Science Learning Packet

BIO B:
Genetics: Inheritance, Part 1 of 2

science learning activities for SPS students during the COVID-19 school closure.

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Due to the COVID-19 closure, teachers were asked to provide packets of home activities. This is not intended to take the place of regular classroom instruction but will help supplement student learning and provide opportunities for student learning while they are absent from school. Assignments are not required or graded. Because of the unprecedented nature of this health crisis and the District’s swift closure, some home activities may not be accessible.

If you have difficulty accessing the material or have any questions, please contact your student’s teacher.
The goals of Genetics: Inheritance are twofold: 1) for students to understand how an organism’s traits are determined by proteins, which in turn are determined by DNA, and 2) to show students how traits are passed between generations. This unit builds on student’s prior learning in Genetics: Development. Students will gather evidence to explain how a trait persists in a family.

Why should you do this?
These materials will help you continue your learning at home. The unit addresses content that is not covered in any other high school science course. Goals are listed for each activity to help you track your learning. Your teacher will provide information on which item(s) will be submitted, when they are due, and how they will be submitted.

This unit is designed to address the following Washington State Science Standards (Next Generation Science Standards):

Performance Expectations

LS1-1: Construct an explanation based on evidence for how the structure of DNA determines the structure of proteins which carry out the essential functions of life through systems of specialized cells.

LS1-2: Develop and use a model to illustrate the hierarchical organization of interacting systems that provide specific functions within multicellular organisms.

LS3-1: Ask questions to clarify relationships about the role of DNA and chromosomes in coding the instructions for characteristic traits passed from parents to offspring. [Assessment Boundary: Assessment does not include the phases of meiosis or the biochemical mechanism of specific steps in the process.]

LS3-2: Make and defend a claim based on evidence that inheritable genetic variations may result from (1) new genetic combinations through meiosis, (2) viable errors occurring during replication, and/or (3) mutations caused by environmental factors. [Clarification Statement: Emphasis is on using data to support arguments for the way variation occurs.] [Assessment Boundary: Assessment does not include the phases of meiosis or the biochemical mechanism of specific steps in the process.]

LS3-3: Apply concepts of statistics and probability to explain the variation and distribution of expressed traits in a population.

Science and Engineering Practices: Constructing Explanations, Developing and Using Models

Crosscutting Concepts: Systems and System Models, Structure and Function

What resources do I need?
This packet, a pencil or pen, and scrap paper. You may find it useful to have a highlighter and markers or colored pencils, but this isn’t required. We recommend that you call a friend to talk through the lessons and/or share your learning with someone in your household.

What about online resources?
This packet references several videos and websites that you can access with a phone. If you don’t have internet access on your phone, you may find it helpful to call or text a friend to ask questions. If this is not possible, just skip those suggestions and use the materials in the packet.

What resources do I have to be successful?
If you can access Schoology, your teacher may be providing resources on their class webpage. If not, everything you need is in this packet. You can also ask questions of your teacher by sending them an email or contacting them using their usual procedure.

Timeline:
This packet will take 5-6 weeks to complete. Below we have provided a suggestion on how you might work through the materials. Your teacher may provide a modified version of this schedule on their Schoology page. Please adjust for you / your family.

Unit Driving Question: How does a fatal disease persist in a family?
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<thead>
<tr>
<th>Day</th>
<th>Activities</th>
<th>Extensions (if time allows)</th>
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<tr>
<td>1</td>
<td>1.1 Inheritance Initial Model PowerPoint lesson</td>
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<td>1.1 Inheritance Initial Model Worksheet</td>
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<tr>
<td>2</td>
<td>1.2 Genetics Review PowerPoint lesson</td>
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<td>00 Genetics Vocabulary Student Worksheet</td>
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<td>3</td>
<td>1.2 Genetics – Development Self-Assessment worksheet</td>
<td><strong>Stated Clearly: What is DNA and how does it work?</strong></td>
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<td></td>
<td>Then:</td>
<td><strong>Search for these videos on YouTube</strong></td>
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<td>For extra review, watch the video in the “Extensions” section and</td>
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<td>communicate with your teacher for assistance and/or</td>
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<td>For a challenge, read 1.1 OPTIONAL Adapted Scientific American SCA NCAA</td>
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<td>article</td>
<td><strong>Stated Clearly: What is a gene?</strong></td>
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<td><strong>Stated Clearly: What is an allele?</strong></td>
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<td>2.4 OPTIONAL Student Questions</td>
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<td>2.1 Protein to Trait PowerPoint lesson</td>
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<td>2.1 Modeling Proteins in Cells worksheet</td>
<td>2.1 OPTIONAL Serotonin Practice</td>
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<td>5</td>
<td>2.2 Genetics Vocabulary PowerPoint lesson</td>
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<td>00 Genetics Vocabulary Student Worksheet</td>
<td><strong>Stated Clearly: What is a gene?</strong></td>
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<td>00 Learning Tracking Tool Inheritance</td>
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<td>6</td>
<td>2.2 Practice - Spirit Bears worksheet</td>
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<td>7</td>
<td>2.3 DNA to Protein PowerPoint lesson</td>
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<td>2.3 DNA to Protein Practice worksheet</td>
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<td>9</td>
<td>2.4 Genotype to Phenotype PowerPoint lesson</td>
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<td>00 Genetics Vocabulary Student Worksheet</td>
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<td><strong>Stated Clearly: What is an allele?</strong></td>
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<td>10</td>
<td>Start 2.4 Investigating Genotype to Phenotype – Bioflower Color worksheet</td>
<td>2.4 OPTIONAL Student Questions</td>
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<td>11</td>
<td>Finish 2.4 Investigating Genotype to Phenotype – Bioflower Color worksheet /</td>
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<td>check work using provided key</td>
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<td>00 Learning Tracking Tool Inheritance</td>
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<td>Make an entry in the Discussion provided on your teacher’s Schoology page</td>
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<td>12</td>
<td>2.5 Zooming into Sickle Cell</td>
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<td>13</td>
<td>Catch-up day: Finish anything from above that you haven’t done yet</td>
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<td>14</td>
<td>2.6 Inheritance Model Revisions Tool</td>
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<td>00 Learning Tracking Tool Inheritance</td>
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<tr>
<td>15</td>
<td>3.1 Chromosomes and Alleles PowerPoint lesson</td>
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<td>00 Genetics Vocabulary Student Worksheet</td>
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<td>16</td>
<td>3.1 Modeling Chromosomes with Chirwibbles</td>
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<td>00 Learning Tracking Tool Inheritance</td>
<td>3.1 OPTIONAL Modeling Chromosomes Review Questions with Chirwibbles</td>
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<tr>
<td>17</td>
<td>3.2 Introduction to Meiosis PowerPoint lesson</td>
<td>3.2 OPTIONAL Reproduction and Meiosis Reading</td>
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<td>18</td>
<td>3.3 Meiosis Demo with Chirwibbles PowerPoint lesson</td>
<td>3.3 OPTIONAL Meiosis Demo Analysis Questions</td>
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<td>00 Learning Tracking Tool Inheritance</td>
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<td>19</td>
<td>4.1 Making Gametes</td>
<td>4.1 OPTIONAL Punnett Square Extension - Dihybrid Cross</td>
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<td>4.1 Making Gametes Worksheet</td>
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<td>20</td>
<td>Make an entry in the Discussion provided on your teacher’s Schoology page</td>
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<td>Catch-up: Finish anything from above that you haven’t done yet</td>
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<td>21</td>
<td>4.2 Inheritance Practice PowerPoint lesson</td>
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<tr>
<td></td>
<td>Start 4.2 Puppy Practice Problems worksheet</td>
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<td>22</td>
<td>Finish 4.2 Puppy Practice Problems worksheet</td>
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<td></td>
<td>00 Learning Tracking Tool Inheritance</td>
<td><strong>Challenge problems on 4.2 Puppy Practice</strong></td>
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<tr>
<td>Day</td>
<td>Task 1</td>
<td>Task 2</td>
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| 23  | 5.1 Mutations Reading
    00 Learning Tracking Tool Inheritance | Optional mutations activity:
    5.1 OPTIONAL Mutation Tables
    5.1 OPTIONAL Mutation DNA Sequences
    Stated Clearly: Point Mutations |
| 24  | 5.2 Explaining Sickle Cell Disease PowerPoint lesson
    5.2 Inheritance Model Revisions Tool
    Start 5.2 Inheritance Final Model | |
| 25  | Finish 5.2 Inheritance Final Model | |
| 26  | 6.1 Explaining Other Examples PowerPoint lesson | |
| 27  | 6.2 Genetics - Inheritance Self-Assessment | |
| 28  | Catch-up day: Finish anything from above that you haven’t done yet | |
Genetics Vocabulary

Add in Lesson 1.2:

1. What is DNA? What are chromosomes?

2. Draw and label an unreplicated chromosome and a replicated chromosome.

Add in Lesson 2.2:

3. Label a gene in the picture below. A gene is the instructions for making a _________________.

Add in Gene Regulation Lesson 2.4:

4. What is an allele? Label the alleles in the picture below.
5. What makes a protein **functional vs. non-functional**? Draw an example.

6. Use the picture below to explain the difference between **genotype** and **phenotype**. **Hint:** _______________ determines _______________.

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**Add in Inheritance Lesson 3.1:**

7. What are **homologous chromosomes**? How are they the same and how are they different? Explain using the picture.

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8. Define and give examples of **homozygous vs. heterozygous**. **Hint:** The person with the two chromosomes show in Question 7 is homozygous for gene ___ and heterozygous for gene ___.

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9. Define and give examples of **dominant vs. recessive alleles**.
# Learning Tracking Tool for Genetics - Inheritance:
How does a fatal disease persist in a family?

