

COURSE PACKET

**Bio 100
Sections 18 & 19
Dr. Jamie Jensen**

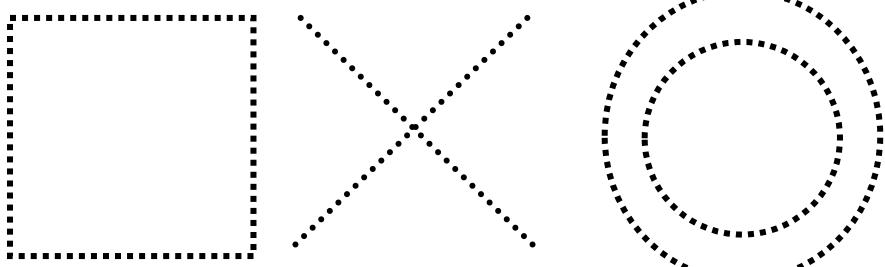
Fall 2021

This packet contains student guides that will be used in class. Please bring it with you to every class period.

Writing in a Mirror

Directions:

Obtain a small mirror. With one hand place the mirror against your forehead, facing down (as shown in the illustration to the right), and with the other hand, trace the shapes below:



Now, write your name on the line below so that it appears correctly IN THE MIRROR.

Expected Learning Outcomes
Bio 100 Fall 2021

Day 1	1. Explain the basic structure of the course. 2. Explain why the course is taught the way that it is. 3. Make observations about natural phenomena.
Day 2	1. Construct plausible hypotheses to explain a phenomenon. 2. Design controlled experiments to test hypotheses. 3. Identify and control variables in an experimental situation. 4. Draw conclusions from data. 5. Outline the basic steps of the scientific method: Puzzling Observations, Causal Questions, Hypotheses, Design Experiments, Make Predictions, Conduct Experiments, Results, and Conclusions. 6. Be able to determine the independent and dependent variables in given experimental scenarios.
Day 3	1. Explain why nothing can ever be ‘proved’ in science. 2. Distinguish the difference between correlation and causation. 3. Evaluate the reliability of evidence. 4. Distinguish sources of evidence: primary, secondary, anecdotal. 5. Evaluate whether or not questions can be answered using science
Day 4	1. Derive the chi-square statistical test equation 2. Create a null hypothesis 3. Use probabilistic reasoning to define a p-value 4. Determine statistical significance using a p-value 5. Determine where on a sampling distribution a test statistic will fall given the statistical significance 6. Use the word “theory” in scientific terms 7. Distinguish a theory from a hypothesis and understand that theories guide the formation of hypotheses. 8. Develop a hypothesis from a theory and likewise identify the theory behind a given hypothesis.
Day 5	Practice – Unit 1
Day 6	1. Determine, from data, whether a given feature is a homology or an analogy (convergent evolution). 2. Explain the principle behind the Law of Superposition. 3. Use evidence (fossils, morphology/anatomy, embryology) to support the theory of evolution. 4. Describe the basic structure of a chromosome, including centromeres and telomeres. 5. Compare and contrast chromosomal features to reconstruct evolutionary history. 6. Explain the significance of vestiges and atavisms in support of the theory of evolution. 7. Identify a trait as a vestige based on an evolutionary history. 8. Clearly state the position of the Church of Jesus Christ of Latter-day Saints on evolution.
Day 7	1. Build a graphical depiction of relatedness.

	<ol style="list-style-type: none"> 2. Distinguish between a monophyletic and paraphyletic grouping. 3. Read and interpret a phylogenetic tree. 4. Use data to build a phylogenetic tree using principles of parsimony. 5. Construct a character matrix. 6. Use a character matrix to determine relationships and construct a phylogenetic tree. 7. Manipulate a model (rotate nodes) to construct alternative forms of a given tree. 8. Determine closest relatives using a tree. 9. Determine the history of a trait (homology v. convergent evolution) given a tree. 10. Explain that all organisms have been evolving for the same amount of time, such that one species is not ‘more advanced’ or ‘more evolved’ than another and one species does not ‘evolve into’ another.
Day 8	<ol style="list-style-type: none"> 1. Use evidence to distinguish species. 2. Differentiate between the morphological, phylogenetic, biological, and ecological species concepts. 3. Understand mechanisms of reproductive isolation. 4. Be able to evaluate the most likely reproductive isolation mechanism in a given scenario. 5. Distinguish between allopatric and sympatric speciation. 6. Predict evidences that would support or not support separate species. 7. Apply species concepts to human race. 8. Interpret data to determine which hypothesis about the evolution of the modern human races is most supported.
Day 9	<ol style="list-style-type: none"> 1. Contrast human skulls and chimpanzee skulls 2. Evaluate different hominid species to determine how closely related they are to modern humans 3. Summarize the evidences that support the relationships of <i>Homo sapiens</i> relative to the extant great apes and to the main extinct hominids. 4. Use chromosomal evidence to support our relationship to great apes. 5. Synthesize an argument for what makes humans unique.
Day 10	<ol style="list-style-type: none"> 1. Predict an outcome of natural selection given a set of circumstances. 2. Predict ecological circumstances given an outcome of natural selection. 3. Hypothesize a potential cause of a change in a population. 4. Use differences in reproduction to determine fitness. 5. Explain how changes in the environment do not cause evolution, but that changes in the environment select upon pre-existing variation. 6. Apply a natural selection explanation to an authentic situation (e.g., Pigweed, Antibiotic resistance). 7. Identify patterns of selection (i.e., directional, stabilizing, and diversifying/disruptive)

Day 11	<ol style="list-style-type: none"> List the postulates (or conditions) of Natural Selection that are necessary for evolution to occur (via this mechanism). Explain the importance of each postulate of the theory of natural selection, and how a violation of this postulate will influence the course of selection. Predict how patterns of variation will change given the violation of each of these postulates. Explain how natural selection is the result of unequal reproductive success. Evaluate hypotheses for the evolution of sexual reproduction. Hypothesize reasons for sexual dimorphism. Use sexual selection to hypothesize mechanisms of speciation.
Day 12	<ol style="list-style-type: none"> List the three components of life history strategies. Predict the effects of environmental stochasticity on life history behaviors. Define terminal investment and use data to defend why it occurs. Explain the relationship between size and quantity of offspring. Hypothesize evolutionary causes of animal behavior.
Day 13	<ol style="list-style-type: none"> Hypothesize mechanisms for the evolution of symbiotic relationships. Determine symbiotic relationships based on ecological circumstances (i.e., cooperation, mutualism, selfishness, predation/parasitism, commensalism, altruism, spite, competition). Predict positive, negative, or neutral effects on species given a symbiotic relationship. Predict genetic relatedness of organisms given a symbiotic relationship. Distinguish between intraspecific and interspecific relationships Predict ways in which predator/prey relationships can influence the evolution of each player. Identify each symbiosis in humans.
Day 14	Practice – Unit 2
Day 15	<ol style="list-style-type: none"> Use the knowledge that for every gene, an individual possesses two alleles and that sometimes one allele is dominant and one allele is recessive, to solve genetics problems. Hypothesize genetic explanations for inheritance patterns. Analyze phenotypic and genotypic data and determine parent genotypes. Predict offspring based on parental genotype and/or phenotype. Use the terms homozygous, heterozygous, dominant, recessive, genotype, and phenotype correctly. Describe human sex determination.
Day 16	<ol style="list-style-type: none"> Derive the Hardy-Weinberg equation. Determine probabilities of each genotype and phenotype given the proportion of alleles in a population.

	<ol style="list-style-type: none"> 3. Determine allele frequencies given genotypes/phenotypes in a sample of individuals. 4. Determine the change in allele frequencies that will occur in a population over each generation. 5. Use the Hardy-Weinberg equation to determine allele frequencies in a population given a genotype and/or phenotype frequency and vice versa (assuming the population is in Hardy-Weinberg equilibrium).
Day 17	<ol style="list-style-type: none"> 1. Predict the outcome of the violation of each of the given assumptions of Hardy-Weinberg. 2. Predict which assumption has been violated given allelic frequencies of two populations. 3. Apply the Hardy-Weinberg equation to real-world scenarios to hypothesize evolutionary causes. 4. Compare expected and actual frequencies to hypothesize evolutionary causes.
Day 18	<ol style="list-style-type: none"> 1. Draw the process of meiosis. 2. Explain where and how the law of independent assortment and the law of segregation are fulfilled by meiosis. 3. Use the principles of meiosis to predict offspring outcomes. 4. Determine ploidy number (e.g., diploid and haploid) of a cell based on a figure or description of the cell. 5. Justify why gametes should have half the number of chromosomes as the parent cell. 6. Determine all possible gametes that can result from a given parent.
Day 19	<ol style="list-style-type: none"> 1. Predict genotypes given phenotypes of family members, from word problems and pedigrees. 2. Solve problems about traits expressing complete dominance, codominance, and incomplete dominance. 3. Identify and predict genotypes for traits with more than two alleles. 4. Perform a monohybrid and dihybrid cross, using Punnett squares and/or multiplication rules. 5. Assess the mode of inheritance for a given trait, using pedigrees or family history.
Day 20	<ol style="list-style-type: none"> 1. Solve problems about traits expressing sex linkage. 2. Solve problems where traits are linked on the same chromosome.
Day 21	<ol style="list-style-type: none"> 1. Explain the process of gamete formation in males and females. 2. Predict genetic results of in vitro fertilization using polar bodies. 3. Determine how to increase the odds of becoming pregnant. 4. Determine the parent in which nondisjunction occurred, and when during meiosis or mitosis nondisjunction occurred, given a genetic outcome of the daughter cells or offspring.
Day 22	<ol style="list-style-type: none"> 1. Compare and contrast meiosis and mitosis. 2. Diagram the key steps in each phase of the cell cycle and in each phase of mitosis. 3. Predict a meiotic or mitotic event in a life cycle given the purpose. 4. Predict when and how cancer might occur in the life cycle of a cell.

	<p>5. Explain and contrast proto-oncogenes, tumor suppressor, and repair genes; predict the effects of a mutation in each.</p> <p>6. Determine at which checkpoint a cancerous cell has experienced a mutation.</p> <p>7. Predict the effect of lengthening telomeres.</p>
Day 23	Practice – Unit 3
Day 24	<p>1. Compare and contrast viruses and bacteria.</p> <p>2. Distinguish between gram positive and gram negative bacteria based on a diagram and/or stained slide.</p> <p>3. Identify the different shapes of bacteria and assign the appropriate name.</p> <p>4. Predict the outcome of antibiotic treatment based on pathogen.</p> <p>5. Predict whether an illness is caused by a lytic or lysogenic viruses.</p> <p>6. Identify enveloped and non-enveloped viruses from a diagram.</p> <p>7. Predict how the immune system will respond to both types of pathogens.</p> <p>8. Determine the cause of symptoms for each type of pathogen.</p> <p>9. Predict the mechanism behind vaccines based on immune system function.</p> <p>10. Predict the mechanism behind autoimmune diseases based on immune system-pathogen interactions.</p> <p>11. Make an informed decision about vaccine use.</p>
Day 25	<p>1. Identify and describe the function of key organelles and cellular structures.</p> <p>2. Discriminate prokaryotic and eukaryotic (plant & animal) cells by discussing their similarities and differences.</p> <p>3. Predict the type of organism (prokaryote v. eukaryote; plant v. animal) given estimates of cell size and structure.</p> <p>4. Explain the structure of the membrane in relation to its proximity to water and predict how it will facilitate the movement of substances into and out of the cell.</p> <p>5. Predict solute concentration based on the movement of water into and out of the cell.</p> <p>6. Explain the difference between passive (both simple and facilitated diffusion), active, and bulk transport.</p> <p>7. Contrast hypertonic, hypotonic and isotonic solutions and predict what happens to cells immersed in each.</p>
Day 26	<p>1. Analyze data and make inference about gut microorganisms on human health.</p> <p>2. Analyze data and draw conclusions about the effect of antibiotic disturbance on diversity and abundance of gut microflora</p> <p>3. Predict the outcome of food consumption on diversity and abundance of gut microflora.</p> <p>4. Evaluate the interactions between the gut microbiome and human immune system development.</p>

	<p>5. Analyze the interactions of organisms in gut microflora and judge how positive and negative interactions change the distribution and abundance of species.</p>
Day 27	<ol style="list-style-type: none"> Predict results of reactions based on enzyme specificity. Identify energy reaction curves with and without an enzyme present and calculate the change in overall energy. Determine experimental outcomes based on the molecular make-up of each of the macromolecules. Identify the name of the monomer and polymer of each macromolecule. Analyze different items of food and evaluate which macromolecules are found in each and in what proportions using the 4-4-9 rule. Determine causes of discrepancies between calculated calorie count (using 4-4-9) and actual calorie count listed. Predict what happens when we have too much or too little of each macromolecule. Respect their own bodies as gifts from our Heavenly Father.
Day 28	<ol style="list-style-type: none"> Model the structure of a DNA molecule. Draw conclusions from historical experiments about DNA structure and function. Apply Chargaff's rule, using word problems. Explain the function of DNA. Apply the principles of macromolecules, cell structure, and disease to the pathophysiology of diabetes. Evaluate treatment and eating plans for diabetic patients.
Day 29	<ol style="list-style-type: none"> Transcribe RNA from DNA using information about directionality (5' and 3' ends). Translate RNA into a protein using the codon table. Determine tRNA anti-codon sequences given an mRNA. Evaluate amino acid and RNA sequences to identify different types of mutations. Discriminate normal from mutated genes, using the original DNA sequences, RNA transcripts and amino acid sequences.
Day 30	<ol style="list-style-type: none"> Compare DNA sequences to determine mutations and predict the effects of those mutations on amino acid sequences.
Day 31	Practice – Unit 4
Day 32	<ol style="list-style-type: none"> Explain the unique differences in our DNA that help differentiate us from others. Draw the process of DNA replication and explain the purpose of each component. Contrast the three historic hypothesis of DNA replication, and predict/evaluate the outcomes of each in a classic experiment. Construct the process of PCR using knowledge of DNA replication and specify the purpose of each component (<i>Taq</i> polymerase, primers, DNA templates, nucleotides, heating and cooling, etc.). Predict the outcome of PCR.

	<ul style="list-style-type: none"> 6. Analyze STR outputs and determine differences in individuals. 7. Calculate the probability of an STR profile.
Day 33	<ul style="list-style-type: none"> 1. Interpret the results of gel electrophoresis. 2. Determine fragment lengths produced by a restriction enzyme. 3. Use a RFLP to determine relatedness. 4. Hypothesize mechanisms for genetically modifying organisms. 5. Defend the benefits of GMOs. 6. List potential drawbacks of GMOs.
Day 34	<ul style="list-style-type: none"> 1. Analyze data from various biotechnology techniques (i.e., qPCR, Northern Blots, Western Blots, Molecular Mass analysis, regulatory sequences) and draw conclusions about mRNA and/or protein characteristics. 2. Hypothesize ways to cure genetic disease. 3. Use Plasmids to clone genes 4. Use restriction enzymes to determine orientation in a plasmid
Day 35	<ul style="list-style-type: none"> 1. Evaluate the plausibility of various genetic modifications (e.g., Jurassic Park). 2. Hypothesize ways in which genetic modification can occur based on the evolutionary history of our genome. 3. Hypothesize ways to use atavism activation to create new organismal features. 4. Employ the bioinformatic tool BLAST to infer relatedness of sequences or identify an unknown. 5. Determine the number of missing amino acids and/or nucleotides in two compared sequences from a BLAST search.
Day 36	<ul style="list-style-type: none"> 1. Determine the atomic structure of a molecule using the periodic table. 2. Predict atomic structure based on atomic weight and vice versa. 3. Determine isotope weight based on neutron numbers and vice versa. 4. Draw the electrons onto an atom in the right energy shells. 5. Predict the number of bonds made, type of bonds made, polarity, and electronegativity, based on the number of electrons. 6. Describe the atomic make-up of a given isotope. 7. Use principles of electron movement to explain potential and kinetic energy. 8. Draw a covalent bond. 9. Predict what types of molecules are hydrophilic, hydrophobic, or amphipathic using information of polarity and electronegativity. 10. Draw an ionic bond. 11. Predict how amphipathic molecules will act around other molecules.
Day 37	<ul style="list-style-type: none"> 1. Explain how energy is released from molecules that we eat. 2. Calculate the amount of energy released from the change in temperature and mass of a food item. 3. Predict whether a reaction is endergonic or exergonic based on an energy diagram. 4. Draw the process of energy coupling. 5. Predict the overall energy loss/gain of a coupled reaction

	6. Describe the purpose of ATP as an energy coupling molecule.
Day 38	<ol style="list-style-type: none"> Using principles of energy transfer, hypothesize a mechanism for the process of cellular respiration, and how each step interacts with another. Explain the purpose of NADH as an electron carrier. Distinguish substrate-level and oxidative phosphorylation. Predict the outcome of missing oxygen in this process. Predict where each reactant ends up in the process of cellular respiration.
Day 39	<ol style="list-style-type: none"> Predict where plant biomass comes from. Identify the reactants and products of photosynthesis and formulate an equation for photosynthesis. Define and contrast heterotrophs and autotrophs. Draw a detailed model of the process of photosynthesis: follow the path of electrons through the systems. Describe the process of photosynthesis to others.
Day 40	<ol style="list-style-type: none"> Diagram the relationship between cellular respiration and photosynthesis. Compare fermentation and cellular respiration, and explain the conditions under which each occur. Contrast C3 (“regular”), C4, and CAM photosynthesis. Draw the process of photorespiration, and describe the conditions under which it occurs over photosynthesis. Compare the anatomy and functions of the mitochondria to that of the chloroplast. Predict what happens to cells when they are deprived of CO₂ or O₂. Relate organismal biomass to metabolism and demonstrate how the inputs manifest in changes to biomass. Draw the pools and fluxes of the carbon cycle. Contrast the relative sizes of major pools of carbon. Demonstrate how to organize concepts of how physical and biological processes cycle matter. Reconstruct how an atom of carbon cycles through every pool.
Day 41	Practice – Unit 5
Day 42	<ol style="list-style-type: none"> Define global warming in the context of climate change. Interpret historical climate data and make conclusions about trends. Using graphical data, defend if our planet has really warmed over time. Understand the impacts, present and future, of biodiversity loss. Evaluate the effects of changing behavior versus adapting to consequences; weigh the benefits of each strategy.

Nature of Science Practice
“How is science done?”
Student Guide

Scenario #1: Burying Beetles and Mites

1. What is the puzzling observation?

The beetles with more mites on their head win more battles.

2. What is the causal question?

Why do the beetles with mites win more carcasses?

3. Can you come up with two potential hypotheses?

1. the mites are scary and so the opponent runs away
2. the mites give strength to the beetles.
3. Beetles are scared of red, so the more (red) mites, the scarier the beetle is.

4. Pick one of your hypotheses. How would you test it?

(3) paint a beetle red and release it into the wild. Control group: regular beetle success rate. Independent variable: surface area of beetle that is red. Independent variable: fights won by beetle.

5. What was the authors'...

- a. Hypothesis:

Mites are a source of heat that makes the beetles more active and better fighters.

- b. Proposed experiment:

control: beetles with mites fighting beetles without mites.

normal beetles fighting beetles that have been warmed.

normal beetles fighting beetles that have been cooled down (both w/ mites)

- c. Independent variable: IV: presence of mites, temperature of the beetle

- d. Dependent variable: DV: Success of the beetle

- e. What results would support the authors' hypothesis?

if the beetles who were warmed

were more successful in battle.

- f. What was the authors' conclusion?

the evidence shows that beetles without mites who were warmed did just as well as beetles who only had mites. Thus, the author's hypothesis was supported.

Scenario #2: Orchestra and ADHD

6. What is the puzzling observation?

Elementary school teachers report that their students with ADHD who played a stringed instrument seem to be more well-behaved than those who do not.

7. What is the causal question?

What is causing this puzzling observation?

8. Can you come up with two potential hypotheses?

1. their parents disciplined them into playing instruments so they're naturally scared of adults.
2. playing instruments develops focusing power of children.

9. Pick one of your hypotheses. How would you test it?

(2) have two groups of ADHD children, one who plays a string instrument, one who plays another kind of instrument (brass), and record the time it takes for each student to complete a puzzle. Average both times and compare.

10. What was the authors'...

- a. Hypothesis:

The training required to play in an orchestra increases a child's inhibitory- and self-control (both of which are implicated as impaired in ADHD children).

- b. Proposed experiment:

treatment 1: participate in a 3-month orchestra training program

treatment 2: an equally matched group of ADHD kids who will not participate in the orchestral training program

- c. Independent variable: IV: time spent orchestra training

- d. Dependent variable: DV: inhibitory control, hyperactivity and impulsivity, teacher-reported

- e. What results would support the authors' hypothesis?

the kids who go through orchestra training would have better control

- f. What was the authors' conclusion?

Scenario #3: Beans and Drought

11. What is the puzzling observation?

12. What is the causal question?

13. Can you come up with two potential hypotheses?

14. Pick one of your hypotheses. How would you test it?

15. What was the authors'...
 - a. Hypothesis:

 - b. Proposed experiment:

 - c. Independent variable:
 - d. Dependent variable:
 - e. What results would support the authors' hypothesis?

 - f. What was the authors' conclusion?

Nature of Science, Internet Falsities
“Can you prove it?”
Student Guide

Visit the following website and read about the Zyto Scan:

<http://zyto.com/Learning/GettingaScan> Then, answer the following questions.

1. What information do you feel is missing?
Lack of data, there's no real description of the purpose of the ZYTO scan, what ZYTO stands for, and what, specifically, it accomplishes. It "helps us make better decisions" but how? It has "no known side effects" but that could just be because nobody has researched it.
2. What evidence would you like to see that would be more convincing that this is something on which you should spend your hard-earned money (in other words, what type of experiment would need to be performed to convince you of the effectiveness of the Zyto Scan)?
Proof of experimentation to determine side effects. Experiments comparing <insert purpose of ZTYO scan here> in people who took the scan vs not. There's straight up no data at all. Anything would be appreciated
3. List three questions you have that you might ask the founders of Zyro technology:
Who hurt you? Why are you the way that you are? How long have you been retarded?

Now read the article entitled “Zyto Scanning: Another Test to Avoid” from this website:

<http://www.devicewatch.org/reports/zyto/overview.shtml>

4. How reliable do you think the first website was? Why?
Very unreliable. 0/10 on reliability. There was no information and there were no statistics.

5. What additional information did you gain from the second website?
Honestly not a whole lot. There was a lot of information but I didn't understand any of it.

6. How do you feel about the validity of Zyro Scan technology? Back up your feelings with evidence from both websites. *It don't think it's very valid. For aforementioned reasons.*

Visit the following website and read about Vitamin O: https://www.rgarden.com/vitamin_o.html. Read the description of the product under “Need Extra Energy?” and then answer the following questions.

7. Do you have any problems with what has been said? Explain.
Yes many problems. It is scientifically incorrect. It says it's ahead of its time but there are no studies to prove such. It is, in a word, very dumb.

8. What additional information would you want to convince you that this is a good supplement to take?
Studies to suggest that what they are saying Vitamin O does is actually what it does. It's just salt water. There's no evidence.

Now read the description of Vitamin O from WebMD at this site:
<https://www.webmd.com/vitamins/ai/ingredientmono-452/vitamin-o>

9. What do you think about the validity of Vitamin O? Back up your feelings with evidence from both websites.

Invalid. reasons mention before.

Briefly review the article, "MMR vaccine and autism: a review of the evidence for a causal association," with your group. It is an evaluation of a scientific study conducted by Wakefield and colleagues that claimed to have found a link between vaccination and autism after studying twelve children with an inflammatory bowel condition that progressed to autism after receiving the MMR vaccine. This created widespread panic in the public that vaccinations would harm their children.

10. What were the four limitations of evidence discussed in the article? Discuss why each is a limitation.

12 children is not a large enough sample size to represent an entire population of kids. Furthermore, we don't know how these children were selected. Thus, they could have been specially selected to support a certain result, or be wildly unrepresentative of the population.

There was no control group.

They didn't address the fact that the relationship could have just been correlation, not causation.

Lack of laboratory evidence.

11. According to scientific evidence, does the Measles, Mumps, and Rubella vaccine cause children to develop autism?

NO, it doesn't.

VACCINES AND AUTISM

MMR vaccine and autism: a review of the evidence for a causal association

Molecular Psychiatry (2002) 7, S51–S52. doi:10.1038/sj.mp.4001181

The hypothesis that combined measles-mumps-rubella (MMR) vaccine may cause autism was advanced by Wakefield and colleagues in a report describing 12 patients with inflammatory bowel conditions and regressive developmental disorders, primarily autism.¹ The authors hypothesized that MMR vaccine may have been responsible for the bowel dysfunction which subsequently resulted in the neurodevelopmental disorders.

Autism has a strong genetic component and associated neurological defects probably occur early in embryonic development. Therefore, in most cases, it is unlikely that a vaccination that is given after birth could cause autism. In a minority of cases, a child can appear to be developing completely normally but then regress and develop autistic characteristics. Theoretically, for cases of regression a biologically plausible link with vaccination could be made. A recent study by Nelson, however, has identified abnormal levels of certain growth factors at birth among children who developed autism, including the subgroup with regression.²

The original cases reported by Wakefield had all been referred for evaluation at the author's gastroenterology clinic. The main evidence of an association with vaccination was that for eight of the 12 cases the child's parents or pediatrician suspected that MMR vaccine may have contributed to the onset of behavioral problems. The authors speculated that persistent measles virus infection of the gastrointestinal tract could have resulted in pathologic changes that allowed gastrointestinal absorption of toxic neuropeptides which then caused central nervous system damage and neurodevelopmental regression.¹

There are a number of limitations to evidence from the case reports. First, the small number of cases referred to a gastroenterology clinic may well have been a biased sample and not representative of children with autism. Second, there was no unaffected comparison group. Third, the possibility of a coincidental, but not causal, temporal association with MMR vaccination was not addressed. Fourth, the postulated link between bowel disease and autism was tenuous, as there was no confirmatory laboratory evidence (ie,

measles virus was not detected in bowel) and bowel disease did not precede onset of autism in any of the cases.

Subsequent studies by Wakefield and colleagues were also not supportive of their hypothesis. For example, Wakefield's group (as well as other researchers) published that highly specific laboratory assays in patients with inflammatory bowel disease—the proposed link between autism after MMR—are negative for measles virus.³ Moreover, a recent study in the US found no association between MMR vaccine and risk of inflammatory bowel disease.⁴ Wakefield and colleagues have since proposed a new syndrome consisting of milder inflammatory bowel disease (eg, ileocolonic lymphonodular hyperplasia and mild intestinal inflammation) associated with behavioral regression.⁵ They have reported identifying laboratory evidence of measles virus genome in the white blood cells⁶ and intestines⁷ of some of these patients.

To evaluate Wakefield's hypothesis, Taylor and colleagues identified all 498 known cases of autism spectrum disorders in a district of London born since 1979 and linked them to a regional vaccination registry.⁸ The study found that, although the number of cases of autism disorders had been increasing since 1979, there was no sharp increase after the introduction of MMR vaccine in 1988. Also, cases vaccinated before 18 months of age, after 18 months of age, or not vaccinated, all had similar ages at diagnosis, indicating that vaccination does not result in earlier expression of autism. In addition, at age 2 years the MMR vaccination coverage among the autism cases was nearly identical to coverage in children in the same birth cohorts in the whole district. Taylor and colleagues then employed a case series methodology to assess the relative incidence of autism within pre-defined time periods after vaccination. No statistically significant associations were found, except for a small increased relative incidence (1.48) for the association of MMR vaccination and initial parental concern. The inability to find a temporal relationship between vaccination and onset of regression, in particular, provides persuasive evidence against the hypothesis that MMR may cause autistic regression or exacerbate autistic symptomatology.

Indirect evidence of a lack of association between MMR vaccine and autism also comes from recent ecological studies conducted in Great Britain⁹ and California.¹⁰ Each of these studies compared temporal trends in measles vaccination coverage with corresponding trends in autism prevalence. Neither found a positive correlation.

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A follow-up study of a national MMR vaccination program in Finland indicates that children who experience gastrointestinal symptoms shortly after vaccination are not at increased risk of neurodevelopmental problems.¹¹ Out of about 3 million vaccine doses administered, reports of gastrointestinal complaints were received from 31 recipients. These individuals were traced 1–15 years (median 10 years) later and none had developed autism. Although the small number of individuals with gastrointestinal problems precludes making firm conclusions about the risk of autism in people experiencing gastrointestinal reactions, the results indicate that any possible association following MMR vaccination would have to be extremely rare.

In conclusion, the initial case reports by Wakefield and colleagues raised a hypothesis that MMR vaccination may cause autistic regression. The one study published to date that addressed this hypothesis found no association between MMR vaccine and onset of regression or the development of autism. Data on temporal trends of autism occurrence and MMR vaccination coverage in different populations also have not revealed an association. The biological plausibility of the association is tenuous because, in most cases, the neuroanatomic abnormalities of autism probably develop *in utero* and recent evidence suggests that this is likely to be true for regression as well. The laboratory findings of measles virus genome in biological samples of some patients all emanate from one group and there has been no independent verification by other investi-

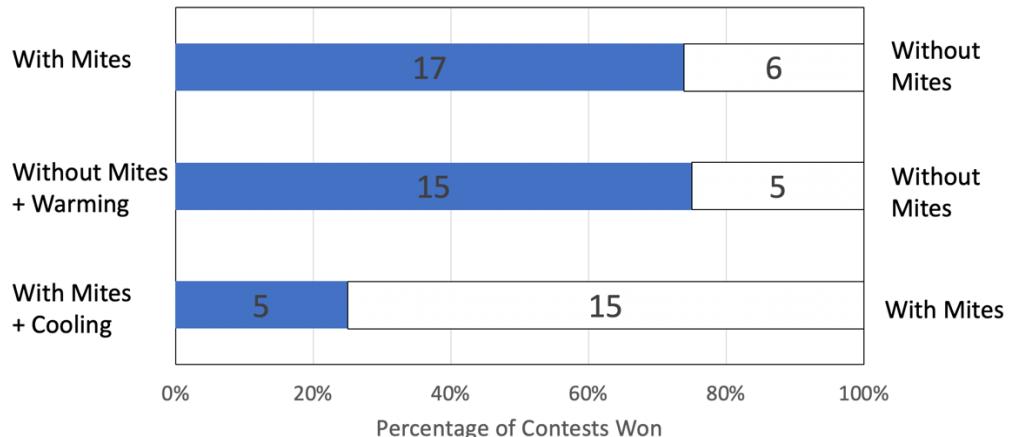
gators in other populations. Moreover, the relevance of the laboratory findings is not clear because no association has been established between vaccination and autism or inflammatory bowel disease. Therefore, the hypothesis that MMR vaccine may cause regression or the onset of autism has little support. The weight of the currently available epidemiological and related evidence argues against a causal association. The possibility of an idiosyncratic reaction in certain susceptible individuals cannot be ruled out, but such occurrences would have to be too rare to cause detectable increased risks on a population level. A review committee of the Institute of Medicine reached similar conclusions.¹²

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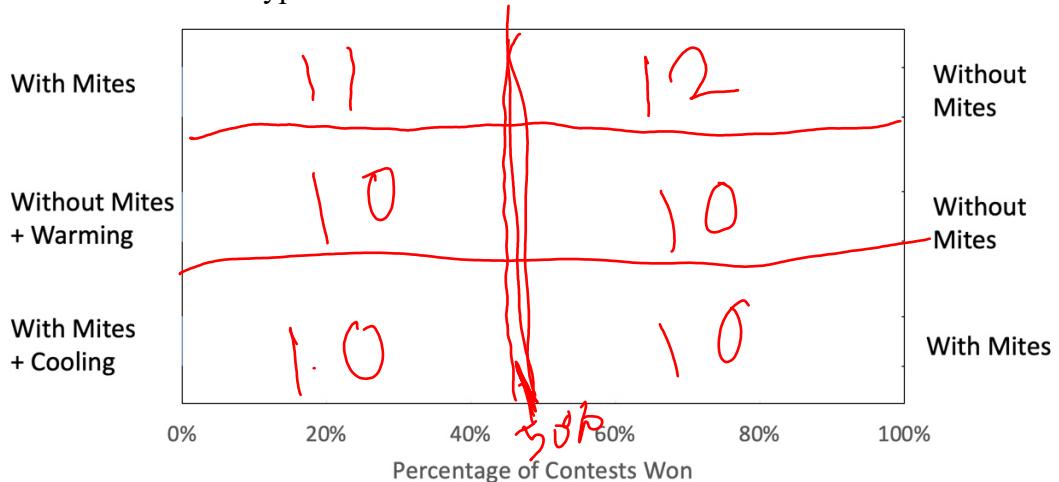
- 1 Wakefield AJ *et al.* *Lancet* 1998; **351**: 637–641.
- 2 Nelson KB *et al.* *Ann Neurol* 2001; **49**: 597–606.
- 3 Chadwick N *et al.* *J Med Virol* 1998; **55**: 305–311.
- 4 Davis RL *et al.* *Arch Pediatr Adolesc Med* 2001; **155**: 354–359.
- 5 Wakefield AJ *et al.* *Am J Gastroenterol* 2000; **95**: 2285–2295.
- 6 Kawashimi H *et al.* *Digestive Diseases and Science* 2000; **45**: 723–729.
- 7 Uhlmann V *et al.* *J Clin Pathol: Mol Pathol* 2002; **55**: 1–6.
- 8 Taylor B *et al.* *Lancet* 1999; **353**: 2026–2029.
- 9 Kaye JA *et al.* *BMJ* 2001; **322**: 460–463.
- 10 Dales L *et al.* *JAMA* 2001; **285**: 1183–1185.
- 11 Peltola H *et al.* *Lancet* 1998; **351**: 1327–1328.
- 12 Stratton K *et al* (eds). *Immunization Safety Review: Measles-Mumps-Rubella Vaccine and Autism*. National Academy Press: Washington, DC, 2001.

Nature of Science, Statistics
“How close does it have to be?”
 Student Guide

Scenario #1: Burying Beetles and Mites



1. Illustrate their null hypotheses below:

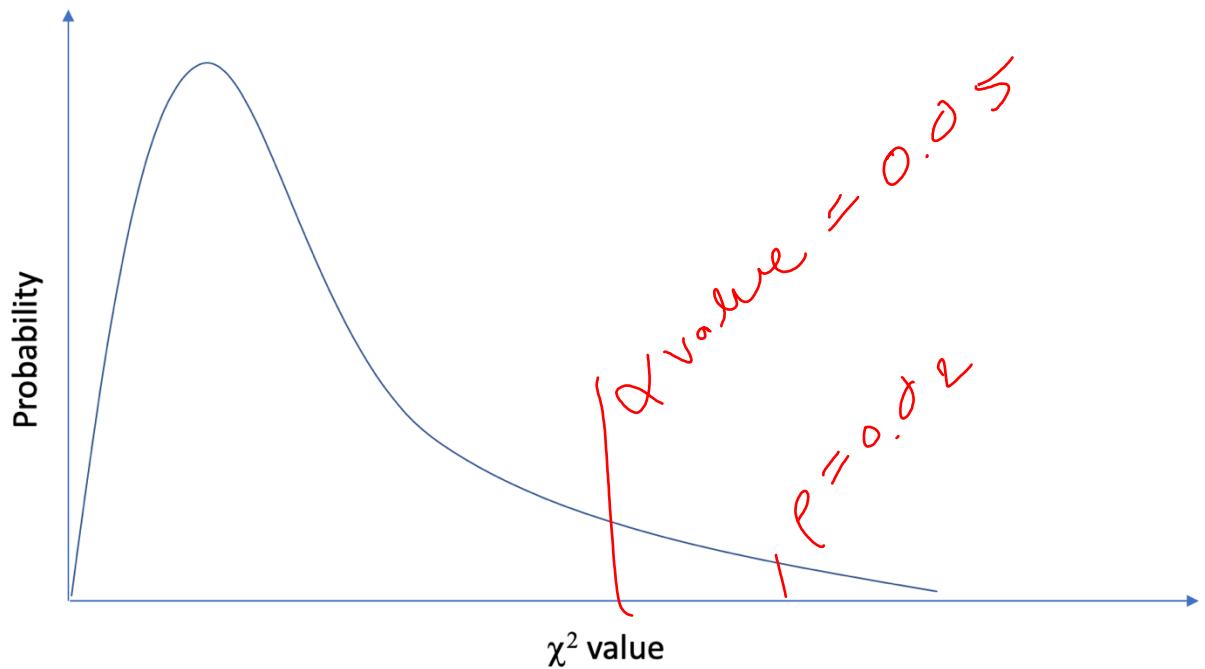


NULL HYPOTHESIS FOR EXPERIMENT ONE

2. Fill in the following table for experiment #1 (with mites vs. without mites):

	Scientific	Statistics
Claim to be tested	<i>If...</i> <small>mites don't have an effect</small>	<i>If...</i> <small>beetles are equally matched (differences are due to chance)</small>
Test	<i>and...</i> <small>we put mites on the beetles</small>	<i>and...</i> <small>I build a sampling distribution and calculate my statistic (from my particular battle) then where should my statistic fall? (p > alpha)</small>
Expected Result	<i>then...</i> <small>the win rates will be roughly equal</small>	<i>then...</i> <small>my statistic will fall well within the sampling distribution i.e. my p-value > alpha value</small>

3. For experiment #1, researchers ran a Chi-square statistical test. They obtained the following values: $\chi^2 = 9.82$, $p = .002$. Given its p-value, illustrate where this chi-square value falls on the sampling distribution below. Also shade in an alpha value of 0.05.



