



3D Models for 3D Instruction: Using Models with Storylines



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Kim Parfitt

Taught for 18 years at
Cheyenne Central High School

And after 11 years of using 3D
models and attending CBM
summer courses, I am now
working full-time for this
amazing company.





Now, about you...



Workshop Learning Goals

You will be able to:

- Explore the connection between existing, and potential storylines, and 3D models.
- Explain how 3D models can support your students making sense of phenomena.



“The storylines approach includes design principles for engaging students with phenomena and problems to elicit their own questions that teachers, with support of curriculum materials, use to guide the trajectory of their sensemaking.”

“...storylines organize cycles of engaging with phenomena, questions, and sensemaking to incrementally build, test, and revise explanatory models...”

Brian J. Reiser
Northwestern University



coherence



Coherence from the student perspective

A Mouse That Howls

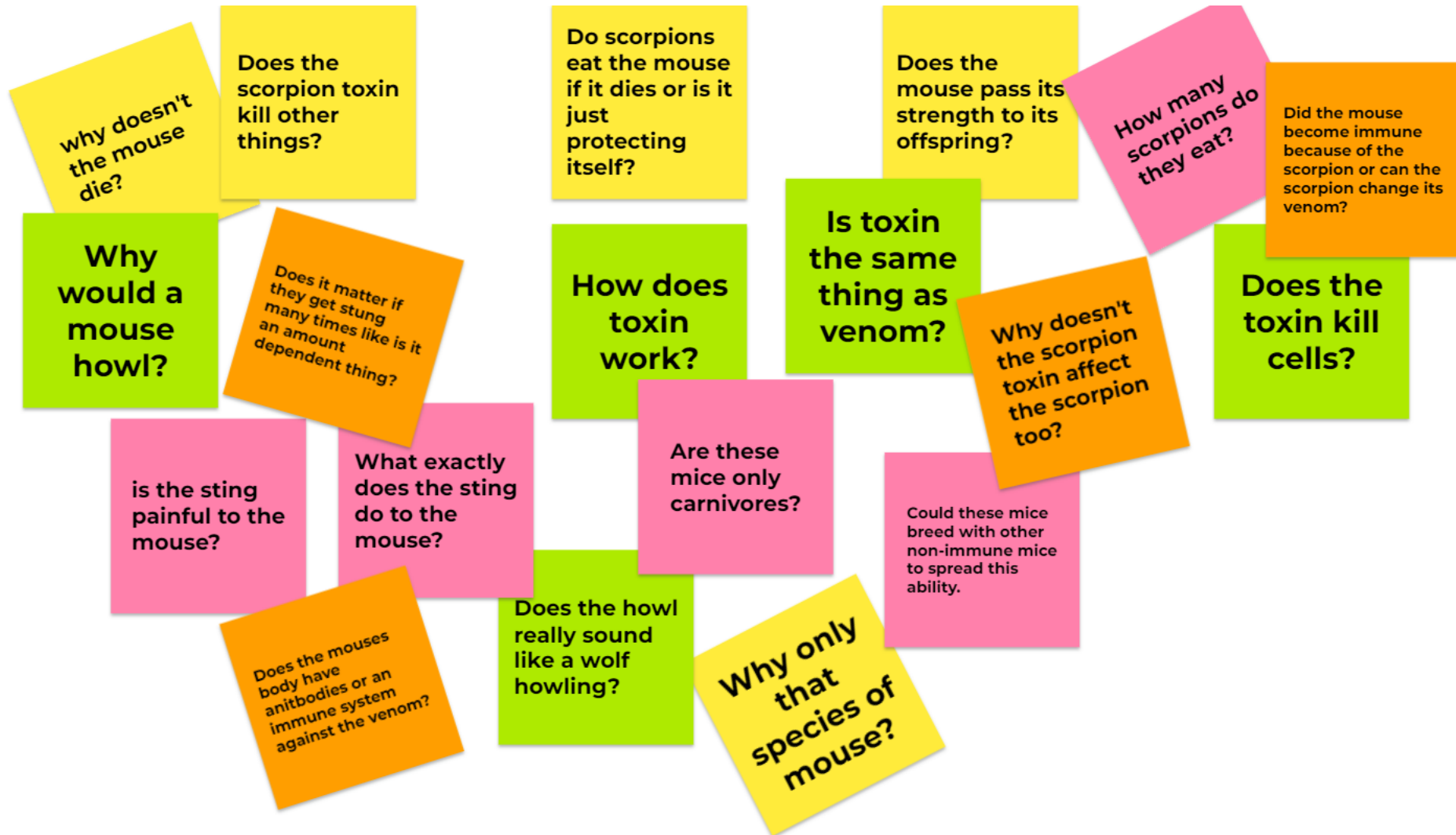


And it is a warrior!





Driving Question Board