<table>
<thead>
<tr>
<th>Lesson</th>
<th>What did we do? What did we figure out? Summarize key information and activities with a description and/or picture.</th>
<th>How can our learning be used to explain the phenomenon? Describe what you will add to your explanation of the phenomenon.</th>
<th>Self-Assess: Where am I with my understanding of the phenomenon? (Example: Ready to explain, starting to get it, need more information)</th>
<th>What questions do I have? What additional information do you need to understand the phenomenon?</th>
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DNA to Amino Acid Codon Wheel

Will be used in multiple lessons
How to use this PowerPoint

- **Work at your own pace.** Your health and your family come first.
- If possible, you might find it helpful to go through activities at the same time as a peer. Then you can communicate through text, email, or a call if you have questions or to share ideas.
- You might find it helpful to have a piece of scrap paper and a pencil or pen to record questions or ideas.
- Read through the slides one at a time. Take your time to explore the images and any links.
- If you come across something you don’t understand, make a note of which slide you are on and come back to it after you go through the whole PowerPoint. If you are still confused, feel free to email your teacher with a question. You could also ask someone in your household or reach out to a peer through text, email, or a call.
- When you finish, consider sharing what you learned with someone in your household or a friend through text, email, or a call. Explaining your thinking will help you to retain and make sense of the information.

Goals

After reviewing this PowerPoint, you should be able to:

1) Identify several things that you notice from a video and several questions that you have about Sickle Cell Disease.
2) Develop an initial model for how a fatal disease persists in a family.
3) Add details to your model at the organism, cellular, and atomic-molecular scales.

1.1 Inheritance Initial Model

How does a fatal disease persist in a family?

Genetics: Inheritance Unit
Driving Question

How does a fatal disease persist in a family?
Video: Shaniya’s Story

Make a T-chart to record your notes:

<table>
<thead>
<tr>
<th>What I notice:</th>
<th>What I wonder:</th>
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What did we learn about Sickle Cell Disease?

On the Schoology Discussion Board for Lesson 1.1 create a post to share your learning and ideas:

1. List as many learnings as you can.
2. What questions do you have about sickle cell disease?
3. How do you think this might relate to things you learned previously about Genetics? *(Think back to February!)*

How did Shaniya get Sickle Cell Disease?

- Pedigrees are a tool to track genetic traits through families.
- Sickle Cell Disease is often fatal.

We will use our understanding of scale to help us figure out how Shaniya got Sickle Cell Disease:

The Genetics Three Questions

- **Organism Scale:** What is the observable trait(s)?
- **Cellular Scale:** What is going on inside cells to produce the observable trait?
- **Molecular Scale:** How is DNA involved in the trait(s)?
This brings us back to our driving question: How does a fatal disease persist in a family?

Complete your initial model:
- Use pictures and written explanations to clearly show your ideas
- Answer the Genetics Three Questions for individuals with and without Sickle Cell Disease
- Write the questions you have about parts of the model you are not able to explain yet

Check Your Understanding
1) Identify several things that you notice from a video and several questions that you have about Sickle Cell Disease.
2) Develop an initial model for how a fatal disease persists in a family.
3) Add details to your model at the organism, cellular, and atomic-molecular scales.

What’s Next?
Finish your Initial Model worksheet if you haven’t yet.
Sickle cell disease (SCD) is an inherited blood disorder that can cause pain, anemia, infection, and other serious health problems. Although the exact number of people with SCD is unknown, estimates suggest that approximately 100,000 people in the United States are affected by SCD.

When both parents have sickle cell trait (SCT), there is a 1 in 4 chance that each of their children will have sickle cell disease (SCD). For Fatimah’s mother and father, who both have SCT, their first daughter (Fatimah’s older sister) was born with SCD; 7 years later, Fatimah was also born with SCD. “I don’t think they were expecting both kids to have SCD. I know it’s hard on them because they feel bad that we have the condition,” said Fatimah, a college senior at the University of Illinois, Urbana-Champaign.

Although both Fatimah and her older sister have SCD, the disease (illness) affects them differently. “My sister would get pain crises (acute episodes of pain) every now and then, but it’s really me who has the real issues with SCD. For me, I would be in and out of hospitals all the time. I had really bad pain crises, and I would get really bad headaches and pain in my stomach. I get sick a lot,” said Fatimah.

When Fatimah was around 7 years old, she experienced a terrible pain crisis. Her parents gave her medicine, but she wasn’t responding to the medication. Her pain and health continued to
worsen. They took her to the emergency department (ED) for treatment. While in the ED, her heart rate dropped, and doctors treated her to bring her heart rate back to normal. “When I woke up in the morning, I needed to go to the bathroom. When I got up, the whole right side of my body gave out; I just collapsed on the floor.” At 7 years old, Fatimah had suffered a stroke, and she was in the hospital for several months. “Somehow, by some miracle, I regained everything. Looking at me now, you wouldn’t know that I had a stroke,” said Fatimah.

For Fatimah, frequent visits to the hospital and ED was a part of her childhood. Growing up, she didn’t realize that other people didn’t experience the same health problems she did. “In high school, I learned that everyone doesn’t get sick the way I do.” The hardest part about growing up was coming to terms with the fact that sometimes it was harder for her to engage in certain activities, like cheerleading, compared to her friends. “My parents always told me to take a break [when cheerleading] if I ever got tired, but I would try to push through it unless it got to the point that I really couldn’t anymore. If it got to that point, then I would let my team and coach know.”

Despite the health challenges she faced, Fatimah never wanted her SCD to influence who she was and what she could achieve. “I want to showcase the fact that you can have SCD but can also do everything else. My older sister is studying to be a doctor because of me. She is studying hematology and doing sickle cell research. She told me she met a patient one day in her clinical rotations, and the patient, who also has SCD, was surprised my sister was going to school and becoming a doctor. She told me the patient was asking her, ‘you’re going to school? You’re going to be a doctor? How do you manage (take care of) it?’ The way my sister described it, the girl was so interested in knowing because she puts so much work into her health that she didn’t know it could be possible to also do other things, too. I don’t think that having a medical condition should keep her from doing what she wants. It’s just one obstacle you have to face, but you don’t have to stop everything you’re doing,” said Fatimah.

When Fatimah was in her junior year of college, she decided to study abroad in Milan, Italy, despite her parents’ concerns. She was interested in fashion and modeling, and she wanted to take the opportunity to explore Milan, a fashion capital, and absorb the rich culture the city had to offer. “My parents were worried about finding a doctor in Italy and how I was going to stay healthy, but I could figure that stuff out. Why miss out on an opportunity to live in another country?” Once in Italy, Fatimah was able to find a doctor, but she admits it was challenging to receive care away from home. “It was the first time I went to a doctor by myself, and it was in a different country, too. They didn’t speak English. I was scared. I was comparing myself, thinking about how my friends don’t have to worry about finding a doctor and going to a hospital. I had to be up at 4am to be on the train and be at the hospital for my appointment.” Despite the challenges she faced abroad, Fatimah doesn’t regret her decision to spend a semester in Italy. She kept her spirits up by putting her healthcare needs into perspective. “I have to do these extra things to stay healthy, but at the same time, it’s that
little sacrifice. It’s just one day out of the month, and I don’t have to let it impact the other
days.”

Fatimah is expected to graduate as a finance major in May 2019. She has already accepted a
full-time job at JP Morgan Chase, which she will begin after graduation, but she plans to
continue pursuing her dreams in travel, blogging, and fashion as well. Fatimah wants to
encourage other people like her who are living with SCD to know that they can live a full life.
“We can still achieve whatever we want to do, whatever we put our minds to. Yeah, you have
to deal with something that not everybody has to, but at the end of the day, you’re still here,
and you’re still healthy for the most part. So if you’re able to go through your day, then know
that it’s one thing you have to deal with, but don’t let it be everything and the deciding factor
for you.”

CDC would like to thank Fatimah for sharing her story.

Mimi’s Story

“When I’m in that moment of pain in the emergency room, I can’t even think for myself. The
pain is debilitating and they’re asking me questions like, ‘Are you sure you have sickle cell?
We need to look into this,’” says Mimi, a 37-year-old lawyer and mom of four.
Mimi’s medical history is similar to that of many individuals with sickle cell disease (SCD): she
has experienced severe pain since a young age, frequently sought care in the emergency
department (ED), and has received numerous blood transfusions (when healthy blood is given to a patient
through one of their blood vessels). But Mimi’s
 genetic (inherited) blood disorder comes as a surprise
to many healthcare providers when they first meet
her because of one detail: she’s not African American.

A commonly held myth about SCD is that it only
affects individuals of African descent. **Although SCD
is most common among African Americans in the
United States, it can also affect Hispanics, and
people whose ancestors come from countries in
South Asia (such as India), southern Europe (such
as Greece and Italy), and the Middle East (such as
Saudi Arabia and Lebanon).**

SCD is a disease one is born with and is now a part of
the newborn screening program for all states. But
because Mimi was born before this program started
in her state, she was not diagnosed until she was 5.
Because of Mimi’s Arab American background, a sickle cell diagnosis did not occur to many of her healthcare providers. “I was about 3 years old when I started presenting with pain in my wrists and ankles. My parents would take me to the emergency room and the doctors would take an X-ray. They wouldn’t be able to find anything; they would just send me back home. My parents had psychologists telling them I was faking the pain,” recalls Mimi. It wasn’t until one particular doctor requested a blood test that her family discovered she has SCD.

Throughout her life, healthcare providers have doubted Mimi when she told them she had SCD. This has caused delays in Mimi getting the treatment she needs. “I’ll have to wait for the blood work to come back for them to help me with anything. I’ll have to wait several hours in the emergency room for any sort of pain relief,” says Mimi.