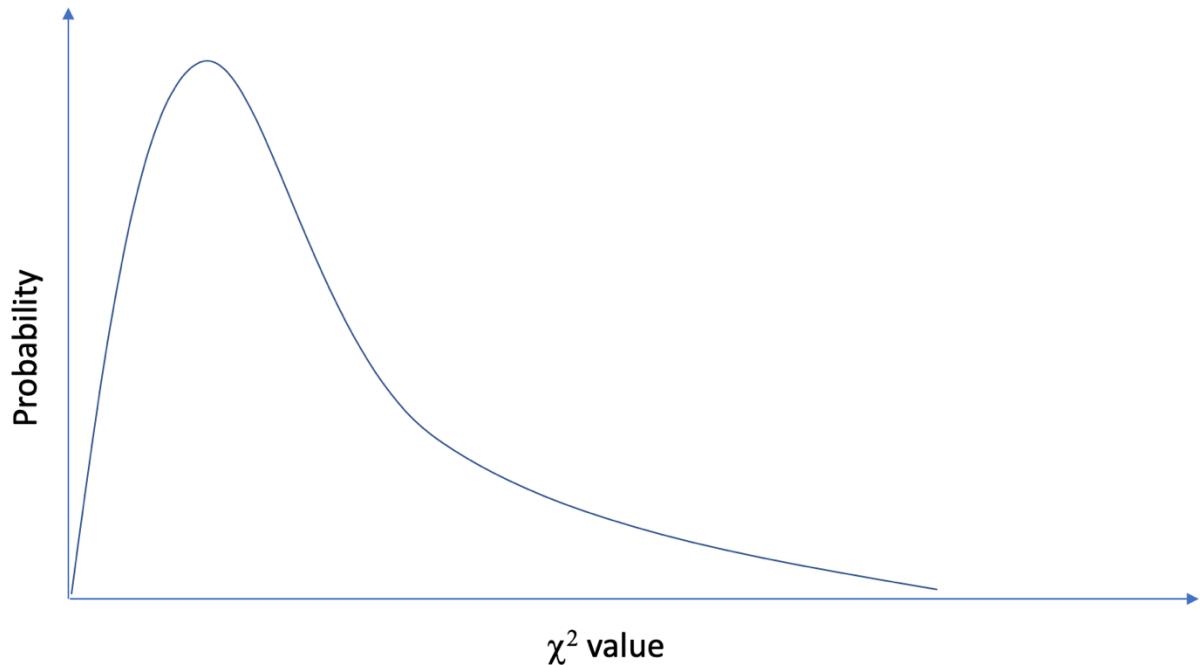
4. Based on these values, what should the authors statistical conclusion be?

The author should conclude that, because the probability of the test statistic is lower than the alpha value, the beetles are not equally matched. (differences are not due to chance).

5. Let's try one more with the mites. Fill in the following table for experiment #3 (with mites vs. with mites + cooling):

	Scientific	Statistics
Claim to be tested	<i>If...</i> temperature has no effect	<i>If...</i> the beetles are equally matched (differences are due to chance)
Test	<i>and...</i> we heat/cool the beetles	<i>and...</i> we calculate my statistic
Expected Result	<i>then...</i> equally matched wins/lose ==	<i>then...</i> my statistic will fall well within the sampling distribution i.e. $p > \alpha$

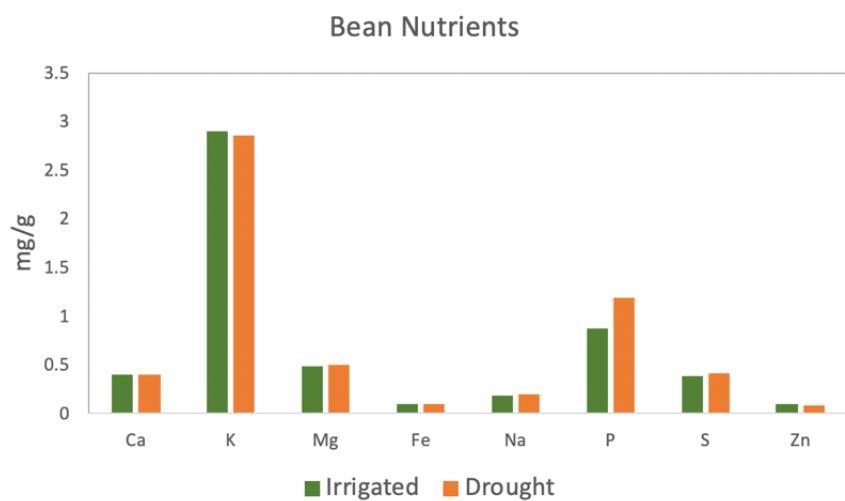
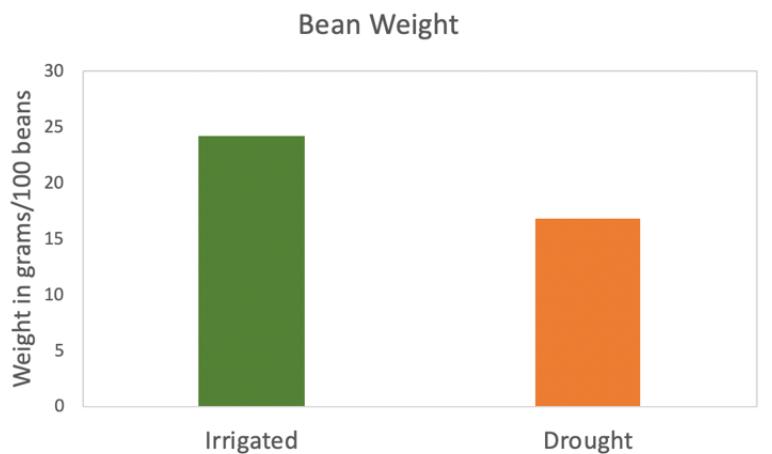
6. For experiment #3, researchers again ran a Chi-square statistical test. They obtained the following values: $\chi^2 = 8.98, p = .003$. Given its p-value, illustrate where this chi-square value falls on the sampling distribution below. Also shade in an alpha value of 0.05.



7. Based on these values, what should the authors statistical conclusion be?

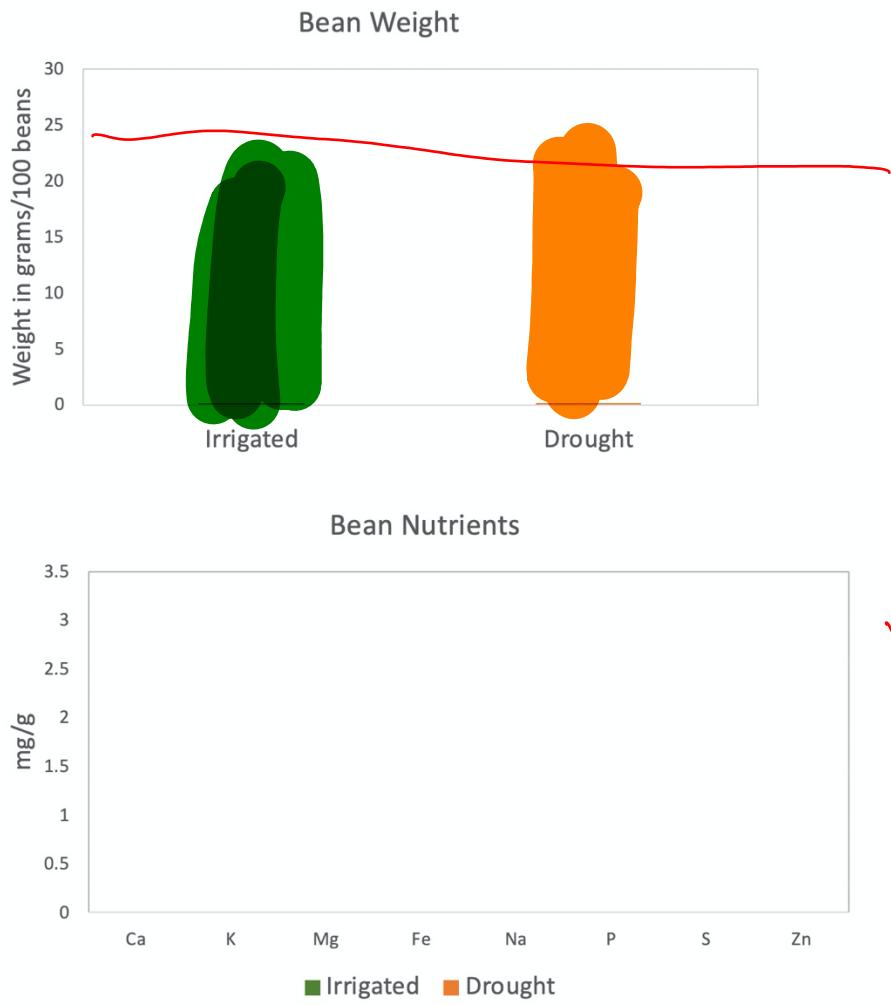
Conclusion: because the p-value was less than the alpha value, it is very unlikely that the beetles were equally matched.

Scenario #3: Beans and Drought



8. Illustrate their null hypotheses below:

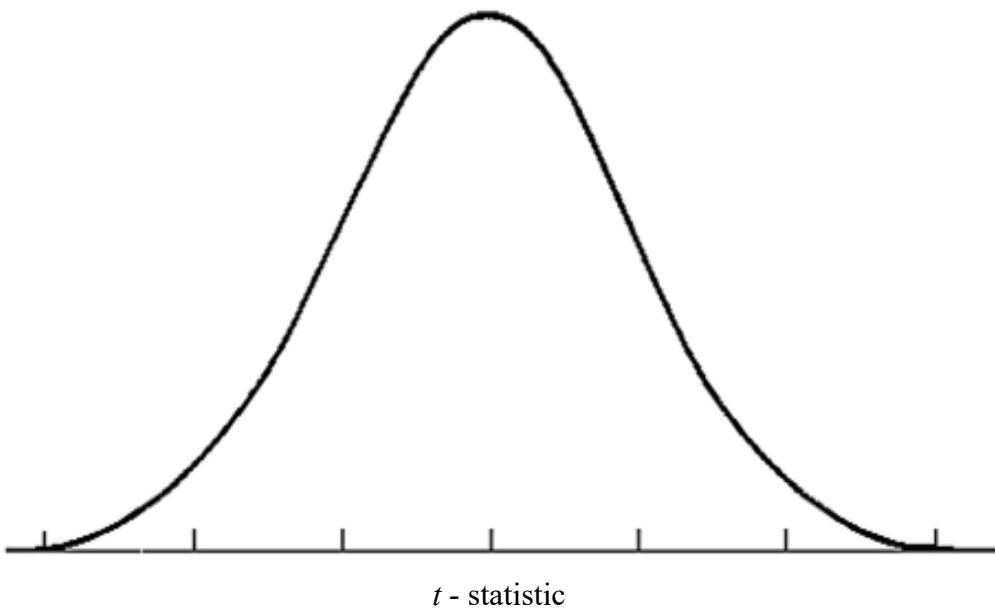
irrigation has no effect on bean weight or nutrients



9. Fill in the following table for experiment #1 (bean weight):

	Scientific	Statistics
Claim to be tested	<i>If...</i>	<i>If...</i> these beans are the same (same population of beans), and differences are due to chance
Test	<i>and...</i>	<i>and...</i> build sampling distribution and I calculate my statistic
Expected Result	<i>then...</i>	<i>then...</i> p less than α

10. For experiment #1, researchers ran a t -statistical test. They obtained the following values: $t = 5.89, p = .001$. Given its p -value, illustrate where this t value falls on the sampling distribution below. Also shade in an alpha value of 0.05.



11. Based on these values, what should the authors statistical conclusion be?

evolution and the origin of man

This packet contains, as far as could be found, all statements issued by the First Presidency of the Church of Jesus Christ of Latter-day Saints on the subject of evolution and the origin of man, and a statement on the Church's attitude toward science. The earliest First Presidency statement, "The Origin of Man," was issued during the administration of President Joseph F. Smith in 1909. This was followed by a First Presidency Message in 1910 that included brief comments related to the study of these topics. The second statement, "Mormon View of Evolution," was issued during the administration of President Heber J. Grant in 1925. Although there has never been a formal declaration from the First Presidency addressing the general matter of organic evolution as a process for development of biological species, these documents make clear the official position of the Church regarding the origin of man.

This packet also contains the article on evolution from the [Encyclopedia of Mormonism](#), published in 1992. The current First Presidency authorized inclusion of the excerpt from the First Presidency minutes of 1931 in the 1992 [Encyclopedia](#) article.

Various views have been expressed by other Church leaders on this subject over many decades; however, formal statements by the First Presidency are the definitive source of official Church positions. It is hoped that these materials will provide a firm foundation for individual study in a context of faith in the restored gospel.

Approved by the Board of Trustees, June 1992.

THE ORIGIN OF MAN

By The First Presidency of the Church

Inquiries arise from time to time respecting the attitude of the Church of Jesus Christ of Latter-day Saints upon questions which, though not vital from a doctrinal standpoint, are closely connected with the fundamental principles of salvation. The latest inquiry of this kind that has reached us is in relation to the origin of man. It is believed that a statement of the position held by the church upon this subject will be timely and productive of good.

In presenting the statement that follows we are not conscious of putting forth anything essentially new; neither is it our desire so to do. Truth is what we wish to present, and truth--eternal truth--is fundamentally old. A restatement of the original attitude of the Church relative to this matter is all that will be attempted here. To tell the truth as God has revealed it, and commend it to the acceptance of those who need to conform their opinions thereto, is the sole purpose of this presentation.

"God created man in his own image, in the image of God created he him; male and female created he them." In these plain and pointed words the inspired author of the book of Genesis made known to the world the truth concerning the origin of the human family. Moses, the prophet-historian, "learned," as we are told, "in all the wisdom of the Egyptians," when making this important announcement, was not voicing a mere opinion, a theory derived from his researches into the occult lore of that ancient people. He was speaking as the mouthpiece of God, and his solemn declaration was for all time and for all people. No subsequent revelator of the truth has contradicted the great leader and lawgiver of Israel. All who have since spoken by divine authority upon this theme have confirmed his simple and sublime proclamation. Nor could it be otherwise. Truth has but one source, and all revelations from heaven are harmonious with each other. The omnipotent Creator, the maker of heaven and earth--had shown unto Moses everything pertaining to this planet, including the facts relating to man's origin, and the authoritative pronouncement of that mighty prophet and seer to the house of Israel and through Israel to the whole world, is couched in the simple clause: "God created man in his own image"(Genesis 1:27; Pearl of Great Price--Book of Moses, 1:27-41).

The creation was two-fold--firstly spiritual, secondly temporal. This truth, also, Moses plainly taught--much more plainly than it has come down to us in the imperfect translations of the Bible that are now in use. Therein the fact of a spiritual creation, antedating the temporal creation, is strongly implied, but the proof of it is not so clear and conclusive as in other records held by the Latter-day Saints to be of equal authority with the Jewish scriptures. The partial obscurity of the latter upon the point in question is owing, no doubt, to the loss of those "plain and precious parts of sacred writ, which, as the book of Mormon informs us, have been taken away from the bible during its passage down the centuries (1 Nephi 13:24-29). Some of these missing parts the Prophet Joseph Smith undertook to restore when he revised those scriptures by the spirit of revelation, the result being that more complete account of the creation which is found in the book of Moses, previously cited. Note the following passages:

And now, behold, I say unto you, that these are the generations of the heaven and of the earth, when they were created, in the day that I, the Lord God, made the heaven and the earth;

And every plant of the field before it was in the earth, and every herb of the field before it grew.

For I, the Lord God, created all things of which I have spoken, spiritually, before they were naturally upon the face of the earth. For I, the Lord God, had not cause it to rain upon the face of the earth.

And I, the Lord God, had created all the children of men, and not yet a man to till the ground; for in heaven created I them, and there was not yet flesh upon the earth, neither in the water, neither in the air.

But, I, the Lord God, spake, and there went up a mist from the earth, and watered the whole face of the ground.

And I, the Lord God, formed man from the dust of the ground, and breathed into his nostrils the breath of life; and man became a living soul, the first flesh upon the earth, the first man also.

Nevertheless, all things were before created, but spiritually were they created and made, according to my word (Pearl of Great Price-Book of Moses, 3:4-8. See also chapter 1 and 2, and compare with Genesis 1 and 2).

These two points being established, namely, the creation of man in the image of God, and the two-fold character of the creation, let us now inquire: What was the form of man, in the spirit and in the body, as originally created? In a general way the answer is given in the words chosen as the text of this treatise. "God created man in his own image." It is more explicitly rendered in the Book of Mormon thus: "All men were created in the beginning after mine own image" (Ether 3:15). It is the Father who is speaking. If therefore, we can ascertain the form of the "Father of spirits," "The God of the spirits of all flesh," we shall be able to discover the form of the original man.

Jesus Christ, the Son of God, is "the express image" of His Father's person (Hebrews 1:3). He walked the earth as a human being, as a perfect man, and said, in answer to a question put to Him: "He that hath seen me hath seen the Father" (John 14:9). This alone ought to solve the problem to the satisfaction of every thoughtful, reverent mind. The conclusion is irresistible, that if the Son of God be the express image (that is, likeness) of his Father's person, then His Father is in the form of a man; for that was the form of the Son of God, not only during His mortal life, but before His mortal birth, and after His resurrection. It was in this form that the Father and the Son, as two personages, appeared to Joseph Smith, when, as a boy of fourteen years, he received his first vision. Then if God made man--the first man--in His own image and likeness, he must have made him like unto Christ, and consequently like unto men of Christ's time and of the present day. That man was made in the image of Christ is positively stated in the Book of Moses: "And I, God, said unto mine Only Begotten, which was with me from the beginning, Let us make man in our image, after our likeness; and it was so....And I, God, created man in mine own image, in the image of mine Only Begotten created I him, male and female created I them."(2:26,27).

The Father of Jesus is our Father also. Jesus Himself taught this truth, when He instructed His disciples how to pray: "Our Father which art in heaven," etc. Jesus, however, is the firstborn among all the sons of God--the first begotten in the spirit, and the only begotten in the flesh. He is our elder brother, and we, like Him, are in the image of God. All men and women are in the similitude of the universal Father and Mother, and are literally the sons and daughters of Deity.

"God created man in His own image." This is just as true of the spirit as it is of the body, which is only the clothing of the spirit, its complement; the two together constituting the soul. The spirit of man is in the form of man, and the spirits of all creatures are in the likeness of their bodies. This was plainly taught by the Prophet Joseph Smith (Doctrine and Covenants 77:2).

Here is further evidence of that fact. More than seven hundred years before Moses was shown the things pertaining to this earth, another great prophet, known to us as the brother of Jared, was similarly favored by the Lord. He was even permitted to behold the spirit-body of the foreordained Savior, prior to His incarnation; and so like the body of a man was gazing upon a being of flesh and blood. He first saw the finger and then the entire body of the Lord--all in the spirit. The Book of Mormon says of this wonderful manifestation:

And it came to pass that when the brother of Jared had said these words, behold the Lord stretched forth His hand and touched the stones one by one with His finger; and the veil was taken from off the eyes of the brother of Jared, and he saw the finger of the Lord; and it was as the finger of a man, like unto flesh and blood; and the brother of Jared fell down before the Lord, for he was struck with fear.

And the Lord saw that the brother of Jared had fallen to the earth; and the Lord said unto him, Arise, why hast thou fallen?

And he saith unto the Lord, I saw the finger of the Lord, and I feared lest he should smite me; for I knew not that the Lord had flesh and blood.

And the Lord said unto him, Because of thy faith thou hast seen that I shall take upon me flesh and blood; and never has man come before me with such exceeding faith as thou hast; for were it not so, ye could not have seen my finger. Sawest thou more than this?

And he answered, Nay, Lord, show thyself unto me.

And the Lord said unto him, Believest thou the words which I shall speak?

And he answered, Yea, Lord, I know that thou speakest the truth, for thou art a God of truth and canst not lie.

And when he had said these words, behold, the Lord showed himself unto him, and said, Because thou knowest these things ye are redeemed from the fall; therefore ye are brought back into my presence; therefore I show myself unto you.

Behold, I am He who was prepared from the foundation of the world to redeem my people. Behold, I am Jesus Christ, I am the Father and the Son. In me shall all mankind have light, and that eternally, even they who shall believe on my name; and they shall become my sons and my daughters.

And never have I shewed myself unto man whom I have created, for never hath man believed in me as thou hast. Seest thou that ye are created after mine own image? Yea, even all men were created in the beginning after mine own image.

Behold, this body, which ye now behold, is the body of my spirit, and man have I created after the body of my spirit; and even as I appear unto thee to be in the spirit, will I appear unto my people in the flesh. (Ether 3:6-16).

What more is needed to convince us that man, both in spirit and in body, is the image and likeness of God, and that God Himself is in the form of man?

When the divine Being whose spirit-body the brother of Jared beheld, took upon Him flesh and blood, He appeared as a man, having "body, parts and passions," like other men, though vastly superior to all others, because He was God, even the Son of God, the Word made flesh: in Him "dwelt the fulness of the Godhead bodily." And why should He not appear as a man? That was the form of His Spirit, and it must needs have an appropriate covering, a suitable tabernacle. He came unto the world as He had promised to come (III Nephi 1:13), taking an infant tabernacle, and developing it gradually to the fulness of His spirit stature. He came as man had been coming for ages, and as man has continued to come ever since. Jesus, however, as shown, was the only begotten of God in the flesh.

Adam, our progenitor, "the first man," was, like Christ, a pre-existent spirit, and like Christ he took upon him an appropriate body, the body of a man, and so became a "living soul." The doctrine of the pre-existence, --revealed so plainly, particularly in latter days, pours a wonderful flood of light upon the otherwise mysterious problem of man's origin. It shows that man, as a spirit, was begotten and born of heavenly parents, and reared to maturity in the eternal mansions of the Father, prior to coming upon the earth in a temporal body to undergo an experience in mortality. It teaches that all men existed in the spirit before any man existed in the flesh, and that all who have inhabited the earth since Adam have taken bodies and become soul in like manner.

It is held by some that Adam was not the first man upon this earth, and that the original human being was a development from lower orders of the animal creation. These, however, are the theories of men. The word of the Lord declares that Adam was "that first man of all men" (Moses 1:34), and we are therefore in duty bound to regard him as the primal parent of our race. It was shown to the brother of Jared that all men were created in the beginning after the image of God; and whether we take this to mean the spirit or the body, or both, it commits us to the same conclusion: Man began life as a human being, in the likeness of our heavenly Father.

True it is that the body of man enters upon its career as a tiny germ embryo, which becomes an infant, quickened at a certain stage by the spirit whose tabernacle it is, and the child, after being born, develops into a man. There is nothing in this, however, to indicate that the original man, the first of our race, began life as anything less than a man, or less that the human germ or embryo that becomes a man.

Man, by searching, cannot find out God. Never, unaided, will he discover the truth about the beginning of human life. The Lord must reveal Himself, or remain unrevealed; and the same is true of the facts relating to the origin of Adam's race-God alone can reveal them. Some of these facts, however, are already known, and what has been made known it is our duty to receive and retain.

The Church of Jesus Christ of Latter-day Saints, basing its belief on divine revelation, ancient and modern, proclaims man to be the direct and lineal offspring of Deity. God Himself is an exalted man, perfected, enthroned, and supreme. By His almighty power He organized the earth, and all that it contains, from spirit and element, which exist co-eternally with Himself. He formed every plant that grows, and every animal that breathes, each after its own kind, spiritually and temporally—"that which is spiritual being in the likeness of that which is temporal, and that which is temporal in the likeness of that which is spiritual." He made the tadpole and the ape, the lion and the elephant but He did not make them in His own image, nor endow them with Godlike reason and intelligence. Nevertheless, the whole animal creation will be perfected and perpetuated in the Hereafter, each class in its "distinct order or sphere," and will enjoy "eternal felicity." That fact has been made plain in this dispensation (Doctrine and Covenants 77:3).

Man is the child of God, formed in the divine image and endowed with divine attributes, and even as the infant son of an earthly father and mother is capable in due time of becoming a man, so the undeveloped offspring of celestial parentage is capable, by experience through ages and aeons, of evolving into a God.

Joseph F. Smith,

John R. Winder,

Anthon H. Lund,

First Presidency of The Church of Jesus Christ of Latter-day Saints

(Improvement Era 13:75-61 [November, 1909])

WORDS IN SEASON FROM THE FIRST PRESIDENCY

In this Christmas message, the First Presidency devoted several sentences to the Church's position with regard to questions raised by science:

Diversity of opinion does not necessitate intolerance of spirit, now should it embitter or set rational being against each other. The Christ taught kindness, patience, and charity.

Our religion is not hostile to real science. That which is demonstrated, we accept with joy; but vain philosophy, human theory and mere speculations of men, we do not accept nor do we adopt anything contrary to divine revelation or to good common sense. But everything that tends to right conduct, that harmonizes with sound morality and increases faith in Deity, finds favor with us not matter where it may be found.

"MORMON" VIEW OF EVOLUTION

A Statement by the First Presidency of

The Church of Jesus Christ of Latter-day Saints

"God created man in his own image, in the image of God created he him; male and female created he them."

In these plain and pointed words the inspired author of the book of Genesis made known to the world the truth concerning the origin of the human family. Moses, the prophet-historian, who was "learned" we are told, "in all the wisdom of the Egyptians," when making this important announcement, was not voicing a mere opinion. He was speaking as the mouthpiece of God, and his solemn declaration was for all time and for all people. No subsequent revelator of the truth has contradicted the great leader and law-giver of Israel. All who have since spoken by divine authority upon this theme have confirmed his simple and sublime proclamation. Nor could it be otherwise. Truth has but one source, and all revelations from heaven are harmonious one with the other.

Jesus Christ, the Son of God, is "the express image" of his Father's person (Hebrews 1:3). He walked the earth as a human being, as a perfect man, and said, in answer to a question put to him: "He that hath seen me hath seen the Father" (John 14:9). This alone ought to solve the problem to the satisfaction of every thoughtful, reverent mind. It was in this form that the Father and the Son, as two distinct personages, appeared to Joseph Smith, when, as a boy of fourteen years, he received his first vision.

The Father of Jesus Christ is our Father also. Jesus himself taught this truth, when he instructed his disciples how to pray: "Our Father which art in heaven," etc. Jesus, however, is the first born among all the sons of God-the first begotten in the spirit, and the only begotten in the flesh. He is our elder brother, and we, like him, are in the image of God. All men and women are in the similitude of the universal Father and Mother; and are literally sons and daughters of Deity.

Adam, our great progenitor, "the first man," was, like Christ, a pre-existent spirit, and, like Christ, he took upon him an appropriate body, the body of a man, and so became a "living soul." The doctrine of pre-existence pours a wonderful flood of light upon the otherwise mysterious problem of man's origin. It shows that man, as a spirit, was begotten and born of heavenly parents, and reared to maturity in the eternal; mansions of the Father, prior to coming upon the earth in a temporal body to undergo an experience in mortality.

The Church of Jesus Christ of Latter-day Saints, basing its belief on divine revelation, ancient and modern, proclaims man to be the direct and lineal offspring of Deity. By his Almighty power God organized the earth, and all that it contains, from spirit and element, which exist co-eternally with himself.

Man is the child of God, formed in the divine image and endowed with divine attributes, and even as the infant son of an earthly father and mother is capable in due time of becoming a man, so that undeveloped offspring of celestial parentage is capable, by experience through ages and aeons, of evolving into a God.

Heber J. Grant

Anthony W. Ivins

Charles W. Nibley

First Presidency

ENCYCLOPEDIA OF MORMONISM

EVOLUTION

The position of the Church on the origin of man was published by the First Presidency in 1909 and stated again by a different First Presidency in 1925:

The Church of Jesus Christ of Latter-day Saints, basing its belief on divine revelation, ancient and modern, declares man to be the direct and lineal offspring of Deity.... Man is the child of God, formed in the divine image and endowed with divine attributes (see Appendix, "Doctrinal Expositions of the First Presidency").

The scriptures tell why man was created, but they do not tell how, though the Lord has promised that he will tell that when he comes again (D&C 101:32-33). In 1931, when there was intense discussion on the issue of organic evolution, the First Presidency of the Church, then consisting of Presidents Heber J. Grant, Anthony W. Ivins, and Charles W. Nibley, addressed all of the General Authorities of the Church on the matter, and concluded,

Upon the fundamental doctrines of the Church we are all agreed. Our mission is to bear the message of the restored gospel to the world. Leave geology, biology, archaeology, and anthropology, no one of which has to do with the salvation of the soul of mankind, to scientific research, while we magnify our calling in the realm of the Church....

Upon one thing we should all be able to agree, namely, that Presidents Joseph F. Smith, John R. Winder, and Anthon H. Lund were right when they said: "Adam is the primal parent of our race" [First Presidency Minutes, April 7, 1931].

WILLIAM E. EVENSON

(Encyclopedia of Mormonism, Vol.2)

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What should I tell a young woman who says she feels pressure to go on a mission?

When President Thomas S. Monson announced that young men could serve at 18 years old and young women at 19, he reiterated that missionary service is a priesthood duty and encouraged all worthy and able young men to serve. He then said, “Many young women also serve, but they are not under the same mandate to serve as are the young men. We assure the young sisters of the Church, however, that they make a valuable contribution as missionaries, and we welcome their service” (“Welcome to Conference,” Oct. 2012 general conference).

If young women feel pressure to serve a mission, it’s not because of a Church policy or program that sets a new expectation for them to serve. Any person exerting this kind of pressure is not in harmony with what President Monson and other Church leaders have taught. The Church’s stance has been the same for a long time: young women are *welcome* to serve missions, but unlike young men, they are not *expected* to serve. NE

What does the Church believe about evolution?

The Church has no official position on the theory of evolution. Organic evolution, or changes to species’ inherited traits over time, is a matter for scientific study. Nothing has been revealed concerning evolution. Though the details of what happened on earth before Adam and Eve, including how their bodies were created, have not been revealed, our teachings regarding man’s origin are clear and come from revelation.

Before we were born on earth, we were spirit children of heavenly parents, with bodies in their image. God directed the creation of Adam and Eve and placed their spirits in their bodies. We are all descendants of Adam and Eve, our first parents, who were created in God’s image. There were no spirit children of Heavenly Father on the earth before Adam and Eve were created. In addition, “for a time they lived alone in a paradisiacal setting where there was neither human death nor future family.” They fell from that state, and this Fall was an essential part of Heavenly Father’s plan for us to become like Him. (See Elder Jeffrey R. Holland of the Quorum of the Twelve Apostles, “Where Justice, Love, and Mercy Meet,” Apr. 2015 general conference.) NE

For further reference, see “The Origin of Man,” *Improvement Era*, Nov. 1909, 78; *Ensign*, Feb. 2002, 29. See also *Encyclopedia of Mormonism*, 5 vols. (1992), “Evolution,” 2:478.

Building 3-D Trees

Part 1. Analyzing Race Maps – Let's do a little review

1. Look at the maps that your classmates have created. Compare them to yours. Notice that the order of the species (in relation to each other) is likely different between trees. Why do they each have a different order? Is one wrong and one right? Or are they the same? Explain.

If they are different, it is because some nodes can be rotated / switched with other nodes and the result would still be the same. Since the end traits were not enumerated, we can't be sure in what order things evolved (and, for example, if two things have homology or analogy).

2. A race implies a winner. If this map is chronological with the starting line being ancient times and the finish line being modern day, is there a ‘winner’? Why or why not?

There is no winner because the magnitude of how much something has evolved is a function of time, and the time spent evolving is the same for all organisms.

3. What do the checkpoints represent? Where should the checkpoints be located and how do they relate to branching points?

checkpoints are the traits, the branching points are the process of obtaining the checkpoints.

Part 2. Pipe Cleaner Trees

Using the pipe cleaners you were given, reconstruct the map you drew to make a tree that shows how these animals evolved [Tip: Start by twisting all the pipe cleaners together at the root of the tree about five times]. At each branch point, separate any species that are branching off and twist together the pipe cleaners of the species following the same path. Label the species with paper and tape.

4. Is a gecko more closely related to a salamander or a cow? Why?
5. Is a crocodile more closely related to a human or a cow? Why?
6. If scientists discovered a new species that has a vertebral column, four limbs, lungs and an amniote egg, where would it be placed on your tree? Would adding this new species change the relationships of the other species?

Part 3. Practice Building a Tree

1. Given the following morphological matrix, build a phylogenetic tree:

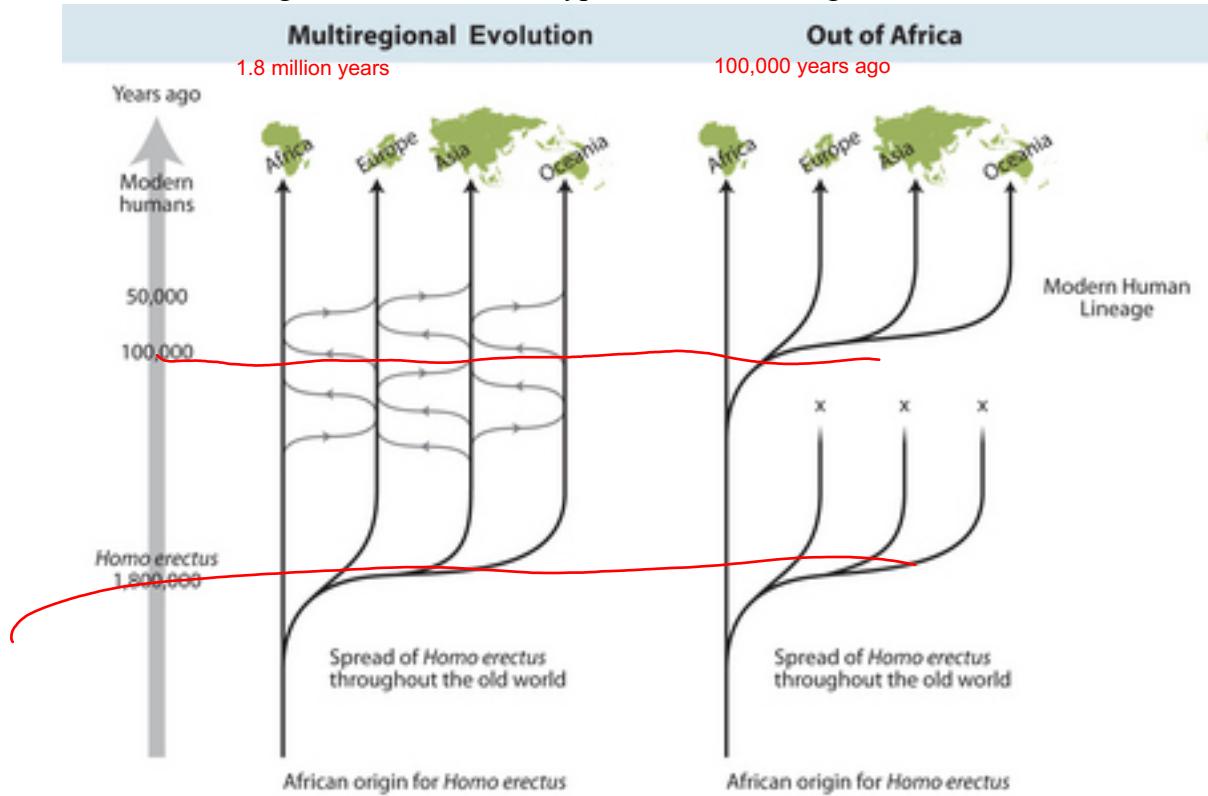
Species	Trait 1	Trait 2	Trait 3	Trait 4	Trait 5	Trait 6	Trait 7	Trait 8	Trait 9	Trait															
O	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
A	0	1	1	0	1	1	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1	0
B	0	0	0	0	1	1	1	0	0	0	0	0	0	0	1	1	0	0	1	0	0	0	1	1	1
C	1	0	0	0	1	1	1	1	1	1	0	0	0	1	0	0	1	0	0	1	1	1	0	1	1
D	0	0	0	0	1	1	1	0	0	1	1	0	0	1	0	0	1	0	0	1	1	0	1	1	0
E	1	0	0	0	1	1	1	0	1	1	0	0	0	1	0	0	1	1	1	1	0	1	1	0	1
F	0	1	0	0	1	1	0	0	0	0	0	0	0	0	1	1	1	1	0	0	0	0	1	1	1
G	0	0	0	0	1	1	1	0	0	1	1	1	0	1	0	0	1	1	0	1	1	0	1	1	0

* Note: Species "O" is the Outgroup

2. On your tree, circle a **monophyletic clade** that includes *at least three* species.
 3. Identify a **homology** for species *A* and *F*: _____
 4. Identify which trait(s) represent **convergent evolution**: _____

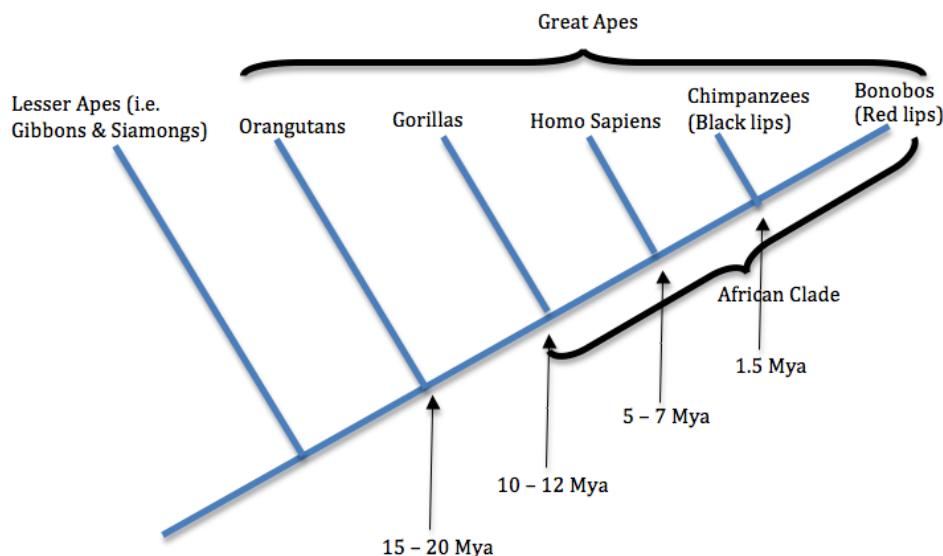
Human Race Student Guide

Part 1. Following are two alternative hypotheses for the origins of modern humans.



Discuss each of these hypotheses with your group and answer the following questions:

1. The Multiregional hypothesis predicts greater differences *between* races than the Out of Africa hypothesis.
 - a. Greater
 - b. Fewer



2. Look at the divergence times shown in the figure above.

The Multiregional hypothesis would predict that there is ~~more / less~~ genetic variation *between* human races than between chimpanzees and bonobos.

