further mutation matching the deficiency due to back mutation)? 5. You have to think about... (i) 250 members in the population of 1000 persons are identical ineffective BB. What is the estimated number of BB and BB individuals if this population shows hardy-weinberg frequencies? [That's the easy part.] (ii) and now the actual question: The actual number of BB persons was found to be 220 and the number of BB persons was 580. If you wanted to do a chi-square test to see if these numbers correspond to hardy-weinberg predictions, what value would you use for DF (#of degrees of freedom)? why? Lectures 36 and 37 6. One of the problems using DNA tests to screen for carriers of cystic fibrosis is that there is only ~70% success rate in detecting carriers in the test (because there are many different mutant alleles in the population). For the following questions, assume that the frequency of carriers = 0.05. (i) If all Matrimony Partners are checking for carrier status, what excerpts of heterozygote/heterozygote. (that is, heterozygote x hetrozygott is not recognized as pairs). (ii) If only one member is tested in a couple, and heterozygote is found, what is the probability that both the members of the couple are heterozygotes? (iii) If one member is found to be heterotropic and another member is also tested and found that the disease is not allies, what is the probability that both the members of the couple are heterozygotes? 7. A young boy has acute familial hypercholesteriemia because both copies of the LDL receptor gene contain promoter mutations that block their transcription. Family physicians are well aware of the use of bile acid binding resins in the treatment of hypercholesteriemia, but he tells the family that in this case the treatment will be ineffective. What was his (correct) argument? 8. As a last resort in treating a major disease, some clinical researchers decided to use an antisense formulation to block disease gene function. Mutant Eli (marks the asterisk mutation) and two possible antisense constructions (Build 1 and Build 2) are shown. Which of these two constructions is a better chance to succeed as an antisense creation, and why? Quiz section lecture 30 #3 for 1, #8 and #13. In Drosophila, both torso and FS are maternal influence genes that produce homogeneous females embryos with phenotypes that lack tail structures. If you were given the strain of Drosophila that showed this phenotype (i.e., women produced tailless offspring), how would you determine which gene was mutated? 2. What changes in the expression pattern of Crepel and Norps would you expect for a mutant that has high levels of both bibide and nanotechnology? (Let's say that the increased transcription of the hunchback corresponds to the increasing blockage of the cochback translation.) 3. The second thoracic segment in Drosophila is believed to produce wings, while the third thoracic segment should produce halter (flight balancer). A new homotic mutation causes the mutation 1 wings to develop on the second and third thoracic segments (no rein anywhere), while a second mutation causes mut2 to develop on both segments (no wings anywhere). Depending on what you know about setting segment identities, tell us if you expect each mutant phenotype to be effective or ineffective, and why. Lectures 31 4. For each of the following symptoms, state that phenotype-genetic variation or environmental factors are more important in determining: characteristic herability (i) by Thomson's gazelle 0.6 (ii) sunflower in seed size 0.65 (iii) turtles in penis (female vs. male) 0.1 (iv) egghel thickness in snapping 0.4 5. In the city of metropolis, all schools are equally good, are objectively encouraged in mental functions, and all Environmental factors relevant to IQ tests are consistently good; That means there is no variation in the environment within the metropolis about school education. In Gotham City (in the same country), all schools are equally poor, learning is never encouraged, and all environmental factors relevant to taking IQ tests are consistently unfavourable; That means there is no variation in the environment within Gotham City. (i) what is the percentage of intelligence within the metropolis and within the city of Gotham? To explain. (ii) What could be the reason for differences in IQ score while comparing between Mahanagar and Gotham city? Lectures 32 Questions 6-9 are reproduced from lecture notes (before page p.133). 6. Assume that the height in a plant is controlled by two gene pairs and each additive elial contributes 5 cm to the base height of 20 cm (i.e., aabb = 20 cm). (i) What is the height of AAB plant? (ii) Predict phenotypic ratio of F1 and F2 plants in a cross between Aabb and AAbb. (iii) List all genotypes that give birth to plants that are 25 and 35 cm in height. 7. In a cross where three gene pairs determine the weight in squash, in which proportion of individuals of Cross ABCCC x AABCC will only have 2 additive alleles? Do genotype(s) fall into this category? 