Like many individuals with SCD, Mimi’s pain has been dismissed in the ED several times. “This has happened as recently as the last 2 years, where they’re extremely dismissive. And I have to explain to them step by step what’s going on, what I need. I just need some pain relief. They may give me a little bit, but they send me home,” she says.

What is going through Mimi’s mind during these frustrating experiences? “It’s just the pain. Like please make it stop. There is nothing else you can think about except, please make it stop, please hurry. I’ve been in tears; I would be crying and waiting. I remember a nurse many, many years ago who told me, ‘Relax, it’s not that much of a big deal.’ That is extremely frustrating,” says Mimi.

Although Mimi has had negative experiences seeking health care for her SCD, she’s also had some great experiences. “I’ve had some amazing providers, some absolutely wonderful doctors and nurses who know my history. My hematologist (a blood disorder specialist) here in Atlanta told me, ‘You just come in and we will see you immediately.’” During her time living in Sydney, Australia, Mimi had a hematologist who was always on standby along with his team ready to take her in during an SCD-related health issue.

While Mimi has been through many challenges as someone with SCD, she says her condition has only encouraged her to be “grateful for every minute.” Growing up, she loved to be active in extracurricular activities at school including ballet, jazz, and swimming, but would often have to miss practices and performances when she ended up in the hospital for pain or other SCD-related health issues. “I hated being in the hospital as a child. I’d be so grateful when I came out. Just be thankful for every moment that you have.”

Mimi credits her motivation in life and her career to having missed out on so much as a child because of her condition. Originally from Atlanta, Georgia, Mimi moved to Sydney, Australia for school because she felt it would be a fun experience. She ended up starting a family there, which includes her four children, and living in Sydney for 18 years.
These days, Mimi is back in Atlanta with her family and getting ready to take the bar exam (a test every lawyer has to pass before becoming licensed to practice law) in February 2020. After passing the bar, she looks forward to practicing family law. “People always say to me, ‘Just relax, take it easy.’ I can’t, I just can’t. I have to follow the next thing,” Mimi says with a laugh.

What is Mimi’s advice for others with SCD?

- **Develop a relationship with your doctor.** “If you don’t have a great relationship with your doctor, move on to someone with whom you can because they will be the person who really has their eye out for you.”
- **Build a strong support system.** “Make sure you have parents, friends, family members, a spouse, whoever. I wasn’t too upset that I didn’t have a community support system as a child because I had a phenomenal one at home.”
- **Ask for help.** “Pick up the phone and say, ‘Please help me out.’ Don’t be afraid to ask for help right away when you need it. Don’t be afraid to tell people exactly how bad it is.”

Mimi hopes for better knowledge of and care for people with SCD. “Rapid intervention makes all the difference. Knowing that just because I look a certain way doesn’t mean that I’m not in the same amount of pain. I would like to see change from the bottom up; I’ve had issues all along at all levels,” she says.

Mimi’s story shows how important it is for healthcare providers, community workers, policy makers, and other supporters of SCD to know that SCD affects diverse groups so that all people with SCD can receive timely treatment for SCD.

**Geno Atkins’ Story**

“My story started when a young man met a young lady on the campus of Florida A&M University. On their first date he asked the young woman if she carried the sickle cell trait! That young man became my dad and the young lady is my mother. My dad carries the sickle cell trait and was well aware that if he married someone who also carries the trait, their kids had a 50% chance of being born with full blown sickle cell disease. He discovered that my mother is not a carrier of the sickle cell trait. The rest is history.

“I am the oldest of three children and the only one with sickle cell trait. The first time I learned I carry the sickle cell trait was as a freshman at the University of Georgia. I called home and my mother said, “Your dad has the trait, but I don’t recall the doctor saying you had the trait when you were born.” All newborns are tested for the trait in Florida, yet I had gone my whole life without knowing.

“Once I learned I had the trait I researched as much as I could and talked with the football training staff. They assured me that the trait would not affect my ability to play. There were
four freshmen who tested positive for the trait along with me and we were assigned a trainer who watched us closely during practice sessions and on game day. I was not treated differently by my teammates and went about my life just as I had before. One day I learned that a football player had died from complications of sickle cell trait while participating in spring practice at another university. That’s when I realized that this is a serious issue and I should not take any chances with my health. I played at the highest level in college and it earned me a spot in the NFL.

“I knew from my research that it would not be good for me to play in high altitude, so I prayed I wouldn’t get drafted by Denver, which is at a high altitude. I ended up in Cincinnati and have played at a very high level without any adverse affects of the sickle cell trait. During the 2011 season we did travel to Denver to play the Broncos and that was the first time I can truly say I felt the effects of the trait. I could not breathe after a 10-play series and had to be given oxygen on the sideline.

“Some of the changes I’ve made in my life include eating healthy, avoiding drugs and alcohol, not smoking, and most importantly getting a lot of rest. Everyone in my family knows that I have to take my daily nap. I drink more water, sports drinks, and coconut water than ever before because it is important to stay well hydrated before and after activities.

“Having the sickle cell trait does not exclude an athlete from participating in sports, however, the training staff and coaches need to take precautions to ensure the athlete is not put in dangerous situations. In high school my coaches would get on me because I was always in the back during running drills and I often got very tired. I think back now and realize that it could have been a dangerous situation for me if over-zealous coaches or I had pushed too much during those hot days in south Florida.

“Each year I am saddened to learn of another young athlete dying from complications of the trait while participating in sports. This vicious cycle lets me know that not enough information, education, and spotlight are given to this issue. My goal is to start a foundation, with the primary focus on offering testing for athletes at the high school level and getting education for trainers and coaches on how to help the athletes be the best they can be.”

CDC would like to thank Geno Atkins for sharing his personal story.
How does a fatal disease persist in a family? Use pictures and words to explain.

- Use the zoom-ins to show what you think is happening at the cellular and atomic-molecular scales
- Explain how information and instructions are passed between generations
- Show how DNA and proteins are involved in the trait(s)
Complete the table below to answer the Genetics Three Questions for individuals with different traits:

<table>
<thead>
<tr>
<th>Individual Without Sickle Cell Disease</th>
<th>Individual with Sickle Cell Disease (⭐)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Organism Scale Question:</strong></td>
<td><strong>Organism Scale Question:</strong></td>
</tr>
<tr>
<td>1. What is the observable trait?</td>
<td>1. What is the observable trait?</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cellular Scale Question:</strong></td>
<td><strong>Cellular Scale Question:</strong></td>
</tr>
<tr>
<td>1. What is going on inside the cell to produce the observable trait?</td>
<td>1. What is going on inside the cell to produce the observable trait?</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Molecular Scale Question:</strong></td>
<td><strong>Molecular Scale Question:</strong></td>
</tr>
<tr>
<td>1. Where is DNA coming from?</td>
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</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>2. How is DNA involved in the trait?</td>
<td>2. How is DNA involved in the trait?</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Question Box:** What are you still puzzled about? What else do you need to know?

1. 
2. 
3. 
How to use this PowerPoint

- Work at your own pace. Your health and your family come first.
- If possible, you might find it helpful to go through activities at the same time as a peer. Then you can communicate through text, email, or a call if you have questions or to share ideas.
- You might find it helpful to have a piece of scrap paper and a pencil or pen to record questions or ideas.
- Read through the slides one at a time. Take your time to explore the images and any links.
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- When you finish, consider sharing what you learned with someone in your household or a friend through text, email, or a call. Explaining your thinking will help you to retain and make sense of the information.

1.2 Genetics Review

How can we apply our understanding of genetics to figure out how a fatal disease persists in a family?

Goals

After reviewing this PowerPoint, you should be able to answer these three questions:

1) Why and how do cells divide?
2) What is DNA and how is it involved in cell division?
3) What makes the different types of cells in your body unique?

If you have it, get out your Genetics: Development Learning Tracking Tool. This is a great way to review!
Question 1: Why and how do cells divide?

Acronym: G-R-R
• Growth of the organism
• Replacement of old cells
• Repair of damaged tissues

Remember the Development Unit?
• Planaria Lab
• Model of a single cell forming a complex, multicellular organism

The Cell Cycle and Mitosis

Cell Cycle – the sequence of events that cells perform in order to divide (from the end of one mitosis to the end of the next)

G = Growth phase
S = DNA replication
M = Mitosis

Mitosis
= the process by which the nucleus and chromosomes are distributed evenly into two daughter nuclei

Animation here or here

http://www.sumanasinc.com/webcontent/animations/content/mitosis.html
https://www.youtube.com/watch?v=DwAFZb8juMQ
The cell nucleus stores DNA and is surrounded by the nuclear membrane (envelope). The nuclear envelope is a phospholipid bilayer with protein channels – just like the cell membrane!

Mitochondria

Chromosomes can get packed together to take up less space. Chromosome = single molecule of DNA.

DNA = Deoxyribonucleic Acid

DNA chromosomes have two strands. (DNA is double-stranded)

WHY?

DNA is made of monomers called nucleotides.

Pairs to

Adenine (A) Guanine (G) Thymine (T) Cytosine (C)
Nucleotides always pair specifically = complementary base pairing

- $A$ - $T$
- $C$ - $G$

Identify the complementary DNA sequence (write it below the sequence given)
- ATCG
- TCGGCTA
- GCCTTT
- TCGAT

Recall mitosis

Step 1
Step 2

How did the DNA go from an unreplicated chromosome to a replicated chromosome?
DNA Replication

Check Your Understanding:
- What are unreplicated and replicated chromosomes?
- When is the DNA copied?

DNA synthesis

chromosome
replicated chromosome (sister chromatids)
daughter chromosomes

Question 3:
What makes the different types of cells in your body unique?