3. Based on Hardy-Weinberg, if two populations (in this case, we are calling these races) are reproductively isolated from one another for many generations, what would you predict about the following (circle the correct answer):

Between races we would expect to see

- a) Similar allelic frequencies
- b) Different allelic frequencies
- c) Different allelic frequencies *and* completely different alleles not found in other races

If races represent different populations, what would we expect to see regarding overall genetic differences within and between races?

- a) Within race differences will be greater than between race differences
- b) Between race differences will be greater than within race differences

Part 2. Why do racial characteristics exist?

Study the following evidences and come up with hypotheses for the existence of human racial characteristics.

Nose Shape

The purpose of the nose is to condition inhaled air to prevent it from damaging our fragile lung tissues. It must warm the air to body temperature and moisten the air; in addition, it serves to retain moisture during exhaling, especially in dry climates.

The following table outlines the purpose of the nose in each climate:

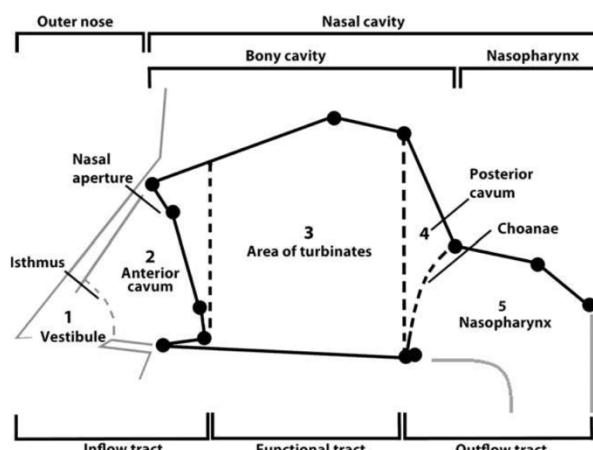
TABLE 1. Overview of air-conditioning demands in different climate types

Climate	Humidity adjustment of air	Temperature adjustment of air	Expected stress level
Cold-dry	Much humidification needed. Moisture conservation during expiration	Much warming needed. Minimization of heat loss during expiration	Very high
Cold-humid	Much humidification needed. Cold air contains little moisture. Moisture conservation during expiration	Warming of air needed. Minimization of heat loss during expiration	High
Temperate	Seasonal fluctuations in humidity, but never extreme	Seasonal fluctuations in temperature, but never extreme	Intermediate
Hot-dry	Humidification needed: hot dry air can extract moisture from the body. Moisture conservation during expiration	Air temperature can be higher than body temperature. Cooling rather than heat preservation	Medium
Hot-humid	No air-conditioning needed	No air-conditioning needed. Cooling rather than heat preservation	Low

Increasing the mucosal contact surface per unit of air volume increases the amount of temperature and moisture exchange that can take place. So how do you increase this surface area?

- Increasing the length of the outer nose and nasal cavity
- Narrowing the outer nose and nasal cavity (to a limit)
- Lengthening the turbinate chamber (where turbulence happens and allows the air to bump into the inside surfaces of the nose, see the figure below)

Figure 1. Anatomy of the nose

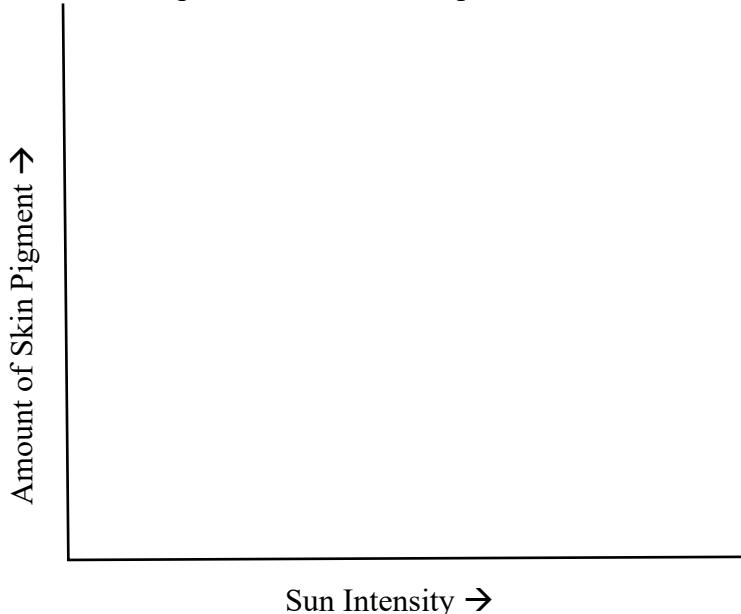


Given this information, what prediction would you make concerning nose shape and climate?

Climate	Nose Shape
Cold-dry	
Cold-humid	
Temperate	
Hot-dry	
Hot-humid	

Skin Color

- Vitamin D is required to maintain proper calcium levels in your bones and blood. Your body can only produce the precursor to vitamin D. This precursor collects in the skin where it is converted by sunlight into usable vitamin D. This is the major source of vitamin D in your body. Only small portions come from your diet.
- Folic acid is required for the production of new DNA every time your cells divide. It is also critical to brain and spinal cord development in the growing fetus.
- Sunlight destroys folic acid if it penetrates the skin
- Melanin, the pigment in your skin, blocks sunlight from penetrating your skin.
- Given this information, predict the relationship between skin color and climate.



Human Evolution
Apply
Student Guide

Chromosomes

What evidence for evolution do the chromosomes of chimps and humans provide?

The patterns on the ends of the DNA strands are similar.

GULO Gene

How does the GULO Gene mutation provide evidence for shared ancestry?

All of our close relatives cannot make vitamin C.

Active GULO genes make it so that we can make vitamin C. Monkeys and humans have inactive GULO genes, but squirrels and tarsiers and stuff have active GULO genes.

Hominid skulls

Use the space below to jot down any thoughts about what you see today.

Natural Selection

Herbicide-resistant Pigweed

Your instructor will play a video for you.

1. Describe how these weeds developed this resistance to herbicides. Was the resistance gene already there? Did the pesticides cause the plant to become resistant? Is the resistance gene heritable?

The herbs had random mutations, and one mutation was resistant to the herbicide so it flourished. It is heritable because they pass down the gene to their weed children.

2. Should farmers avoid using pesticides in order to prevent the selection of resistant strains? Is there an alternative solution?

I don't know. I don't think so. I think just switch up the herbicide sauce every once in a while. Or don't overuse it.

Tuberculosis in India

Your instructor will play a video for you.

3. In the video, doctors mention that mistreatment of patients is causing the development of these resistant strains. Explain how this is happening.

Most of the TB that doesn't have these resistant strains are being killed by the drugs. So the resistant TB is flourishing.

4. If patients are treated with inadequate amounts of antibiotics or the wrong kind of antibiotic, it will not eliminate the pathogen and the more resistant strains will thrive. In your opinion, what part does poverty play in this epidemic?

Impoverished people have a harder time affording enough antibiotics. A lack of education can result in incorrect application / kinds of antibiotics used.

Misuse of Antibiotics

Read the article from Mayo Clinic.

5. List three things you can do to prevent antibiotic resistance.

Take the full dose of antibiotics

6. In your opinion, what is the biggest problem that is driving the development of these 'super bugs'?

Multi-Drug Resistant Bacteria

Your instructor will play a video for you.

7. In your opinion, what is the role of each of the following groups of people in this problem?
 - a. Doctors

- b. Pharmaceutical Companies

- c. Patients

- d. The Media



Antibiotics: Misuse puts you and others at risk

Antibiotics can be lifesavers, but misuse has increased the number of drug-resistant germs. See how this affects you and what you can do to help prevent antibiotic resistance.

By Mayo Clinic Staff

If you think antibiotic resistance isn't a problem or doesn't affect you, think again. A prominent example of the dangers of antibiotic resistance is the spread of methicillin-resistant *Staphylococcus aureus* (MRSA). MRSA was once a concern only for people in the hospital, but a newer form of MRSA is causing infections in healthy people in the community.

Antibiotic resistance occurs when antibiotics no longer work against disease-causing bacteria. These infections are difficult to treat and can mean longer lasting illnesses, more doctor visits or extended hospital stays, and the need for more expensive and toxic medications. Some resistant infections can even cause death.

Although experts are working to develop new antibiotics and other treatments to keep pace with antibiotic-resistant strains of bacteria, infectious organisms can adapt quickly. Antibiotic-resistant bacteria will continue to be a global health concern — and using antibiotics wisely is important for preventing their spread.

Antibiotics are effective against bacterial infections, certain fungal infections and some kinds of parasites. Antibiotics don't work against viruses. The chart shows common illnesses and whether they're caused by bacteria or viruses. Taking an antibiotic when you have a viral infection won't make you feel better — and can contribute to antibiotic resistance.

Bacterial infections	Viral infections
<ul style="list-style-type: none">• Bladder infections	<ul style="list-style-type: none">• Bronchitis

- | | |
|---|---|
| <ul style="list-style-type: none">• Many wound and skin infections, such as staph infections• Severe sinus infections that last longer than 2 weeks• Some ear infections• Strep throat | <ul style="list-style-type: none">• Colds• Flu (influenza)• Most coughs• Most ear infections• Most sore throats• Stomach flu (viral gastroenteritis) |
|---|---|

If antibiotics are used too often for things they can't treat — like colds, flu or other viral infections — not only are they of no benefit, they become less effective against the bacteria they're intended to treat.

Not taking antibiotics exactly as prescribed also leads to problems. For example, if you take an antibiotic for only a few days — instead of the full course — the antibiotic may wipe out some, but not all, of the bacteria. The surviving bacteria become more resistant and can be spread to other people. When bacteria become resistant to first line treatments, the risk of complications and death is increased.

The failure of first line antibiotics also means that doctors have to resort to less conventional medications, many of which are more costly and associated with more-serious side effects. For instance, the drugs needed to treat drug-resistant forms of tuberculosis (TB) are much more expensive than are the drugs used to treat nonresistant TB. The course of treatment is long — up to two years — and the side effects can be severe.

Other consequences are the increased costs associated with prolonged illnesses, including expenses for additional tests, treatments and hospitalization, and indirect costs, such as lost income.

Repeated and improper use of antibiotics is the primary cause of the increase in the number of drug-resistant bacteria. Here's what you can do to promote proper use of antibiotics:

- **Understand when antibiotics should be used.** Don't expect to take antibiotics every time you're sick. Antibiotics are effective in treating most bacterial infections, but they're not useful against viral infections, such as colds, acute bronchitis or the flu. And even some common bacterial ailments, such as mild ear infections, don't benefit much from antibiotics.
- **Don't pressure your doctor for antibiotics if you have a viral illness.** Instead, talk with your doctor about ways to relieve your symptoms — for instance, a saline nasal spray to clear a stuffy nose or a mixture of warm water, lemon and honey to temporarily soothe a sore throat.
- **Take antibiotics exactly as prescribed.** Follow your doctor's instructions when taking

medication. Don't stop treatment a few days early because you're feeling better. Taking the full course of antibiotics is the only way to kill all of the harmful bacteria. A shortened course of antibiotics, on the other hand, often wipes out only the most vulnerable bacteria while allowing relatively resistant bacteria to survive.

- **Never take antibiotics without a prescription.** If you didn't complete a full course of antibiotics, you might be tempted to use the leftover medication the next time you get sick or to pass it along to someone else. But this isn't a good idea. For one thing, the antibiotic might not be appropriate for another illness. And even if it is, you're not likely to have enough pills to combat the germs making you sick, which can lead to more resistant bacteria.
- **Prevent the spread of germs.** Good hygiene goes a long way in preventing infection. Wash your hands thoroughly with soap and water, especially after using the toilet, changing a diaper, or handling raw meat or poultry. Keep food preparation areas clean. Although special antibacterial cleaners and soap are widely available, they aren't necessary. Plain soap and water work fine to kill germs in most settings.
- **Get recommended vaccinations.** Ask your doctor if you have all of the vaccinations you need to protect yourself from illness. Getting vaccinated will help prevent having to take more medications.

Antibiotic resistance is a global health problem. Nearly all significant bacterial infections in the world are becoming resistant to commonly used antibiotics. When you misuse antibiotics, you help create resistant microorganisms that can cause new and hard-to-treat infections. That's why the decisions you make about using antibiotics — unlike almost any other medicine you take — have far-reaching consequences. Be responsible in how you use antibiotics to protect your health and that of your family, neighbors and community.

References

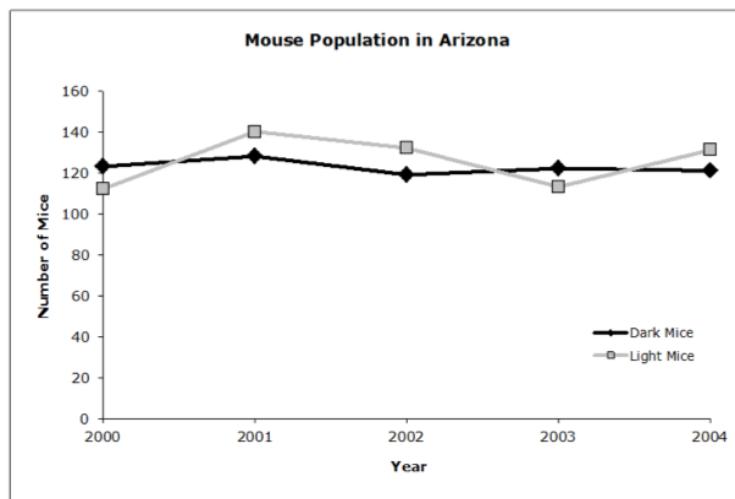
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“The Making of the Fittest.” – Pocket Mice
Student Guide

The following questions come from an HHMI Biointeractive module that your instructor will show you. It can be viewed at the following site: <http://www.hhmi.org/biointeractive/making-fittest-natural-selection-and-adaptation>

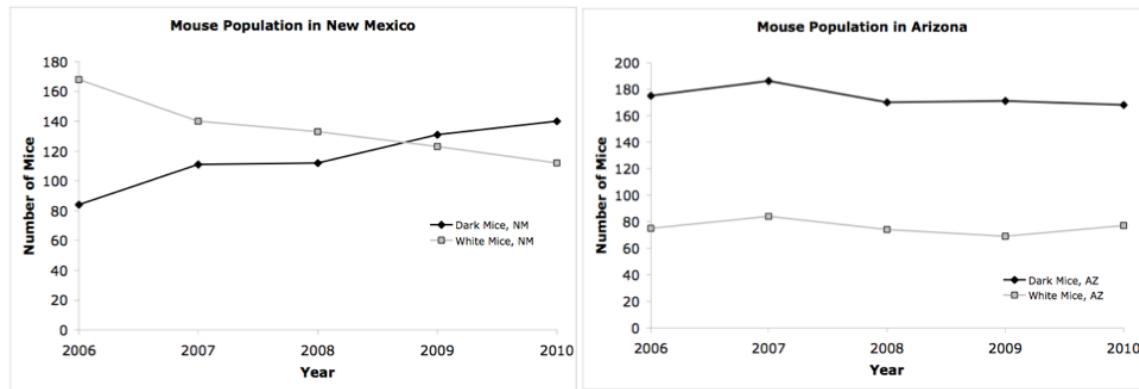
1. T/F: Mutations are caused by selective pressure in the environment. Explain your answer:
False. Mutations are random, their enflourishment is determined by selective pressure.
2. T/F: The same mutation could be advantageous in some environments but deleterious in others.
True.
3. T/F: The appearance of dark-colored volcanic rock caused the mutation for black fur to appear in the rock pocket mouse population. Explain your answer:
False. Mutations are random. The extent to which it was a useful mutation was a result of the dark-colored volcanic rock.
4. As you saw in the film, rock pocket mice evolved to have dark-colored fur in certain habitats. Explain how this trait increased in frequency in the population. Include the following key terms in your explanation: "fitness" (or 'fit'), "survival" (or 'survive'), "selection" (or 'selective'), and "evolution" (or 'evolve').
A random mutation occurred to give the mice dark-colored fur. This mutation made the mice more fit to survive. Thus, natural selection favored these mice and they were able to reproduce. So yeah they evolved.
5. Suppose you are studying a recently discovered population of rock pocket mice with dark-colored fur that lives on volcanic rock. You take a DNA sample from a member of this new population and determine the DNA sequence of a gene known to play a role in fur color. The sequence you get is identical to that of the same gene in another rock pocket mouse population with dark-colored fur that lives on a different patch of volcanic rock. Which of the following could explain this observation?
 - a. The mice in the two populations evolved from the same ancestral population.
 - b. The volcanic rock caused the same mutation in each rock pocket mouse population, resulting in dark coloration.
 - c. The same mutation spontaneously arose in the two different populations
 - d. Both (a) and (c) are possible. (although 'a' is more likely)
 - e. All of the above are possible.

6. Suppose you are studying a new population of rock pocket mice in Arizona. These mice live on a recently discovered patch of dark-colored volcanic rock. You observed the following numbers of light- and dark-colored mice on this new patch of rock.



In one or two sentences, summarize the data presented in the graph.

7. You next decide to move 50 of these newly discovered light-colored rock pocket mice from Arizona to a colony in New Mexico that also lives on dark-colored volcanic rock. You also move 50 dark-colored mice from the New Mexico colony to the Arizona colony. You monitor the populations for five years and observe the following:



In one or two sentences, summarize the data presented in the graphs above. Then, provide an explanation for the observations.

Applying Life History

Student Guide

As you do this activity, remember that in life history strategies, organisms are allocating their resources to three main things: Growth, Maintenance, and Reproduction.

1. Female hummingbirds abandon their young in the nest when food supplies decline. What does this imply about their allocation priorities?

Their highest priority is growth or maintenance.

2. What does this suggest about their potential to have another breeding round?

Probably quite high.

3. Ecologists added predatory fish to some streams, leaving other streams as controls. Which of the following would you predict to happen to the breeding age of the prey fish species in the presence of a predator? (*go up* or *go down*) Why?

It would go down because it's less likely they'll live long enough to reproduce.

4. Consider two rivers: One is spring fed and has a constant water volume and temperature year-round; the other drains a desert landscape and floods and dries out at unpredictable intervals. Which river would you predict is more likely to support many species of animals who have repeated reproductive cycles? Why?

The first would support more because it has less stochasticity.

5. Mice that experience stress such as a food shortage will sometimes abandon their young, or even eat their young. Explain how this behavior could have evolved?

They probably can breed a lot, so their one set of babies doesn't outweigh the potentially multiple sets of babies the mouse could make in the future.

Applying Symbiosis

Student Guide

Part 1. Invisible World

Your instructor will show you a video clip from *The Invisible World*.

1. How would you classify the relationship between the bacteria on our skin and us?
Mutualism

2. How would you classify the relationship between dust mites and us? (barring dust mite allergies)
Commensalism

3. How would you classify the relationship between yeast and us? (If you don't know, google 'thrush')
Predation or Parasitism

4. How would you classify the relationship between yeast and bacteria?

Mutualistic

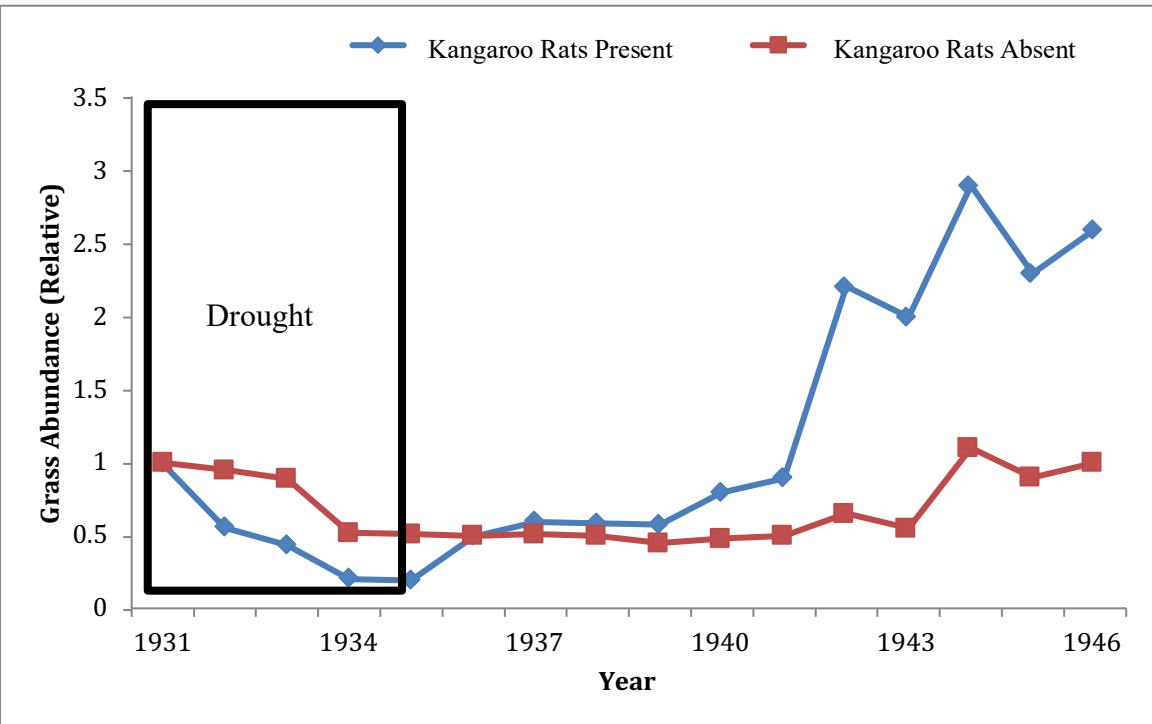
5. Coevolution refers to the idea that organisms evolve in response to each other due to interspecific interactions. Which of the following interactions would be least likely to cause coevolution?

- a. Parasitism
- b. Predation
- c. Mutualism
- d. **Commensalism**

6. Defend your answer:

Commensalism is the only one where one party is not affected and so evolutionary changes won't occur as a result of the other party's actions.

7. Due to the uncertainty of food in the desert, kangaroo rats have evolved the practice of gathering and hiding as many seeds as possible even if it is more than they can eat. In years when seeds are scarce, the rats eat all the seeds they store; in years when seeds are plentiful, the rats store more than they can eat and left-over seeds are likely to germinate and produce more grass plants. Scientists divided a portion of the desert into two segments, one of which they did not allow kangaroo rats to enter. They then gathered kangaroo rat and grass numbers over the course of 14 years during which time there was a drought. Analyze the graph below (taken from p.862 of *Principles of Life* by Hillis) and answer the questions that follow.



- From 1931 to 1935, how would you classify the relationship between the kangaroo rats and the grass? Why?
Herbivory
- From 1940 to 1945, how would you classify the relationship between the kangaroo rats and the grass? Why?
Mutualism
- Can you come up with a hypothesis as to why we see this shift when water is abundant?

When there's enough water, both the kangaroo rats and the grass benefit because the rats keep the grass population in check while also spreading the seed around. So the rats get food and the grass gets embiggened.

8. The tapeworm *Echinococcus multilocularis* is a mouse parasite. It sets up shop in the mouse's intestines and consumes the food that the mouse eats. Normally, you would predict that this would cause the mouse to become thin and emaciated. However, the opposite is true. The tapeworm releases certain chemicals that change the metabolism of the mouse causing its mouse host to become obese and sluggish, making it easy pickings for predators, notably foxes. Not coincidentally, the foxes constitute the next phase in the tapeworm's life cycle!

On the other hand, in a golden jackal pack, young males are banished and forced to live on their own. To survive, the young jackals follow tigers at a safe distance and feed off the leftover meat from a kill.

We consider the tapeworm a parasite. Write a paragraph comparing the relationship of the tapeworm and mouse to that of the jackal and tiger. Is the jackal a parasite? Or is it a different relationship? Could you classify the tapeworm as a mutualistic invader? Why or why not?

9. Here is another example (taken from DAVID P. BARASH. **The Chronicle of Higher Education**. Washington: Nov 7, 2008. Vol. 55, Iss. 11; pg. B.18): "The life cycle of a trematode worm, known as *Dicrocoelium dentriticum*, involves doing time inside an ant, followed by a sheep. Getting from its insect host to its mammalian one is a bit of a stretch, but the resourceful worm has found a way: Ensconced within an ant, some of the worms migrate to its formicine brain, whereupon they manage to rewire their host's neurons and hijack its behavior. The manipulated ant, acting with zombielike fidelity to *Dicrocoelium*'s demands, climbs to the top of a blade of grass and clamps down with its jaws, whereupon it waits patiently and conspicuously until it is consumed by a grazing sheep. Thus transported to its desired happy breeding ground deep inside sheep bowels, the worm turns, or rather, releases its eggs, which depart with a healthy helping of sheep poop, only to be consumed once more, by ants." How would you classify the relationships here?

Mendelian Genetics

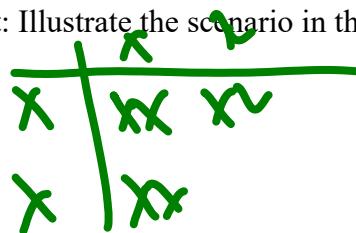
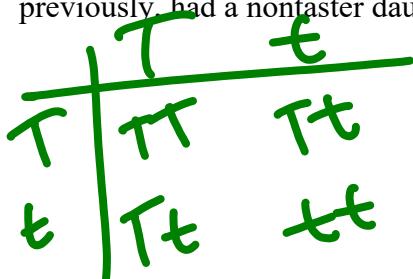
Student Guide

Below are several questions to practice what you learned about Mendelian genetics (traits that are controlled by one gene in a normal dominant/recessive fashion).

1. Phenylthiocarbamide (PTC) tasting is dominant (T) to nontasting (t).

If a taster woman with a nontaster father produces children with a taster man who, previously, had a nontaster daughter (Hint: Illustrate the scenario in the space below)...

Both the taster daughter and the taster man have to be heterozygous



- a. What is the probability that their first child would be a nontaster?

$$tt = \frac{1}{4} = .25$$

- b. What is the probability that their first child would be a nontaster AND a girl?

$$\frac{1}{2} \cdot \frac{1}{2} \cdot \frac{1}{2} = 0.125$$

2. A condition called polydactyly (the presence of an extra finger) is *dominant* to the presence of only five fingers. Not all 'bad' traits are recessive. Use the letter 'E' for dominant and the letter 'e' for recessive in the following questions:

- a. What is/are the possible genotype(s) for a person with polydactyly?
EE, Ee

- b. What is/are the possible genotype(s) for a person with only 5 fingers on each hand?
ee

- c. A polydactyly person and a 'normal' person are married and decide to have children. If the genotype of the polydactyly person is unknown, what are the overall chances of having a child with extra fingers?



So $\frac{1}{4}$ are ee, $\therefore \frac{3}{4}$ will have extra fingers.

- d. This couple's first child has only five fingers on each hand. What is the genotype of the polydactyly parent? Why?

Ee because if the parent was EE then it would be a 100% chance that their kid would have extra fingers.

Another way to do it is: there's 1/2 probability it's EE (and 100% will have extra fingers) or it's a 1/2 probability that there's Ee and in that case only half of them will have extra fingers.

3. If both of your number 9 chromosomes have a certain genetic anomaly, what does this mean about your parents' chromosomes?
- Both of one parent's number 9 chromosomes, but none of the other parent's, must have the defect.
 - Both of your parents have two defective number 9 chromosomes.
 - At least one of your mom's and one of your dad's number 9 chromosomes have the defect.
 - Neither of your parents have defective chromosomes, but both of your grandparents have at least one chromosome number 9 with the defect.

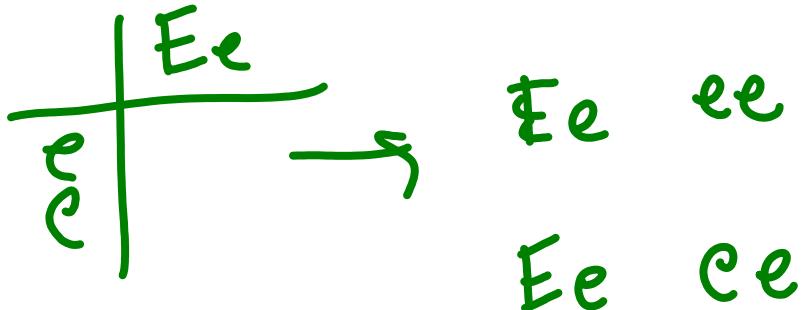
4. In humans, the presence of a widow's peak is a dominant trait.
- What is/are the possible genotype(s) for an individual with a widow's peak?

Ee EE ee

- What is/are the possible genotype(s) for an individual without one?

ee

- If a woman with a widow's peak, whose father did not have a widow's peak, mated with a man who did not have a widow's peak (Again, DRAW it out!), what are the possible genotypes of their offspring, and in what ratios would you expect to see them?



5. A dimpled chin is dominant to a non-dimpled chin.

- If your father has a dimpled chin and your mother does not, but you do, what is your genotype?

Dd

- What is your phenotype?

A dimpled chin

- c. What is the probability that your first child will have a dimpled chin if you marry a person with a non-dimpled chin?

$$\begin{matrix} D & d \\ d & \end{matrix} \quad \frac{1}{2} = .5$$

- d. If you marry a person with a dimpled chin but you do not know his/her genotype, what is the probability that your first child will have a non-dimpled chin?

$$\frac{1}{8} = \frac{0}{4} + \frac{1}{4}$$

6. Phenylketonuria (PKU) is an autosomal (meaning it is on a chromosome other than the sex chromosomes) recessive disorder characterized by the deficiency of an enzyme responsible for processing the essential amino acid phenylalanine. You and your mate are both normal. But, if your mother had PKU (your father was 'PP'), and your mate's father was a carrier (but his/her mother was 'PP'), what is the probability that your child will have PKU?

Your mom had PKU and your father was PP so you are Pp.
 Your mate's father was Pp and his mother was PP so he's either PP or Pp

If your mate is PP, then you have PP & Pp which is a 0/4 chance of PKU.
 If your mate is Pp, then you have Pp & Pp then there is a 1/4 chance of PKU.

Overall chance = 1/8

7. Cystic Fibrosis is an autosomal recessive disease. If you and your mate are both carriers for cystic fibrosis (Cc), what is the probability that your child will have cystic fibrosis?

1/4 = 25%

8. If your father died of Huntington's disease (an autosomal dominant disease),
- what is the probability that you will get the disease (assume that your father's mother was normal and assume *your* mother is normal)? Remember back to the polydactyly example...this is a 'bad' trait that is dominant, so both homozygous dominant and heterozygous individuals will have the disease.

Father was Hh because he had it but his mom was hh. Your mom is normal (hh) so the chance you get it is 1/2.

- What is the probability that your child will get Huntington's if you married a homozygous recessive individual (again, you still don't know your genotype)?

If you have it you'll be Hh. Your mate is hh. In this case there's a 1/2 chance.

If you don't have it you're hh, your mate is hh, so there's a 0/2 chance.

Thus the overall chance is 1/4.

- What if you married a person who was heterozygous for the gene?

You: Hh, your mate: Hh, chance: 3/4

You: hh, your mate: Hh, chance: 1/2 = 2/4

So overall chance = 5/8

Hardy-Weinberg Equation Application

Student Guide

Applying the Hardy-Weinberg Equation

Below are several problems designed to help you learn how to use the Hardy-Weinberg equation. They represent real-life applications of this theorem.

1. An autosomal dominant disease is found in 5% of the population (remember, this is where the allele responsible for the disease is dominant). (Hint: Think about which genotypes will have the disease if the disease is dominant; then, find out how many people are 'normal'). Assuming the population is in Hardy-Weinberg equilibrium,

- (a) What is the frequency of the dominant allele?

3%

q = 0.15
p = 0.85

- (b) What is the frequency of the recessive allele?

q = 0.174

2. An autosomal recessive disease is found in 1 out of every 4000 people. If the population is in Hardy-Weinberg Equilibrium, what percentage of the population is a carrier (Aa) for the disease?

3%

3. Brown hair is dominant to white hair in rats. 7 out of 12 rats have brown hair. The rest have white. What is the brown hair allele frequency in the population if it is in Hardy-Weinberg Equilibrium?

35%

4. Approximately what percentage of rats (referring to the previous problem) would be homozygous dominant?

12.5% ≈ 13%

5. I have a population of beetles whose wings vary in coloration from light brown to black. In forested areas, the approximate allelic frequencies are 90% brown to 10% black; whereas, in the lava fields between forest glades, the allelic frequency is 10% brown to 90% black. Give a hypothesis that would explain the difference in allelic frequencies between these two populations.

This is likely because the brown fur increases fitness to a degree than the black in the forest (and the opposite is true in lava fields).

6. If a predator was introduced into this area that hunted beetles by detecting their movement, rather than their color, how might this affect the allelic frequencies in each area and why?

Frequencies will NOT change because the trait no longer effects their fitness.

7. Let's say that a new volcano erupted and decimated the lava field's population of beetles and the original color-sensing predator is back. Over the next five years, the lava fields were re-populated with the beetles from the forest glades. Predict allelic frequencies in the lava fields starting at the end of these five years and extending ten years into the future. Describe your prediction.

Begin looking mostly brown, selection will make it mostly black, then it will stay that way.

8. In a biology class, 64% of the students were able to roll their tongues. Assume that the ability to tongue roll is influenced by a dominant allele (T). Assuming our classroom is a good representation of our overall population and that our population is in Hardy-Weinberg equilibrium with respect to this trait,

- (a) What is the frequency of the tongue roller and the non-tongue roller alleles (T,t) in this class?

$$P(T) = 0.4 \\ P(t) = 0.6$$

- (b) What do you expect the frequency of tongue rolling to be in the next generation?

same

9. There are two alleles for corn kernel color: Yellow and Green. Yellow (Y) is dominant to green (y). Let's say that the frequency of the yellow allele, p = .2 and the frequency of the green allele, q = .8. Given that the yellow allele is dominant, what will happen to the allele frequencies over the next few generations?

won't change

10. If the population is in Hardy-Weinberg equilibrium, what is

- (a) the expected genotype frequencies in the next generation?
 (b) the expected phenotype frequencies in the next generation?

$$(a) YY = 4\%$$

$$Yy = 32\%$$

$$yy = 54\%$$

(b) Phenotype - Dom
= 36%

Phenotype - Rec
= 64%

Phenotype - Rec
= 64%

11. Cystic fibrosis (CF) is a disabling and often fatal disease inherited as an autosomal recessive characteristic. CF affects cell membrane function and as a consequence causes problems with the glands that produce mucus, digestive enzymes and sweat. Affected individuals often have digestive, respiratory, and reproductive problems. CF occurs in about 1 of every 2000 births in the US white population.

- (a) From this data, what would you estimate to be the proportion of the CF allele in the US white population given that we are in Hardy-Weinberg equilibrium?

 - (b) What is the frequency of heterozygous carriers of the CF allele who are most often parents of children with CF?
12. In a hypothetical bug population in Hardy-Weinberg equilibrium, 58% of the bugs are black and 42% of the bugs are white. Considering that black is dominant to white, what percentage of the bugs are heterozygous?

Hardy-Weinberg Application

Student Guide

1. Tharus People

- a. Provide a hypothesis to explain the difference in Malarial susceptibility between these two populations:
 - b. Can you devise a way to test your hypothesis?
 - c. If the people in Terai were **one** population in H-W equilibrium, what pattern would you expect for their genotypic frequencies?
 - d. Given the genotypic frequencies, calculate the allelic frequencies for each of the four populations (p & q):
 - e. Looking at their frequencies, do all the people living in Terai:
 - a. Represent one population living in Hardy-Weinberg equilibrium, OR
 - b. Differential selection caused them to be two different populations
 - f. What do you predict will happen to the genotype frequencies of the Tharus people over time?

2. Kenya Lowlands

- a. What is a reasonable hypothesis to explain the difference of morbidity rates?

 - b. If lowland and highland people were one population in H-W equilibrium, what pattern would you expect?

- c. Use their results to calculate allelic frequencies for each population:

- d. Why do we see **zero** HbSS individuals?

- e. Considering what you now know regarding HbAS and HbSS, what do you suppose this will do to their genotype frequencies over time?

3. West Africa

- a. What is a reasonable hypothesis to explain the situation in Burkina Faso?
- a. Based on the genotype frequencies provided, calculate allele frequencies in healthy subjects and in malaria patients.

- b. Is this one population in H-W equilibrium?
- c. What assumption of H-W is being violated?
- d. What is causing Natural Selection to occur?

- e. What must happen to the HbS allele and to malarial rates for this prediction to come to pass?

- f. If α -thal, HbS, and HbC give selective advantages in malaria-endemic areas, why haven't all populations evolved the same resistance gene?
- g. If instead of a novel Hemoglobin gene we were talking about a novel mechanism for egg fertilization by sperm, could this population-specific evolution lead to speciation? How?

Applying Genetics, Day 1

Student Guide

Mendel came up with two laws to describe the patterns he saw in genetic inheritance.

- **The Law of Segregation:** For any one gene, a person has two alleles. During the formation of gametes (egg or sperm), these two alleles are separated so that each gamete ends up with only one allele.
 - **The Law of Independent Assortment:** During this separation of alleles that occurs, each gene separates independently of the other genes.
1. Draw the process of Meiosis below using $2n=8$. Label on your diagram where each of these laws is fulfilled.

2. Explain the difference between the homologous pairs and sister chromatids.
3. Explain why sperm cells need one copy of each chromosome in terms of genetics.
4. Describe meiosis using the following terms: gene, chromosome, allele, genotype, phenotype, sister chromatid, homologous pair.

Applying Genetics, Part 2

Student Guide

- At this point, you have discovered two inheritance patterns: complete dominance and codominance. To review, fill in the first two sections of the table below:

Type of Inheritance	Potential Genotypes (i.e., what alleles could an individual have?)	Associated Phenotype (i.e., how does an individual appear?)	Pictorial Representation (i.e., illustrate their phenotype)
Complete Dominance			
Codominance			
Incomplete Dominance			

- Notice that the table contains a third inheritance pattern called **incomplete dominance**. It is a mode of inheritance where neither allele is dominant and, as a consequence, heterozygous individuals end up with a phenotype half-way in between the two extreme phenotypes. An example would be coloration in snapdragons. If you cross a red-flowered plant with a white-flowered plant, the offspring have pink flowers. Fill in the third section of the table above.