Behavior/ecological relationships

Why would a mouse howl?

Does the howl really sound like a wolf howling?

Are these mice only carnivores?

How many scorpions do they eat?

Do scorpions eat the mouse if it dies or is it just protecting itself?

How come it doesn't die from the toxin from eating the scorpion?

Does the mouse pass its strength to its offspring?

Why only that species of mouse?

Could these mice breed with other non-immune mice to spread this ability.

Did the mouse become immune because of the scorpion or can the scorpion change its venom?

Genetics/natural selection & evolution

Toxins and function or nonfunction

How does toxin work?

Does the scorpion toxin kill other things?

What exactly does the sting do to the mouse?

Why doesn't the mouse die?

Why doesn't the scorpion toxin affect the scorpion too?

Is toxin the same thing as venom?

Does the mouse's body have antibodies or an immune system against the venom?

Is the sting painful to the mouse?

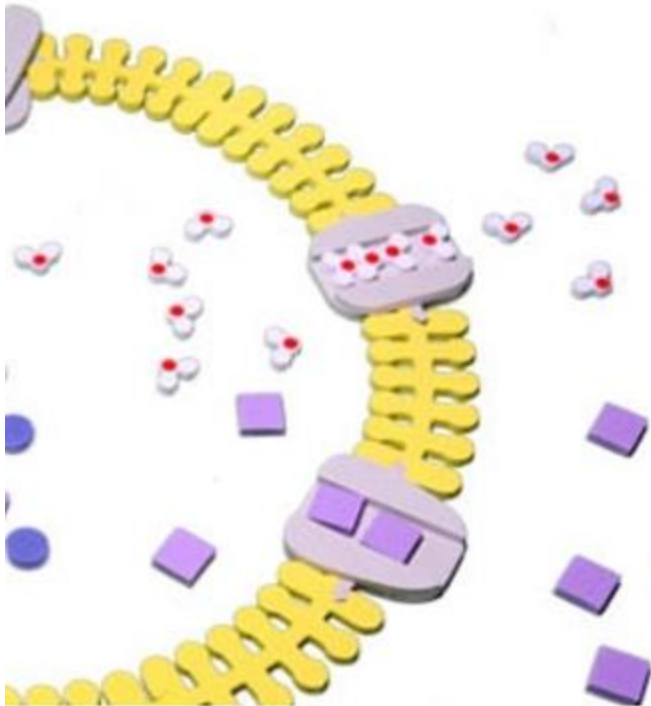
Does the toxin kill cells?

Does it matter if they get stung many times like is it an amount dependent thing?