- Muscle cells and neurons in your body have the same DNA, but they express (use) different genes.
- Gene expression causes cells to look unique and to perform different functions.

Different Cell Type Use Different Instructions
A gene is a section of DNA that has instructions for making specific proteins. If you imagine that DNA is like a library full of cookbooks, then a chromosome is a specific cookbook, and a gene is one recipe in a book.

Nearly all of the cells in an organism contain the same DNA, but the cells do not all look alike nor perform the same function in that organism. This is because different cells produce different proteins.
Remember the cell differentiation activity?

• Every cell in your body has the same DNA instruction manual (the same chromosomes).
• Genes are sections of DNA located on chromosomes, like the bands shown to the right.
• Different genes are turned on or off in different cell types.

When do cells differentiate?

• During development and growth
  • As a single cell divides and forms many different types of cells

• Over your life as cells are replaced
  Examples:
  • Blood cells are replenished
  • Intestinal cells are shed and replaced

You explained planaria regeneration

• If you have your Planaria Explanation Tool, please review it.
• Could you still explain this process? Try talking it through with someone. Call a classmate or talk to someone in your household.
Check Your Understanding

1) Why and how do cells divide?
2) What is DNA and how is it involved in cell division?
3) What makes the different types of cells in your body unique?
4) How does this information help us understand the persistence of a fatal disease in a family?

What’s Next?
1) Add notes to the first section of your Genetics Vocabulary worksheet.
2) Complete the “1.2 Genetics – Development Self-Assessment.”
3) For extra review, watch the video in the “Extensions” section and communicate with your teacher for assistance and/or for a challenge, read 1.1 OPTIONAL Adapted Scientific American SCA NCAA article
Genetics: Development Self-Assessment

Reflect on the standards addressed in the Genetics: Development unit. Check which box describes your current understanding:

4  I know this well enough to teach it to someone.
3  I can do this with almost no mistakes.
2  I can do much of this, but I have questions.
1  I can do this, but only with help.
0  I can’t do this, even with help.

<table>
<thead>
<tr>
<th>Development Unpacked Standards</th>
<th>Rating (0-4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I can describe how a complex organism is organized by:</td>
<td></td>
</tr>
<tr>
<td>the same type of cells that make tissues (differentiated cells), tissues that make organs, and organs that comprise parts of an organ system.</td>
<td></td>
</tr>
<tr>
<td>I can describe how organ systems work together to maintain a complex organism.</td>
<td></td>
</tr>
<tr>
<td>I can describe how systems of specialized cells within organisms help them live.</td>
<td></td>
</tr>
<tr>
<td>I can explain how the DNA in a cell contains a complete set of instructions for making the proteins needed by the organism.</td>
<td></td>
</tr>
<tr>
<td>I can explain that genes are regions on DNA that code for proteins.</td>
<td></td>
</tr>
<tr>
<td>I can explain how proteins carry out essential functions of life.</td>
<td></td>
</tr>
<tr>
<td>I can describe chromosomes as long, condensed strands of DNA that contain many genes.</td>
<td></td>
</tr>
<tr>
<td>I can describe how in mitosis, a cell divides to form 2 new cells that have identical DNA. Their DNA is also identical to that of the original cell.</td>
<td></td>
</tr>
<tr>
<td>I can explain that for a complex organism to maintain itself, its cells must continually undergo mitosis so that the organism can grow and replace dead cells.</td>
<td></td>
</tr>
<tr>
<td>I can explain how cells are different based on which genes are expressed in the cell.</td>
<td></td>
</tr>
<tr>
<td>I can explain that during development, different genes are regulated differently in different cells.</td>
<td></td>
</tr>
<tr>
<td>I can explain that when genes are turned on, that protein is made. When genes are turned off, that protein is not made.</td>
<td></td>
</tr>
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Notes / Comments:
Exercising Caution: Intensive Athletic Activity Could Be Fatal to Those with Sickle-Cell Trait (adapted)
By Rose Eveleth

At 5:30 A.M. on February 19, 2010, 20-year-old Bennie F. Abram started his first day of football practice as a junior at the University of Mississippi. Several hours later he collapsed and died. Last month Abram’s family settled their wrongful-death lawsuit with Ole Miss, receiving $275,000 from the National Collegiate Athletic Association (NCAA) and $50,000 from the university.

Later in 2010, 20-year-old Jospin Milandu and 15-year-old Oliver Louis also died unexpectedly while working out with their teams. Autopsies of all three showed one striking similarity: long, sickle-shaped blood cells that indicated they had at least one non-functional version of the gene that leads to sickle-cell anemia (SCA). SCA is a genetic condition that primarily affects some people of African descent but also can be found in Caucasians, Hispanics and Mediterranean populations.

Sickle-cell anemia and a related condition called sickle-cell trait follow classic patterns of genetic inheritance. People who receive two non-functional copies of the gene from their parents have sickle-cell anemia, a condition in which their red blood cells are always sickle-shaped. Those who inherit just one non-functional copy of the gene are said to have sickle-cell trait. Their blood cells look normal and they experience no side effects—most of the time. But there are a handful of situations that puts those with sickle-cell trait in danger—circumstances when their body is exposed to low amounts of oxygen, such as climbing a mountain or intense exercise.

In these low-oxygen conditions something changes and those with sickle cell trait become more likely to die. Researchers do not know exactly what causes death under these conditions, although they have some ideas. Possibilities include the breakdown of skeletal muscle, heart failure, and/or kidney failure. Football makes sickle-cell trait particularly risky for a number of reasons: practice tends to start during some of the hottest months of the year and players often practice in pads, which can add to the heat stress.

Three years ago in an attempt to avoid more deaths like Abram’s, the NCAA adopted a policy that requires mandatory testing of all Division I athletes for the sickle-cell trait. But the move was controversial. Simply testing everyone for the trait won’t necessarily save players, and most athletes with sickle-cell trait will never need saving. Understanding how to protect against the occasional side effects of sickle cell trait is complicated by the fact that it can be hard to know for sure whether the non-functional gene is the source of a particular problem in the first place.

Another complicating factor is that not all athletes with sickle-cell trait face the same degree of risk. Between 2004 and 2008 there were 273 athlete deaths in the NCAA, five of which occurred among players with sickle-cell trait. Researchers found that football could be especially dangerous for players with the trait. The data showed that an NCAA Division I football player with sickle-cell trait was 37 times more likely to die during practice than a player without it. In fact, all sickle-cell trait deaths recorded in the past 20 years in the NCAA have been in men, and all but one played football, says Kim Harmon, a professor of sports medicine at the University of Washington.

Part of the reasoning behind the NCAA policy to test athletes was the idea that if coaches knew which of their players had sickle-cell trait, they could watch them for signs of an episode, and listen to them when they asked

Adapted from Scientific American, published August 13, 2013
for a break. But some argue that rather than singling out sickle-cell trait players, universal changes should be made to reduce the risk of heat stroke and exhaustion for all players. Others worry that coaches might bench sickle-cell trait players more or pull them out of practices, putting them at a disadvantage.

This fear isn’t totally unreasonable. When De Vaughan Darling died from a sickle-cell trait episode during practice at The Florida State University, his identical twin Devard was barred from playing by the team’s medical staff. And players themselves are worried about their NFL draft prospects. In the end, De Vaughan Darling’s twin Devard went on to play for the NFL without incident. And although no one knows the exact number of players in the NFL with sickle-cell trait, estimates put the number at around 90 out of over 1,600 players, including the New York Jets wide receiver Santonio Holmes, Cincinnati Bengals lineman Geno Atkins and six-time Pro Bowl receiver Terrell Owens. None of them have ever had trouble. Ryan Clarke, a safety for the Pittsburgh Steelers, nearly died during a sickle-cell trait episode in Denver in 2007, however. He still plays but sits out when his team goes back to the mile-high city, and hasn’t had an incident since.

Like many genetic tests the question of sickle-cell trait comes down to whether knowing a person’s health status will actually help save them. Do doctors and coaches know enough about the risks of sickle-cell trait to make testing an effective prevention approach? Do scientists know enough about sickle cell trait to recommend a prevention strategy? So far, three years into the NCAA testing program no one has died from a sickle-cell trait episode.

In July, after Abram’s family settled with the University of Mississippi, the NCAA agreed to add more detailed information about sickle-cell trait in their 2012–2013 NCAA Sports Medicine Handbook. With more information available, coaches should be better prepared to keep their sickle-cell trait players safe—and with more research, scientists hope to better understand exactly what creates the risk these players face. Because whereas coaches certainly want to win, Harmon says, “they do not want to kill these kids.”

Answer the following questions with complete answers (2-4 sentences). Explain each answer with a thoughtful response.

1. What is the genotype of the athletes in the article?
2. How is it possible that some players with the trait, like Terrell Owens, “never [have] trouble”?
3. How does “climbing a mountain or intense exercise” cause symptoms in heterozygous individuals?
4. Should athletes be tested for Sickle Cell Trait?
   a. Would you want to be tested? Why?
   b. What would be the issue with forced testing? Why is it controversial? Who is most impacted by this policy?

Adapted from Scientific American, published August 13, 2013
How to use this PowerPoint

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Goals

After reviewing this PowerPoint, you should be able to:
1) Describe the role of proteins in determining traits.
2) Explain how functional and nonfunctional versions of proteins will result in different traits.
3) Model the function of proteins in cells and predict the traits produced by those cells.

2.1 Protein to Trait

What determines an organism’s traits?