A group of strangers have embarked on an African safari in Tanzania. Each couple joined the safari group knowing that a world-renowned geneticist (Dr. *insert your name here*) would be on the trip and each of them have some burning genetics questions they want answered. You are up to the task. So, buckle your seatbelts; here we go!

Wanda & Miles

1. Wanda and Miles have joined the trip from Ireland. Wanda comes from a long line of curly, red-haired Irish women and she is curious if their children will have curly hair. Miles happens to have a rare condition in which he never grew hair, so it is impossible to know his phenotype. Miles and all his family have red hair, but his mother has wavy hair, while his father has curly hair. (NOTE: This hair texture follows an incomplete dominant pattern.) Given what you know about Miles' parents' phenotype, what is the probability that Miles would have had curly hair had he been able to grow it?

For curly and straight, wavy is in the middle. Let straight be a let curly be b, then Miles' mother is ab, and his father is bb. possible genotypes: ab, ab, bb, bb

50% wavy, 50% curly

2. Not knowing his genotype, what is the overall probability that Wanda and Miles could have kids with curly hair?

Wanda has bb.

If miles has ab, it's 50% chance of kid with curly

If miles has bb, it's 100% chance... overall chance: 0.75

Jacques & Sabrine

3. Jacques and Sabrine have joined the group from France. Sabrine's mother, Antoinette, died of Huntington's disease as did Sabrine's maternal grandfather (Huntington's Disease is an autosomal dominant disease that causes neurodegeneration and is 100% fatal; people with the disease usually start showing symptoms as middle-aged adults). However, her maternal grandmother did not have the disease. Sabrine's father also does not have the disease and is alive and well. What is the probability that Sabrine has also inherited Huntington's disease?

mother could have Aa, grandfather could be Aa or AA. grandmother had to be aa for the gene. father is also aa.

So mother is Aa and father is aa which means Sabine has a .5 probability to get it (Aa)

4. What is the probability that Sabrine and Jacques' child will get Huntington's disease if Jacques is a homozygous recessive individual? $(0.5 \times 0.5) + (0.5 \times 0) = 0.25$
5. What if Jacques were heterozygous for the diseased gene?
 $(0.5 \times 0.75) + (0.5 \times 0.5) = 5/8$

Mindy & Joseph

6. Mindy and Joseph joined the safari from West Virginia. In reading her late mother's journals, Mindy discovered that her mother had a sister who died of cystic fibrosis (CF), an autosomal recessive disease that frequently causes death due to lung failure (NOTE: assume that if a person makes it to adulthood without symptoms, they do not have CF).

Mindy's mother never had any symptoms of CF, but Mindy wants to know what the odds are that her mother was a carrier of the disease (i.e., heterozygous, Aa)?

mother's sister was aa, which means parents had to be Aa or aa. So the chances her mother had it are:

7. What are the odds that Mindy is a carrier? (NOTE: Mindy's father was homozygous and did not have CF and we know Mindy's mother did not have the disease.)
8. Joseph has a brother with CF. What are the odds that Joseph is a carrier?
9. Knowing that neither Mindy nor Joseph have CF, if they decide to have children, what are the odds that their child could have CF?

Gunnar & Einar

10. Gunnar and Einar are brothers from Iceland who joined the safari as part of a wild adventure across Africa. They are both rather daring and a little reckless. During the expedition, the group spotted a northern white rhino, which was previously thought to be extinct. Understandably excited, the group moved closer and closer to get pictures. Gunnar and Einar decided they wanted to touch the rhino. Unfortunately, you can guess what happened: Gunnar got gored by the rhino! He has lost a great amount of blood on his way to the hospital tent. He is in need of an immediate blood transfusion. Gunnar served in the military and knows that his blood type is A-. What type(s) of blood can he safely receive as a transfusion?
11. None of the guests know their blood types and no blood is on hand in the hospital tent. Einar, who does not know his blood type, volunteers to donate, hoping that due to his sibling relationship, he's Gunnar's best bet. They know that their parent's blood types were O- and A+, and that their sister is O-. Quickly, you do out the odds of whether or not Einar's blood will work, obviously not wanting to take the risk if the odds are less than 100%. How confident are you that Einar's blood will work? Will you do the transfusion?

Thankfully, just before anyone decides to take the risk, a helicopter arrives to whisk Gunnar off to the closest hospital where he will make a full recovery. Phew! The guests are amazed at your knowledge and want to know more about blood types. You teach them and then decide to give them a little quiz. Help them solve it:

Given these individuals and their blood types, answer the following questions:



Travis
AB+



Jane
A-



Bob
A+



Bertha
O+



Andy
O-



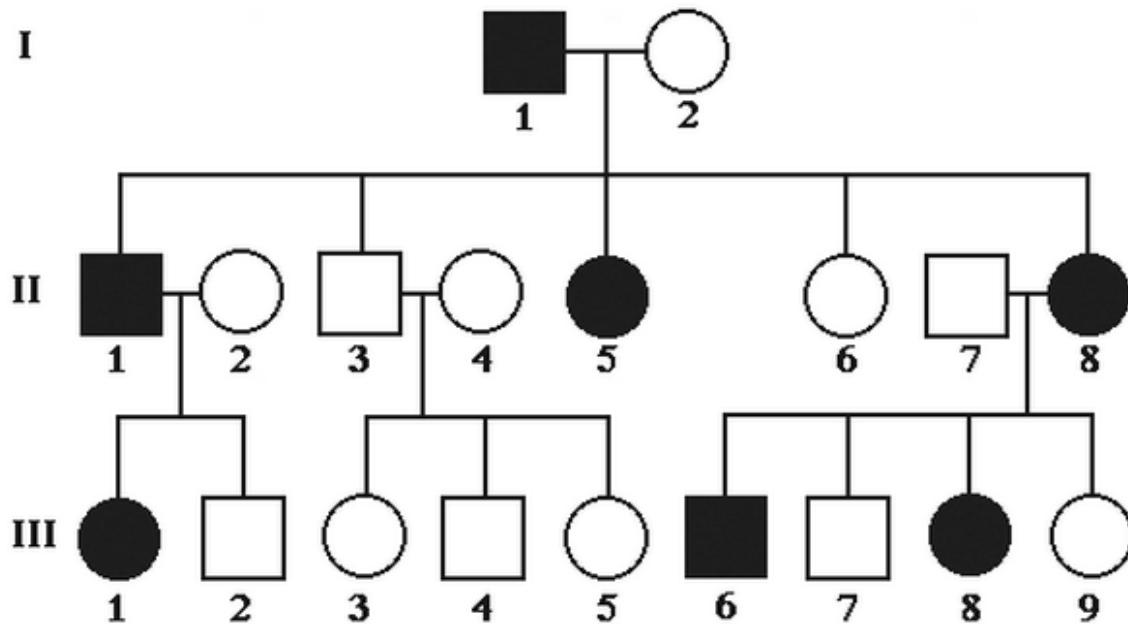
Alice
B+

12. Which man could NOT produce a child with O- blood? Why not?
13. The +/- symbol stands for the presence or absence of the Rh factor on the red blood cell. Women who are Rh- who become pregnant with an Rh+ child must receive a special shot to avoid rejecting their fetus. Which woman will potentially need this shot? Why?
14. Who could she marry to avoid this shot altogether? Why?
15. Which three couples could produce children with type-AB+ blood?
16. Which couple will ONLY produce children with type-O blood?

Applying Genetics, Part 3

Student Guide

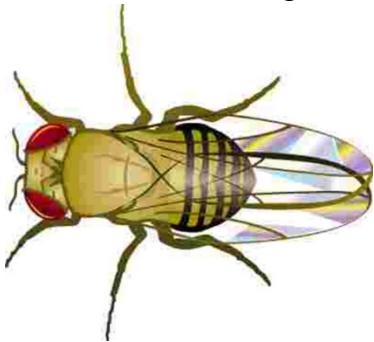
Given the following pedigree chart for an autosomal trait, answer the following questions (remember, squares represent boys, circles represent girls, lines represent a mating and offspring, and if the individual is filled in it indicates that they display the trait being mapped):



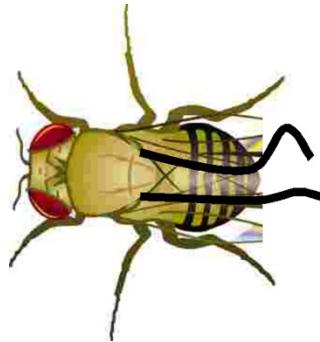
1. If individual I2 is homozygous, is this an autosomal dominant or autosomal recessive trait?
2. What is the genotype of individual II3?
3. What is the genotype of individual III6?
4. If individual I2 is heterozygous, is this an autosomal dominant or autosomal recessive disease?
5. Given that individual I2 is heterozygous, what must be the genotype of individual III7?

6. In Loch Ness Monsters, three humps are dominant to two humps, and fire-breathing ability is dominant to non-fire-breathing. I crossed a three-humped, fire-breathing monster with a two-humped, non-fire-breathing monster. All of my baby monsters were three-humped and fire-breathing.
- What were the genotypes of the mom and dad monsters? _____
 - What are the genotypes of the babies? _____
 - If I crossed a baby brother with a baby sister, what would the expected genotypic and phenotypic frequencies be?
- d. If I got a ratio of three three-humped, fire-breathing monsters, for every one two-humped, non-fire-breathing monster, what would I suspect about these genes? _____
7. Explain in one paragraph why males inherit X-linked recessive traits more commonly than females

8. The two fruit flies shown below were mated. Red eyes and normal wings are dominant to black eyes and vestigial wings. Answer the following questions about this mating:



Female: Red Eyes, Normal Wings



Male: Red Eyes, Vestigial Wings

- a. What are the possible genotypes of the female:
- b. What are the possible genotypes of the male:
- c. Pick one of the possible genotypes for each parent and perform a cross.

- d. Report the genotypes of the offspring:
- e. Report the phenotypes of the offspring:

9. MendAliens were found at a crash site in Roswell, New Mexico and were taken to Area 51. Scientists are now trying to map the chromosomes of these MendAliens. They are doing so by performing test crosses (a test cross is when you cross an individual of unknown genotype to an individual who is homozygous recessive). The current chromosome of interest is MendAlien Chromosome 2. Scientists believe that the genes for eye size and antenna are found on MendAlien Chromosome 2. Large eyes are dominant to beady eyes and normal antenna is dominant to vestigial antenna. The scientists crossed heterozygous individuals with homozygous recessive individuals and got the following table:

Phenotype	# of offspring
Large Eyes, Normal Antenna	205
Beady Eyes, Vestigial Antenna	202
Large, Eyes, Vestigial Antenna	45
Beady Eyes, Normal Antenna	43
Total	495

- a. What is the recombination frequency for these two genes?

- b. This alien species also has a telepathic gene on Chromosome 2. Given that the recombination frequency between the eye size gene and the telepathic gene is 20.4%, and the frequency between the antenna and telepathic gene is 38.2%, can you tell the order of the genes on the chromosome?

Nondisjunction
Student Guide

1. Illustrate Nondisjunction in Meiosis I

2. Illustrate Nondisjunction in Meiosis II

REMEMBER: homologous pairs are not necessarily identical; sister chromatids are identical. This will help you as you answer the following questions.

Sex Aneuploids. As we also discussed in class, most aneuploids (conditions with 'not the true number' of chromosomes) are not survivable. Exceptions include trisomy of the 21st chromosome (Down Syndrome, can live a long life), trisomy of the 18th chromosome (Edwards Syndrome, death usually occurs within the first few days), and trisomy of the 13th chromosome (Patau Syndrome, death usually occurs within the first month). However, nondisjunction of the sex chromosomes (the X and Y) are much more survivable.

1. The first we will discuss is Poly X or Triple X Syndrome. This is where a girl has three X chromosomes (XXX). Triple X syndrome often has no associated physical features or medical problems. A small proportion of women with the condition may have menstrual irregularities as well as learning disabilities, delayed speech, and compromised language skills. Most, however, are normal. How and when does Poly X occur—mom? Dad? Meiosis I? Meiosis II?
2. The second is Klinefelter Syndrome. This is where a male has an extra X chromosome (XXY). Most individuals don't even know they have it. The main problems seen are small testicles and reduced fertility. How and when does Klinefelter Syndrome occur—mom? Dad? Meiosis I? Meiosis II?
3. The third is called Jacob Syndrome. This is where a male has an extra Y chromosome (XYY). Most of the time there are no unusual features. Sometimes they are taller than average and may have some learning disabilities as well as delayed speech and language skills. How and when does Jacob Syndrome occur—mom? Dad? Meiosis I? Meiosis II?

4. The last one is actually a Monosomy. It is when a child gets only one X chromosome (X0) and it is called Turner Syndrome. (If you are wondering, Y0 is not survivable.) Turner's individuals are female but often they do not develop secondary sex characteristics (e.g., breasts, hips). Thus, it is often difficult to determine their gender just by looking at them. Anatomically, however, they are definitely a female. How and when does Turner Syndrome occur—mom? Dad? Meiosis I? Meiosis II?
5. Let's say that you have a couple: James and Janette. Neither of them are colorblind; however, they have a child with Klinefelter's (XXY) who happens to be colorblind. (Remember, colorblindness is an X-linked **recessive** trait). In which parent and at what phase did nondisjunction occur?
6. Let's say that James has hemophilia, an X-linked **recessive** disorder. Let's say they have a second child who is Turner's Syndrome (X0) and the child has hemophilia (this is one unlucky couple!). In which parent and in which phase could nondisjunction have occurred?

Cancer Application

Student Guide

Part 1. Regulations of the Cell Cycle

Review the Cell Cycle that you learned in your homework:

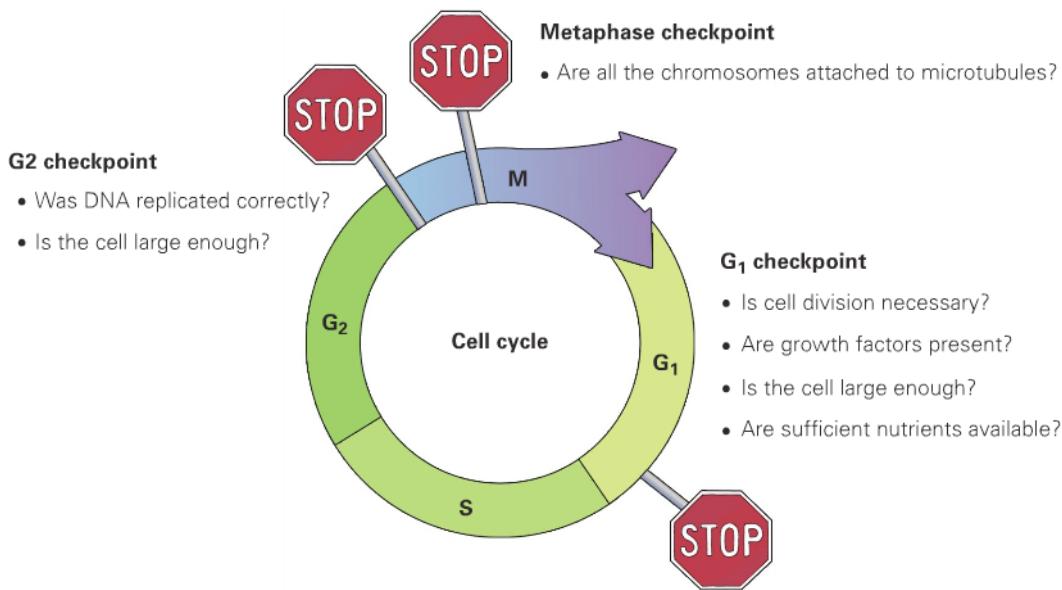


Figure 5.10 Controls of the cell cycle. Checkpoints at G₁, G₂, and metaphase determine whether a cell will continue to divide.

1. For each condition below, decide which checkpoint would likely catch the error:
 - a. DNA damage from UV irradiation (i.e., being in the sun): **G₁**
 - b. DNA replication error: **G₂**
 - c. Spindle fibers ('ropes) not connected properly: **M**
 - d. The cell is not needed: **G₁**
2. Let's discuss the genes involved in these checkpoints. Define the following:
 - a. Tumor Suppressor Genes:
Suppresses tumors, regulates cell growth.
 - b. Proto-Oncogenes:
3. Based on these definitions, which kind of mutation in a **tumor suppressor gene** would lead to cancer?
 - a. A mutation that makes it inactive
 - b. A mutation that makes it overactive

4. Given that you have two copies of each of these genes (i.e., you are AA, or Aa, or aa), how many mutations in a **tumor suppressor** would you need for it to lead to cancerous tendencies?
 - a. Only one; therefore, it is dominant
 - b. Two; therefore, it is recessive
 5. Based on these definitions, which kind of mutation in a **Proto-oncogene** would lead to cancer?
 - a. A mutation that makes it inactive
 - b. A mutation that makes it overactive
 6. Given that you have two copies of each of these genes (i.e., you are AA, or Aa, or aa), how many mutations in a **proto-oncogene** would you need for it to lead to cancerous tendencies?
 - a. Only one; therefore, it is dominant
 - b. Two; therefore, it is recessive
 7. How do **telomeres** play a role in preventing cancer?
-
8. Luckily, we have several tumor suppressor genes, several proto-oncogenes, and other back-up mechanisms. Thus, a single mutation is not likely to cause cancer (we have a back-up). So, how, do you suppose, cancer occurs?

 9. What is the difference between a benign and malignant tumor?
-
-
-
10. How can we kill cancer cells if they look just like our cells!

You gotta almost kill yourself and hope the group you kill is the cancer? What is the one thing cancer cells do more often than regular cells? Mitosis. 3 treatments:

1. Slash
2. Burn
3. Poison

Part 2. Cancer WebQuest

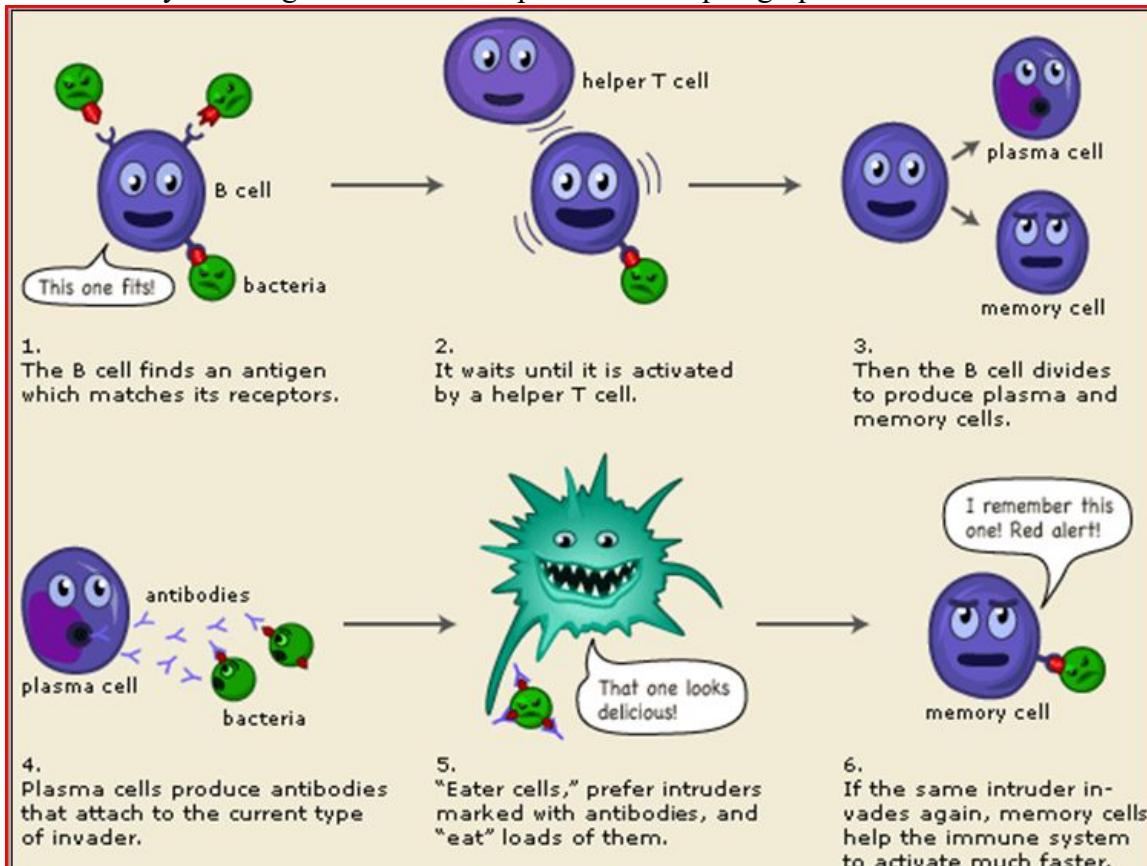
Use the American Cancer Society's website, Google "Cancer Facts and Figures, 20__" and download the PDF, to answer the following questions:

11. What state has the most New Cancer Cases?
12. What is the most common cancer by death rates for
 - a. Men?
 - b. Women?
13. What is the most common cancer by New Cases for
 - a. Men?
 - b. Women?
14. Scroll down to lung cancer. What are the top two risk factors?

Applying Principles of Disease

Student Guide

- Analyze the figure below and explain it in one paragraph:



Answer the following questions:

- How does a B cell know which antibodies to produce?
- What is the purpose of a memory cell?

- Using this information, is it possible to design a way to produce memory cells (i.e., immunity) *without* getting sick? With your group, brainstorm how you might do this. Remember the information you learned about the structure of viruses and bacteria from your explore activity.

Skim the following article: <http://www.jennermuseum.com/vaccination.html>

- Describe below, how the cowpox vaccination method works:
- Choose **2** of these myths and see if you can figure out what the truth is.
 - Myth 1: Getting so many vaccines will overwhelm my child's immune system.
 - Myth 2: As long as other children are getting vaccinated, mine don't need to be.
 - Myth 3: Now that major illnesses have largely disappeared, we really don't need vaccines anymore.
 - Myth 4: Vaccines cause autism and other disorders.
 - Myth 5: My baby might get the disease it's supposed to prevent.
 - Myth 6: Vaccines can contain preservatives that are dangerous.
 - Myth 7: You shouldn't give a vaccine to a child who has a cold.
 - Myth 8: I had chicken pox when I was a kid and it isn't a big deal.
 - Myth 9: Vaccines can provide 100 percent disease protection.
 - Myth 10: It's best to wait until children are older before starting to give them vaccines.

Herd Immunity

Together as a class, you will visit the following site:

<http://www.software3d.com/Home/Vax/Immunity.php> This website is simulating what we call, “Herd Immunity”. The screen is divided into two halves (there is no line indicating the divide). When an individual is green, they are vaccinated. If they are red, they have not been vaccinated. By clicking on an individual, you are infecting them and they will turn black. If they infect others, others will turn black. You can choose the following parameters:

- Immunization rate on the left: This is the percentage of the population who are vaccinated on the left-side of the screen
- Immunization rate on the right: This is the percentage of the population who are vaccinated on the right-side of the screen (if you check the box “Same as left”, it will automatically match them)
- Infection rate if not vaccinated: This is specific to any given pathogen. It is the virulence rate or the chances you will catch the disease if you are exposed to it.
- Infection rate if vaccinated: If you looked up myth 9, you will have realized that vaccines are never 100% effective. So, depending on the vaccine, you still have a small chance of being infected, even if you are vaccinated.
- Infection speed: This is the spread rate and really depends on how crowded the population is and how often people come in contact with one another. I usually leave this at the default of 10.

7. We will try a simulation. According to Immunize-Utah.org, Utah county’s vaccination rate for MMR (Measles, Mumps, and Rubella) is 60.4% (compared to Utah state’s average of 92.6% and the US average of 91.9%). So, let’s make the left side of the simulation the United States and the right side Utah county. Also, according to the CDC, you have a 90% chance of getting the virus if exposed an unvaccinated, but only a 3% chance if you have been properly vaccinated. Input the correct numbers into the simulation and try infecting 5 people on each side. Record the following information

- a. Total percentage of the population infected: _____
- b. Percentage of vaccinated population infected: _____
- c. Percentage of unvaccinated population infected: _____
- d. Percentage of infected that had been vaccinated: _____

With your group, answer the following questions:

8. How do vaccinated individuals protect unvaccinated individuals? (Pay particular attention to the left-hand-side of the screen)
9. How do unvaccinated individuals hurt vaccinated individuals? (Pay particular attention to the right-hand-side of the screen)
10. What can you conclude about the importance of vaccinations in a community where we come into contact with others?

11. (This is an opinion question; therefore, there is no right or wrong answer. It is just something I want you to think about): Is the decision to vaccinate yourself or your children a decision that only affects you? Should this be a decision that you are allowed to make on your own if you interact with the public (e.g., your children attend public schools, you go to the movies in a public theater, you eat out at restaurants, etc.)? Explain your reasoning.

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February 29, 2008 / 57(08);203-206

Outbreak of Measles --- San Diego, California, January--February 2008

On February 22, this report was posted as an MMWR Early Release on the MMWR website (<http://www.cdc.gov/mmwr>).

Measles, once a common childhood disease in the United States, can result in severe complications, including encephalitis, pneumonia, and death. Because of successful implementation of measles vaccination programs, endemic measles transmission has been eliminated in the United States and the rest of the Americas. However, measles continues to occur in other regions of the world, including Europe (1). In January 2008, measles was identified in an unvaccinated boy from San Diego, California, who had recently traveled to Europe with his family. After his case was confirmed, an outbreak investigation and response were initiated by local and state health departments in coordination with CDC, using standard measles surveillance case definitions and classifications.* This report summarizes the preliminary results of that investigation, which has identified 11 additional cases of measles in unvaccinated children[†] in San Diego that are linked epidemiologically to the index case and include two generations of secondary transmission. Recommendations for preventing further measles transmission from importations in this and other U.S. settings include reminding health-care providers to 1) consider a diagnosis of measles in ill persons who have traveled overseas, 2) use appropriate infection-control practices to prevent transmission in health-care settings, and 3) maintain high coverage with measles, mumps, and rubella (MMR) vaccine among children.

The index patient was an unvaccinated boy aged 7 years who had visited Switzerland with his family, returning to the United States on January 13, 2008. He had fever and sore throat on January 21, followed by cough, coryza, and conjunctivitis. On January 24, he attended school. On January 25, the date of his rash onset, he visited the offices of his family physician and his pediatrician. A diagnosis of scarlet fever was ruled out on the basis of a negative rapid test for streptococcus. When the boy's condition became worse on January 26, he visited a children's hospital inpatient laboratory, where blood specimens were collected for measles antibody testing; later that day, he was taken to the same hospital's emergency department because of high fever 104°F (40°C) and generalized rash. No isolation precautions were instituted at the doctors' offices or hospital facilities.

The boy's measles immunoglobulin M (IgM) positive laboratory test result was reported to the county health department on February 1, 2008. During January 31--February 19, a total of 11 additional measles cases in unvaccinated infants and children aged 10 months--9 years were identified. These 11 cases included both of the index patient's siblings (rash onset: February 3), five children in his school (rash onset:

January 31--February 17), and four additional children (rash onset: February 6--10) who had been in the pediatrician's office on January 25 at the same time as the index patient. Among these latter four patients, three were infants aged <12 months. One of the three infants was hospitalized for 2 days for dehydration; another infant traveled by airplane to Hawaii on February 9 while infectious.

Two generations of measles cases were identified. The first generation (eight cases) included the index patient's two siblings, two playmates from his school, and the four children from the pediatrician's office. The second generation cases included three children from the index patient's school: a sibling of a child from the first generation and two friends of one of the index patient's siblings ([Figure](#)).

California allows personal beliefs exemptions (PBEs) to vaccinations required of schoolchildren[§]; parents can request exemptions if all or some vaccinations are contrary to their beliefs. The index patient and one of his siblings attended a school with 376 children, who ranged in age from 5 to 14 years. Thirty-six (9.6%) of the children had PBEs on file at the school. Among the nine patients aged \geq 12 months, including the index patient, eight were unvaccinated because of PBEs. Among the 36 schoolchildren with PBEs, four had documentation of previous measles vaccination, 11 were vaccinated during the outbreak, and the remaining 21, who did not have evidence of immunity to measles, were placed under voluntary quarantine for 21 days after their last exposure. Overall, approximately 70 children exposed to children with measles in the school, a day care center, the pediatrician's office, and other community settings were placed under voluntary home quarantine because their parents either declined measles vaccination or they were too young to be vaccinated.

As part of the public health response in San Diego, surveillance has been enhanced to identify additional rash illnesses, and outbreak response measures in the community are ongoing. In Hawaii, ongoing response measures include following up airplane and other contacts of the infant who traveled to Hawaii to inform them of their potential exposure and refer them to their physicians regarding their susceptibility to measles. Five exposed infants, four airplane contacts, and one personal acquaintance were administered immune globulin within 72 hours of exposure. No secondary cases have been identified in Hawaii to date.

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Editorial Note:

Once ubiquitous, measles now is uncommon in the United States. In the prevaccine era, 3 to 4 million measles cases occurred every year, resulting in approximately 450 deaths, 28,000 hospitalizations, and 1,000 children with chronic disabilities from measles encephalitis. Because of successful implementation of measles vaccination programs, fewer than 100 measles cases are now reported annually in the United States and virtually all of those are linked to imported cases ([2,3](#)), reflecting the incidence of measles globally and travel patterns of U.S. residents and visitors. During 2006--2007, importations were most common from India, Japan, and countries in Europe, where measles transmission remains endemic and large outbreaks have occurred in recent years (CDC, unpublished data, 2008). Since November 2006, Switzerland has

experienced that country's largest measles outbreak since introduction of mandatory notification for measles in 1999 (1).

The San Diego import-associated outbreak, affecting exclusively an unvaccinated population and infants too young to be vaccinated, serves as a reminder that unvaccinated persons remain at risk for measles and that measles spreads rapidly in susceptible subgroups of the population unless effective outbreak-control strategies are implemented. Although notable progress has been made globally in measles control and elimination, measles still occurs throughout the world. U.S. travelers can be exposed to measles almost anywhere they travel, including to developed countries. To prevent acquiring measles during travel, U.S. residents aged ≥ 6 months traveling overseas should have documentation of measles immunity before travel (4). Travel histories should be obtained and a diagnosis of measles should be considered by physicians evaluating patients who have febrile rash illness within 3 weeks of traveling abroad.

Measles virus is highly infectious; vaccination coverage levels of >90% are needed to interrupt transmission and maintain elimination in populations. The ongoing outbreak in Switzerland, which has resulted in hospitalizations for pneumonia and encephalitis, has occurred in the context of vaccination coverage levels of 86% for 1 dose at age 2 years and 70% for the second dose for children aged <12 years. In the United States, vaccination coverage levels for at least 1 dose of MMR vaccine have been >90% among children aged 19--35 months and >95% among school-aged children during this decade. Although not measured routinely, 2-dose vaccine coverage is extremely high among U.S. schoolchildren because of school vaccination requirements.

Measles transmission in schools was common in the era before interruption of endemic-disease transmission, and school requirements for vaccination have been a successful strategy for achieving high vaccination coverage levels in this age group and decreasing transmission in school settings. In the United States, all states require children to be vaccinated in accordance with Advisory Committee on Immunization Practices recommendations before attending school (4). However, medical exemptions to immunization requirements for day care and school attendance are available in all states; in addition, 48 states offer nonmedical religious exemptions, and 21 states (including California) offer nonmedical PBEs.^J These exemptions are defined differently by each state. The PBE allowed by California requires only a parental affidavit (5). Compared with vaccinated persons, those exempt from vaccination are 22 to 224 times more likely to contract measles (5--7).

The community transmission that has occurred during the San Diego outbreak is consistent with previous observations that the frequency of vaccination exemptors in a community is associated with the incidence of measles in that community; in addition, imported measles cases have demonstrated the potential for sizeable outbreaks in U.S. communities with suboptimal vaccine coverage (5,6,8). The public health response to this outbreak has included identification of cases, isolation of patients and vaccination, administration of immune globulin, and voluntary quarantine of contacts who have no evidence of measles immunity. Costs associated with control of these outbreaks can be substantial. In Iowa, the public health response to one imported measles case cost approximately \$150,000 (9).

This outbreak also illustrates the risk for measles transmission in health-care settings. Airborne transmission of measles has been reported in emergency departments, physician offices, and pediatric ambulatory care-settings (10). Persons exposed to measles should be instructed to inform all health-care providers of their exposure before entering a health-care facility. Health-care personnel providing care to suspected measles patients (i.e., patients with febrile illness and generalized maculopapular rash or known contacts with

prodromal symptoms) should apply appropriate isolation practices, including airborne precautions, in addition to taking standard precautions for such patients.**

Once a suspected measles case has been identified, prompt isolation of the potentially infectious patient and implementation of appropriate infection-control measures can help to decrease risk for transmission. Patients with suspected measles should be placed in an examination room, preferably an airborne-infection isolation room, as soon as possible and should not be permitted in patient waiting areas. Until placed in an airborne-infection isolation room, the patient should wear a surgical mask. If a surgical mask cannot be tolerated, other practical means to contain respiratory aerosols should be implemented. The door to the examination room should be kept closed, and all health-care personnel in contact with the patient should be documented as immune to measles. Health-care personnel and visitors without evidence of immunity (i.e., documentation of adequate vaccination, laboratory evidence of immunity, born before 1957, or documentation of physician-diagnosed measles) should be restricted from entering the rooms of patients known or suspected to have measles (4,10). The examination room should not be used for 2 hours after the infectious patient leaves. Suspected measles patients should not be referred to other locations for laboratory tests unless infection-control measures can be implemented at those locations.

Measles morbidity and mortality can be reduced through vaccination with MMR vaccine. Vaccination of U.S. travelers can reduce measles importations. Sustained high population immunity through vaccination, effective surveillance, and robust public health preparedness and response capacity are needed to keep the United States free from indigenous measles transmission and control any outbreaks associated with importations.

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* Available at http://www.cdc.gov/ncphi/disss/nndss/casedef/measles_current.htm.

† One case was identified in a girl aged 2 years whose vaccination was delayed. The girl had received a dose of single antigen measles vaccine routinely. However, investigators later determined that she had been exposed to measles 6 days before vaccination. Because postexposure vaccination is only considered effective if administered within 3 days of exposure and because immunity takes several weeks to develop, investigators considered the girl unvaccinated.

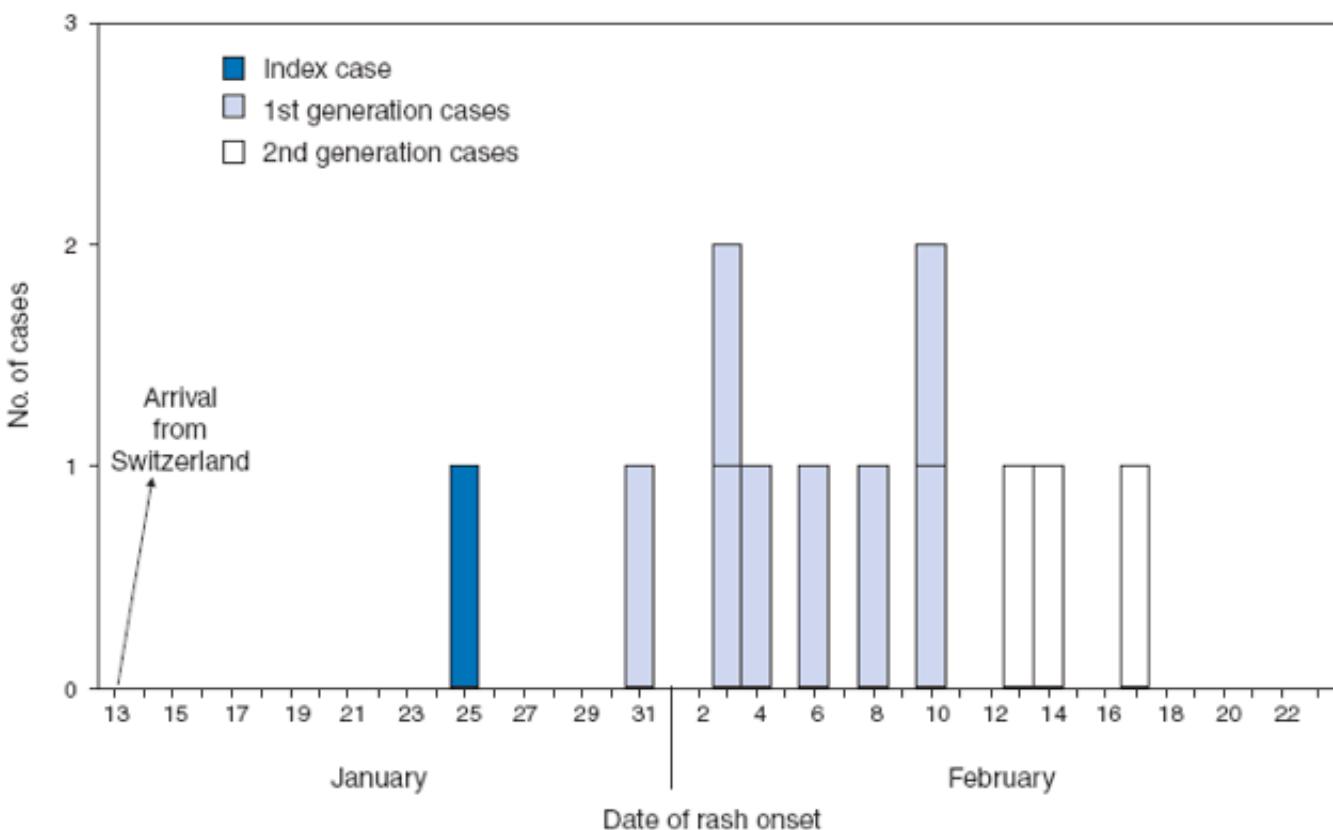
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Figure

FIGURE. Number of epidemiologically linked cases (N = 12) in a measles outbreak, by date of rash onset — San Diego, California, January–February 2008



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HEALTH GUIDES

VACCINE MYTHS DEBUNKED

GUIDE SECTIONS



VACCINE MYTHS DEBUNKED

U.S. public health officials and physicians have been combating misconceptions about vaccine safety for over twenty years. They've had [mixed success](#). Despite the fact that numerous studies have found no evidence to support the notion that vaccines cause autism and other chronic illnesses, a [growing number](#) of parents are refusing to vaccinate their children.