8. An inbred tension of plants has an average height of 24 cm. A second strain of the same species from a different country also has an average height of 24 cm. F1 plants are also 24 cm higher than a cross between these two strains. However, the F2 generation shows a wide range of heights; The majority are like P1 and F1 plants, but about 4 of the 1000 are only 12 cm higher, and 4 36 cm more than 1000. (i) What mode of inheritance is taking place here? 2 How many gene pairs are included? (iii) To what extent each gene contributes to the height of the plant? (iv) Indicate a possible set of genotypes of P1 and F1 plants that can explain their heights. (v) Indicate a possible set of genotypes in the account of 18 cm or 33 cm high F2 plants. 9. Plants can be 10, 20, 30, 40, 50, 60 or 70 cm high where the plant height is under polygenic control. A 10 cm true breeding plant crosses a 50 cm true breeder. How many gene pairs are involved? Can F1 and F2 results be predicted? Lectures 33 10. In a certain population of 20 million people, an autosomal ineffective characteristic in 500 individuals, dissatisfied intolerance is found. Assuming that this number reflects the Hardy-Weinberg frequencies, how many individuals in the population are expected to be carriers of the speciality? 11. Island iguanas come in two varieties, beach love (major phenotype) and bridge love (ineffective phenotype). The frequency of bridge lover iguana on an island is 0.04. On a neighboring island that has a similarly sized population of iguanas, the bridge-loving frequency of iguanas 0.16. One day some bridge lovers iguanas build a bridge between two islands, so now iguanas can move freely between the islands. (i) Suppose the iguana on each island is exclusively with iguana from another island, what will be the frequency of the bridge-loving iguana in the next generation? How about the generation that followed? (ii) Suppose, instead, the beach-loving iguana on one island was only with beach lover Iguana on another island, and the bridge-loving iguana on an island only got matched with the bridge lover Iguana on another island, what would be the frequency of beach lover and bridge lover iguana in the next generation? 12. What is the relationship between genotype frequency and allie frequency in women versus men for the ineffective characteristic associated with an X? 13. Pattern baldness is an autosomal feature in humans that reflects the inheritance affected by sex - it is prominent in men (BBBB and BBBB men become bald) but ineffective in women (only BBBB women become bald). In a certain population that exhibits hardy-weinberg frequencies, 9% of women become bald. (i) What percentage of men goes bald? (ii) What would be the frequency of Ail BB among men in the next generation? Questions from old 1-1998 diagram is a series of crosses that will allow you to screen for maternal effects mutations. For each of the following pairs of population 2-1998, the state which will show you high heritability (in the broad sense) for the characteristic T, and why: (i) a population that is mostly identical to the gene that controls characteristic T, or one that is mostly odd (ii) in a population that is mostly in similar environments in relation to factors affecting characteristic T, or which is in more heterogeneous environments. 3-1998 The frequency of an ineffective elyse in the population is 0.2. Assuming that major and ineffective Allils are in hardy-weinberg frequencies, the following predict the possibilities of each of the mating: (i) Homojigus major x samosadar major (ii) odd x homogenous ineffective (iii) odd x odd 4-1998 [Warning: Warning requires actual alzara [horrors!] You choose to examine the aesthetic appeal of common Seattle slug. The aesthetic appeal in the slug is determined by an autosomal locus slim, on which there are three alleles. Allils are Icky (SI), Yucky (Sy), and Gross (SG). Icky is dominant on both yucky and gross. Yucky is dominant at Gross. After a long walk through the rain, you see that the phenotypes of the slug are in the following ratio: 50% are icky, 30% are yuki, and 20% are gross. (Don't worry about statistical error in the measurement of these numbers.) Assuming alles are in hardy-weinberg ratios, what are allele frequency P(SI), P(SI), and P (SG)? Which slugs would you move on instead, and why? #2, #4 and #6 for quiz Lectures 27, 28 1. What phenotype would you predict would result from each of the following mutations in E. coli? In each case let's say the cell is otherwise wild type. (a) Lac - (Promoters removed) (b) Lakhosi (c) Lacs (c) Lacs - (Lakha removed) (d) Lacy - (Missens mutation) (e) Lacy - (Stop coding near the beginning of genes) (f) Cap - (Cap gene delete) (g) Eno - (eno - phosphoral is required for synthesis of pyruvate) 2. For each partly biloved stress shown below, indicate whether beta-galactocytase activity will be persuasion (normal induction), less constitution (i.e., less in the absence of appearance or inductive), or constitutional high (high in the absence of appearance or inductive). Give a brief explanation for each answer. Suppose glucose is never present. (i) i-p+oc z+i+p+o+z-(ii) i+p+o+o+(iii) i-p-oc z+ (iv) p-o+ z+ i-p+1-p+oc z-p+oc z+oc z+oc z+oc mute operator to prevent the binding of oppressive; Is the mutation resulting in a super-suppressor that cannot be uprooted from the operator; P mutations prevention transcription initiation from the promoter; OC mutations prevent the operator's binding of oppressive, and Z-mutations cause the production of non-functional beta-gal. 