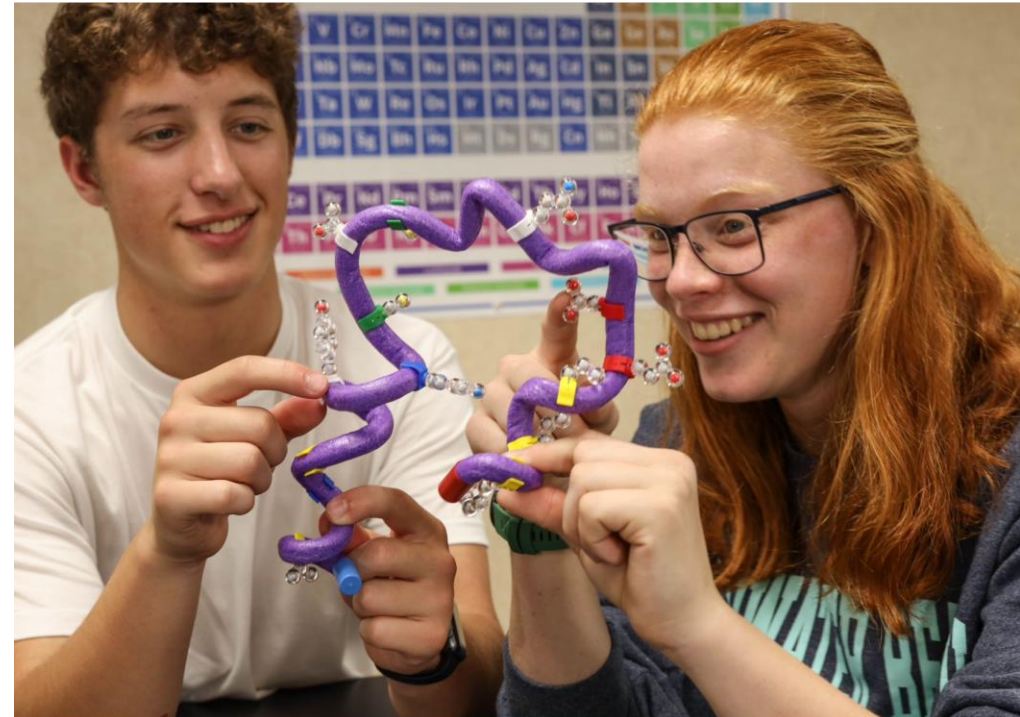


How can grasshopper mice take on scorpions, but other mice species can't, and does it matter? Why does the venom work on some species and not others?