Researchers [now link](#) falling immunization rates to recent resurgences of vaccine-preventable diseases. In 2010, California saw 9,120 cases of whooping cough, more than any year since the whooping cough vaccine was introduced in the 1940s. Ten infants too young to be vaccinated died of whooping cough during the outbreak. [The CDC warns](#) that events like these will become more frequent and harder to control if vaccination rates continue to fall.

Fears over the safety of vaccines are understandable. The CDC [vaccination schedule](#) calls for children to receive up to 14 inoculations by the age of six – many of them vaccines developed within the last twenty years. Many parents distrust these vaccines; worried about the potential for risks and long-term side effects. Research, however, shows that most of our biggest fears about vaccinations are unfounded. These eight major vaccine myths that research has shown to be baseless:

Myth #1: Vaccines cause autism.

The widespread fear that vaccines increase risk of autism originated with a 1997 study published by Andrew Wakefield, a British surgeon. The article was published in *The Lancet*, a prestigious medical journal, suggesting that the measles, mumps, rubella (MMR) vaccine was increasing autism in British children.

The paper has since been completely discredited due to serious procedural errors, undisclosed financial conflicts of interest, and ethical violations. Andrew Wakefield lost his medical license and the paper was retracted from *The Lancet*.

Nonetheless, the hypothesis was taken seriously, and several other major studies were conducted. None of them found a link between any vaccine and the likelihood of developing autism.

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Today, the true causes of autism remain a mystery, but to the discredit of the autism-vaccination link theory, several studies have now identified symptoms of autism in children well before they receive the MMR vaccine. [And even more recent research](#) provides evidence that autism develops in utero, well before a baby is born or receives vaccinations.

Myth #2: Infant immune systems can't handle so many vaccines.

Infant immune systems are stronger than you might think. Based on the number of antibodies present in the blood, a baby would theoretically have the ability to respond to around 10,000 vaccines at one time. Even if all 14 scheduled vaccines were given at once, it would only use up slightly more than 0.1% of a baby's immune capacity. And scientists believe this capacity is purely theoretical. The immune system could never truly be overwhelmed because the cells in the system are constantly being replenished. In reality, babies are exposed to countless bacteria and viruses every day, and immunizations are negligible in comparison.

Though there are more vaccinations than ever before, today's vaccines are far more efficient. Small children are actually exposed to [fewer immunologic components overall than children in past decades](#).

Myth #3: Natural immunity is better than vaccine-acquired immunity.

In some cases, natural immunity — meaning actually catching a disease and getting sick— results in a stronger immunity to the disease than a vaccination. However, the dangers of this approach far outweigh the relative benefits. If you wanted to gain immunity to measles, for example, by contracting the disease, you would face a [1 in 500 chance](#) of death from your symptoms. In contrast, the number of people who have had severe allergic reactions from an MMR vaccine, is less than [one-in-one million](#).

Myth #4: Vaccines contain unsafe toxins.

People have concerns over the use of formaldehyde, mercury or aluminum in vaccines. It's true that these chemicals are toxic to the human body in certain levels, but only trace amounts of these chemicals are used in FDA approved vaccines. In fact, according to the FDA and the CDC, formaldehyde is produced at higher rates by our own metabolic systems and there is [no scientific evidence](#) that the low levels of this chemical, mercury or aluminum in vaccines can be harmful. See [section III](#) of this guide to review safety information about these chemicals and how they are used in vaccines.

Myth #5: Better hygiene and sanitation are actually responsible for decreased infections, not vaccines.

Vaccines don't deserve all the credit for reducing or eliminating rates of infectious disease. Better sanitation, nutrition, and the development of antibiotics helped a lot too. But when these factors are isolated and rates of infectious disease are scrutinized, the role of vaccines cannot be denied.

One example is [measles](#) in the United States. When the first measles vaccine was introduced in 1963, rates of infection had been holding steady at around 400,000 cases a year. And while hygienic habits and sanitation didn't change much over the following decade, the rate of measles infections dropped precipitously following the introduction of the vaccine, with only around 25,000 cases by 1970. Another example is [Hib disease](#). According to CDC data, the incidence rate for

this malady plummeted from 20,000 in 1990 to around 1,500 in 1993, following the introduction of the vaccine.

Myth #6: Vaccines aren't worth the risk.

Despite parent concerns, children have been successfully vaccinated for decades. In fact, there has never been a single credible study linking vaccines to long term health conditions.

As for immediate danger from vaccines, in the form of allergic reactions or severe side effects, the incidence of death are so rare they can't even truly be calculated. For example, only one death was [reported to the CDC between 1990 and 1992](#) that was attributable to a vaccine. The overall incidence rate of severe allergic reaction to vaccines is usually placed around one case for every one or two million injections.

Myth #7: Vaccines can infect my child with the disease it's trying to prevent.

Vaccines can cause mild symptoms resembling those of the disease they are protecting against. A common misconception is that these symptoms signal infection. In fact, in the small percentage ([less than 1 in one million cases](#)) where symptoms do occur, the vaccine recipients are experiencing a body's immune response to the vaccine, not the disease itself. There is only one recorded instance in which a vaccine was shown to cause disease. This was the [Oral Polio Vaccine \(OPV\)](#) which is no longer used in the U.S. Since then, vaccines have been in safe use for decades and follow [strict Food and Drug Administration \(FDA\) regulations](#).

Myth #8: We don't need to vaccinate because infection rates are already so low in the United States.

Thanks to "herd immunity," so long as a large majority of people are immunized in any population, even the unimmunized minority will be protected. With so many people resistant, an infectious disease will never get a chance to establish itself and spread. This is important because there will always be a portion of the population – infants, pregnant women, elderly, and those with weakened immune systems – that can't receive vaccines.

But if too many people don't vaccinate themselves or their children, they contribute to a collective danger, opening up opportunities for viruses and bacteria to establish themselves and spread.

Not to mention, as the Centers for Disease Control (CDC) [warn](#), international travel is growing quickly, so even if a disease is not a threat in your country, it may be common elsewhere. If someone were to carry in a disease from abroad, an unvaccinated individual will be at far greater risk of getting sick if he or she is exposed.

Vaccines are one of the great pillars of modern medicine. Life used to be especially brutal for children before vaccines, with huge portions being felled by diseases like measles, smallpox,

whooping cough, or rubella, to name just a few. Today these ailments can be completely prevented with a simple injection.

So as science continues to advance and tackle new challenges, people should not forget how many deaths and illnesses vaccines have prevented, and how they continue to protect us from potentially devastating forms of infectious disease.

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Cells and Membranes Apply

Student Guide

The case of the dead gerbils

Let's make a preliminary guess. Who do you think is responsible for killing our gerbils?

Clostridium piliforme

Philodendron

Fluffy

What does **Experiment #1** tell us?

Cells do expand, but they do not rupture.

So we think the culprit has a cell wall.

Since fluffy is a cat, he is OUT.

What does **Experiment #2** tell us?

We are now gonna measure CO₂ levels.

We're testing photosynthesis. In photosynthesis, the plants turn CO₂ into sugar. So if the cell is photosynthesizing, we would expect that CO₂ levels in the same system as the cells decrease over time.

Results: CO₂ levels increased in both dark and light rooms, so it CAN'T be the plant (Philodendron)

What does **Experiment #3** tell us?

Amoxicillin - blocks enzyme that normally creates links between amino acids in peptidoglycan molecules.

Streptomycin - blocks protein synthesis in prokaryotes.

Ciprofloxacin hydrochloride (cipro) - blocks the enzyme needed to counteract excessive twisting of DNA that occurs when circles of DNA are unwound to be copied into DNA or RNA.

Would these kill a strand of bacteria? Yes.

2 of these 3 work on our cell, but not all 3.

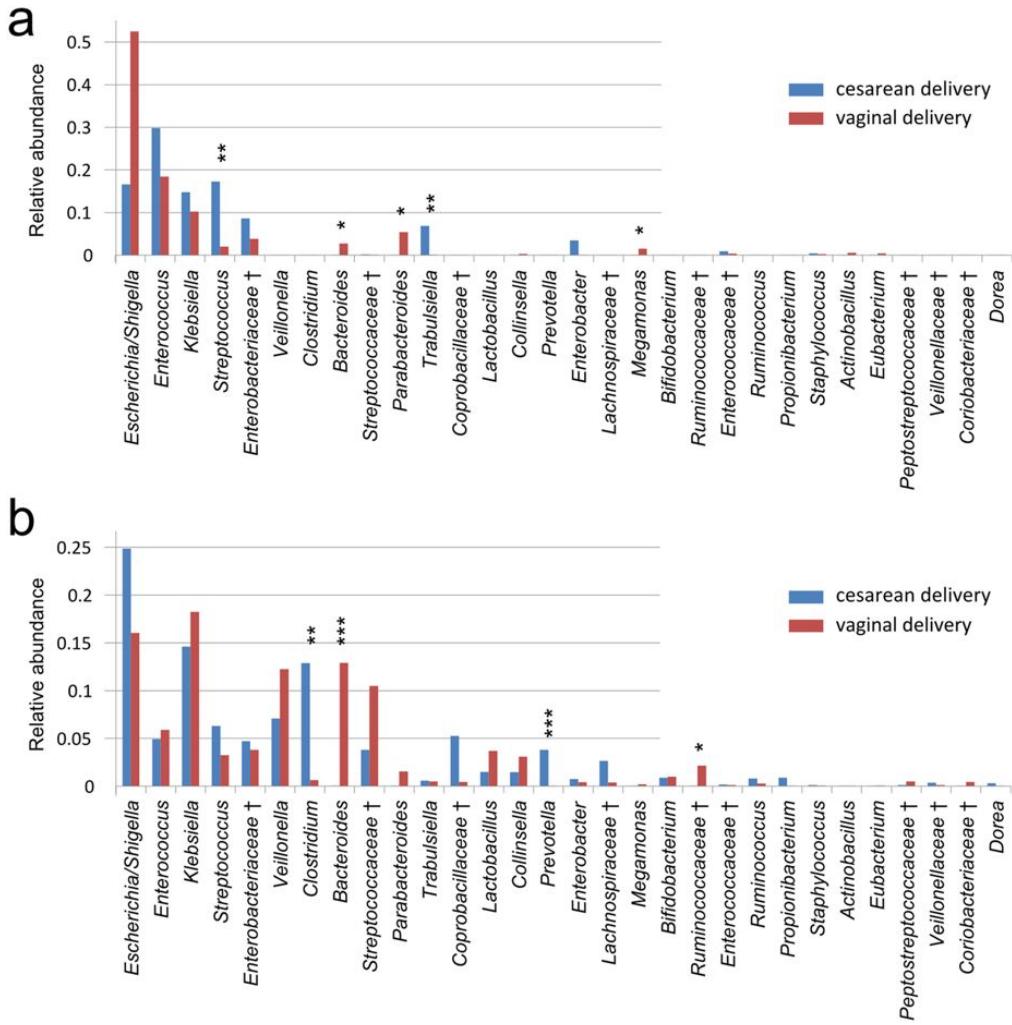
Microbiome Application

Student Guide

Question 1.

Analyze the following three graphs and come up with hypotheses about where your microbiome originally came from:

Graph 1.

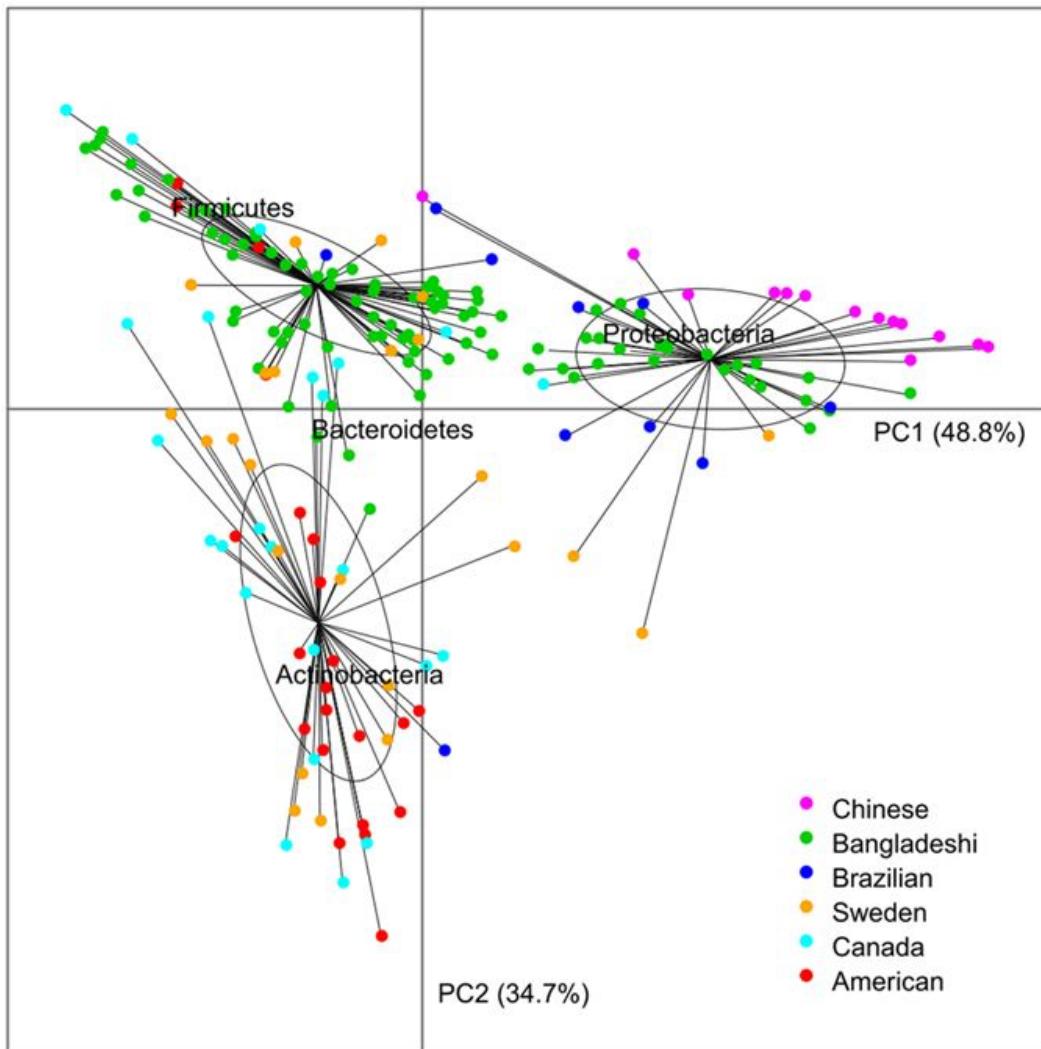


Comparison of the microbial community of vaginal and cesarean delivery subjects for neonates (a) and 2-month-old infants.

(b) Only the top 30 genera are shown for clarity. Genera with significant differences between neonates and 2-month-old infants are marked by asterisks: “*” denotes $P < 0.05$; “**” denotes $P < 0.01$; “***” denotes $P < 0.001$. Unclassified genera under a higher rank are marked by “†”.

Conclusions:

Graph 2.

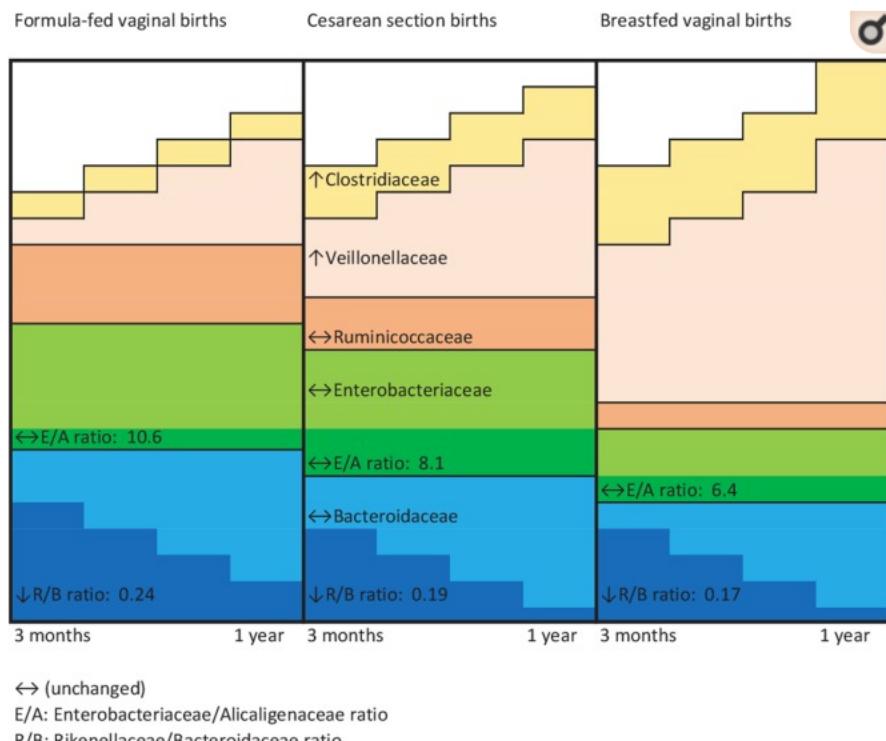


The first two principle components (PCs) and their ratio of variance contribution are shown. Nodes represent the individuals, lines connect individuals in the same group, and colored circles cover the individuals near the center of gravity for each group.

Conclusions:

Graph 3

Figure 2



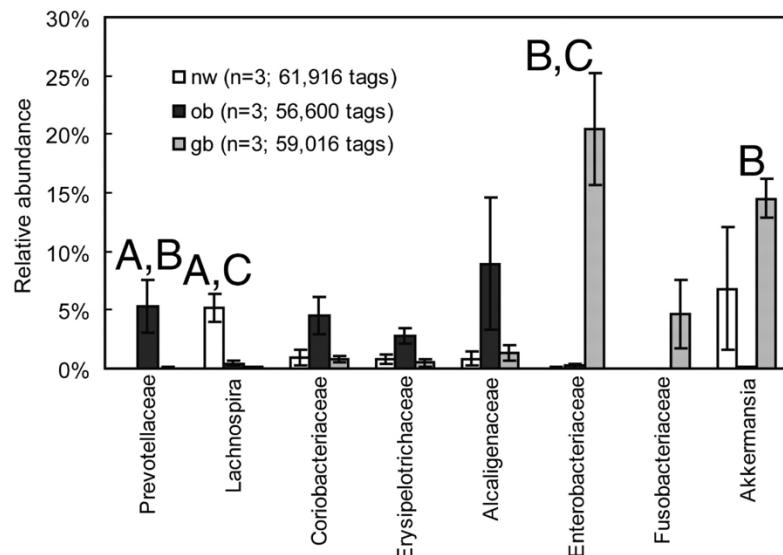
Schematic representation of microbial family changes by perinatal group. ↔, Unchanged; E/A, *Enterobacteriaceae/Alcaligenaceae* ratio; R/B, *Rikenellaceae/Bacteroidaceae* ratio.

Conclusions:

Summarizing all three graphs, from where does your microbiome originate?

Question 2

Can the microbiome affect your health?



The graph above shows results from the analysis of the gut microbes in 9 people: 3 normal weight, 3 obese, and 3 who have had gastric bypass surgery (a surgery in which they bypass a large portion of your stomach, designed to help you lose weight). What interpretations can you make about this data?

Question 3

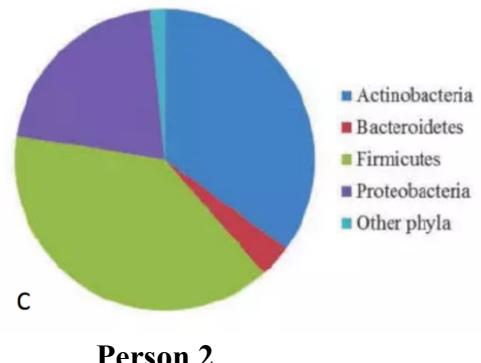
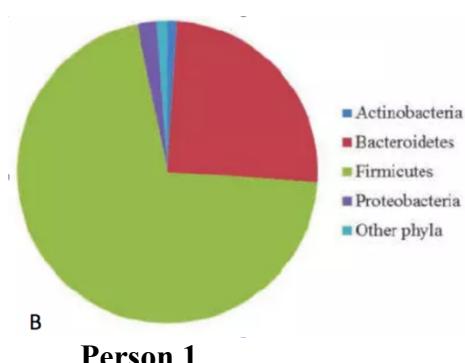
Following are two people's diets and their associated microbiome species in their guts. What conclusion can you draw about the relationship between your diet and your microbiome diversity?

Person #1 diet

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
Scrambled eggs, Sandwich, Caesar salad	Scrambled eggs, Sandwich Burger and fries	Scrambled eggs	Scrambled eggs	Scrambled eggs	Scrambled eggs	Scrambled eggs
		Pizza	Pizza	Burger and fries	Pizza	Popcorn, ice cream

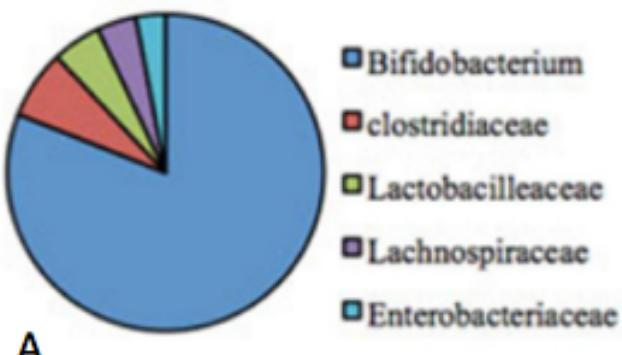
Person #2 diet

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
Oatmeal PBJ sandwich, Caesar salad	Yogurt and fruit, Sandwich (lettuce, meat, cheese, tomato) Chicken noodle soup	Apples and peanut butter Burger and fries, Broccoli stir-fry	Scrambled eggs and bacon, salad, Salmon And rice	Yogurt and fruit, Sandwich with lettuce, meat, tomato Bean Chili	Apples and peanut butter, Quesadilla with beans, Chinese stir-fry vegetables	Waffles, roast potatoes, and carrots, popcorn, ice cream

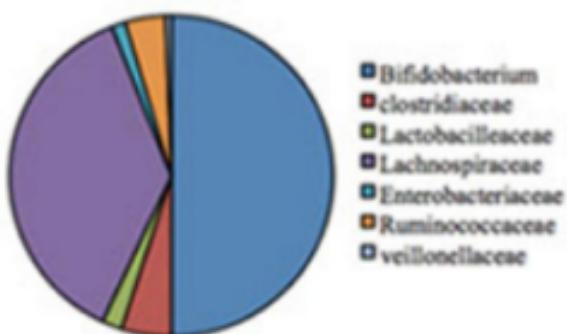


Question 4

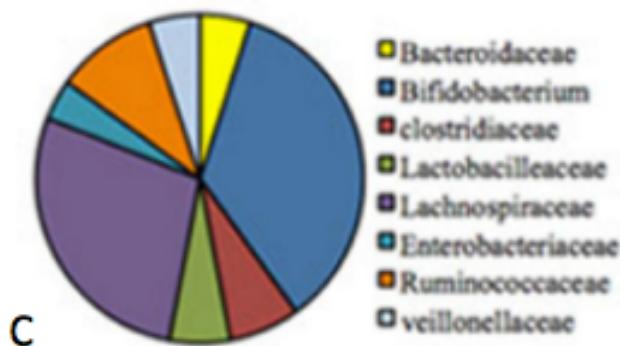
Pictured below are three gut microbiome profiles of three different children. Martha lives in the city with no pets. Jayesh lives in the poverty-stricken slums of Mumbai, India. Parker lives on a farm in Kansas. First, looking at the profiles, which profile would you predict to belong to each child? Second, from this, what overall conclusion can you draw about the environment and the development of your microbiome? Last, who do you think has the strongest immune system?



A



B



C

First, which profile belongs to each child?

Martha –

Jayesh –

Parker –

Second, what conclusions can you draw about the environment and development of the microbiome?

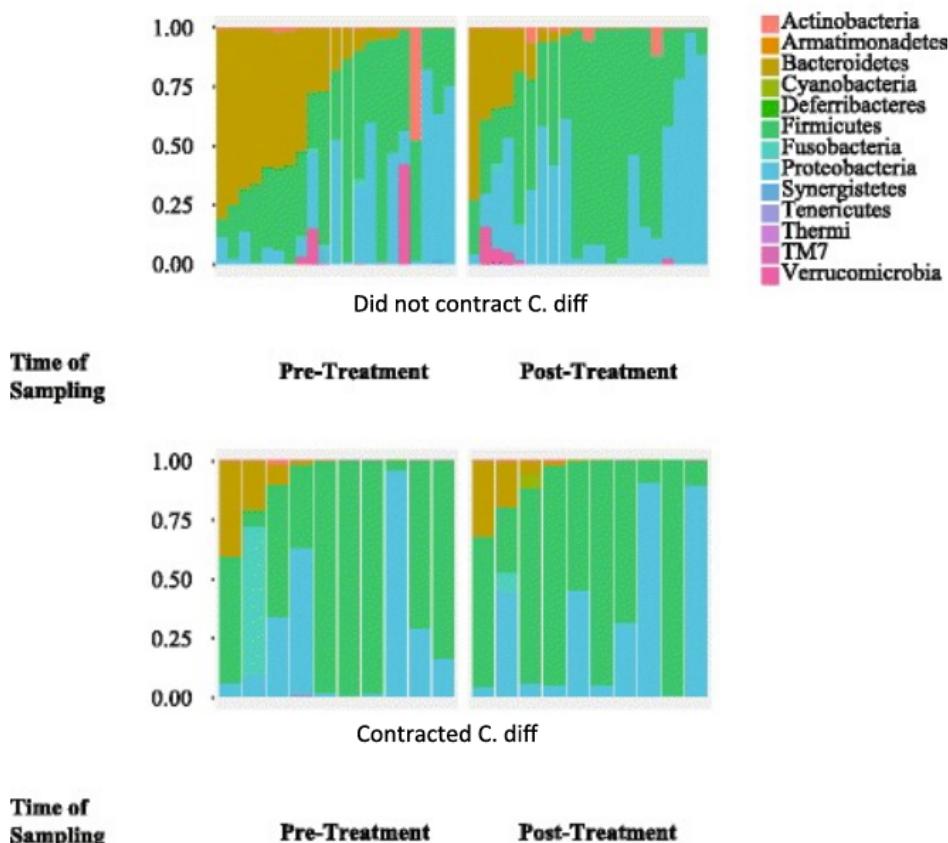
Third, who do you think has the strongest immune system and why?

Question 5:

Scientists were studying the incidence of *C. difficile* (a bacterial infection common in hospitals that causes severe, and often life-threatening diarrhea) in hospital patients receiving antibiotic treatment. They sampled the gut microbiome of two groups of patients who all received antibiotics treatment. One group contracted *C. difficile* while in the hospital, the other did not.

Looking at the graphs below, what can you conclude about the ability of *C. difficile* to outcompete the native population in the two different patient groups?

Fig. 4



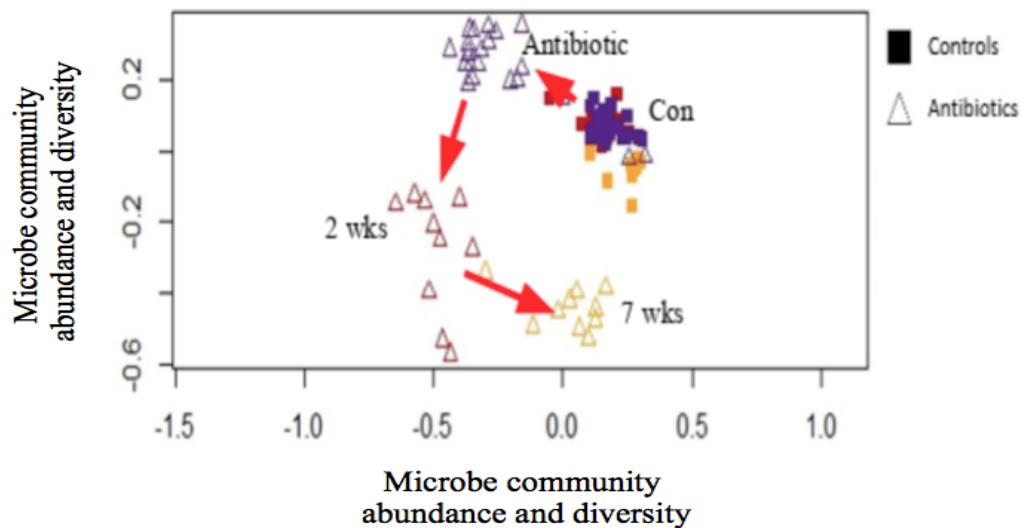
Relative abundance of bacterial phyla in patients who contracted *C. difficile* and patients who did not, within a hospital setting where antibiotics were administered.

What can you conclude about the effects of antibiotics on a patient's microbiome diversity?

Question 6:

The graph below is the result of a study looking at mouse gut microbes after administering antibiotics. The control (squares) shows the microbe communities in the gut never given antibiotics. The X and Y axes are a measure of deviations of the microbial community abundance (numbers) and diversity from the controls. The triangles indicate the transition of mouse gut microbes after being exposed to antibiotics at the time of exposure, 2 weeks after exposure, and 7 weeks after exposure.

From these results, what conclusions could you draw?



As part of this study, scientists then paired up control mice with each other (control-control), antibiotic mice with each other (antibiotic-antibiotic), and control mice with antibiotic mice (control-antibiotic) to see if living together could help them return to good health. Their results are shown below:

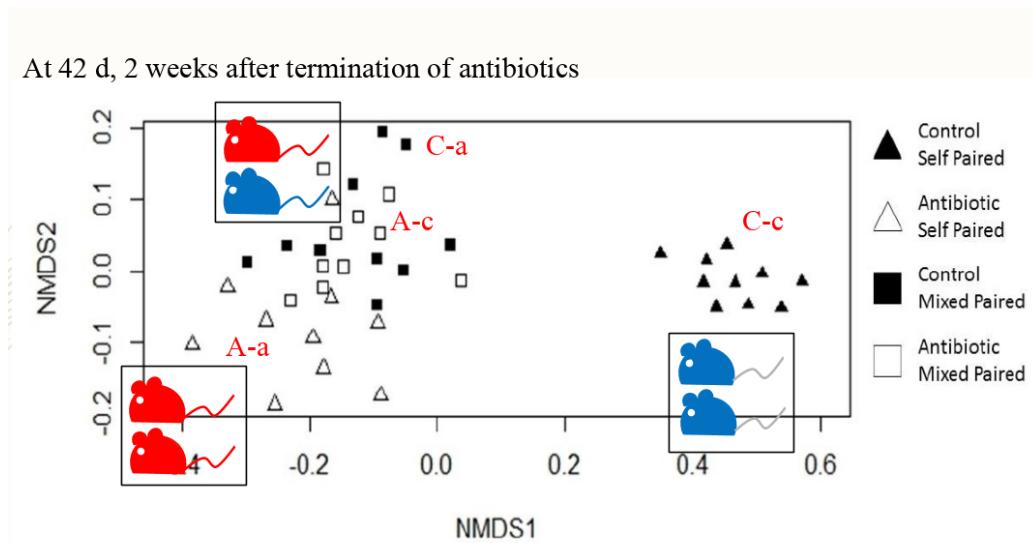
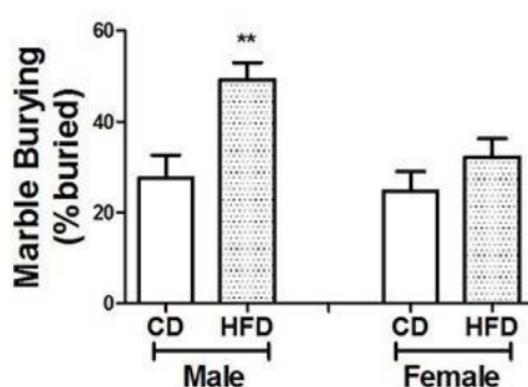
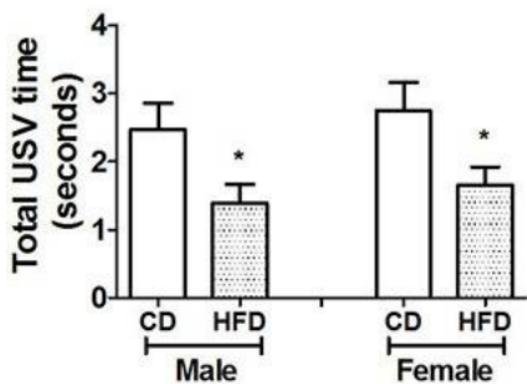


Figure 1.
Ordination of bacterial communities based on OTUs on Day 42 for mice given the cornstarch diet.

Question 7:

To understand how the microbiome affects mental health, scientists studied mice pups and measured anxious and obsessive-compulsive behavior. To measure anxiety, they measured the amount of time it took for the pups to start yelping when they were separated from their mothers (Total USV time). To measure obsessive-compulsive behavior, they looked at the number of marbles the pups buried (OCD in mice!). They compared pups from mothers who had a microbiome consistent with a high fat diet (HFD) to pups from mothers who had a microbiome consistent with a normal balanced chow diet (CD). The data are shown below. What can you conclude about the relationship between diet, microbiome, and mental health of offspring?



Nutrition
Student Guide

Nutrient	How much should you have in a day? (Give a food example that would equal this amount)	What happens if you consume <u>too much</u>?	What happens if you consume <u>too little</u>?
Water			
Carbohydrates			
Proteins			
Fats			
Vitamins			
Minerals			

Using the 4-4-9 method

Carbohydrates have, on average, 4 calories per gram.

Proteins have, on average, 4 calories per gram.

Fats have, on average, 9 calories per gram.

Use the label below to calculate what percentage of the calories in Doritos come from each type of macromolecule:

 <p>Doritos Cool Ranch FLAVORED TORTILLA CHIPS</p>	<p>Nutrition Facts Serving Size 1 oz (28g/About 12 chips)</p> <table border="1"><thead><tr><th colspan="2">Amount Per Serving</th></tr></thead><tbody><tr><td>Calories</td><td>150 Calories from Fat 70</td></tr><tr><td colspan="2">% Daily Value*</td></tr><tr><td>Total Fat</td><td>8g 12%</td></tr><tr><td>Saturated Fat</td><td>1g 5%</td></tr><tr><td>Trans Fat</td><td>0g</td></tr><tr><td>Cholesterol</td><td>0mg</td></tr><tr><td>Sodium</td><td>180mg</td></tr><tr><td>Total Carbohydrate</td><td>18g 6%</td></tr><tr><td>Dietary Fiber</td><td>2g 6%</td></tr><tr><td>Sugars less than</td><td>1g</td></tr><tr><td>Protein</td><td>2g</td></tr><tr><td>Vitamin A</td><td>0% Vitamin C 0%</td></tr><tr><td>Calcium</td><td>2% Iron 0%</td></tr><tr><td>Vitamin E</td><td>6% Thiamin 4%</td></tr><tr><td>Riboflavin</td><td>2% Vitamin B6 4%</td></tr><tr><td>Phosphorus</td><td>4% Magnesium 4%</td></tr></tbody></table> <p>* Percent Daily Values are based on a 2,000 calorie diet. Your daily values may be higher or lower depending on your calorie needs:</p> <table border="1"><thead><tr><th></th><th>Calories: 2,000</th><th>2,500</th></tr></thead><tbody><tr><td>Total Fat</td><td>Less than</td><td>65g</td></tr><tr><td>Sat Fat</td><td>Less than</td><td>20g</td></tr><tr><td>Cholesterol</td><td>Less than</td><td>300mg</td></tr><tr><td>Sodium</td><td>Less than</td><td>2,400mg</td></tr><tr><td>Total Carbohydrate</td><td></td><td>300g</td></tr><tr><td>Dietary Fiber</td><td></td><td>25g</td></tr></tbody></table> <p>Calories per gram: Fat 9 • Carbohydrate 4 • Protein 4</p>	Amount Per Serving		Calories	150 Calories from Fat 70	% Daily Value*		Total Fat	8g 12%	Saturated Fat	1g 5%	Trans Fat	0g	Cholesterol	0mg	Sodium	180mg	Total Carbohydrate	18g 6%	Dietary Fiber	2g 6%	Sugars less than	1g	Protein	2g	Vitamin A	0% Vitamin C 0%	Calcium	2% Iron 0%	Vitamin E	6% Thiamin 4%	Riboflavin	2% Vitamin B6 4%	Phosphorus	4% Magnesium 4%		Calories: 2,000	2,500	Total Fat	Less than	65g	Sat Fat	Less than	20g	Cholesterol	Less than	300mg	Sodium	Less than	2,400mg	Total Carbohydrate		300g	Dietary Fiber		25g
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DNA Structure

Before we jump into our application activity, compare your DNA drawing to a neighbor's. Draw your finished sketch here. With the help of your instructor, label your strands with 5' and 3'.

The Case of Diabetes Mellitus

Peter, a white male, aged 8, presents in the emergency room with abdominal pain and vomiting; he is also slipping in and out of consciousness. Your immediate impressions of the patient tell you that he is experiencing cognitive impairment and you can smell what seems to be acetone (fingernail polish remover) on his breath. His mother informs you that Peter was recently (within the last month) diagnosed with Type I Diabetes Mellitus and that it has been difficult to keep her rambunctious 8-year-old on his correct meds. You quickly do a finger stick and check Peter's blood only to find that his glucose levels are well above the physiological limit (at 260 mg/dL). Being the brilliant physician that you are, you diagnose Peter with Diabetic Ketoacidosis and immediately start him on IV administration of insulin and fluids. Within an hour, Peter is back to his normal self again—disaster averted! Way to go, you! The question is, what happened to Peter??? What is wrong inside his little body? That is what we are going to find out.