3. You have heard in the lecture that galactose can neutralize gal80 proteins, allowing transcription of galactose-orbital genes. In fact, Gal80 does not directly respond to galactose; Rather, in response to galactose, Gal3 binds proteins and neutralizes Gal80 proteins. State do you expect to give a major or ineffective phenotype to each of the following GAL3 mutations: (a) gal3c: Binds and disables Gal80 whether galactose exists or absent (b) gal3: unable to binding to Gal80 protein 4. The diagram below represents an unspeakable operand in a certain bacterium; A, B and C represent three structural genes. The control elements contain promoters, operators, etc. Control element gene A gene B gene C ----- (a) A mutation in Gene B results in a deficiency of functional proteins B and C, but protein A is functional. Explain this phenotype. (b) Regulatory mutations were found in a separate gene reggae. The reg-1 mutation results in a high expression of operan ABC; The Reg-2 mutation results in a constitution low expression of ABC. Partly the use of diploid bacteria suggests that reg-1 phenotype is dominant, while reg-2 phenotype is ineffective. Offer a mechanism for regulating the oper ABC. Lectures 29 5. A certain mutation in toad xenopus is fatal. The examination of fetal development shows that early development is indistinguishable from normal and wild types, but the development stalls after the first few cell divisions. Based on what you know about the development in Drosophila, a suggestion For the delayed effect of xenopus mutation. 6. Predict the phenotype of the following homojigus faucet mutation in Drosophila. (a) Nano - (b) Hunchback - (Here suppose it is a purely zygotic gene) Also try: 1998 Problem Set 5, Q. 6 (Behind lecture notes) (Some of these are again from 1998. There are only two lectures scheduled for 9 weeks, so the lecture will be included in questions 27 questions week 9.) Quiz section #3, #5, and #12 for Lecture 24 1. Two true breeding, blind strains of crickets were crossing each other. F1 crickets all showed normal vision. When these F1 crickets crossed each other, the resulting F2 progeny included 178 crickets with normal vision and 142 blind crickets. (i) Are the strains of two original parents mutated in the same gene? If not, what is the mm number of genes involved? (ii) Explain the results (why F1 crickets can see, and why this ratio of F2 phenotype was achieved). What progeny ratio would you expect if F1 crickets completely surpassed homogenous ineffective cricket? (iii) Which part of F2 would you expect true breeding? 2. In a certain breed of dogs, B and B determine the color of black (dominant) and brown (ineffective) coats respectively. Ineffective elil e of a different (independently categorized) gene blocks the expression of both B and B alleles, giving a yellow coat. (The major E Eli quote does not affect color.) Determine the parent's genotype (give cause) for each of these crosses: (i) Brown dogs gave x yellow dogs - 1/2 yellow pups 1/4 black pups 1/4 brown pups (ii) brown dogs gave x black dogs - 3/8 black pups 3/8 brown pups 1/4 yellow pups 3. A new species of great Northwestern pygmy rats was discovered in some caves near Ellensburg. Analysis of blood samples showed that like humans, these Ellensburg mice came in A, B, AB or O blood types; Like humans, blood type is determined by the addition of a type or B-type sugar to a protein on the surface of blood cells. When scientists crossed a true breeding B bloodtype rat with a true breeding o rat, however, they were surprised to learn that the offspring all had AB blood types. F2 progeny received by crossing these F1 AB mice to a second had the following blood groups: 3/16 there were a 3/16 b 3/8 the AB 1/4 were o explain the result, using Punnett squares to describe your answer. Tip: Are the results similar to a monohybrid ratio or a diybrid ratio? Lectures 25 4. As you have heard before, a common ADE gene in yeast cells (i.e., ade + cells) can grow in the absence of adenine in the medium, and produce white colonies on agar plates. Cells that are ade-require adenine in the medium, and produce red colonies on plates. A yeast strain that a frameshift in ADE gene Given (gene non-functional rendering), identify the identity of revertants for a selection and outline of a screen There is a functional ADE gene. 5. A laboratory in Seattle is interested in understanding the genetics of flower petal evolution in petunias. After mutations on the plant and screening of mutant products, they identify three homogeneous mutant strains that fail to make all petals; They call these mutant strains p1 through P3. Meanwhile, they hear from their friends at Sedro Woolley, who had similar screens and identified four ineffective mutant alleles r1 through R4 that also failed to make flower petals. laboratories exchange strains and cross between different strains; The ability of progeny to make petals (+) or inability (-) is indicated: p1 p2 p3 r1 r2 r3 r4 p1 -r1 -r2 -r3 - + r3 - + r4 - R4 - How many genes are represented in this collection of mutant stress? Which mutants represent allies of the same gene? Why is half the table left empty up? Lectures 26 6. The following is the pathway for the synthesis of E, an essential metabolite of yeast: Which compound or compound (between A-E) will allow the development of yeast deficiency -- (i) Enzyme E3? (ii) Enzyme E4? (iii) Enzymes E2 and E3? You think which compound will accumulate in each of the above mutants? 