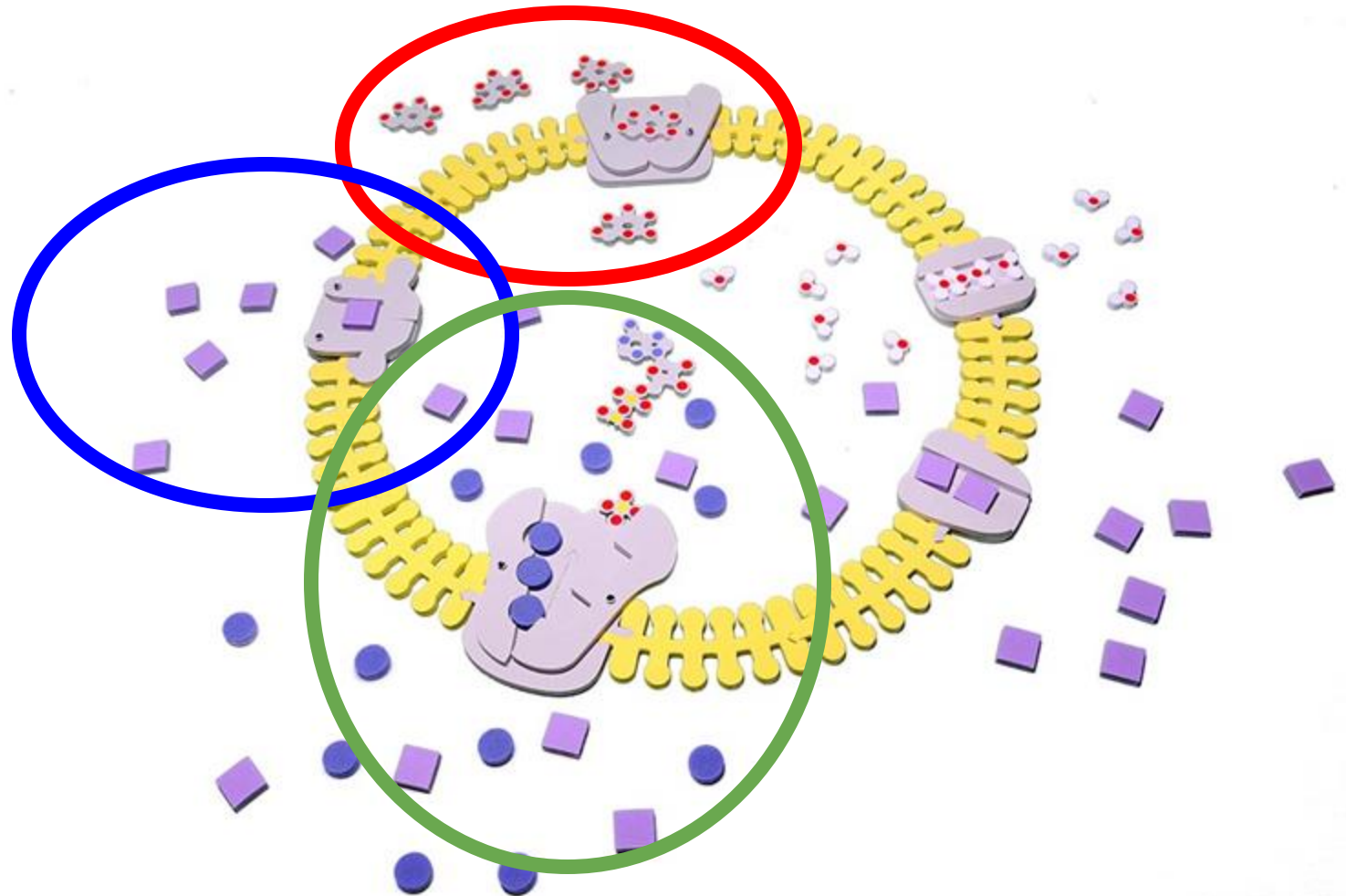
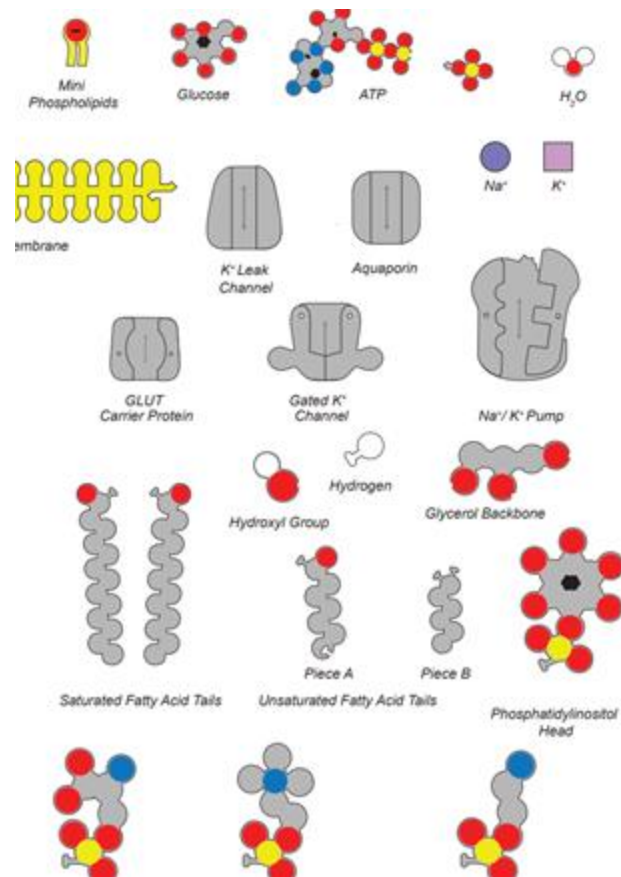
Phospholipid & Membrane Transport Kit