First, a little bit about the disease: Diabetes Mellitus, Type I, is referred to as Insulin-Dependent Diabetes, and most often is juvenile onset (diagnosed during childhood). It is an *autoimmune* disease caused by the destruction of pancreatic β -cells *by your own immune system*. Pancreatic β -cells are responsible for insulin production in your body; without them, you produce no insulin.

1. Based on what we discussed about disease and the immune system, briefly give an explanation as to *why* these β -cells are being destroyed:

The cause of diabetes mellitus is suspected to be a combination of genetic susceptibility and environmental triggers. Scientists have found that two particular mutations in a gene responsible for building the complex of proteins on the outside of the cells that identify them as 'self' (called *HLA class II DR3* and *DR4*) make people more susceptible to diabetes. They discovered this by sampling the general population and looking at the incidence of the *DR3* and *DR4* alleles among those with and without diabetes and found the incidence of the mutations to be much higher among diabetic patients.

2. This study would be considered
 - a. Correlational
 - b. Causational

3. In this study, what were the independent and dependent variables?

Independent: _____

Dependent: _____

Environmental triggers include viral infection, as the most likely trigger, and diet as the second most likely trigger. Regarding the viral causes, it is hypothesized that the introduction of particular viruses into the individual causes the immune system to attack the β -cells along with the virus.

4. If Peter's diabetes was indeed triggered by exposure to a particular virus, would administration of antibiotics during the infection have been effective? Why or why not?

5. If Peter had been vaccinated against that particular virus before getting infected with it, would that have prevented his diabetes? Why or why not?

β -cells are responsible for the production and export of insulin.

6. Which organelle is most likely responsible for *building* the insulin molecule?

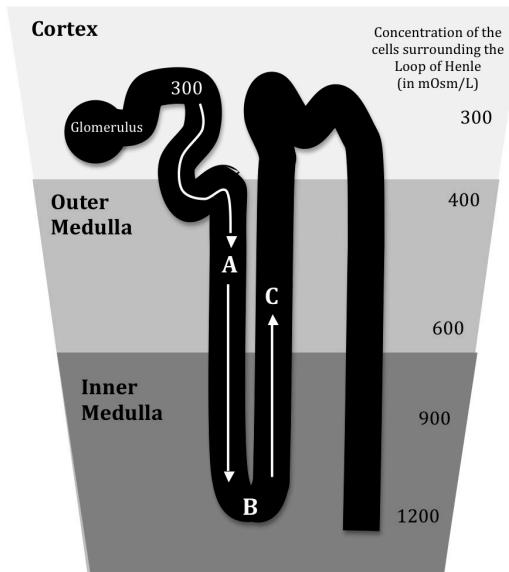
7. Insulin is a protein; therefore, it is made up of individual _____

Returning to our poor patient, Peter...Why is his apparent lack of β -cells so devastating to his body? It just so happens that glucose is too big of a molecule to pass through the cell membrane. Thus, it must pass through a particular protein embedded in the membrane, called GLUT4. When glucose is not needed by the cells, insulin is not actively produced by the β -cells and the GLUT4 protein is sequestered in vesicles that are brought back inside the cell. When glucose is needed, insulin is released from β -cells and causes the GLUT4 proteins to be transported back to the cell's surface so they can allow glucose to pass into the cells.

8. If it is first thing in the morning and you have just eaten breakfast after a long night of no food, what type of transport is the GLUT4 receptor performing?
- Passive diffusion
 - Facilitated diffusion
 - Active transport
 - Osmosis
9. If, in the case of Peter, you do not produce insulin, then the glucose in your bloodstream cannot enter the cells because the GLUT4 receptors are not at the surface. This causes the blood to become
- Hypertonic
 - Hypotonic
 - Isotonic

If you feed a person with diabetes mellitus, but do not administer an insulin injection, the person ends up in a hyperglycemic state (too much glucose in the blood stream)—this is most likely the cause of Peter's distress. All of this glucose in the blood is sent to the kidneys. The normal job of the kidneys is to reabsorb needed glucose from the blood and to reabsorb water. However, the kidney has a limit to how much it can reabsorb and that limit is 180 mg/dL. Thus, at 260 mg/dL, Peter has way more glucose than his kidneys can tolerate.

To the right is pictured the nephron of a human kidney. Inside of the kidney are many tiny loops of tubules, called "Loops of Henle" (pictured in black). They start in the cortex where diluted waste fluid (about 300mOsm/L) from the blood is deposited through filtration in the glomerulus. This diluted fluid then travels down from the outside of the kidney (the cortex) to the inside of the kidney (the medulla; from point A to point B) and then back up again (from point B to point C), before making a final trip back down and depositing the concentrated urine into a collecting area in the kidney. From point A to point B, the tubule only allows water to pass through the membrane. From point B to point C, the tubule only allows salt to pass through the membrane. Glucose can at NO point pass through the membrane of the tube.



10. In a *non-diabetic* person, the filtrate coming in has a concentration of 300 mOsm/L, which is _____ to the cortex.
 - Hypertonic
 - Hypotonic
 - Isotonic
11. Therefore, from point A to point B, water should be moving into/out of the tube.
12. However, in Peter, the filtrate is entering the loop of Henle at 1600 mOsm/L because of all of the unabsorbed glucose it is carrying. This means that from point A to point B, water will be moving into/out of the tube.
13. This means that a diabetic person, in a hyperglycemic state, will have
 - Really concentrated urine and not much of it
 - Really diluted urine and a ton of it

Thus, it appears that Peter was not given enough insulin for his body's glucose needs. However, treating diabetes is a tricky business, because you can also administer too much insulin!

14. What would the effects of *too much* insulin be?

15. Many adults with adult-onset diabetes (which is caused by a resistance to insulin, not a lack of it) treat it through a diet low in carbohydrates and high in proteins (commonly referred to as the “Atkins” diet). However, this diet is NOT recommended for children who are on insulin therapy. Why not?

16. Another dietary suggestion for children is to feed them a high-carbohydrate snack right before bed and especially after prolonged exercise. Why might this be necessary?

Lastly, it is suspected that diabetes is genetic. This means that you must have the gene to produce the ill-fated antibody that destroys your pancreatic β -cells.

17. Let's say that the beginning of the DNA sequence for this gene is:

ATGCCGAGACTAGATTCAAGGGTCTTATTACATAG

18. DNA is usually double-stranded. What would the sequence be of the other strand? (draw it above)

19. Now label the strands with 5' and 3'

20. If this codes for a protein antibody, how many codons are in this length of DNA? _____

21. How many amino acids long would this be if you used your genetic code to translate it?

22. If *E. coli*'s DNA is 26% A's, what percentage of the DNA is G's? _____

Applying Gene Expression

Now that you know how transcription, RNA processing, and translation works, we are going to do some practice. You will be using the same DNA molecule as we used in class, but we are now going to create the myosin protein on the other strand of DNA.

1. Transcribe the *Myosin* gene into an mRNA. Write the sequence of the full, *unprocessed*, mRNA below (Remember that DNA is read from 5' to 3'!):
 2. What enzyme performs the function of building an mRNA? _____
 3. When building an mRNA, which strand of the DNA is used by the enzyme you listed above in #2, Coding or Template? _____
 4. If the letters that are in bold are exons, write the mRNA resulting *after* RNA processing has occurred below:
 5. What two other components are added to the mRNA before it leaves the nucleus?
 - a.
 - b.
 6. Where is the only place in a cell where you would find *unprocessed* mRNA?

 7. What organelle is going to *translate* this mRNA? _____
 8. If a cell were missing tRNA, what would NOT occur (*please be specific*)?

 9. Using the genetic code, translate your mRNA **from #4** into the appropriate amino acids and write them below:
 10. Now write the sequence of tRNA **anti-codons** that appeared on the tRNA's that built the protein in #9:
 11. Let's look at what happens when mistakes are made. Let's use a hypothetical gene, *Levi-501*, as an example. The sequence for a *Levi-501* mRNA is as follows:

5'-AUGGGGCUCUGGGCGUUGCUCGUUUUCUACGUGA-3'

For each of the following questions, tell me if the mistake is a *missense mutation*, *nonsense mutation*, *frameshift mutation*, or *silent mutation*. **Start back with the original mRNA each time (do not do mutations on top of mutations).

12. Addition of an **A** between Bases #4 and #5: _____
13. Base #9 changed from **C** to **G**: _____
14. Base #12 changed from **G** to **A**: _____
15. Base #4 changed from **G** to **C**: _____

DNA.

Hemoglobin Gene →

5' - CTTGAGATTACATAGGATACCATCGTATTACGGTATGCTTGACCTATAAAGCCGTAATGCG**ATGCACTTTCA**TGCCGTA..
3' - GAACTCTAATGTATCCTATGGTAGCATAATGCCATACGAACGGATATTCGGCATTACGC**TACGTGAGAAGTACGGC**AT...

...**ATAGTTTGGCCTAGCGTGCAGTCGTATACCGTGTAG**TCGGACGCCAATAAATTGTTATTCCGCTGTAC**TTAACCCATCTCTA**..
...**TATCAAAACCGGATCGCACGTCAGCAT**TGGCACATCAGCCTGCGTTATTAACAAATAAGGCACATG**AATTGGTAGAGAT**..

← *Myosin Gene*

...ATTGATCTACTGTAGATTACCTATC-3'
...TAACTAGATGACATCTAATGGATAG-5'

Second mRNA base					
	U	C	A	G	
U	UUU Phe UUC UUA UUG	UCU UCC UCA UCG	UAU Tyr UAC Ser UAA Stop UAG Stop	UGU Cys UGC UGA Stop UGG Trp	U C A G
C	CUU CUC CUA Leu CUG	CCU CCC CCA CCG	CAU His CAC Pro CAA Gln CAG	CGU CGC CGA CGG	U C A G
A	AUU AUC Ile AUA AUG Met or start	ACU ACC ACA ACG	AAU Asn AAC Thr AAA Lys AAG	AGU Ser AGC AGA Arg AGG	U C A G
G	GUU GUC Val GUA GUG	GCU GCC GCA GCG	GAU Asp GAC Ala GAA Glu GAG	GGU GGC GGA Gly GGG	U C A G

The Genetic Code

Gene Expression Review

1. Explain the following observations:
 - a. The gene copies (mRNA) in the cytoplasm are far shorter than the DNA from which they were copied.
 - b. The number of subunits in the protein (amino acids) is *just less* than 1/3 the number of subunits in the mRNA (nucleotides).
2. We are going to create an exact copy of the SMN1 gene as it appears in this coding strand (like a photocopy; keep in mind, the recipe is only on one strand of the DNA). Remember, the cell uses the **complimentary strand** to make this copy through a process called **transcription**. This complimentary strand is referred to as the _____ strand.
3. What does the **Template** strand of Exon 7 (only) look like? Write it below:

4. What does the **mRNA** for Exon 7 look like? Write it below:

5. What **3 things** must occur before the unprocessed mRNA can leave the nucleus?
 - a.
 - b.
 - c.
6. Translate your Exon 7 mRNA into a polypeptide:

7. Compare your sequence to your neighbors'. Were there mistakes? What were they?

8. Review:
- a. What is the job of the ribosome?

 - b. What is the job of the tRNA?

 - c. How does the tRNA recognize the codon and bring the correct amino acid?

 - d. How does the ribosome know when to stop translating?

Comparing Sequences

9. What difference(s) do you see between SMN1 and SMN2?

10. What kind of mutation is it?

11. How might this affect one's phenotype?

Forensic Mystery

Student Guide

Human Identification via STR

The DNA in every one of your cells is 99.9% identical to every other human on earth. The remaining tenth of a percent of the human genome contains many non-coding introns. Scientists use some of these regions, called STRs or “short tandem repeats,” as aggregate markers to differentiate among individuals. STR sites are repeated units of two to six nucleotides (e.g., GATA), which occur near chromosomal centromeres (see Box 1). The length of these STRs, or number of copies of the repeated units, is heritable. Since humans are diploid, we have two alleles of each STR, one donated to us by each of our parents.

Box 1. The four-nucleotide repeat of STR site D7S280 is “GATA”. Different alleles of this locus have from 6 to 15 tandem repeats of the “GATA” sequence. Below is an example of the “15” allele, corresponding to the 15 tandem repeats (shown in bold).

```
1 aattttgtattttttag agacggggtt tcaccatgtt ggtcaggctg actatggagt  
61 tatttaagg ttaatatata taaagggtat gatagaacac ttgtcatagt ttagaacgaa  
121 ctaacgatag atagatagatagatagatagatagatagatagatagatagata  
181 agatatgttt ttttatctc actaaatagt ctatgtaaa catttaatta ccaatatttg  
241 gtgcaattct gtcaatgagg ataaaatgtgg aatcggtata attcttaaga atatatattc  
301 cctctgagtt ttgataacct cagatttaa ggcc
```

In the 1990s, the FBI selected 13 core STR loci for inclusion in a nationwide database for forensic purposes. These 13 loci are internationally recognized as the standard in human identification. Although STR alleles are not unique to every individual (clearly, as you share STRs with your close relatives), the combination of all 13 ensures the likelihood of any two individuals sharing the same 13-loci STR profile is over one in 500 trillion.

How it works...

- 1) DNA is isolated from any human cell (e.g., blood, saliva, hair, skin).
- 2) Specific regions of DNA, STR sites, are amplified using Polymerase Chain Reaction (successive heating and cooling of DNA doubles the quantity of original DNA). This step allows trace amounts of material to be effectively used for forensic purposes.
- 3) DNA is separated and detected using electrophoresis and fluorescence.
- 4) The number of tandem repeats is counted, and alleles are determined using known genotypes of each STR site.
- 5) The resulting STR profile for an individual shows the number of repeats (the allele) at each STR loci.

		Parent 1	
		16	18
Parent 2	15	15, 16	15, 18
	18	16, 18	18, 18
D3S1358			

Once the costly, lab-intensive work is complete (see steps above), you have a STR profile. STR alleles follow classic Mendelian inheritance, of which you are now an expert (see example Punnett square to the right). STRs are commonly used for paternity investigations, missing person cases, and crime scenes. Today we have a case to help solve.

Crime Scene Investigation

1. How many crime scenes do we have?
2. What biological evidence should we be looking for?
3. What other evidence should we collect?
4. Where are we going to find DNA evidence that will help find her killer?

Determining Guilt

5. Based on STR analysis, who did you decide was the killer?
6. Let's determine the probability that we got it wrong. To do this, we are going to compare the perpetrator's STR profile to known frequencies of these alleles in the population. On the next page is a table of known frequencies of the STR regions in our printout among Caucasians:

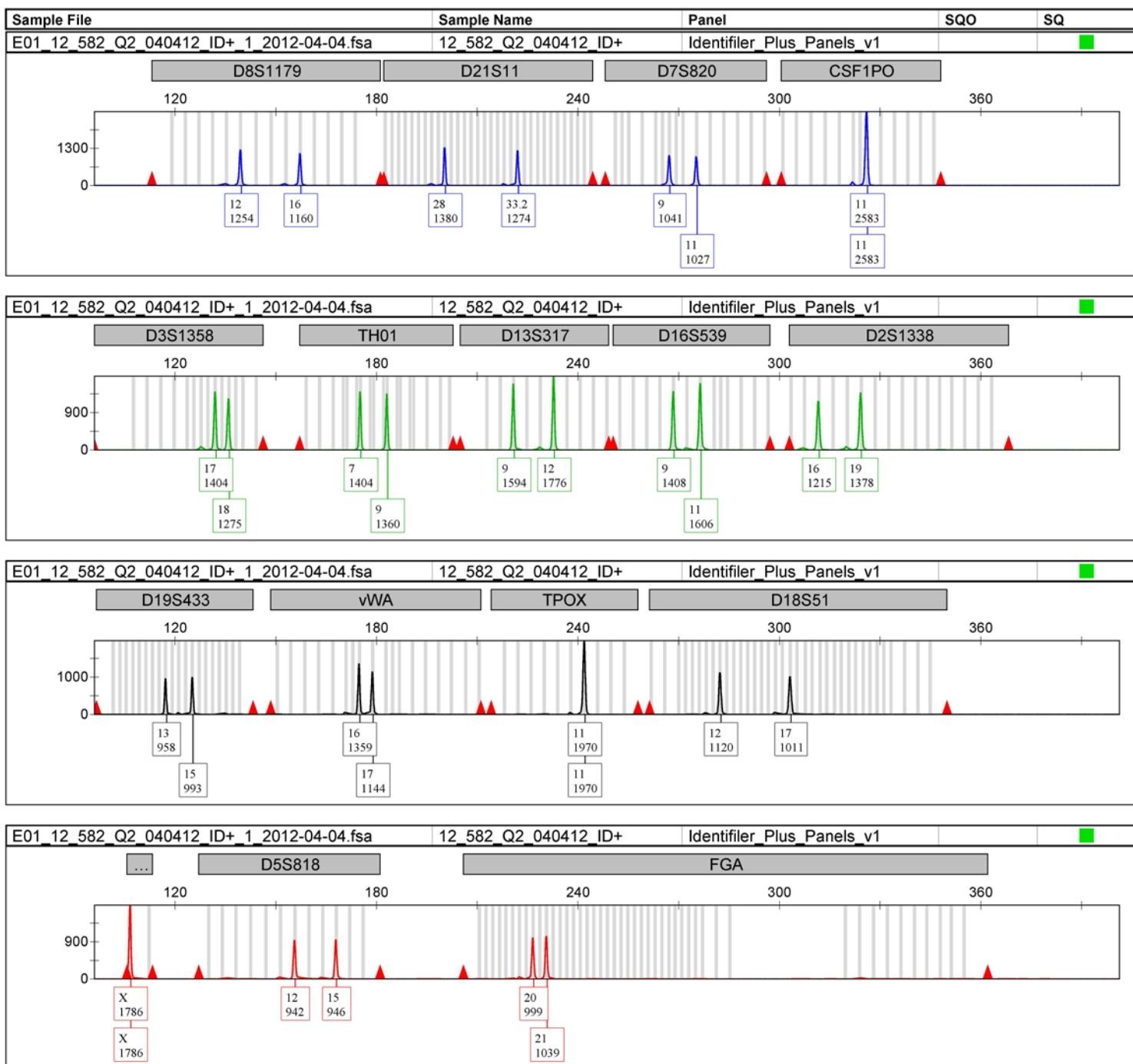
Results for Population: Cauc (Sample Size = 361)																
Allele	CSF1PO	D13S317	D16S539	D18S51	D19S433	D21S11	D2S1338	D3S1358	D5S818	D7S820	D8S1179	FGA	TH01	TPOX	vWA	
5														0.00	0.00	
6														0.24	0.00	
7									0.00	0.03				0.19		
8	0.01	0.12	0.02						0.01	0.14	0.01			0.10	0.52	
9	0.01	0.08	0.11						0.04	0.17	0.01			0.12	0.13	
10	0.22	0.05	0.06	0.01	0.00				0.06	0.26	0.10			0.01	0.05	
11	0.31	0.33	0.31	0.01	0.01			0.00	0.36	0.20	0.08			0.00	0.25	
12	0.36	0.27	0.31	0.11	0.07				0.39	0.16	0.17			0.04	0.00	
13	0.08	0.12	0.16	0.12	0.25			0.00	0.14	0.03	0.33			0.00	0.00	
14	0.01	0.04	0.03	0.13	0.36			0.11	0.01	0.00	0.17				0.09	
15		0.00		0.17	0.16		0.00	0.27	0.00		0.10					0.11
16				0.15	0.06		0.04	0.24			0.03					0.20
17				0.14	0.01		0.19	0.21			0.00					0.28
18				0.08			0.07	0.15				0.02				0.20
19				0.04			0.12	0.02				0.05				0.10
20				0.02			0.16	0.00				0.12				0.01
21				0.01			0.04					0.18				0.00
22				0.01			0.03					0.20				
23							0.11					0.15				
24							0.11					0.13				
25							0.10					0.08				
26							0.03					0.03				
27					0.02							0.00				
28					0.16											
29					0.20											
30					0.28											
31					0.07											
32					0.01											

In the following table, determine the overall probability that another random stranger has the same STR profile as your perpetrator:

			Overall Probability (Multiply)	