Traits

• We all have different traits. Think eye color, hair color, and height.
• Traits have different versions
  — Example: Different colors of flowers (red, white, pink)
  — Example: Having Sickle Cell Disease or not
• Proteins make things happen to determine our traits

protein ➔ traits
Role of Proteins

Proteins can ...
– Transport substances (ex: hemoglobin)
– Allow substances in and out of a cell (ex: channels)
– Signal (ex: hormones)
– Receive those signals (ex: receptors)
– Build parts of our cells and bodies (ex: collagen)
– Break down or change other molecules (ex: enzymes)

Protein Example: Enzymes

• Enzymes are proteins that control the rate of chemical reactions
• The shape of the enzyme allows molecules to bind (fit) allowing a chemical change to happen

Example of protein (enzyme) influencing a trait:
Human Skin Color – No Albinism

• When the protein is a functional shape, we see a particular trait
  – The skin has pigment
Example of protein (enzyme) influencing a trait: Human Skin Color – **Albinism**

- When the protein is a non-functional shape, we see a different trait
  - The skin does **not** have pigment

Where is this happening?

- Biosynthesis occurs inside of cells
  - *Remember 1st semester?*
- Therefore, proteins live and work inside of cells

What is different about the two proteins?

What do you think might cause this difference?
Let’s practice:
Modeling Proteins in Cells Worksheet

Proteins on your worksheet:
• A pigment enzyme from imaginary bioflowers
• The receptor protein that can detect PTC
• Collagen, a structural protein, necessary for “normal” movement and coordination in C. elegans
• MAO enzyme that converts serotonin into another signaling molecule

Complete the worksheet – draw in the proteins where necessary and explain the relationship between the observed traits and proteins present.

Check Your Understanding

1) Describe the role of proteins in determining traits.
2) Explain how functional and nonfunctional versions of proteins will result in different traits.
3) Model the function of proteins in cells and predict the traits produced by those cells. (Complete the Modeling Proteins in Cells Worksheet.)

What’s Next?
1) Check your work on the Modeling Proteins in Cells Worksheet using the provided key.
2) Consider completing “2.1 OPTIONAL Serotonin Practice.”
Modeling Proteins in Cells – A Molecular Model

Scientists frequently use models to help understand complex processes. Here, we will use a model to help understand the traits of an organisms. As we continue to learn about traits, this model will help you understand how genetic information flows in organisms and between generations.

**Trait and associated proteins:** Pigment enzyme Q converts colorless pigment to red pigment in bioflowers.

<table>
<thead>
<tr>
<th>Proteins in the Cell</th>
<th>Molecular Model</th>
<th>Trait</th>
</tr>
</thead>
<tbody>
<tr>
<td>Draw a cell with ALL <strong>functional</strong> pigment enzyme proteins.</td>
<td><img src="image" alt="Molecular Model" /></td>
<td>What trait would a bioflower with these proteins have?</td>
</tr>
<tr>
<td>Draw a cell with ALL <strong>nonfunctional</strong> pigment enzyme proteins.</td>
<td><img src="image" alt="Molecular Model" /></td>
<td>What trait would a bioflower with these proteins have?</td>
</tr>
</tbody>
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**Trait and associated proteins:** The TSR cell membrane receptor sticks to bitter molecules and signals bitter taste. This protein is present on the cell surface, inserted into the cell membrane.

<table>
<thead>
<tr>
<th>Proteins in the Cell</th>
<th>Molecular Model</th>
<th>Trait</th>
</tr>
</thead>
<tbody>
<tr>
<td>Draw a cell with ALL <strong>functional</strong> TSR receptor proteins.</td>
<td><img src="image" alt="Molecular Model" /></td>
<td>What trait would an individual with these proteins have?</td>
</tr>
<tr>
<td>Draw a cell with ALL <strong>nonfunctional</strong> TSR receptor proteins.</td>
<td><img src="image" alt="Molecular Model" /></td>
<td>What trait would an individual with these proteins have?</td>
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</table>
**Trait and associated proteins:** The structural protein collagen allows worms to move smoothly when the protein is functional.

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<tbody>
<tr>
<td>What proteins would be present in the cells of a <em>C. elegans</em> with this trait?</td>
<td><img src="image1" alt="Molecular Model" /></td>
<td><em>C. elegans</em> worm has normal movement.</td>
</tr>
<tr>
<td>What proteins would be present in the cells of a <em>C. elegans</em> with this trait?</td>
<td><img src="image2" alt="Molecular Model" /></td>
<td><em>C. elegans</em> worm must roll to move.</td>
</tr>
</tbody>
</table>

**Trait and associated proteins:** The MAO enzyme changes serotonin into another molecule. Low amounts of serotonin are one contributor to depression.

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<th>Proteins in the Cell</th>
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<tbody>
<tr>
<td>Draw a cell with ALL partially functional MAO enzyme proteins.</td>
<td><img src="image3" alt="Molecular Model" /></td>
<td>What trait would an individual with these proteins have?</td>
</tr>
<tr>
<td>What proteins would be present in the cells of an individual with this trait?</td>
<td><img src="image4" alt="Molecular Model" /></td>
<td>An individual struggles with depression.</td>
</tr>
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**Challenge Question:** What do you think would happen if a cell made BOTH functional and non-functional proteins?
Modeling Proteins in Cells – A Molecular Model

Scientists frequently use models to help understand complex processes. Here, we will use a model to help understand the traits of an organisms. As we continue to learn about traits, this model will help you understand how genetic information flows in organisms and between generations.

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<td>Draw a cell with ALL nonfunctional pigment enzyme proteins.</td>
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<th>Molecular Model</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Draw a cell with <strong>fully functional</strong> MAO enzyme proteins.</td>
<td><img src="image" alt="Diagram" /></td>
<td>What trait would an individual with these proteins have? Higher serotonin levels. Less likely to have depression.</td>
</tr>
<tr>
<td>What proteins would be present in the cells of an individual with this trait?</td>
<td><img src="image" alt="Diagram" /></td>
<td>An individual struggles with depression.</td>
</tr>
</tbody>
</table>
Mental Health is a Trait

How can proteins determine how we feel? What is the protein to trait relationship?

2. Class Practice:
Serotonin

- Serotonin is a molecule called a neurotransmitter used by cells in the brain to communicate
- Part of its function is to regulate emotions
- Abnormally low amounts of serotonin in the brain can cause depression

How is serotonin produced, and how can we use our understanding of it to treat depression?

Some enzymes involved in serotonin synthesis

Your proteins determine your characteristics...
Your proteins determine your characteristics...

Low amounts of AAAD – more or less serotonin?

Low amounts of MAO – more or less serotonin?

High amounts of MAO – more or less serotonin?

So do you want lots of MAO or a little bit of MAO?

Antidepressants

- One type of drug to treat depression works by decreasing the activity of the MAO enzyme. These are called MAO inhibitors or MAOIs.

- The drug molecules have a shape that fits into the active site of the MAO enzyme and blocks it.
How to use this PowerPoint

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2.2 Genetics Vocabulary

Goals
After reviewing this PowerPoint, you should be able to:
1) Define “gene.”
2) Describe how different versions of a gene will result in different proteins.
3) Describe how different proteins will result in different traits.

Get out your vocabulary sheet and make notes on the next section:
What are genes?

- Regions of chromosomes called **genes** are instructions for building proteins
- Traits result from which proteins are made

Human chromosome # 7

- Contains about 1,800 genes
- Contains over 150 million nucleotides

A **gene** is a section of a chromosome that tells an organism how to make a specific protein

Practice: Spirit Bears

Use the provided worksheet or answer on a separate sheet of paper.

A population of bears in British Columbia is famous for their white fur— they are called ghost bears, or spirit bears. Genetically, the bears are identical to black bears. Draw a Molecular Model that shows a skin cell of a ghost (white) bear and the skin cell of a black bear.

Label the following parts of your modeling: cell, cell membrane, nucleus, protein(s), enzyme(s).

Write an explanation for the relationship between the trait of each bear and the activity of proteins. Include an explanation of the relationship between the structure and function of any associated proteins. (**at least three sentence explanation**)

5 6 7 8
Check Your Understanding

1) Define “gene.”
2) Describe how different versions of a gene will result in different proteins.
3) Describe how different proteins will result in different traits.

What’s Next?
1. Make an entry in your Learning Tracking Tool titled “2.2 Protein to Trait” that includes your learning from the last two lessons.
2. Check your work on the “Practice – Spirit Bears” worksheet using the key on the next slide.

Answer Key - Spirit Bears

The two bears have different alleles (versions) of the gene that produces the pigment enzyme. The nucleotide sequence of each allele is different, leading to the protein produced to be different. In spirit bears, the pigment enzyme protein does not have an active site to convert colorless (white) pigment into the dark brown/black pigment so the traits of the bears are different.
**Practice: Spirit Bears**

A population of bears in British Columbia is famous for their white fur— they are called ghost bears, or spirit bears. Genetically, spirit bears are identical to black bears.

1. Draw a Molecular Model that shows a skin cell of a spirit (white) bear and the skin cell of a black bear.

2. **Label** the following parts of your modeling: cell, cell membrane, nucleus, protein(s), enzyme(s).

3. Write an explanation for the relationship between the trait of each bear and the activity of proteins. Include an explanation of the relationship between the structure and function of any associated proteins. *(at least three sentence explanation)*

<table>
<thead>
<tr>
<th><strong>Black Bear</strong></th>
<th><strong>Spirit (white) Bear</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular Model:</td>
<td>Molecular Model:</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Explanation:</th>
<th>Explanation:</th>
</tr>
</thead>
</table>
Modeling Protein Activity Exit Ticket

A population of bears in British Columbia is famous for their white fur—they are called ghost bears, or spirit bears. Genetically, spirit bears are identical to black bears.