7. The following branch represents the synthesis of passages E and F, two essential amino acids in a certain mold. (i) Name two compounds (between A-F) which will allow the development of mutants lacking enzyme E3. (ii) Name of a complex that will allow the development of mutants with enzyme E1 deficiency. 8. The purple flower color in a plant species requires the conversion of a white precursor to the red pigment by enzyme E1 and for the blue pigment by enzyme E2. The combination of two pigments gives the purple color, as indicated: (i) What would be the phenotype of a plant homozygus for a zero allele of genes for E1? (ii) What would be the phenotype of a plant homojigus for a zero oil of genes for E2? (iii) What would F1 phenotype(s) be if (i) crossed the plant (ii) in F1 phenotype(s)? (iv) What would be the F2 phenotype and ratio if F1 plants cross each other? 9. Yeasts that were able to synthesize amino acid histidine were mutantized and incapable mutants in histidine biosynthesis were isolated. [Note: The original, normal stress that may increase in the absence of added histidine, is called prototropic for histidine, while mutants are oxtroph. The mutation fell into four complementary groups, the M1-M4. The ability of various compounds to protect the growth of mutants is shown when added to the minimum development medium (+ indicates development, - indicates a lack of growth): Hastidine L-Histidinol Phosphate L-Histidinol Imidazole Acetol Phosphate M1+ M2+ + M3 + M4+ - - A pathway for biociness of its tidine. (M1 - M4 to denote the gene represented by these mutations Use. 10. 10. The table shows the ability of various mutant strains of neurospora that grow at moderate reduction on the intermediate medium in the deficiency of thiamine (vitamin) or synthesis of thiamine. Then, + indicates growth, - indicates a lack of growth. The complementary mutation was none thiamine pyrimidine thiazole-1-+++tha-2-+--tha-3-+-(i) Why do these data support the pathway by a branch for the synthesis of thiamine instead of a linear pathway? (ii) Propose a route for biosynthesis of thiamine. (To denote the genes represented by these mutations was-1. thi-2 and use thi-3.) 11. A plant that usually produces purple flowers was mutantogized and the following phenotypes of tap mutations were observed: mutation phenotype p-blue flowers R-red flower p-r-blue flowers propose a genetic pathway for the color of flowers in this plant. 12. A separate plant produces flowers that are part white and part orange. Phenotypes of various tap mutations are indicated: mutation phenotype A- red and white flower C- completely orange flower A-B- completely white flower A- completely red flower B-c- completely white flowers suggest a genetic pathway for the color of flowers in this plant instead of orange and white B. 13. The general regulation of DNA synthesis in yeast depends on three genes, CLN, CLB and SIC. Phenotypes of various zero mutations are indicated: Mutation Phenotype CLB- No DNA synthesis CLB- Excessive DNA synthesis CLB-SIC-no DNA synthesis CLN-SIC-excessive DNA synthesis Clan-CLB-no DNA synthesis [Note: In real life, experiments will be complicated by the fact that these mutations will probably have fatal consequences. Don't let this happen that you're upset.] (i) Which gene is absolutely required for DNA synthesis? (ii) Propose a genetic pathway for regulation of DNA synthesis in yeast. [Some of these ovens are less than fresh — they're recycled from 1998, for crunch time. My apologies.] Quiz section #2 questions for Lecture 191, #3, and #9. In the fruit fly, black body (B) and low hairding (RD) are ineffective alleles of linked autosomal genes. In a strain that was B+/- rd, it was noted that flies sometimes showed small patches of ineffective phenotype — mostly black bodies and lone patches of twin patches — next to black-body patches less bristle patches. The lone black patch and twin patch occurred in a ratio of 5:6. (i) Explain how patches occurred, required with pictures of chromosomes and multiples (including centromere). (ii) Prepare a map of the chromosome showing the relative distance between centromere and each of the two genes. (iii) Close examination of flies revealed rare, lone patches of low colic. Suggest two different mechanisms do not include point mutations by which these lone patches might arise. 2. Mold (e.g. aspergillus) grows as disk expansion, new developments are being made On the outer edge of the colony. One consequence of this pattern of development is that myotic recombination, instead of giving patches in Drosophila, causes the area shown below. Genes for mold color (y += normal, Y = yellow, ineffective), colony morphology (R + = normal, R = rough edges, ineffective), growth density (g + = normal, g = sparse, ineffective), and highfall texture (M+= normal, M = mold, ineffective) are known to be added to one such mold. A haploid strain that shows all four common phenotypes, all four shows ineffective phenotypes. When the resulting diploids were grown, most colonies showed major phenotypes, but some areas of ineffective