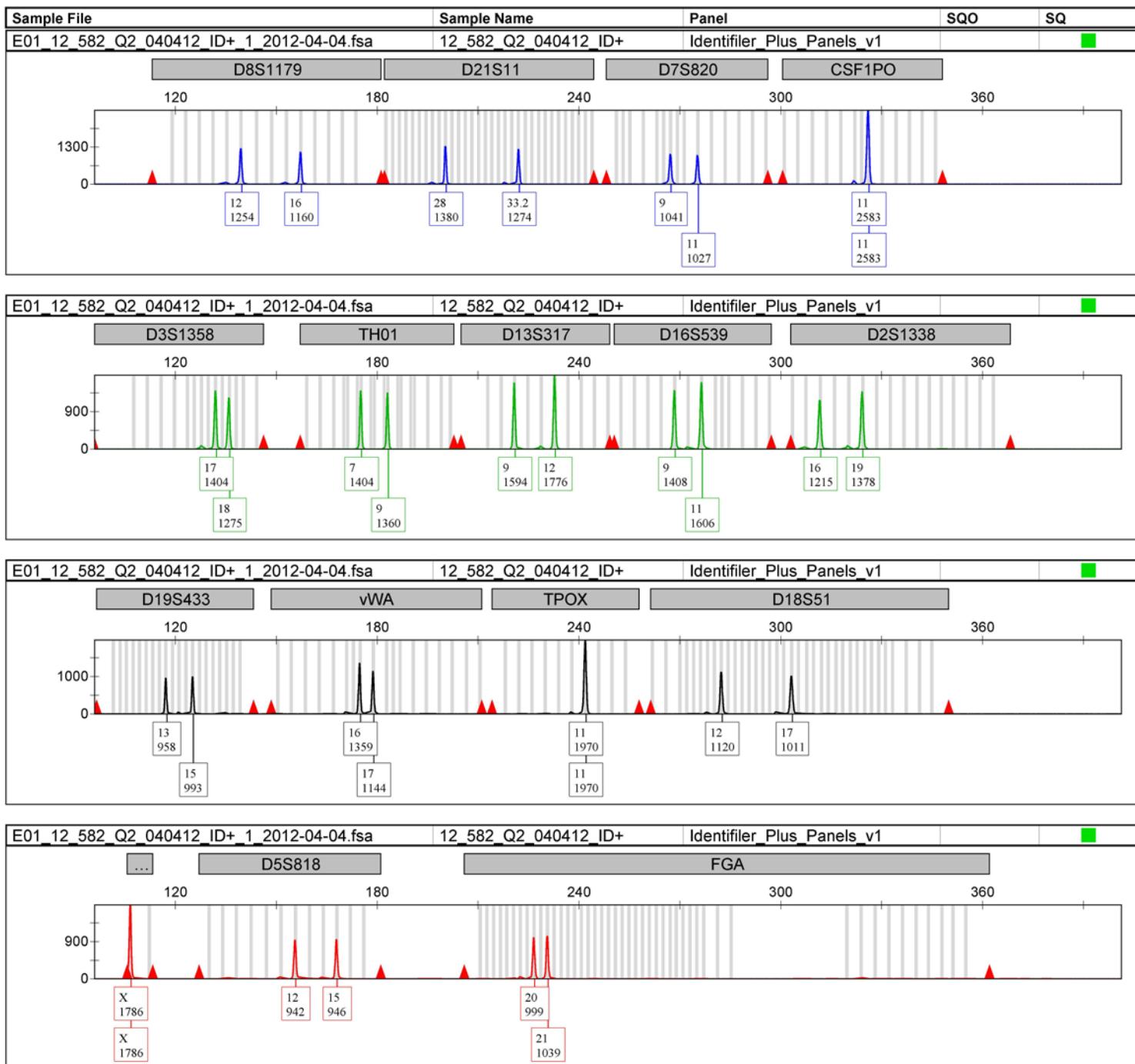
Blood from the house

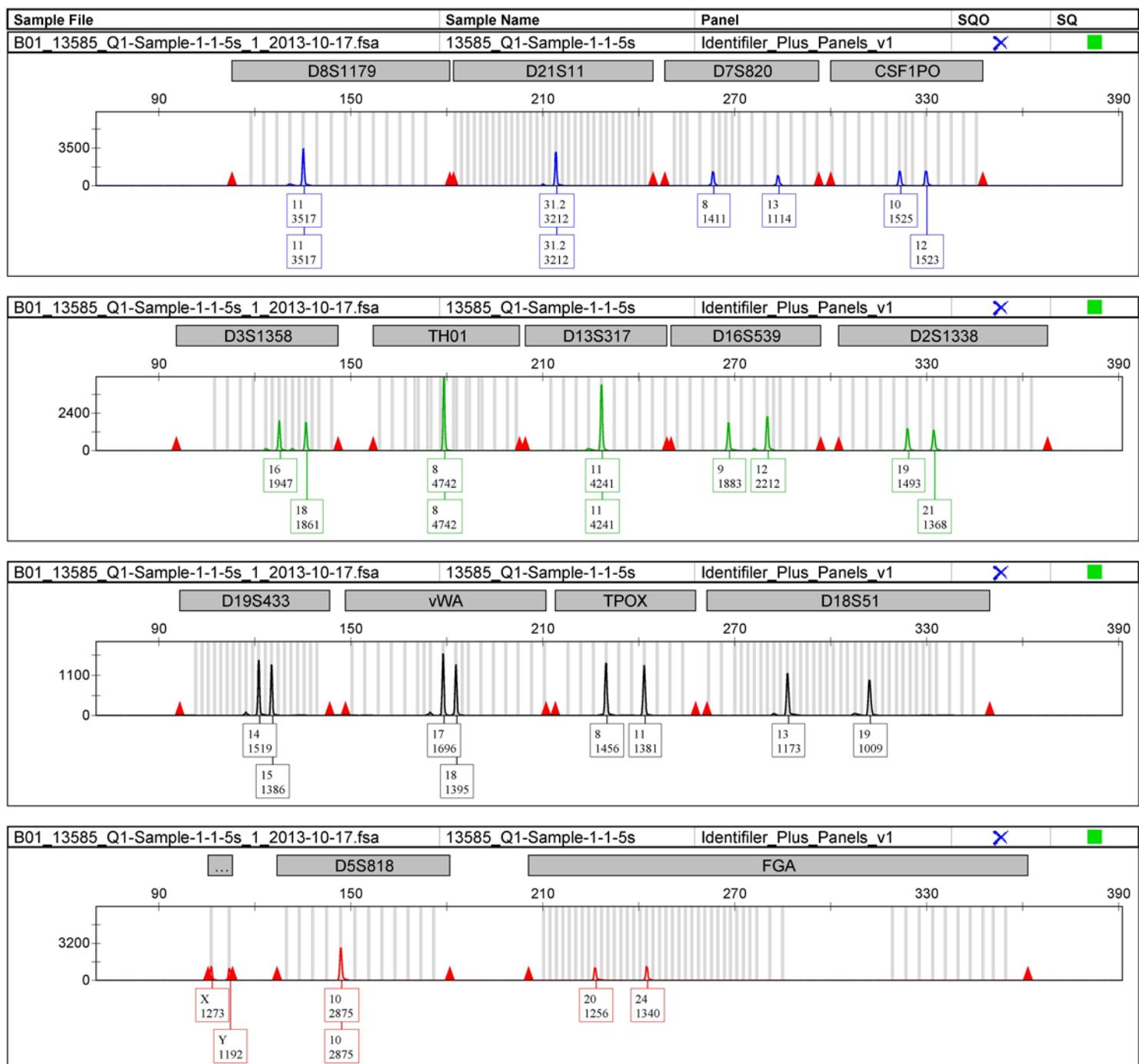
GM_12582_RB

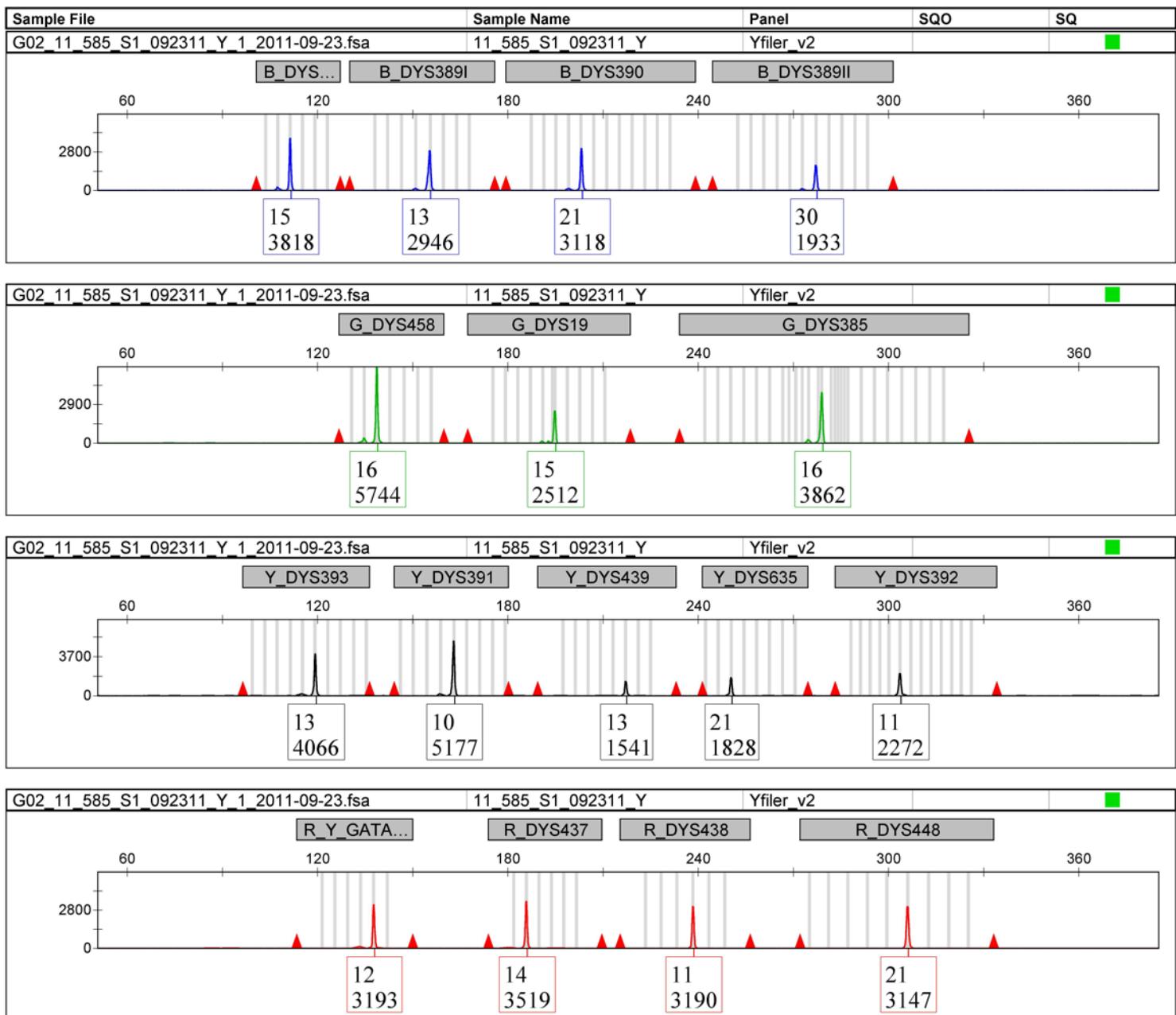


Blood from the vehicle

GM_12582_RB



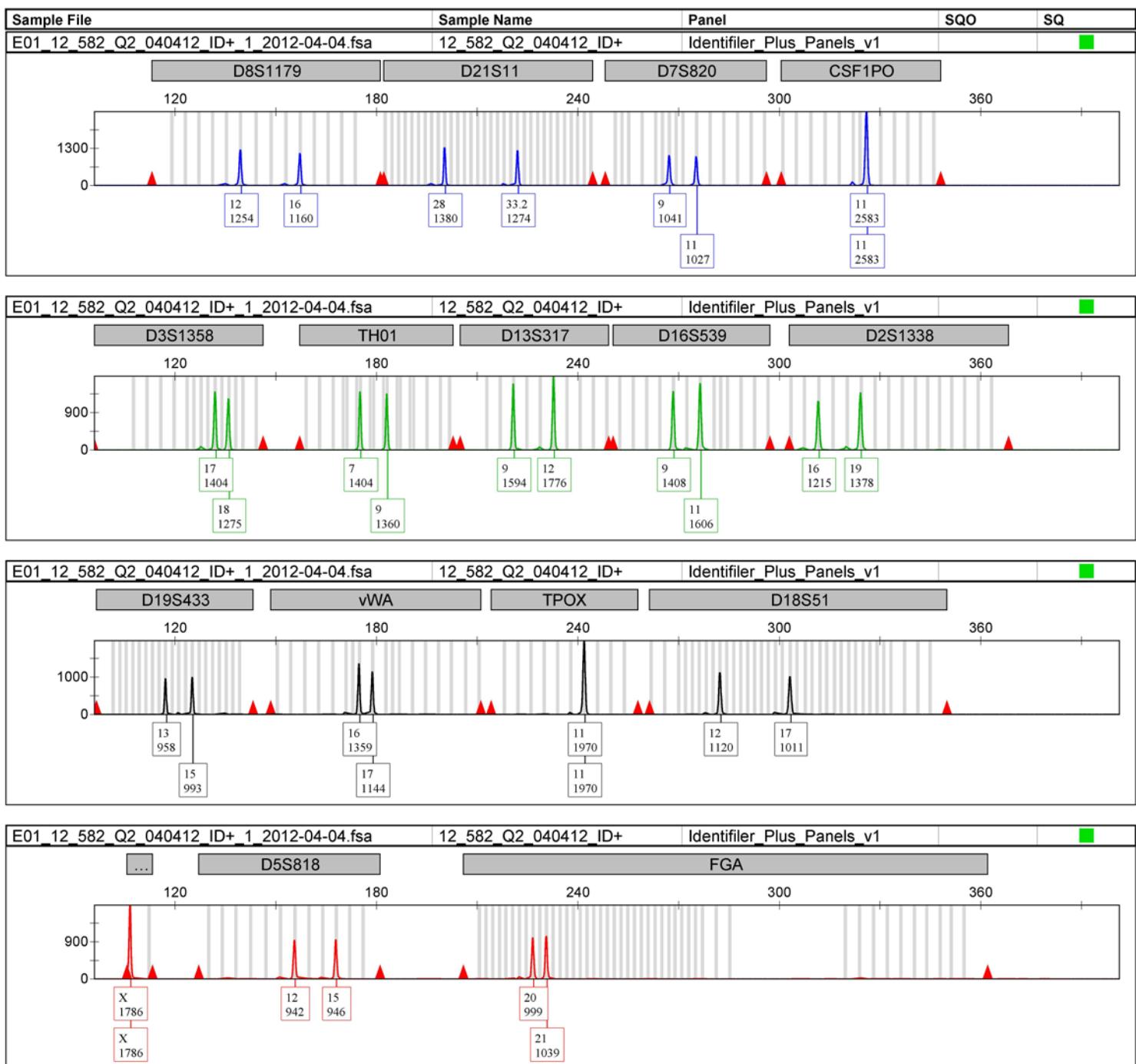




DNA from under the fingernails of the victim

GM_12582_RB

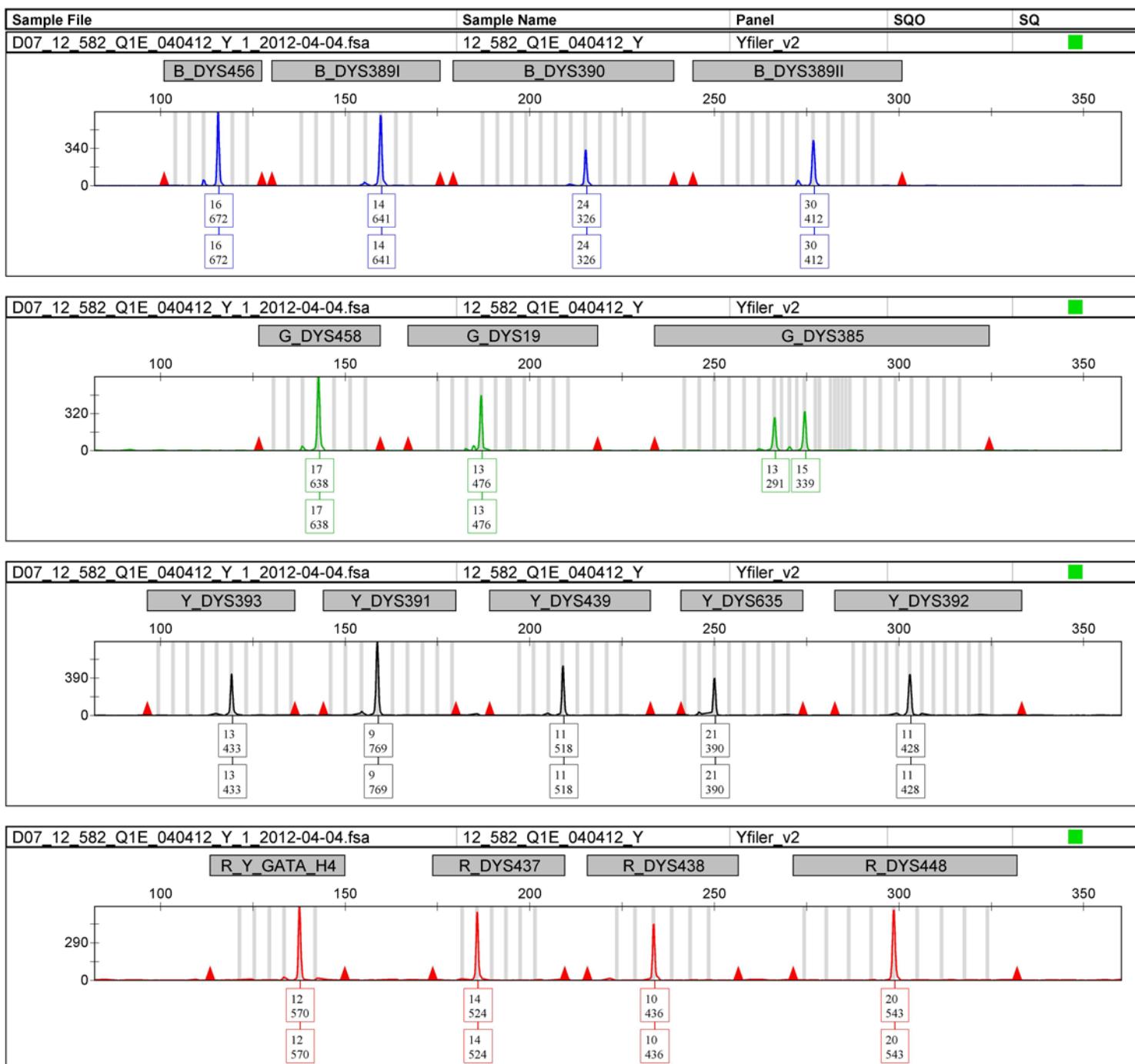
GeneMapper ID v3.2.1



Y-chromosome analysis from under the victim's fingernails

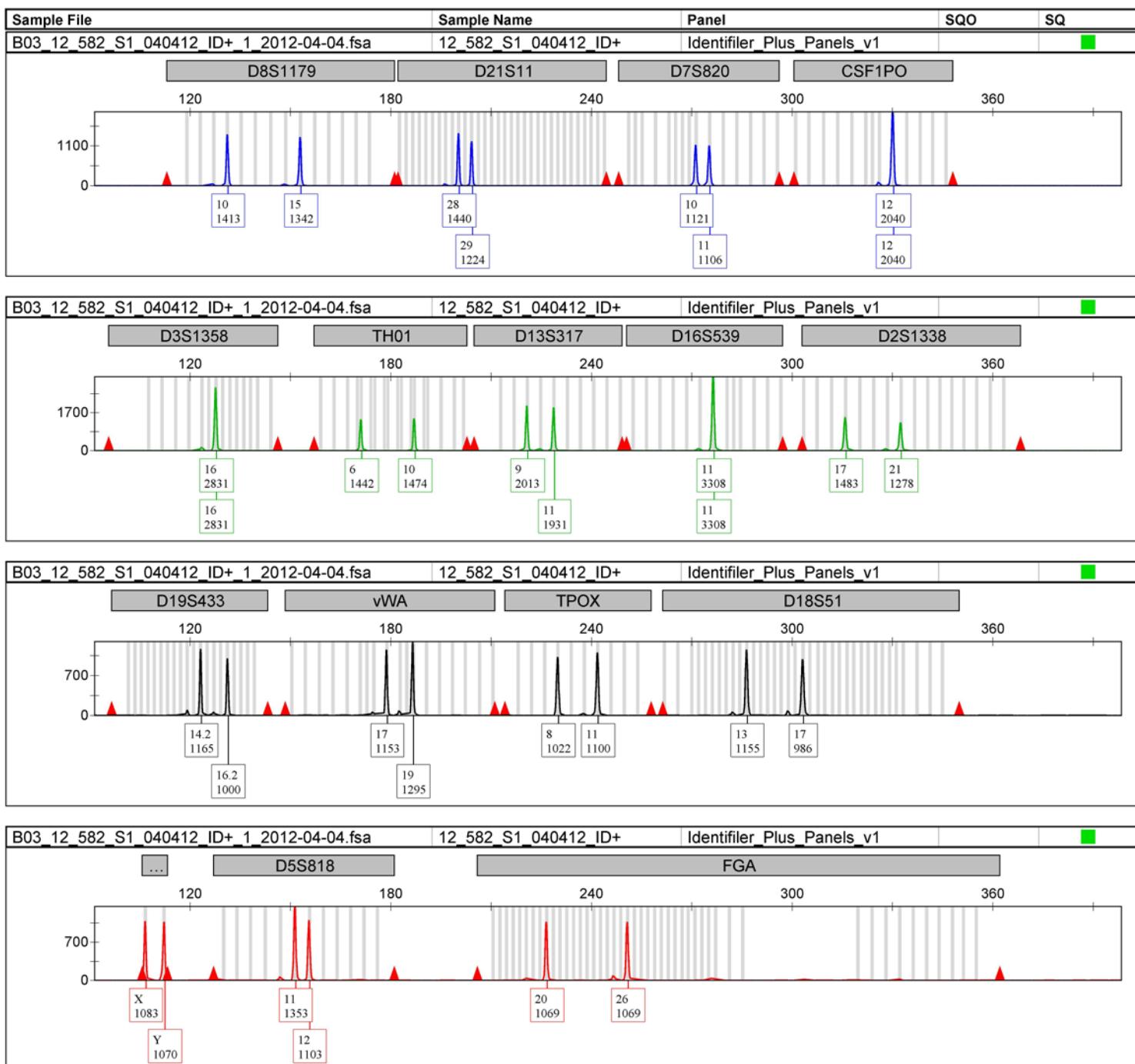
GM_12582_RB

GeneMapper ID v3.2.1



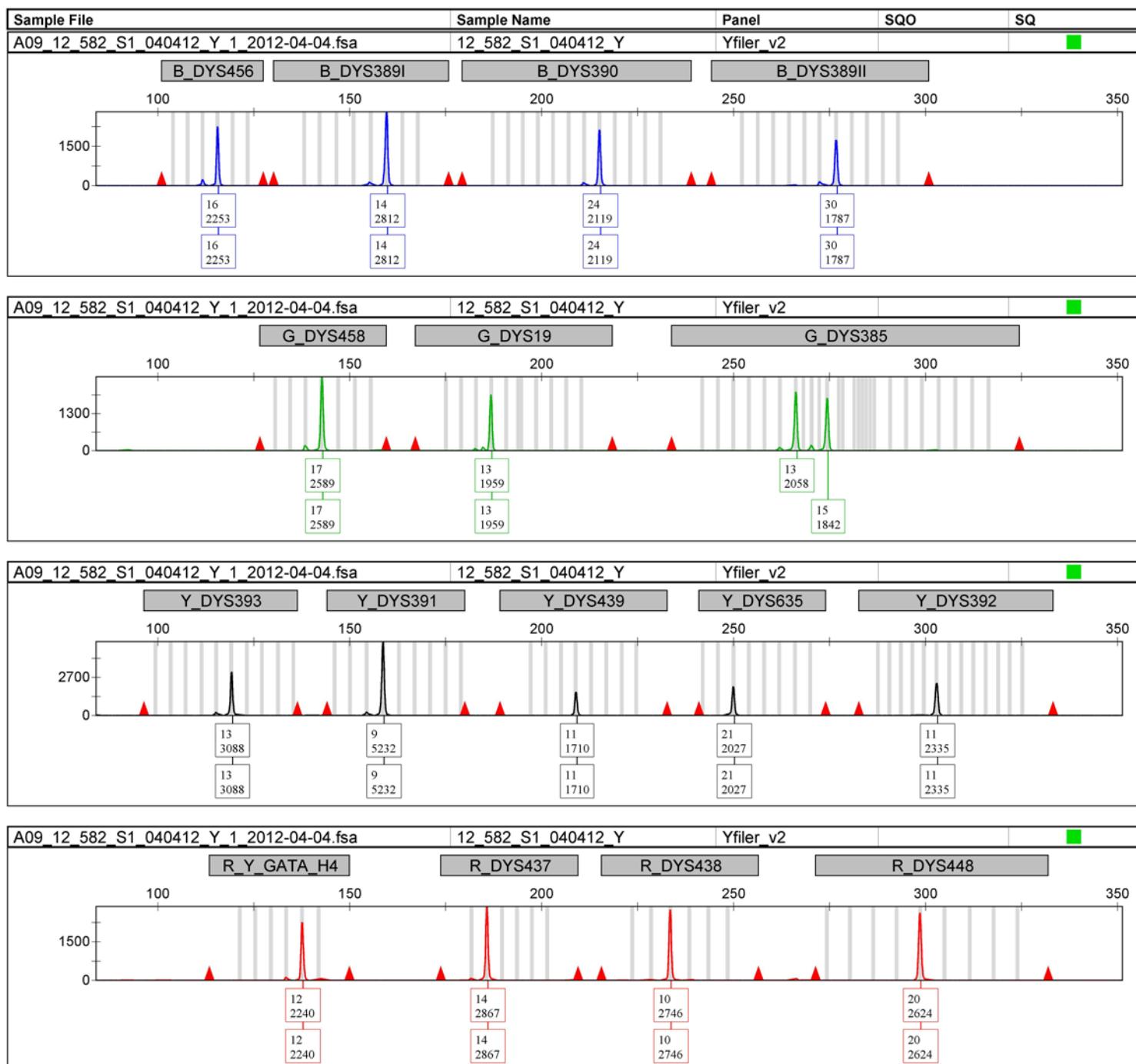
Roommate Standard

GM_12582_RB



Roommate Y chromosome

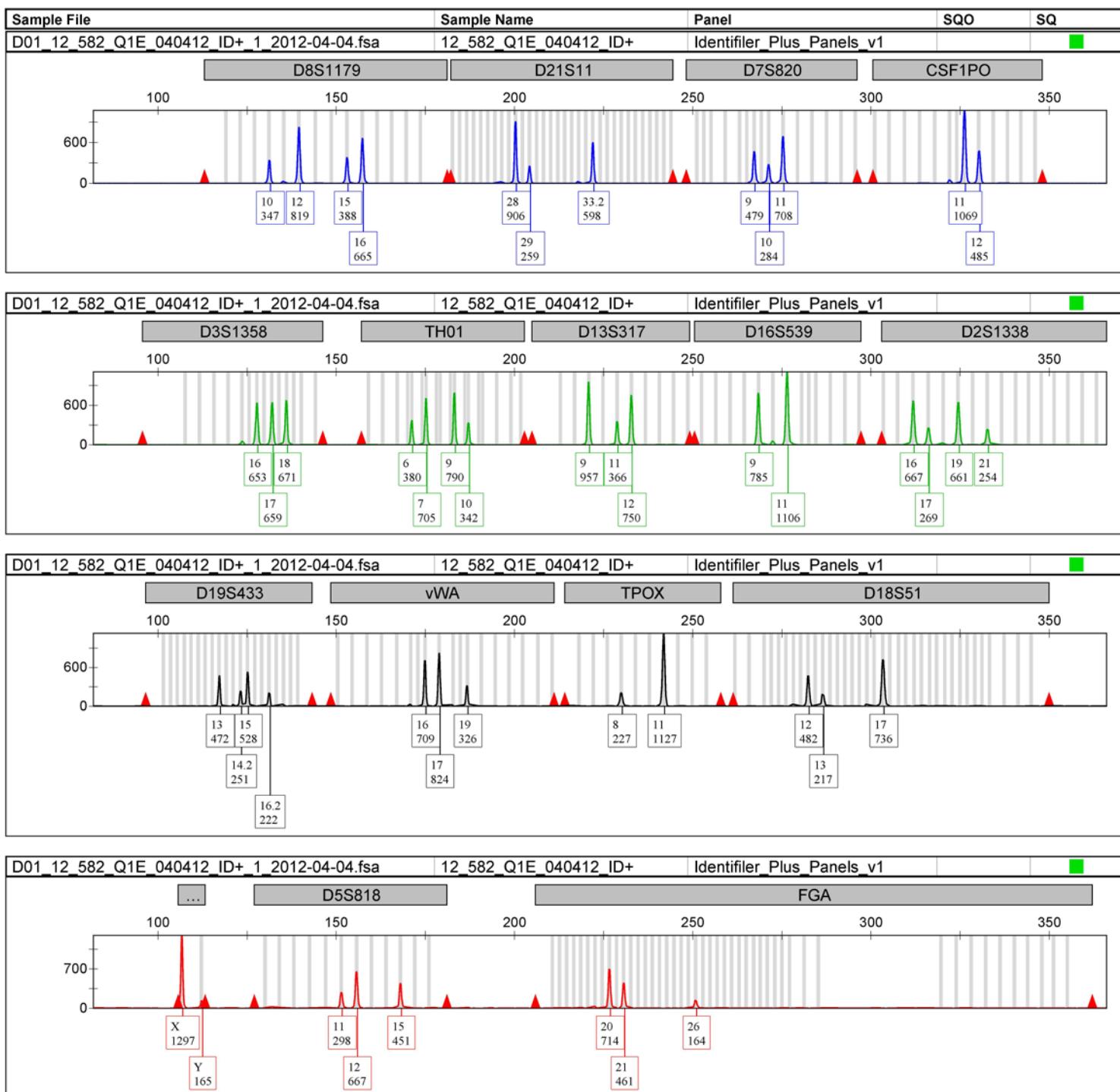
GM_12582_RB



Semen Stain

GM_12582_RB

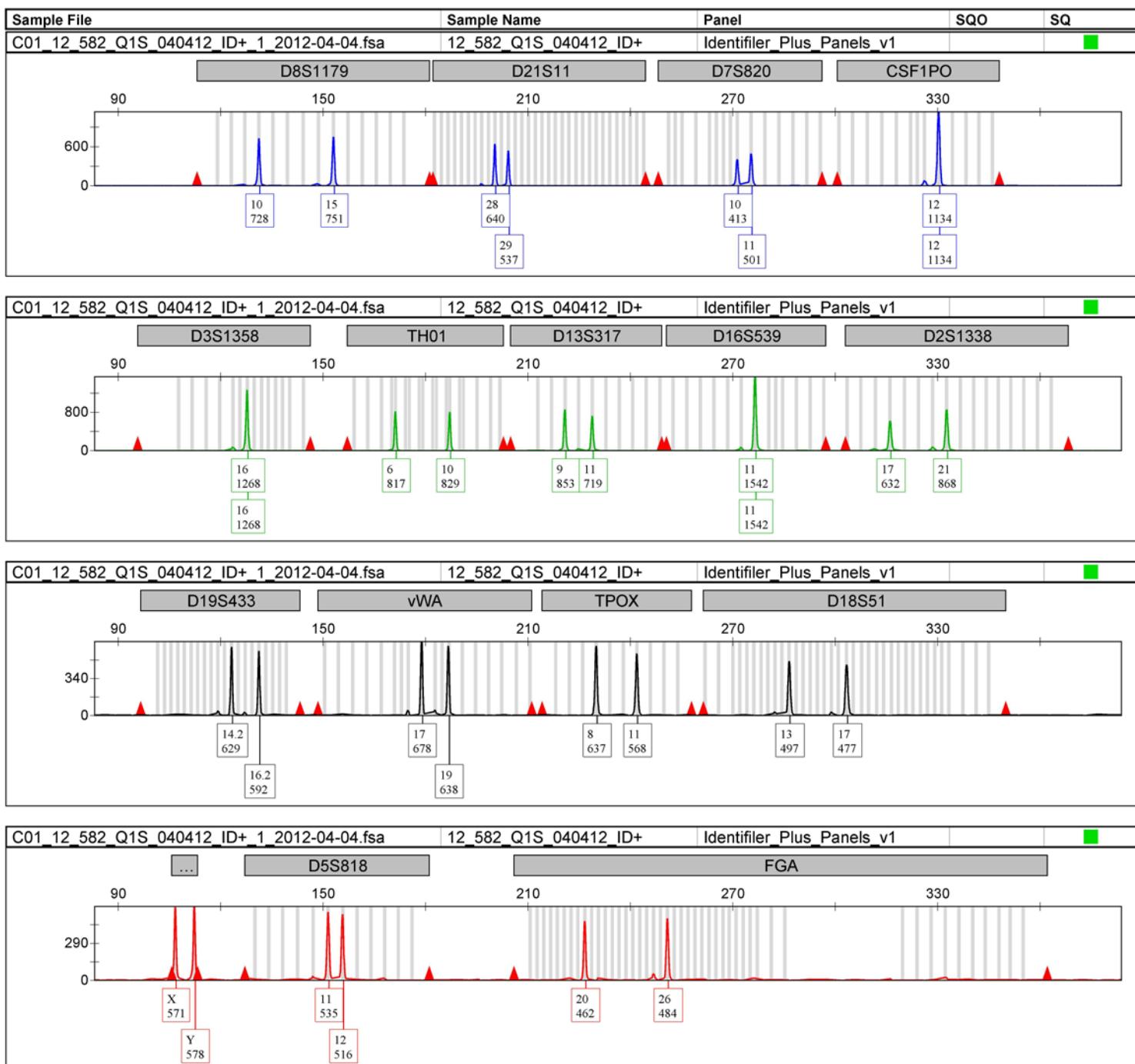
GeneMapper ID v3.2.1



Swab from steering wheel

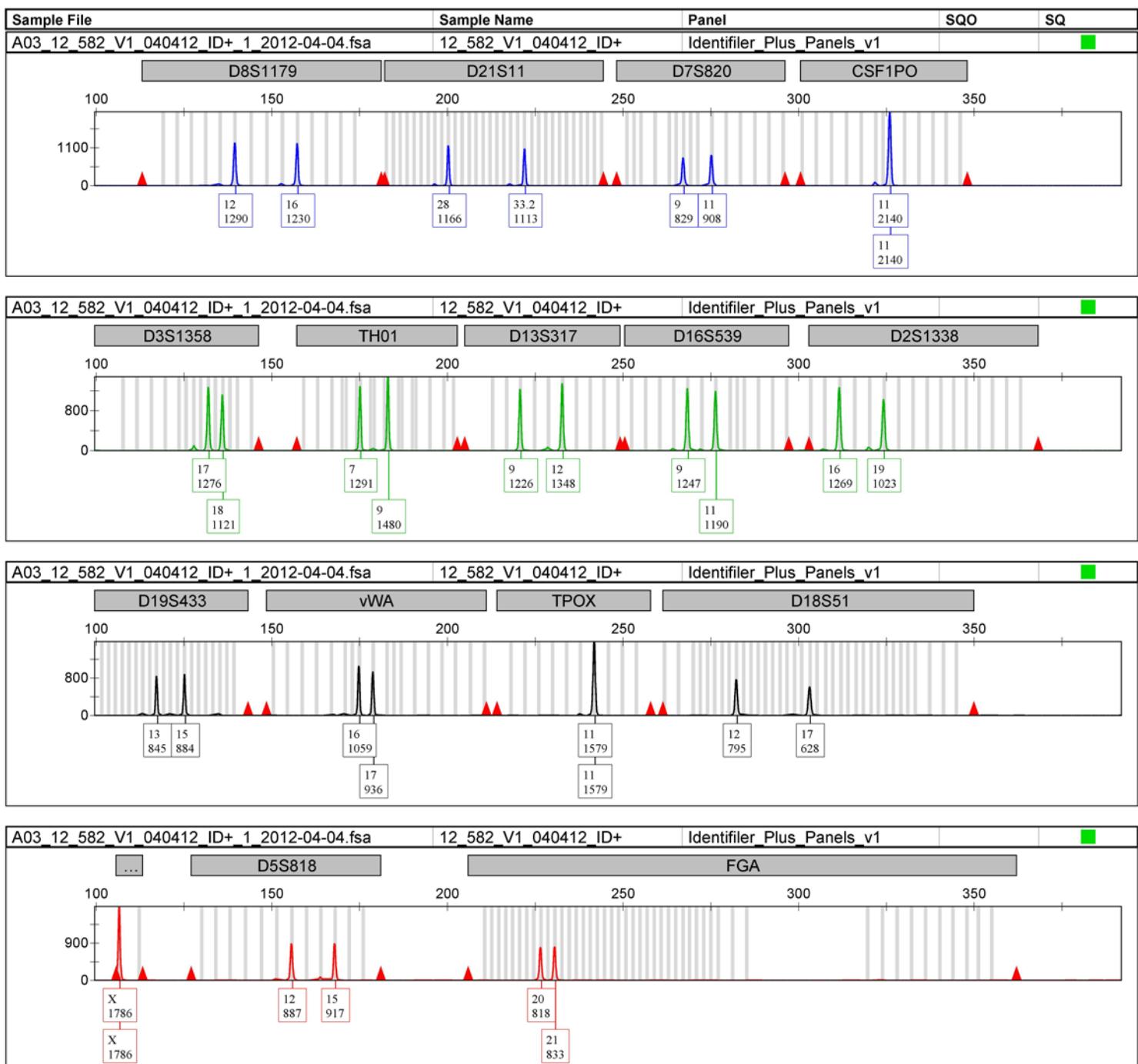
GM_12582_RB

GeneMapper ID v3.2.1



Victim Standard

GM_12582_RB



Biotechnology Application

Student Guide

Problem #1: Cholera Calamity

According to the World Health Organization (WHO), Cholera is an acute diarrheal infection caused by ingestion of food or water contaminated with the bacterium *Vibrio cholerae*. Researchers have estimated that every year, there are roughly 1.4 to 4.3 million cases, and 28, 000 to 142, 000 deaths per year worldwide¹ due to cholera. The short incubation period of 2 hours to 5 days, is one factor that triggers the potentially explosive pattern of outbreaks.



Sub-Saharan Africa has the largest cholera outbreaks. Cholera can be prevented by broad-scale vaccination, however, that is expensive and hard to orchestrate. Scientists would like to find a way to easily administer vaccines to children in Africa. One of the ideas they have come up with is to make a banana that can deliver the vaccine.

Read the attached article “Yes, we have vaccinating bananas”:

Questions:

1. Remember back to how vaccines work, and keep in mind the techniques we have been learning, how do you make a banana produce the benefits of a vaccine?

2. What are the potential downsides to creating a modified banana plant? If you don't know, feel free to Google it!

Problem #2: Reinventing Rice

In large parts of Asia and Africa, rice is a staple diet of the poor. Children are weaned from their mother's milk on rice gruel. Rice lacks beta-carotene, a precursor to vitamin A. The World Health Organization (WHO) estimates that 250,000 – 500,000 children go blind each year due to vitamin A deficiency; half of them will die by their fifth birthday. 668,000 children under the age of 5 die each year from this deficiency.



Read the attached article “Golden Rice is safe to eat, says FDA”

Questions:

1. Using what you have learned about biotechnology, how might you make a rice plant that contains beta-carotene?
 2. What are the potential dangers of producing modified rice plants? If you don't know, Google it!

Problem #3: Defending against Drought

Corn is a staple in much of Africa. However, corn is susceptible to the frequent droughts that occur in the region. In 2008, drought killed thousands of people in Ethiopia. In 2010 – 2012, drought in Somalia killed and estimated 260,000 people who died of starvation.



Read the attached article “Cross-bred crops get fit faster”

Questions:

1. Using what you have learned about biotechnology, how do we get corn to be more drought resistant? Scientists have found several genes in *Arabidopsis* (rock cress) that allow it to be more heat tolerant:
 - Cold stress proteins, CspA and CspB, allow a plant to resist water loss in times of drought
 - HARDY increases photosynthesis rates with lower water
 - DRO1 causes their roots to grow deeper to access water.
2. What are potential drawbacks for creating drought-resistant plants? If you don't know, Google it!

Problem #4: Capturing Carbon Dioxide

Global climate change is causing a variety of consequences:



- Rising ocean temperatures lead to coral bleaching
- Polar ice caps are melting destroying the habitat of polar bears
- Human disease are spreading into areas where they have never previously been



- Droughts are killing millions of people
- And more...

This is largely caused by an excess of CO₂ in the air coming from the burning of fossil fuels. Plants take in CO₂ during photosynthesis and turn it into sugars that they use to build their own bodies. The enzyme they use to do this is called Rubisco. The Rubisco in photosynthetic cyanobacteria is more efficient at sequestering CO₂ from the air and incorporating it into sugars than plant Rubisco.

Read the attached article “Increasing plant enzyme efficiency may hold key to global warming”

Questions:

1. Using your knowledge of biotechnology, how can we make plants more efficient and thus use them to remove excess CO₂ from the air?
2. What are the potential drawbacks or dangers of creating plants that increase CO₂ use?

Yes, we have vaccinating bananas

20 September 1996

By **Vincent Kiernan**

Washington DC

BANANAS, genetically engineered to carry vaccines, could provide developing countries with a cheap way to protect children from life-threatening diseases, according to researchers in the US.

Biotechnologists at the Boyce Thompson Institute for Plant Research, an independent research centre at Cornell University in New York State, are genetically engineering a banana to produce an antigen found in the outer coat of the hepatitis B virus. Banana vaccines would be ideal for developing countries because they would cost just a few cents per dose, compared to the \$100 to \$200 per dose for traditional vaccines, says Charles Arntzen, president of the institute.

Last year, the team showed that hepatitis B antigens produced by genetically engineered potatoes triggered an immune response in rats. But because potatoes are not eaten raw, and cooking them would destroy the vaccine, they are unsuitable for vaccinating people. So the researchers switched their efforts to bananas, which are already grown extensively throughout the developing world.

Arntzen says the genetically engineered bananas could be grown on special plantations. For example, just 10 hectares of the fruit would be enough to vaccinate all children under five years old in Mexico. The vaccine would be delivered not as raw bananas, but in a purée similar to baby food. "We would probably put 10 doses of vaccine in a bottle," says Arntzen. This would ensure that it was of consistently high quality and that each child received the proper dose.

The researchers are working with colleagues in Mexico on a series of experiments with banana-vaccines. They hope that the Mexican government and the US Food and Drug Administration will approve the vaccines at the same time. The scientists will also experiment with banana vaccines for diarrhoea, which kills millions of children in developing countries each year. They have already engineered potatoes to produce antigens from *Escherichia coli*, a common

cause of diarrhoea in developing countries, and the Norwalk virus, which is often the culprit in the developed world. These produced an immune response in mice fed raw potatoes.

Arntzen hopes they will be able to develop bananas that can vaccinate against a range of different diseases, such as measles, yellow fever, diphtheria and polio. "I don't see any reason why we can't make this work," he says.

Magazine issue 2048 , published 21 September 1996

Golden Rice is safe to eat, says FDA

Golden Rice, the staple food genetically designed to contain beta carotene, a precursor to vitamin A, has been judged safe to eat by the US Food and Drug Administration (FDA). A letter from the FDA on May 24 stated that the agency had no further questions about the safety of the rice, originally developed to provide a rich source of vitamin A for children whose diets are deficient in this nutrient.

The rice has now been declared safe in four countries, including Australia, New Zealand and Canada. None of these decisions, including the FDA's, is a formal approval, but rather the local regulators reviewed data submitted by the Philippines-based International Rice Research Institute (IRRI) and declared they had no further questions about the rice's safety. Many, however, will see this latest move from the US agency as a stamp of approval, says Jennifer Kuzma, who studies attitudes to genetic engineering at North Carolina State University in Raleigh. And the regulators' positive opinion will be incorporated into the Organisation for Economic Co-operation and Development's consensus documents on bio-safety, which other countries consult to help guide their own decisions on food safety.

Although the US has no plans to grow or import the rice, the FDA's decision is still important, says Kuzma. "Other countries often look to the FDA as the first mover," she says. And it will protect against legal issues and help defuse any controversy should imports of regular rice ever become accidentally contaminated with Golden Rice.

Golden Rice was created in response to a major nutritional crisis affecting some of the poorest countries in the world. But because it is genetically modified (GM), the beta-carotene-enriched crop has struggled to overcome public

fears over genetically modified organisms (*Nat. Biotechnol.* **30**, 1017–1019, 2012). Opposition has been fierce throughout, even though the Golden Rice Project has a humanitarian board that included the crop's creators, and set out to make the nutritionally enriched rice available to low-income farmers and researchers throughout the developing world. The hope is that now, with the FDA's endorsement and other approvals, regulators in countries like Bangladesh and the Philippines, which are in the process of considering applications, will be emboldened to allow large-scale cultivation of the rice.

Work on Golden Rice began in the late 1990s by plant scientist Ingo Potrykus at the Swiss Federal Institute of Technology in Zurich, and biochemist Peter Beyer, at the University of Freiburg in Germany, as a way to combat vitamin A deficiency in the developing world. The technology, first reported in 2000 (*Science* **287**, 303–305, 2000) and later in 2005 (*Nat. Biotechnol.* **23**, 482–487, 2005), consists of inserting genes that control the biosynthetic pathway for beta carotene, a precursor to vitamin A, into rice. Initially, Potrykus and Beyer added two genes to the plant—a phytoene synthase from daffodils and a phytoene desaturase from a common soil bacteria—to turn on the beta-carotene-synthesis pathway in the grains. The beta-carotene-rich grains turned a deep golden color, giving the rice its name.

Vitamin A deficiency affects 250 million children, causing blindness in an estimated 250,000–500,000 children each year. This nutrient deficiency also compromises the immune system, leading to death from common childhood diseases like measles or diarrhea. By improving access to vitamin A, the enriched crop could prevent around 1–2 million childhood deaths each year, says Adrian Dubock,

First migraine-prevention antibody approved

On May 17, the US Food and Drug Administration approved a first-in-class monoclonal antibody drug to prevent migraine headache. Amgen's Aimovig (erenumab) is the first biologic drug to target the calcitonin-gene-related peptide (CGRP) receptor. CGRP signaling contributes to migraine pain, by inducing blood vessel dilation and pain sensitization on the trigeminal ganglion, outside the central nervous system. The antibodies act to prevent migraine pain by blocking CGRP. Unlike existing small-molecule CGRP antagonists used to treat acute migraine episodes, direct targeting of the peptide or its receptor with a monoclonal antibody is more specific, with few or no apparent adverse effects, and can be used as prevention (*Nat. Biotechnol.* **36**, 207–208, 2018). In phase 2 and 3 studies in chronic and episodic migraine, Aimovig significantly reduced monthly migraine days and use of acute migraine medications compared with placebo. In an ongoing open-label extension study in episodic migraine (4–14 headache days per month), these effects were sustained for up to 15 months. Also, a dedicated phase 3b study (LIBERTY) in individuals with episodic migraine who had failed two to four prior treatments showed that those taking Aimovig had nearly threefold higher probability of cutting their migraine days by half or more compared with placebo. Anticipating a crowded field for CGRP biologics, Amgen set Aimovig's price at \$6,900 per year—considerably lower than analysts' expectations. Other monoclonal antibodies targeting the CGRP pathway in late-stage development include Petach Tikva, Israel-based Teva Pharmaceuticals' eptinezumab, Alder Biopharmaceuticals' fremanezumab and Eli Lilly's galcanezumab. Unlike Aimovig, these antibodies target the peptide itself. Amgen is partnering with Novartis to co-commercialize Aimovig in the US. In the deal, the Basel-based pharma also gained exclusive commercialization rights to the drug in Europe, Canada and elsewhere. Following on the heels of the US approval, on June 1, the European Medicines Agency's Committee for Medicinal Products for Human Use recommended granting a marketing authorization for Aimovig.

“[The thrill] is tainted with the knowledge that people are sick and dying. I think that tempers the excitement, because there's a reality that accompanies that that's very, very sad.” Nancy Sullivan of the NIH Vaccine Research Center expresses mixed feelings about the start of a long-awaited vaccine trial with the recent Ebola outbreak in the Democratic Republic of the Congo.

(STAT, 22 May 2018)

New money flows to CARB-X for antimicrobials

The UK government and the Bill & Melinda Gates Foundation in May joined a partnership dedicated to the development of new antibiotics, vaccines, diagnostics and other products against drug-resistant bacterial infections. The UK's Global AMR (antimicrobial resistance) Innovation Fund is committing up to £20 (\$26.6) million and the Bill & Melinda Gates Foundation up to \$25 million over the next three years to CARB-X, a non-profit, public-private partnership set up with the mission of averting the threat of drug-resistant diseases. With the new support, CARB-X has now amassed more than \$500 million to invest through 2021 to accelerate innovation by supporting early-stage projects and phase 1 clinical trials. Geneva-based CARB-X was formed in 2016 with support from UK charity Wellcome Trust, the US Department of Health and Human Services Biomedical Advanced Research and Development Authority (BARDA) and the National Institute of Allergy and Infectious Diseases (NIAID). It currently has 33 projects ongoing in seven countries, including five drug candidates advanced to phase 1 trials and two diagnostics now in the system-integration and testing phase. Of the 27 current drug projects, 11 focus on new targets and 9 on new antibiotic classes. Four are biologics: one is a vaccine against *Staphylococcus aureus* and the other three, against *Pseudomonas aeruginosa*, include recombinant lysis protein, a multispecific antibody and an antibody-drug conjugate. Two are small molecules targeting the microbiome: *Clostridium difficile* and carbapenem-resistant Enterobacteriaceae/vancomycin-resistant Enterococci. Of the six device and/or diagnostic projects, three focus on development of hospital-based systems for identifying bloodstream infections. May 2018 also marked the official launch of the Global AMR Research & Development Collaboration Hub, announced at the 71st World Health Assembly in Geneva. The initiative was conceived in 2017 with initial support from the Bill & Melinda Gates Foundation and the Wellcome Trust. The secretariat of the Global AMR R&D Hub will initially be based in Berlin, at the German Center for Infection Research. Its 18 members also include Russia, China, the US, France and the European Commission.

“What genetic difference is it that we are going to root for anyway—the immune system differences between Switzerland and Egypt?” Ethicist Arthur Caplan of NYU School of Medicine takes exception at Fox Sports and 23andMe's ad campaign “Root for Your Roots”, where they team up to generate interest in the World Cup. (*leapsmagazine*, 4 May 2018)

who worked on the early stages of the rice's development and is now executive secretary of the Golden Rice Humanitarian Board, set up to provide strategic guidance to the Golden Rice Project. “That's more mortality than is associated with tuberculosis, malaria or HIV,” he says.

But the development of Golden Rice, the first crop engineered with traits to benefit consumers rather than enhance production, has taken far longer than anyone anticipated says Dubock. “I originally thought in 2001 that it would be available by 2003,” he says. “Instead it has taken almost 20 years.”

In 2001, the Swiss agricultural biotech Syngenta, where Dubock worked, took on Golden Rice development, in a complicated licensing arrangement, involving 23 agreements with 16 licensees, aimed at ensuring the rice would be distributed free to subsistence farmers in the developing world. The company's scientists made several improvements to the rice, such as substituting the daffodil phytoene synthase with an equivalent gene from maize that improved beta-carotene production. The new strain produced up to 37 µg per gram, such that a single serving could deliver more than half the recommended daily intake of beta carotene. In 2004, Syngenta withdrew from the project and since then, development has been carried out at independent research institutes, such as the non-profit research and educational institute IRRI, supported by charities including the Rockefeller Foundation and the Bill and Melinda Gates Foundation.

The rice has overcome a number of technical hurdles and controversies along the way. A 2012 study in China, which found that Golden Rice was as effective as beta-carotene oil at providing vitamin A to children, was retracted in 2015 due to a failure to obtain informed consent from the children's parents and faked ethics approval documents.

And in 2017, a study in India that crossed Golden Rice with a local variety found that the resulting plants were stunted and pale, and yield was reduced by as much as 60–70%. Many opponents of genetically modified crops leapt on that result as proof that the project was doomed. But, in general, when researchers insert genes into a plant genome, they must ensure it is done in a way that allows their expression without interfering with other genes. Ashok Singh, a plant geneticist at the Indian Agricultural Research Institute in New Delhi who led the study, identified the problem.

The stunted rice came from a genetic transformation known as the R event, in which the insertion disrupted an important native membrane transport gene called *OsAux1*. A different transformation event, called the E event,

did not disrupt any native genes and has similar agronomic characteristics to native rice. “The problem had nothing to do with Golden Rice itself, it was event-associated,” he says. It was data from rice based on the E event that were submitted to the FDA and other regulators.

Most scientists expect technical difficulties, says Russell Reinke who leads the healthier rice program at IRRI. “That's just the process of science happening as it should,” he says. Singh is concerned, however, about the low carotenoid content of the E-event rice (just 11 µg per gram of grain), and its stability in the grain during processing and storage.

The FDA also noted that the levels of beta carotene were too low to make any claims about nutrient content. At such concentrations, the rice is not a viable solution for vitamin deficiency, says Paul Johnston, head of the environmental charity Greenpeace's science unit at the University of Exeter, and puts paid to any nutritional claims. “Given that that's what it is intended to solve, you've got to question what the point of it is,” he says.

Greenpeace and other opponents of GM crops say that other interventions, such as increasing consumption of conventionally bred sweet potatoes rich in beta carotene, and ensuring people eat a diverse diet, are more effective at combating vitamin A deficiency than Golden Rice.

But IRRI's Reinke says the FDA's nutritional value calculation was based on the relatively small amount of rice people eat in the US. Based on consumption levels in Asia, where rice makes up to 70% of the daily calorie intake, Golden Rice could provide as much as half the average daily requirement of vitamin A, he says. Johnston is not sure Golden Rice will ever make it to the field. “It's always in the future, the time span never seems to compress,” he says.

But the rice's proponents say they are getting closer to full deployment. The most important next step is receiving regulatory approval from Bangladesh and the Philippines to cultivate and allow people to eat the rice. IRRI submitted applications to both countries last year. Once approved, the Golden Rice trait will still need to be bred into local varieties, and feeding trials will then be necessary to ensure it has the desired nutritional result. Singh estimates it will take at least five to six years after approval to finally get the rice onto people's plates.

Even if regulators give a green light, changing political or social attitudes could add further twists and turns into Golden Rice's long road to acceptance. “If it was just the regulations, I would say it will be very soon. But the politics are very hard to control,” says Dubock. “But it is clear that we are getting closer.”

Brian Owens St. Stephen, New Brunswick, Canada



Loss of crops to drought contributed to a food crisis in Ethiopia in 2008.

AGRICULTURE

Cross-bred crops get fit faster

Genetic engineering lags behind conventional breeding in efforts to create drought-resistant maize.

BY NATASHA GILBERT

Old-fashioned breeding techniques seem to be leading genetic modification in a race to develop crops that can withstand drought and poor soils.

As the climate warms and rainfall becomes more erratic, farmers worldwide will increasingly need crops that can thrive in drought conditions. And the high costs of fertilizers — along with the environmental damage they can cause — are also pushing farmers to look for crop varieties that can do more with less.

The need for tougher crops is especially acute in Africa, where drought can reduce maize (corn) yields by up to 25%. The Drought Tolerant Maize for Africa project, which launched in 2006 with US\$33 million, has developed 153 new varieties to improve yields in 13 countries. In field trials, these varieties match or exceed the yields from commercial seeds under good rainfall conditions, and yield up to 30% more under drought conditions.