1. Draw a Molecular Model that shows a skin cell of a spirit (white) bear and the skin cell of a black bear.

2. Label the following parts of your modeling: cell, cell membrane, nucleus, protein(s), enzyme(s).

3. Write an explanation for the relationship between the trait of each bear and the activity of proteins. Include an explanation of the relationship between the structure and function of any associated proteins. *(at least three sentence explanation)*

<table>
<thead>
<tr>
<th>Black Bear</th>
<th>Spirit (white) Bear</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Molecular Model:</strong></td>
<td><strong>Molecular Model:</strong></td>
</tr>
<tr>
<td>cell</td>
<td>non-functional enzyme</td>
</tr>
<tr>
<td>gene A</td>
<td></td>
</tr>
<tr>
<td>colorless molecules</td>
<td></td>
</tr>
<tr>
<td>pigment molecules</td>
<td></td>
</tr>
<tr>
<td>enzyme (functional)</td>
<td></td>
</tr>
<tr>
<td>nucleus</td>
<td></td>
</tr>
<tr>
<td>cell membrane</td>
<td></td>
</tr>
</tbody>
</table>

**Explanation:**
Black-bears have allele A1 for gene A (or a version of the gene that codes for functional enzyme A). The enzyme produced is functional and converts a colorless molecule into a pigment molecule. That pigment molecule produces the trait of black fur.

**Explanation:**
- Different version of gene A
- Non-functional enzyme
- Colorless molecule is not changed/no pigment made
- Lack of pigment molecules produces the appearance of white fur.
How to use this PowerPoint

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2.3 DNA to Protein

So how are instructions in DNA used to build proteins?

Goals

After reviewing this PowerPoint, you should be able to:

1) Explain how DNA is used to produce a protein and how proteins determine the traits we see.
2) Explain how codons in the DNA code for amino acids that make up a protein.
3) Describe how a change in a nucleotide base in the DNA could change the protein produced.

How the DNA information is used to produce an organism
Watch this demo:
https://www.youtube.com/watch?v=0o7pIbnb9wo&feature=youtu.be

Reviewing Bead Proteins

- **Where** are the instructions for making proteins?
- **What** are the instructions for making protein?
- **What** did the list of colors represent?
- **Where** are proteins made?
- **What** do the beads represent?

**Information flow in cells**

DNA → protein → traits

Instructions → Beads on a string → traits

**How does DNA code for amino acids?**

Start at the inside of the wheel and work your way towards the outside to find the amino acid coded for by the DNA codon.

Example:
ATG is the start codon that codes for the methionine amino acid. This is the first amino acid in every protein.
Codon → Amino Acids

- Each codon has the information for a specific amino acid
- Some code for the same amino acid
  **the sequence of nucleotides = sequence of codons = sequence of amino acids**

### Let’s Practice!

What amino acid sequence will this code for?

ATGCGATACGCAAAAGTAG

Protein:

Met-Arg-Tyr-Ala-Lys-STOP

### Let’s Practice!

What happens if we make this change?

What amino acid sequence will this code for?

ATGCGATACTCAAAGTAG

Protein:

Met-Arg-Ty...
What happens if we make this change?

What amino acid sequence will this code for?

ATGCgATACTCAAGTAG

Protein:
Met-Arg-Tyr-Ser-Lys-STOP

How does this affect protein shape?

Protein:
Met-Arg-Tyr-Ala-Lys-STOP

Protein:
Met-Arg-Tyr-Ser-Lys-STOP

Check Your Understanding
1) Explain how DNA is used to produce a protein and how proteins determine the traits we see.
2) Explain how codons in the DNA code for amino acids that make up a protein.
3) Describe how a change in a nucleotide base in the DNA could change the protein produced.

What’s Next?
1) Complete the DNA to Protein Exit Ticket.
2) Make an entry in your Learning Tracking Tool titled, “2.3 DNA to Protein.”
DNA to Protein

In Biology, information in a cell flows from **DNA to Protein**, and proteins are responsible for the traits of an organism.

**Proteins have Many Roles**
All living organisms contain hundreds to tens of thousands of different kinds of proteins. Each different protein has a unique, three-dimensional structure that corresponds to a specific function. Proteins are responsible for most of the day-to-day functioning of organisms.

**Structure = Function**
A **protein** is a polymer built from **amino acids**. There are 20 different amino acids in cells that arrange in various combinations to form thousands of unique proteins. Think of how you can form thousands of different English words by using different combinations of the 26 letters in the alphabet. Though the amino acid alphabet is slightly smaller, the protein “words” are much longer. Most proteins are at least 100 amino acids in length, with many reaching into the 1,000s and some into the 10,000s. Just as each word in the English language is formed from a unique sequence of letters, each protein has a unique sequence of amino acids.

While the structure of a protein is based on the sequence of amino acids it is made of, the amino acids don’t remain in a simple straight line. The chain folds into a particular shape based on the sequence of amino acids. Each amino acid has its own chemical properties that determines how they interact with each other and the environment inside the cell. Accordingly, each protein is an amino acid chain that has precisely twisted, folded, and coiled into a unique shape, and the overall shape of the protein molecule determines its function. For proteins to function properly, they must retain their three-dimensional shape. When the structure of a protein (its shape) is changed, it usually loses its function.

**Proteins and the Genetic Code**
The proteins that make the unique **traits** (characteristics) of each individual are coded for by the unique DNA sequence of the individual. DNA is the genetic information passed from parent to offspring. Proteins are built by cells based on sequences of DNA called **genes**. In general, one gene contains the instructions to build one protein.

**DNA Holds Instructions for Making Proteins**
The DNA is protected because it is kept in the nucleus. In order to make a protein from the instructions written in the sequence of DNA, cells first “transcribe” the sequence of DNA. This is like making a photocopy of the instructions in the DNA. In the cytoplasm, the ribosome uses the copy of the DNA instructions to build a protein.

**Translation: Nucleic Acid Language into the Language of Amino Acids**
At the ribosome, the information from the DNA sequence is “translated” into the language of amino acids and a protein is made. The rules that determine which nucleotide sequence codes for specific amino acids are known as the **genetic code**. The ribosome reads groups of three nucleotides called codons. Each **codon** is like a word: its letters name a particular amino acid. The ribosome then catalyzes the reaction that adds the amino acid to the growing protein molecule. The nucleotide sequence includes codons that signal the ribosome to start and stop translation.
DNA → PROTEIN PRACTICE

1. What protein (amino acid sequence) will be built using this gene sequence?


2. What is different between sequence #1 and sequence #2?


3. What will happen to the structure of the protein, given the change you identified above? Explain how this may or may not affect the function of the protein. (Hint: tyrosine is hydrophobic, aspartic acid is hydrophilic)

4. Describe the relationship between the nucleotide (DNA) sequence of a gene, and the structure of the protein coded for by that gene.
DNA → PROTEIN PRACTICE  KEY

1. What protein (amino acid sequence) will be built using this gene sequence?


Met – Arg – Arg – Tyr – Gly – Ala – Lys - STOP

2. What is different between sequence #1 and sequence #2?


Met – Arg – Arg – Asp – Gly – Ala – Lys – STOP

3. What will happen to the structure of the protein, given the change you identified above? Explain how this may or may not affect the function of the protein. (Hint: tyrosine is hydrophobic, aspartic acid is hydrophilic)

The structure will change because there is a different order of amino acids, and this will cause the protein to fold/form different, leading to a different shape. This may affect the function.

4. Describe the relationship between the nucleotide (DNA) sequence of a gene, and the structure of the protein coded for by that gene.

The nucleotide sequence of a gene leads to the amino acid sequence of the protein coded for by that gene. The amino acid sequence determines the structure, and therefore the function of the protein.
How to use this PowerPoint

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2.4 Genotype to Phenotype

Goals

After reviewing this PowerPoint, you should be able to:

1) Define these terms: allele, genotype, phenotype.
2) Describe how genotype determines phenotype using the term “allele” in your description.
3) Model how genotype determines phenotype using Bioflowers.

Get out your vocabulary sheet and make notes on the next section:
Review: A gene is a section of a chromosome that tells an organism how to make a specific protein.

An allele is a different version of a gene – it has a slightly different nucleotide sequence.

Example: Bioflowers have two alleles for the enzyme that converts blue pigment into red pigment.
Bioflower Type 1: Two functional alleles

Bioflower Type 2: One functional allele and one nonfunctional allele

Bioflower Type 3: Two nonfunctional alleles

The combination of alleles determines the observed trait (characteristics)
The combination of alleles determines the observed characteristics

**Phenotype** – the observed (or measurable) trait of an organism that relates to one gene

**Genotype** – the two alleles that an organism has for a trait = the combination of alleles

Example: Trait = “flower color”
Phenotype = “RED” or “BLUE”
Genotype = “Functional / Functional” or “Functional / Nonfunctional” or “Nonfunctional /Nonfunctional”

The combination of alleles determines the observed trait (characteristics)

Information flow in cells

DNA → protein → traits

Information flow in cells

DNA → protein → traits
Genotype determines phenotype

**Let’s practice:**
**Genotype to Phenotype Worksheet**
Use the DNA to amino acid codon wheel to complete the worksheet.

**Check Your Understanding**
1) Define these terms: allele, genotype, phenotype.
2) Describe how genotype determines phenotype using the term “allele” in your description.
3) Model how genotype determines phenotype using Bioflowers (complete the Genotype to Phenotype worksheet).

**What’s Next?**
1. Check your work on the Genotype to Phenotype worksheet using the provided key.
2. Consider completing the OPTIONAL Student Questions to clarify and deepen your understanding.
3. Make an entry in your Learning Tracking Tool titled “2.4 Genotype to Phenotype.”
Investigating Genotype to Phenotype – Bioflower Colors

**Part 1:** In imaginary Bioflowers, there are 3 alleles (versions) of gene A and 3 alleles of gene B. Gene A codes for enzyme A and gene B codes for enzyme B. Determine the amino acid sequence of each allele of each gene. Note that these are only portions of the gene sequences – most proteins are made of thousands of amino acids.

<table>
<thead>
<tr>
<th>Gene A</th>
<th>DNA sequence</th>
<th>Amino Acid Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1 allele</td>
<td>AGG TTG GGC GGG GGT GAA CTT TGC</td>
<td></td>
</tr>
<tr>
<td>A2 allele</td>
<td>AGA TAG GCG GTG GTG TCG ATA CG</td>
<td></td>
</tr>
<tr>
<td>A3 allele</td>
<td>AGG TTA GGC GGG GGT GAA TAA TGA</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Gene B</th>
<th>DNA sequence</th>
<th>Amino Acid Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>B1 allele</td>
<td>TGC GAG GGA AGA TGC GGG TTA GAA</td>
<td></td>
</tr>
<tr>
<td>B2 allele</td>
<td>AGA GAG GGA AGA TGC GGG TTA GAA</td>
<td></td>
</tr>
<tr>
<td>B3 allele</td>
<td>TGC GAG GGA AGA TGC GGG TAA GAA</td>
<td></td>
</tr>
</tbody>
</table>

**Part 2:** Use the Protein Key to match the amino acid sequence to the cartoon shape of each protein.

<table>
<thead>
<tr>
<th>Allele</th>
<th>A1</th>
<th>A2</th>
<th>A3</th>
<th>B1</th>
<th>B2</th>
<th>B3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cartoon shape of protein produced by the allele.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Is the protein functional (F) or non-functional (N)?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Explain how you determined if the protein is functional or nonfunctional. Include ideas about structure and function in your explanation.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Part 3:
Protein pathway for Bioflowers:
Draw the enzymes and pigment molecules present in the cells of each type of flower below.

Alleles for flower type 1
Gene A = Allele A1
Gene B = Allele B1

Trait of flower type 1:
What color is this type of flower?

Alleles for flower type 2
Gene A = Allele A1
Gene B = Allele B2

Trait of flower type 2:
What color is this type of flower?

Alleles for flower type 3
Gene A = Allele A1
Gene B = Allele B3

Trait of flower type 3:
What color is this type of flower?

Alleles for flower type 4
Gene A = Allele A2
Gene B = Allele B1

Trait of flower type 4:
What color is this type of flower?
Use this key to match the amino acid sequence to the cartoon shape of each protein. Consider how sequence affects protein structure and function.

<table>
<thead>
<tr>
<th>Amino Acid Sequence</th>
<th>Cartoon Shape</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>arg – leu – gly – gly – gly - glu</td>
<td></td>
<td>Colorless molecule → blue pigment molecule</td>
</tr>
<tr>
<td>cys – glu – gly – arg – cys – gly</td>
<td></td>
<td>Blue pigment molecule → red pigment molecule</td>
</tr>
</tbody>
</table>

Active sites of enzymes A and B
Investigating Genotype to Phenotype – Bioflower Colors KEY

Part 1: In imaginary Bioflowers, there are 3 alleles (versions) of gene A and 3 alleles of gene B. Gene A codes for enzyme A and gene B codes for enzyme B. Determine the amino acid sequence of each allele of each gene. Note that these are only portions of the gene sequences – most proteins are made of thousands of amino acids.

Gene A

<table>
<thead>
<tr>
<th>Allele</th>
<th>DNA Sequence</th>
<th>Amino Acid Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>A2</td>
<td>AGA TAG GCG GTG GTG TCG ATA CG</td>
<td>ARG - STP</td>
</tr>
</tbody>
</table>

Gene B

<table>
<thead>
<tr>
<th>Allele</th>
<th>DNA Sequence</th>
<th>Amino Acid Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>B3</td>
<td>TGC GAG GGA AGA TGC GGG TAA GAA</td>
<td>CYS – GLU – GLY – ARG – CYS – GLY - STP</td>
</tr>
</tbody>
</table>

Part 2: Use the Protein Key to match the amino acid sequence to the cartoon shape of each protein.

<table>
<thead>
<tr>
<th>Allele</th>
<th>A1</th>
<th>A2</th>
<th>A3</th>
<th>B1</th>
<th>B2</th>
<th>B3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cartoon shape of protein produced by the allele.</td>
<td>![Shape]</td>
<td>![Shape]</td>
<td>![Shape]</td>
<td>![Shape]</td>
<td>![Shape]</td>
<td>![Shape]</td>
</tr>
<tr>
<td>Is the protein functional (F) or non-functional (NF)?</td>
<td>F</td>
<td>NF</td>
<td>F</td>
<td>F</td>
<td>NF</td>
<td>F</td>
</tr>
<tr>
<td>Explain how you determined if the protein is functional or nonfunctional. Include ideas about structure and function in your explanation.</td>
<td>Shape of active site of protein fits colorless molecule</td>
<td>No protein is formed</td>
<td>Shape of active site of protein fits colorless molecule</td>
<td>Shape of active site of protein fits blue molecule</td>
<td>Doesn’t appear to have an active site</td>
<td>Shape of active site of protein fits blue molecule</td>
</tr>
</tbody>
</table>
Part 3: Protein pathway for Bioflowers:
Draw the enzymes and pigment molecules present in the cells of each type of flower below.

**Alleles for flower type 1**
Gene A = Allele A1
Gene B = Allele B1

Trait of flower type 1:
What color is this type of flower?

   **RED**

**Alleles for flower type 2**
Gene A = Allele A1
Gene B = Allele B2

Trait of flower type 2:
What color is this type of flower?

   **BLUE**

**Alleles for flower type 3**
Gene A = Allele A1
Gene B = Allele B3

Trait of flower type 3:
What color is this type of flower?

   **RED**

**Alleles for flower type 4**
Gene A = Allele A2
Gene B = Allele B1

Trait of flower type 4:
What color is this type of flower?

   **WHITE**
Analysis Questions: Investigating Genotype to Phenotype – Bioflower Color

1. Explain how each of the following are different from each other:
   a. different genes
   b. different alleles
   c. different proteins

2. Describe how a protein is produced. Use protein A3 as an example.

3. How are enzymes and proteins related? Which molecules in this activity are proteins? Which are enzymes? You may want to refer to 2.1 Protein to Trait.

4. Explain what makes a protein functional or nonfunctional. You might want to draw a picture to help with your explanation.

5. What color will the bioflower be for each of the following allele combinations?
   a. A1, B1:
   b. A1, B2:
   c. A1, B3:
   d. A2, B1:
   e. A2, B2:
   f. A2, B3:
   g. A3, B1:
   h. A3, B2:
   i. A3, B3:

6. On the back of this sheet, create a concept map that includes the following terms to show how the bioflower obtains its color: allele, amino acids, chromosome, function, gene, nucleotide, protein, structure, trait. You may also include additional terms. Explain why you included those terms.
Zooming into Sickle Cell: Does Steve have Sickle Cell Disease?

Steve goes into the doctor’s office because he has been having the following issues: shortness of breath, dizziness, feeling tired all of the time, headaches, times of sudden and intense pain, a rapid heart rate, and cold hands and feet.

The doctor took a sample of his blood and sent it off to the lab. The technicians look at Steve’s blood under a microscope and compared it to healthy blood. This is what they see:

Some of Steve’s red blood cells are sickled, meaning that they have a crescent or bent shape. Based on this finding, Steve’s doctor orders a genetic test of Steve’s DNA. The doctor knows that Sickle Cell Disease is caused by changes in the DNA sequence of the gene that codes for hemoglobin, the oxygen-transporting protein found in red blood cells. Here’s what they found:

Sequence of Steve’s DNA:
ATG GTG CAT CTG CCT GTG GAG AAG TCT

Sequence of healthy DNA:
ATG GTG CAT CTG CCT GAG GAG AAG TCT

1) Find and circle the differences in the DNA sequences above of the two patients.

DNA to protein:
2) Use your codon table to translate the DNA from above into an amino acid sequence (a protein).

What is Steve’s amino acid sequence?

________ _______ _______ _______ _______ _______ _______ _______ _______ _______

What is the healthy amino acid sequence?

________ _______ _______ _______ _______ _______ _______ _______ _______ _______

3) Circle the difference between the two amino acid sequences.

4) What caused this difference in amino acids?
Steve wanted to understand why his cells sickle when other people’s do not, so he did some research. Here’s what he learned:

The hemoglobin protein found in red blood cells is actually made up of several smaller protein subunits. The image to the right shows what these hemoglobin protein subunits look like when they are carrying oxygen and when they are not carrying oxygen.

5) How do the hemoglobin protein subunits compare:

   a) Healthy vs. Steve’s hemoglobin protein subunits when carrying oxygen (shapes 1 and 3):

   b) Healthy hemoglobin when carrying oxygen vs. when not carrying oxygen (shaped 1 and 2):

6) How could you explain the difference in protein shape based on what you learned in Questions 1-4?