An analysis published earlier this year reported that by the project's end in 2016, the extra yields from drought-tolerant maize could help to reduce the number of people living in poverty in the 13 countries by up to 9%

(R. La Rovere *et al.* *J. Dev. Areas* **48**, 199–225; 2014). In Zimbabwe alone, that effect would reach more than half a million people.

The project's success is due in large part to its access to a large seed bank managed by one of its partners, the International Maize and Wheat Improvement Center (CIMMYT) in Mexico City. Breeders from CIMMYT and the International Institute for Tropical Agriculture in Ibadan, Nigeria, searched the collection for maize varieties that thrive in water-scarce regions. The researchers cross-bred these varieties and then mated the most drought-tolerant of their offspring. Several cycles of this process led to seed that was better adapted to water-scarce conditions. In a final step, project scientists cross-bred these plants with varieties that have been successful in Africa.

"It is a painstaking and expensive process," says Kevin Pixley, director of CIMMYT's genetic resources programme.

The CIMMYT researchers established that certain characteristics predict how a maize plant will fare in drought. One of the most telling is the number of days between when the plant's male organs shed pollen and when the female silks emerge. When water is scarce, the silks emerge late. If the delay is long enough,

they emerge after the plants have released their pollen and are not fertilized.

"Finding out this relationship was very important to be able to select for drought tolerance," says Pixley. By favouring plants with shorter intervals between pollen release and silk emergence, breeders were able to produce maize that was more resistant to drought.

Drought tolerance is a complex trait that involves multiple genes. Transgenic techniques, which target one gene at a time, have not been as quick to manipulate it. But CIMMYT and six other research organizations are also developing genetically modified (GM) varieties of drought-resistant maize, in collaboration with agricultural biotechnology giant Monsanto in St Louis, Missouri. Coordinated by the African Agricultural Technology Foundation in Nairobi, the Water Efficient Maize for Africa project aims to have a transgenic variety ready for African farmers by 2016 at the earliest.

Like drought resistance, maize's ability to grow in nitrogen-poor soils is genetically complex, and the need for varieties that do well with little fertilizer is pressing. Most African farmers can afford only one-tenth the amount of fertilizer recommended for their crops. This is one of the biggest problems they face, says Biswanath Das, a maize breeder at CIMMYT.

Researchers at CIMMYT are working to address that problem through the Improved Maize for African Soils (IMAS) project, a collaboration with the Kenya Agricultural Research Institute in Nairobi; the South African Agricultural Research Council in Pretoria; and DuPont Pioneer in Johnston, Iowa. The 10-year, US\$19.5-million project is pursuing conventional and transgenic approaches.

Since its launch in 2010, IMAS has developed 21 conventionally bred varieties. Over the next year the project's leaders hope to commercialize these varieties and introduce them in eight countries. In field tests, IMAS varieties yielded up to 1 tonne per hectare more in nitrogen-poor soils than did commercially available varieties. By contrast, the project's researchers say that they are at least 10 years from developing a comparable GM variety.

Conventional breeding will probably have a greater impact, says Das, "but it is important to consider all options". ■

CORRECTIONS

The News Feature 'Survival of the fittest' (*Nature* **513**, 157–159; 2014) referred to the wrong Possession Island. The penguin work was done on the French Base d'Alfred Faure in the Crozet archipelago.

The World View by Casparus J. Crous (*Nature* **513**, 7; 2014) implied that Saudi Arabian scientists on highlycited.com were all at a single university. In fact, most were at one institution but several came from three other universities in Saudi Arabia.

Increasing Plant Enzyme Efficiency May Hold Key To Global Warming

Date: January 31, 2006

Source: Emory University Health Sciences Center

Summary: Global warming just may have met its match. In research recently completed at Emory University School of Medicine, scientists have discovered a mutant enzyme that could enable plants to use and convert carbon dioxide more quickly, effectively removing more greenhouse gasses from the atmosphere.

FULL STORY

Global warming just may have met its match. In research recently completed at Emory University School of Medicine, scientists have discovered a mutant enzyme that could enable plants to use and convert carbon dioxide more quickly, effectively removing more greenhouse gasses from the atmosphere.

The findings were published online on January 19 and will appear in the February issue of the journal "Protein Engineering Design and Selection." Ichiro Matsumura, PhD, assistant professor of biochemistry at Emory University School of Medicine, is the senior author and principal investigator. The first author is research specialist Monal R. Parikh.

During photosynthesis, plants, and some bacteria, convert sunlight and carbon dioxide into usable chemical energy. Scientists have long known that this process relies on the enzyme rubulose 1,5-bisphosphate carboxylase/oxygenase, also called RuBisCO. While RuBisCO is the most abundant enzyme in the world, it is also one of the least efficient. As Dr. Matsumura says, "All life pretty much depends on the function on this enzyme. It actually has had billions of years to improve, but remains about a thousand times slower than most other enzymes. Plants have to make tons of it just to stay alive."

RuBisCO's inefficiency limits plant growth and stops organisms from using and assimilating all the carbon dioxide in the atmosphere. Since the spread of photosynthesis has not kept pace with the amount of gas in the atmosphere, the gas builds up. The resulting gas buildup is one cause of global warming. A 2004 report by the National Science Foundation estimates that atmospheric carbon dioxide concentrations remained steady at between 200 and 280 parts per million for thousands of years, but that carbon dioxide levels have risen dramatically since the Industrial Revolution of the 1800s, leading to 380 parts per million of carbon dioxide in the atmosphere today.

For decades, scientists have struggled to engineer a variant of the enzyme that would more quickly convert carbon dioxide. Their attempts primarily focused on mutating specific amino acids within RuBisCO, and then seeing if the change affected carbon dioxide conversion. Because of RuBisCO's structural complexity, the mutations did not have the desired outcome.

For their own study, Dr. Matsumura and his colleagues decided to use a process called "directed evolution" which involved isolating and randomly mutating genes, and then inserting the mutated genes into bacteria (in this case *Escherichia coli*, or *E. coli*). They then screened the resulting mutant proteins for the fastest and most efficient enzymes. "We decided to do what nature does, but at a much faster pace." Dr. Matsumura says. "Essentially we're using evolution as a tool to engineer the protein."

Because *E. coli* does not normally participate in photosynthesis or carbon dioxide conversion, it does not usually carry the RuBisCO enzyme. In this study, Matsumura's team added the genes encoding RuBisCO and a helper enzyme to *E. coli*, enabling it to change carbon dioxide into consumable energy. The scientists withheld other nutrients from this genetically modified organism so that it would need RuBisCO and carbon dioxide to survive under these stringent conditions.

They then randomly mutated the RuBisCO gene, and added these mutant genes to the modified *E. coli*. The fastest growing strains carried mutated RuBisCO genes that produced a larger quantity of the enzyme, leading to faster assimilation of carbon dioxide gas. "These mutations caused a 500 percent increase in RuBisCO expression," Dr. Matsumura says. "We are excited because such large changes could potentially lead to faster plant growth. This results also suggests that the enzyme is evolving in our laboratory in the same way that it did in nature."

Story Source:

Materials provided by **Emory University Health Sciences Center**. Note: Content may be edited for style and length.

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Biotechnology Application, part 2

Student Guide

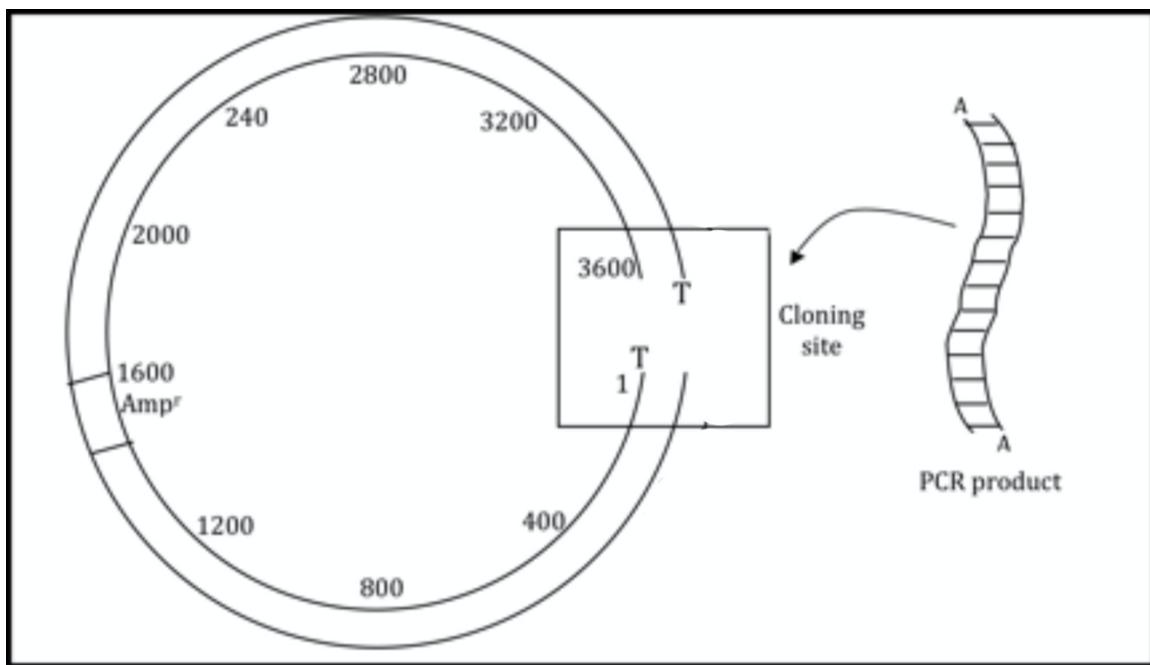
HIV is a nasty virus that specifically targets helper-T cells in the immune system in effect destroying the only defense our bodies have against the virus. Scientists have recently discovered a protein that seems to block HIV infection. It is called a *chemokine*. In effect the chemokine protein competes with the HIV pathogen for the receptor on the helper-T cell. In other words, if the chemokine protein binds to the receptor first, then HIV can't bind and can't get inside the cell. This is a possible promising treatment for HIV infection. In order to test this treatment, we need to isolate the chemokine gene, make multiple copies of it, and figure out a way to express it (similar to the activity we did in class). For this exercise, let's say that the sequence of the chemokine gene is the following:

5'-ATGGAATTCCCGCTATGGCTAACTATAGATAATGGATACGAATATGATTAG-3'

1. If we look in the nucleus of a cell, this gene would be double stranded. Write the complementary strand of DNA just below the sequence above to make it double-stranded DNA.
2. Label your new strand of DNA with 5' and 3' on the appropriate ends.
3. Polymerase Chain Reaction (PCR) works in much the same way as semi-conservative DNA replication. However, in PCR, the DNA strands are heated to separate the strands of the double-helix. What enzyme is the heat a substitute for?
4. *Taq* polymerase in PCR is a substitute for what enzyme in our bodies?
5. In the double-stranded DNA strand above, if the double helix was being “unzipped” from left to right, which strand would be the leading strand and which would be the lagging strand?
 - a. The top strand: _____
 - b. The bottom strand: _____
6. Why must we add primers when doing PCR? (Be specific!)

We now want to clone our PCR product (our chemokine gene) into a plasmid. **Plasmids** are naturally occurring, self-replicating extragenomic DNA in bacteria. Scientists are able to artificially generate plasmids with desired traits, such as antibiotic resistance (in this one, that is the **Amp^r gene**, for Ampicillin resistance) and RNA polymerase promoter sites. Scientists can also insert desired pieces of DNA (for example, the human chemokine gene) into plasmids and place them into bacterial cells. This process is called **DNA cloning**. Bacteria replicate (by asexual reproduction) about every 20 minutes, thus replicating the plasmids within.

During PCR, the *Taq* polymerase enzyme adds additional adenine residues (A's) to the ends of the PCR product. Scientists have exploited this fact and produced plasmids with overhanging thymine residues (T's), like the one pictured below.



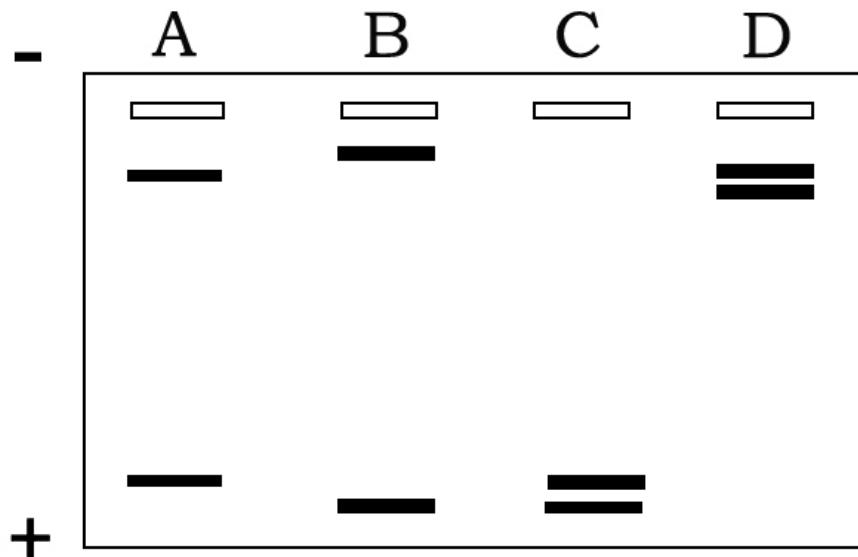
7. If we are going to use bacterial plasmids to produce the *human* chemokine protein, what assumptions are we making about bacteria (Come up with at least two assumptions)?

8. What enzyme from DNA replication might be useful in getting your PCR product fused into a bacterial plasmid?

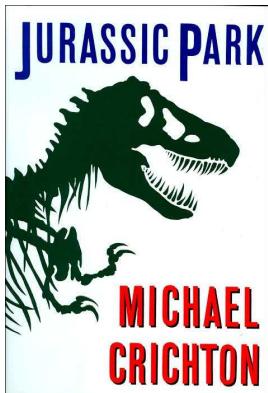
9. Once you create your transformed plasmid, you can use various methods (called *transformation* methods) to get the plasmid inside your bacterial cells. Looking at the plasmid above, and knowing that you likely had far more bacterial cells than available plasmids when you did the transformation, how will you select for ONLY bacteria containing your plasmid of interest (i.e., how do you kill off all of the other bacteria that didn't get a plasmid)?
10. Looking back at our original gene (shown below), can you tell me the length of fragments we will get if we cut *just* the chemokine gene with EcoRI (Remember, the restriction site for EcoRI is G^AAATTC)?
- ATGGAATTCCCGCTATGGCTAACTATAGATAATGGATACGAATATGATTAG
11. If the gene was cloned into the vector with the start codon (ATG) next to the #1 on the plasmid, what size fragments will we get if we cut the cloned product (plasmid with our gene in it) with EcoRI?
12. What size fragments will we get if it is in the opposite orientation (with the ATG next to the #200)?

We are going to run a gel electrophoresis on each of the products from our two plasmids we built (the one with ATG next to #1 and the one with ATG next to #200). Look at the gel below.

13. Of the four possibilities below, which lane would result from cutting the one with the ATG next to #1:
14. Of the four possibilities below, which lane would result from cutting the one with the ATG next to #200:



Bioinformatics
"Jurassic Park" Dino-DNA Analysis
Student Guide



In 1990, Michael Crichton published the book "Jurassic Park" and at one point in the book, Dr. Henry Wu is asked to explain some of DNA techniques used in reconstructing the extinct dinosaur genomes. He also alludes to the fact that they don't have the entire genome but that they "fill in the gaps" with modern day frog DNA.

In 1992, Dr. Mark Boguski, at the National Center for Biotechnology Information, NCBI, having read the book "Jurassic Park" entered this sequence into a text editor and searched all of the known DNA sequences at the time. This collection of sequences make up a database referred to as GENBANK. Mark wrote up his findings and submitted a manuscript to the journal BioTechniques, as a tongue-in-cheek joke. His manuscript was accepted and published. ([Boguski, M.S. A Molecular Biologist Visits Jurassic Park. \(1992\) BioTechniques 12\(5\):668-669.](#))

You will reproduce Mark's experiment by using select, copy and paste to send this sequence for comparison against the GENBANK database just as Mark did in 1992.

To learn more about an “unknown” sequence, one of the first steps is to use a bioinformatics program such as BLAST (Basic Local Alignment Search Tools) to search databases for similar known sequences.

1. Before carrying out the search hypothesize what living organisms should be the best "hits" - organisms that have the highest match value. In other words, which organisms do you expect to have the most similar DNA to the purported dinosaur DNA.

Go to the NCBI BLAST website (<http://blast.ncbi.nlm.nih.gov/Blast.cgi>). Click on “Nucleotide BLAST” (under Basic BLAST). Select, copy, and paste the sequence provided into the area labeled “Enter accession number, gi, or FASTA sequence”. Just keep the default settings and then click the BLAST button at the bottom of the page. It will take a few seconds for the analysis to complete. Once it has completed the search, familiarize yourself with the results a bit by scrolling down. Note the three main sections: Graphic Summary, Descriptions, and Alignments. In the Descriptions section.

2. What are a few of the organisms that had sequences that best matched the dinosaur DNA sequence from “Jurassic Park?” (Note that sequences with equal “E values” are equally similar to your query sequence)
3. Did you recognize any of these organisms? Did they match the organisms you hypothesized from above? What do you think is going on?

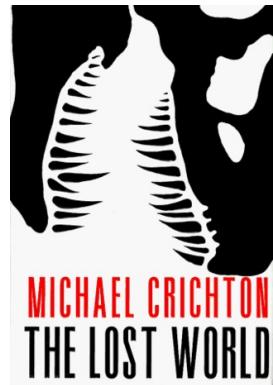
So, did you perhaps come up with the idea that Michael Crichton just made up the letters - he is an author after all. Let's test this idea.

4. Put your fingers on the letters "A", "C", "G", and "T" on your keyboard and randomly enter 100 nucleotides into the area labeled "Enter accession number, gi, or FASTA sequence". What kind of BLAST results do you get with random sequence?

5. So now what do you think? Do you think he just made the sequence, which is much longer than 100 nucleotides, up in his head?

Mark's published article was brought to Michael Crichton's attention. In his second book, "The Lost World", Mr. Crichton used Mark as a consultant. Mark chose a DNA sequence from a living organism which is much more closely related to the dinosaurs. However, Mark played a little trick on Mr. Crichton by embedding a message in the protein translation of the DNA sequence which he submitted for use in the book.

Use the sequence provided for the "Lost World" and BLAST it just as you did before.



6. What are the two top hits from this search? Identify the scientific names and common names.

Now we are going to back to the main [BLAST page](#). But this time we will be doing a different kind of search called a "BLASTx". So click on "BLASTx." The "x" stands for translation. This type of search translates the DNA sequence to six protein sequences (all three reading frames and forward or reverse) and searches the protein database. This search takes longer but is much informative about the relationship between the probe DNA sequence and the hits in the database.

Select, copy, and paste the "Lost World" sequence into the Query Sequence form. Wait for the search to complete - if you see "Putative conserved domains have been detected, click on the image below for detailed results." keep waiting, this is not the end result of the search. After the search is completed you will see a similar results page as before. Recall that proteins use 20 letters of amino acids instead of 4 letters of DNA. So when you scroll down to the alignments you will no longer see only AGCT, you will also see 16 other letters being used in the alignments.

7. How many DNA letters correspond to one amino acid?

8. Mark added extra DNA letters that when translated would create a hidden message. What was Mark's hidden message?

9. How many extra DNA letters did he have to include, unbeknown to Michael Crichton, in order to have his secret message, when translated, present itself?

10. Summarize why it is possible that a BLAST search can be used to identify a stretch of DNA and match it to an organism. In your summary use the words: Nucleotides, Variation, Mutation, Differences, Aligned, Similar, Comparison

Periodic Table of the Elements

IA

hydrogen 1 H 1.0079	
lithium 3 Li 6.941	beryllium 4 Be 9.0122
sodium 11 Na 22.990	magnesium 12 Mg 24.305

IIA

potassium 19 K 39.098	calcium 20 Ca 40.078
rubidium 37 Rb 85.468	strontium 38 Sr 87.62

Key:

element name
atomic number
symbol
atomic weight (mean relative mass)

scandium 21 Sc 44.956	titanium 22 Ti 47.867	vanadium 23 V 50.942	chromium 24 Cr 51.996	manganese 25 Mn 54.938	iron 26 Fe 55.845	cobalt 27 Co 58.933	nickel 28 Ni 58.693	copper 29 Cu 63.546	zinc 30 Zn 65.39	gallium 31 Ga 69.723	germanium 32 Ge 72.61	nitrogen 7 N 14.007	oxygen 8 O 15.999	fluorine 9 F 18.998	helium 2 He 4.0026		
yttrium 39 Y 88.906	zirconium 40 Zr 91.224	niobium 41 Nb 92.906	molybdenum 42 Mo 95.94	technetium 43 Tc [98]	ruthenium 44 Ru 101.07	rhodium 45 Rh 102.91	palladium 46 Pd 106.42	silver 47 Ag 107.87	cadmium 48 Cd 112.41	indium 49 In 114.82	tin 50 Sn 118.71	antimony 51 Sb 121.76	tellurium 52 Te 127.60	bromine 17 Cl 35.453	argon 18 Ar 39.948		
caesium 55 Cs 132.91	barium 56 Ba 137.33	57-70 * Lu 174.97	lutetium 71 Hf 178.49	hafnium 72 Ta 180.95	tantalum 73 W 183.84	tungsten 74 Re 186.21	rhenum 75 Os 190.23	osmium 76 Ir 192.22	iridium 77 Pt 195.08	platinum 78 Au 196.97	gold 79 Hg 200.59	mercury 80 Tl 204.38	thallium 81 Pb 207.2	lead 82 Bi 208.98	bismuth 83 Po [209]	polonium 84 At [210]	astatine 85 Rn [222]
francium 87 Fr [223]	radium 88 Ra [226]	89-102 ** Lr [262]	lawrencium 103 Rf [261]	rutherfordium 104 Db [262]	dubnium 105 Sg [266]	seaborgium 106 Bh [264]	bohrium 107 Hs [269]	hassium 108 Mt [268]	meitnerium 109 Uun [271]	ununnilium 110 Uuu [272]	ununbium 111 Uub [277]	ununquadium 112 Uuq [289]					

*lanthanoids

**actinoids

lanthanum 57 La 138.91	cerium 58 Ce 140.12	praseodymium 59 Pr 140.91	neodymium 60 Nd 144.24	promethium 61 Pm [145]	samarium 62 Sm 150.36	europerium 63 Eu 151.96	gadolinium 64 Gd 157.25	terbium 65 Tb 158.93	dysprosium 66 Dy 162.50	holmium 67 Ho 164.93	erbium 68 Er 167.26	thulium 69 Tm 168.93	ytterbium 70 Yb 173.04
actinium 89 Ac [227]	thorium 90 Th 232.04	protactinium 91 Pa 231.04	uranium 92 U 238.03	neptunium 93 Np [237]	plutonium 94 Pu [244]	americium 95 Am [243]	curium 96 Cm [247]	berkelium 97 Bk [247]	californium 98 Cf [251]	einsteinium 99 Es [252]	fermium 100 Fm [257]	mendelevium 101 Md [258]	nobelium 102 No [259]

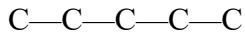
Hydrocarbon chemistry

Student Guide

Most of the macromolecules that we deal with are long chains or rings of carbon atoms with various other atoms stuck in there occasionally. Glucose molecules are 6-carbon rings with a few oxygen's attached. Glucose makes up carbohydrates. Proteins are made out of amino acids, which are carbons and a nitrogen group. Lipids have long tails of carbon-carbon bonds. Even DNA has a backbone of 5-carbon rings (deoxyribose) and carbon-rich nitrogenous bases (A, T, C, and G). Carbon seems to be the ultimate atom for building the blocks of living things and there is good reason for that. Let's take a look at carbon.

1. Looking at the periodic table, can you draw a carbon atom?

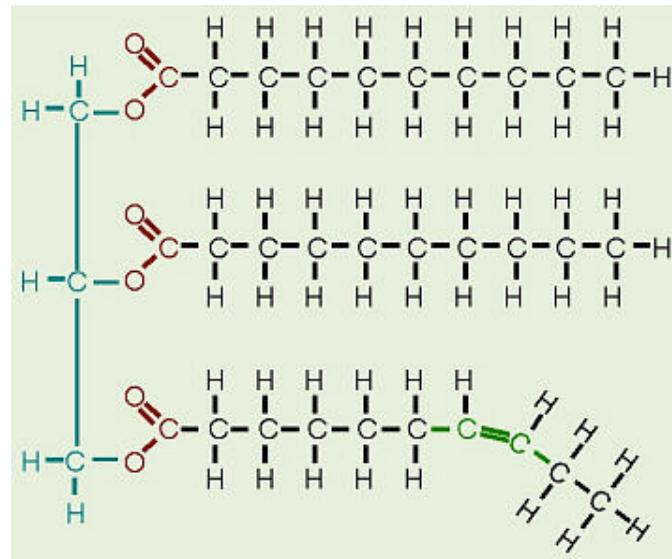
2. How many bonds is Carbon likely to make _____ and will they be ionic or covalent? _____
3. Now, let's try making some carbon-based molecules. We will start very simple and just do a 5-carbon molecule. Let's indicate a bond by just drawing a line, so two carbons bound together would look like this: C—C How many different ways can you draw a 5-carbon molecule? I'll start you off:



Draw as many as you can (keeping in mind how many bonds a carbon-atom can make):

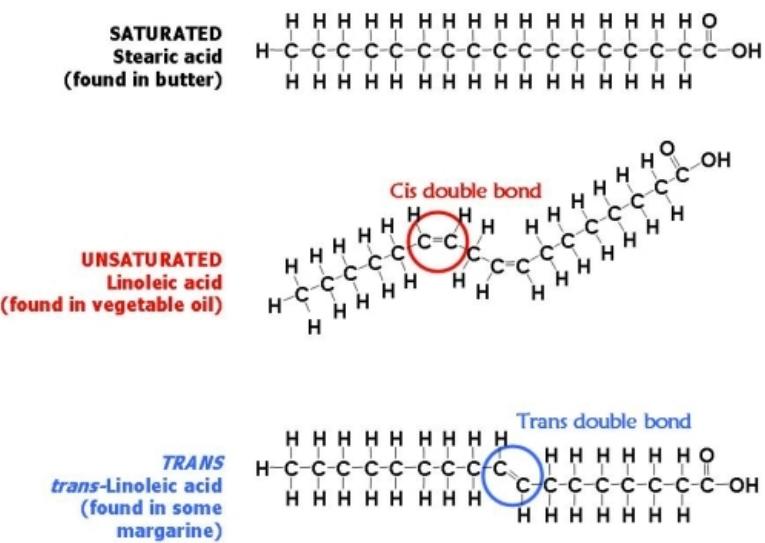
4. This figure shows an **unsaturated** fatty acid tail compared to two saturated ones. What is the difference between a saturated and unsaturated fat?

5. Can you see how the difference you see above would make one solid at room temperature and one liquid at room temperature? Explain.

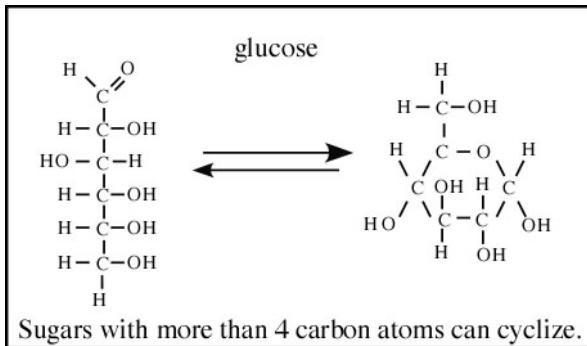


6. This figure shows the difference between a **cis**-double bond and a **trans**-double bond: Describe the difference between a cis- and trans- double bond.

7. How might this affect the consistency of these fats at room temperature?



8. You can see that in fats, the only other atom involved, besides carbon and hydrogen, is oxygen. The same is true for carbohydrates, which are long chains of glucose, right?
What is the same and what is different between glucose and fats?



9. Let's look at proteins. An amino acid is made by attaching 4 different molecules to a central carbon atom. The four different molecules are

 - An 'amino' group: NH₂ (when you draw this, make sure you look at a periodic table to see how many bonds nitrogen can make)
 - An 'acid' group: COOH (one of these oxygens is double-bonded to the carbon and the other is bonded to the carbon *and* the hydrogen; again, look to see how many bonds an oxygen makes)
 - An H
 - An R-group (just label it as 'R' and assume it makes *one* bond)

Draw an amino acid below:

10. Draw each of the following using our Lewis-dot structures, be sure to label them as either polar covalent, non-polar covalent, or ionic:

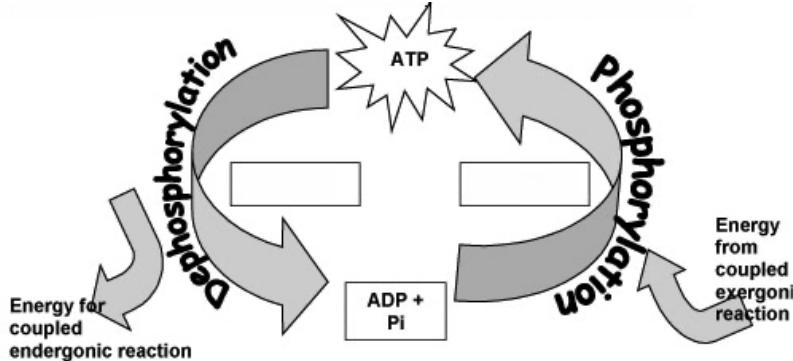
 - a. Calcium chloride
 - b. H₂S (dihydrogen sulfide)
 - c. Sulfur gas (fill its shell by adding more S)
 - d. Carbon dioxide (make sure to fill both the carbon and oxygen shells without adding any additional elements)

11. Draw a hydrogen bond between two molecules of H₂S (be sure to include all of the electrons):
12. Draw a Phosphorus (P) isotope that weighs 33 g/mole. (Be sure to differentiate between protons and neutrons by color, size, or label somehow)
13. If a water molecule were to lose a hydrogen atom but keep the electron it was sharing what kind of charge will the molecule now have?

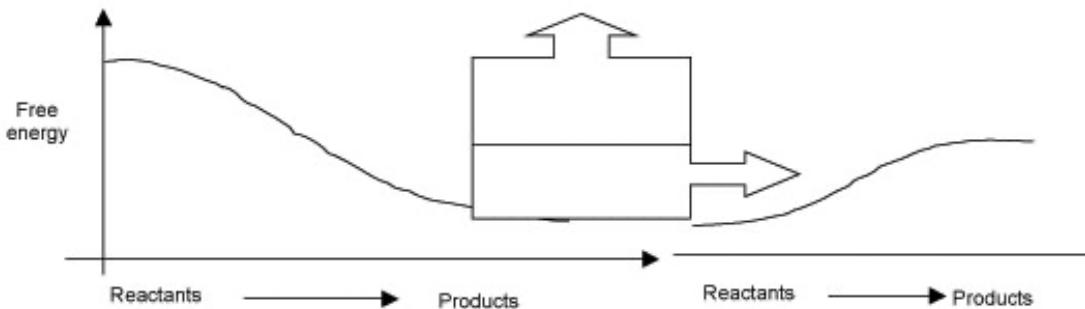
Applying Energy Principles

Student Guide

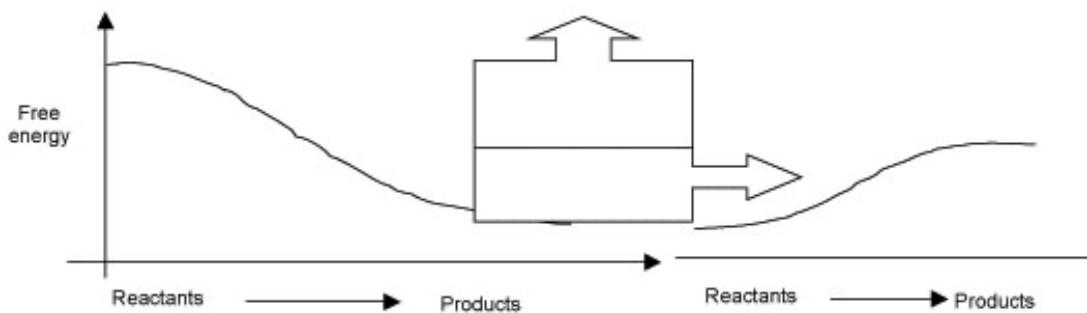
1. Which of the following statements is/are true about an exergonic reaction?
 - a. The reactants contain more free energy than the products.
 - b. Energy is given off or released to the surroundings during the reaction.
 - c. The total energy of the universe remains the same.
2. Complete the following for an endergonic reaction:
 - a. Products have _____ energy than the reactants.
 - b. Reactants are _____ stable than the products.
 - c. Energy is stored/released during this reaction.
3. Label the following reactions as exergonic or endergonic:
 - a. Gasoline burning _____
 - b. Breaking down of starch _____
 - c. Active transport _____
 - d. Building proteins _____
4. Study the diagram below and label which side is endergonic and which side is exergonic in the boxes provided:



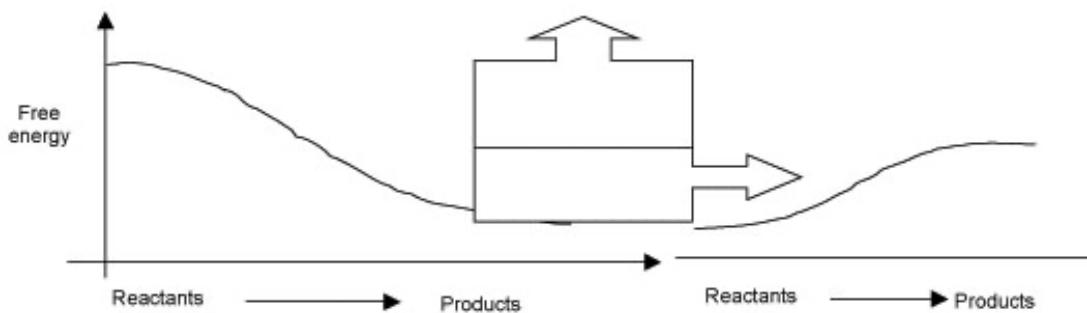
-
5. Label this diagram for the energy transfers involved in **driving a car uphill**. Label the following in your diagram: gasoline at start, gasoline at end, car at bottom of hill, car at top of hill, heat loss, transfer of usable energy. Label the exergonic and the endergonic reaction.



6. Label this diagram for the energy transfers involved in **active transport**. Label the following in your diagram: random distribution of molecules, more concentrated molecules, ATP, ADP + P, heat loss, transfer of usable energy. Label the exergonic and the endergonic reaction.



7. Label this diagram for the energy transfers involved in **building proteins**. Label the following in your diagram: amino acids, polypeptide, ATP, ADP + P, heat loss, transfer of usable energy. Label the exergonic and the endergonic reaction.



Let's say that we ran the same food calorimetry exercise (burning food) that you saw in the video and we obtained the following results.

Food Item	Final Temp (T_f) °C.	Initial Temp (T_i) °C.	Change in Temp ($T_f - T_i$)	Initial Mass (M_i) g	Final Mass (M_f) g	Change in Mass ($M_f - M_i$)	Calories per gram (C/g)
Peanut	43°	19°		1.08g	0.71g		
Corn Chip	88°	19°		1.31g	0.18g		
Honey-comb cereal	57°	19°		0.91g	0.11g		

8. Calculate the Calories (kilocalories) per gram (C/g) for each food type. What might explain any differences observed in the caloric content of these different foods?
9. According to a nutritional database, a Peanut has 6.17 C/g, a Corn Chip has 5.39 C/g, and Honeycomb has 3.91 C/g. Excluding the possibility that the nutritional information is wrong, what could explain any differences observed?

10. Traditionally the caloric content of foods was measured in a bomb calorimeter. Today, however, most caloric determination of foods is done by analyzing the nutritional content of the food and multiplying these values by the corresponding number of calories per gram for these types of molecules.

Proteins and carbohydrates have approximately 4 Calories/g

Fats have approximately 9 Calories/g

For example, 1 gram of a Snickers candy bar contains 0.1 g of protein, 0.22 g of fat and 0.60 g of carbohydrates. Based on the caloric information for proteins, fats, and carbohydrates listed above, how many Calories would you expect 1 g of Snickers to contain?

11. One Snickers bar has a mass of 61 grams. How many Calories would a Snickers bar contain?

The actual listed calories on a Snickers bar is 278 Calories
How close were your calculations?

12. Now let's look at another situation. The "Nutritional Facts" information from a box of All Bran cereal is shown below. Calculate the Calories per serving based on this nutritional information (show your work on the following page).

Nutrition Facts	
Serving Size	_ cup (31 g; 1.1 oz)
Servings per package	About 17
Amount per Serving	
Total Fat.....	1 gram
Total Carbohydrate.....	24 grams
Fiber.....	11 grams
Sugars.....	6 grams
Other carbohydrates.....	7 grams
Protein.....	4 grams

13. The Calories per serving listed on the box are 80. How does this compare with your calculation?

14. How might you explain any difference observed?

Cellular Respiration

Photosynthesis

Nutrient Cycling

Student Guide

Now that we have learned about combustion, cellular respiration, and photosynthesis, we are equipped to apply this to an understanding of nutrient cycling, specifically the cycling of **Carbon** through the environment.

1. Define the following and list them for the **Carbon** cycle:

a. Pools:

b. Fluxes:

Grandma Johnson Problem

Making it real: Grandma Johnson had very sentimental feelings toward Johnson Canyon, Utah, where she and her late husband had honeymooned long ago. Because of these feelings, when she died, she requested to be buried under a creosote bush in the canyon.



2. On the back side of this paper, trace the path of a **CARBON** atom from Grandma Johnson's remains, to inside the leg muscle of a coyote.

Conservation Ecology

Student Guide

1. According to *National Geographic*, what was the ultimate cause of the downfall of Easter Island inhabitants?
 2. What does the “New Story” suggest and why is this a scarier scenario?
 3. What caused the WHO to parachute cats into Borneo? What lessons can we learn from this?
 4. What is our responsibility as God’s stewards?