7) The picture to the right shows how the hemoglobin protein subunits connect together to form a complete hemoglobin molecule inside red blood cells. How does the Sickle Cell hemoglobin compare to normal (healthy) hemoglobin at the molecular scale? (Look at the picture!)
Steve learns that everyone has two copies of the gene that codes for hemoglobin protein subunits. The two versions of the gene (alleles) are:

- A – Normal/healthy hemoglobin
- S – Sickle Cell hemoglobin

In Sickle Cell hemoglobin, the change in the amino acid sequence from glutamine to valine results in a different protein shape. It turns out valine is a *hydrophobic* molecule, meaning it wants to be away from the watery environment of the cell (think way back to when you learned about the cell membrane in first semester). Because of this, the hemoglobin protein subunits will try to fit together in a way that “hides” the valine away from the water. Here’s what that looks like:

<table>
<thead>
<tr>
<th>Oxygen-rich environment</th>
<th>Hemoglobin A (HbA)</th>
<th>Hemoglobin S (HbS)</th>
<th>Hemoglobin A and Hemoglobin S (HbA and HbS)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Shape (circle one)</strong></td>
<td>Round</td>
<td>Sickle</td>
<td>Round</td>
</tr>
<tr>
<td><strong>Reasoning</strong></td>
<td>Because all shapes fit into the circle w/o stacking</td>
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</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Oxygen-poor environment</th>
<th>Hemoglobin A (HbA)</th>
<th>Hemoglobin S (HbS)</th>
<th>Hemoglobin A and Hemoglobin S (HbA and HbS)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Shape (circle one)</strong></td>
<td>Round</td>
<td>Sickle</td>
<td>Round</td>
</tr>
<tr>
<td><strong>Reasoning</strong></td>
<td>Because all shapes fit into the circle w/o stacking</td>
<td>The shapes stacked because the valine wanted to “hide” to avert water</td>
<td>Three shapes stacked to “hide” the valine, one did not. The cell will sometimes sickle.</td>
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<table>
<thead>
<tr>
<th>Predicted Genotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>AA</td>
</tr>
<tr>
<td>SS</td>
</tr>
<tr>
<td>AS</td>
</tr>
</tbody>
</table>

8) Explain the phenotype (trait) of individuals with AA, SS, and AS genotypes.

AA:

SS:

AS:
9. Complete the table below to show the relationship between DNA, protein, and trait for individuals with the genotypes AA, SS, and AS.

<table>
<thead>
<tr>
<th>Genotypes</th>
<th>Diagrams of the cells</th>
<th>What proteins are in the cells?</th>
<th>Organism phenotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allele #1 = A normal hemoglobin protein</td>
<td><img src="image1" alt="Diagram" /></td>
<td><img src="image2" alt="Diagram" /></td>
<td>(draw proteins here)</td>
</tr>
<tr>
<td>Allele #2 = A normal hemoglobin protein</td>
<td><img src="image3" alt="Diagram" /></td>
<td><img src="image4" alt="Diagram" /></td>
<td>(draw proteins here)</td>
</tr>
<tr>
<td>Allele #1 = A normal hemoglobin protein</td>
<td><img src="image5" alt="Diagram" /></td>
<td><img src="image6" alt="Diagram" /></td>
<td>(draw proteins here)</td>
</tr>
<tr>
<td>Allele #2 = S sickle hemoglobin protein</td>
<td><img src="image7" alt="Diagram" /></td>
<td><img src="image8" alt="Diagram" /></td>
<td>(draw proteins here)</td>
</tr>
</tbody>
</table>

**Key:**

- **A** normal hemoglobin protein
- **S** sickle hemoglobin protein

10. What is the genotype for an individual with Sickle Cell Disease? Predict the genotype of their parents. Explain your prediction.
Zooming into Sickle Cell: Does Steve have Sickle Cell Disease?

Steve goes into the doctor’s office because he has been having the following issues: shortness of breath, dizziness, feeling tired all of the time, headaches, times of sudden and intense pain, a rapid heart rate, and cold hands and feet.

The doctor took a sample of his blood and sent it off to the lab. The technicians look at Steve’s blood under a microscope and compared it to healthy blood. This is what they see:

![Steve's blood and Healthy blood images]

Some of Steve’s red blood cells are sickled, meaning that they have a crescent or bent shape. Based on this finding, Steve’s doctor orders a genetic test of Steve’s DNA. The doctor knows that Sickle Cell Disease is caused by changes in the DNA sequence of the gene that codes for hemoglobin, the oxygen-transporting protein found in red blood cells. Here’s what they found:

**Sequence of Steve’s DNA:**
ATG GTG CAT CTG ACT CCT GTG GAG AAG TCT

**Sequence of healthy DNA:**
ATG GTG CAT CTG ACT CCT GAG GAG AAG TCT

1) Find and circle the differences in the DNA sequences above of the two patients.

DNA to protein:
2) Use your codon table to translate the DNA from above into an amino acid sequence (a protein).

What is Steve’s amino acid sequence?

__MET__ VAL__ HIS__ LEU__ THR__ PRO__ VAL__ GLU__ LYS__ SER__

What is the healthy amino acid sequence?

__MET__ VAL__ HIS__ LEU__ THR__ PRO__ GLU__ GLU__ LYS__ SER__

3) Circle the difference between the two amino acid sequences.

4) What caused this difference in amino acids?

GTG codes for valine and GAG codes for glutamic acid. The difference in the DNA sequence codes for a different amino acid.
Steve wanted to understand why his cells sickle when other people’s do not, so he did some research. Here’s what he learned:

The hemoglobin protein found in red blood cells is actually made up of several smaller protein subunits. The image to the right shows what these hemoglobin protein subunits look like when they are carrying oxygen and when they are not carrying oxygen.

5) How do the hemoglobin protein subunits compare:

   a) Healthy vs. Steve’s hemoglobin protein subunits when carrying oxygen (shapes 1 and 3):

   Steve's hemoglobin protein subunits have an extension sticking out of the bottom.

   b) Healthy hemoglobin when carrying oxygen vs. when not carrying oxygen (shaped 1 and 2):

   When carrying oxygen the hemoglobin is a rectangle shape, but when not carrying oxygen there is an open space/notch.

6) How could you explain the difference in protein shape based on what you learned in Questions 1-4?

   The change in DNA codes for a change in amino acid sequence which results in a different protein shape.

7) The picture to the right shows how the hemoglobin protein subunits connect together to form a complete hemoglobin molecule inside red blood cells. How does the Sickle Cell hemoglobin compare to normal (healthy) hemoglobin at the molecular scale? (Look at the picture!)

   Normal hemoglobin subunits connect to form a square shape hemoglobin while the sickle cell hemoglobin subunits form a stretched out chain.
Steve learns that everyone has two copies of the gene that codes for hemoglobin protein subunits. The two versions of the gene (alleles) are:

- **A** – Normal/healthy hemoglobin
- **S** – Sickle Cell hemoglobin

In Sickle Cell hemoglobin the change in the amino acid sequence from glutamine to valine results in a different protein shape. It turns out valine is a **hydrophobic** molecule, meaning it wants to be away from the watery environment of the cell (think way back to when you learned about the cell membrane in first semester). Because of this, the hemoglobin protein subunits will try to fit together in a way that “hides” the valine away from the water. Here’s what that looks like:

<table>
<thead>
<tr>
<th></th>
<th><strong>Hemoglobin A (HbA)</strong></th>
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<td><strong>Oxygen-rich environment</strong></td>
<td><img src="image1.png" alt="Diagram" /></td>
<td><img src="image2.png" alt="Diagram" /></td>
<td><img src="image3.png" alt="Diagram" /></td>
</tr>
<tr>
<td><strong>Shape (circle one)</strong></td>
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<td><strong>Reasoning</strong></td>
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<td><img src="image4.png" alt="Diagram" /></td>
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<td><img src="image6.png" alt="Diagram" /></td>
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8) Explain the phenotype (trait) of individuals with AA, SS, and AS genotypes.

- **AA**: Normal hemoglobin / doesn’t sickle
- **SS**: Sickle Cell Disease / cells sickle when they aren’t carrying oxygen
- **AS**: Cells sometimes sickle when they are not carrying oxygen
9. Complete the table below to show the relationship between DNA, protein, and trait for individuals with the genotypes AA, SS, and AS.

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<td>Allele #1 = A normal</td>
<td><img src="image1" alt="Diagram" /></td>
<td><img src="image2" alt="Proteins" /></td>
<td>Normal / no symptoms</td>
</tr>
<tr>
<td>hemoglobin protein</td>
<td></td>
<td>(draw proteins here)</td>
<td></td>
</tr>
<tr>
<td>Allele #2 = A normal</td>
<td><img src="image3" alt="Diagram" /></td>
<td><img src="image4" alt="Proteins" /></td>
<td>Carrier / symptoms only during</td>
</tr>
<tr>
<td>hemoglobin protein</td>
<td></td>
<td>(draw proteins here)</td>
<td>oxygen stress</td>
</tr>
<tr>
<td>Allele #1 = S sickle</td>
<td><img src="image5" alt="Diagram" /></td>
<td><img src="image6" alt="Proteins" /></td>
<td>Sickle Cell Disease</td>
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Key:  
- A normal hemoglobin protein
- S sickle hemoglobin protein

10. What is the genotype for an individual with Sickle Cell Disease? Predict the genotype of their parents. Explain your prediction.

An individual with Sickle Cell Disease has the genotype SS. Their parents could be:  
SS and AS  
or  
AS and AS

This is because each parent contributes one allele to their offspring (more on this in Lesson 3!).
How Does a Fatal Disease Persist in a Family? Model Revisions

Changes:
- Identify **TWO** changes you made and *explain why* you made those changes (revisions, additions, etc).
- Explain which evidence(s) support your argument. At least one evidence should be at the atomic-molecular scale.
- Identify the scientific principle(s) that support your argument – this is the reasoning.

<table>
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<tr>
<th>Part of your Argument</th>
<th>Explanation why...</th>
<th>Evidence(s)</th>
<th>Supporting Scientific Principle(s)</th>
</tr>
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<td>What you changed</td>
<td></td>
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Things you kept the same:
- Identify **TWO** parts of your argument that you did NOT change and *explain why* you did not change those parts.
- Explain which evidence(s) support your argument. At least one evidence should be at the atomic-molecular scale.
- Identify the scientific principle(s) that support your argument – this is the reasoning.

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