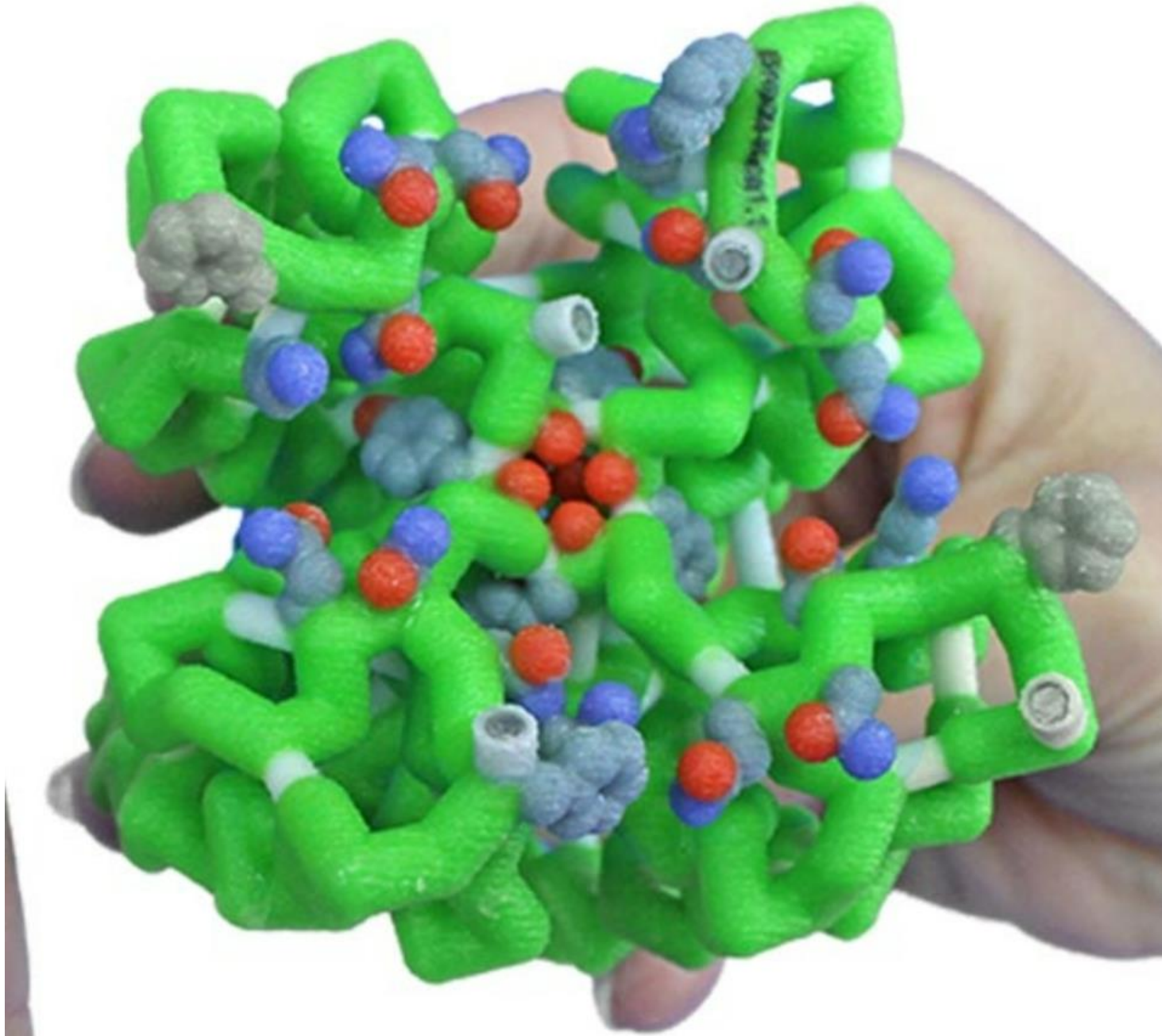
Amino Acid Starter Kit

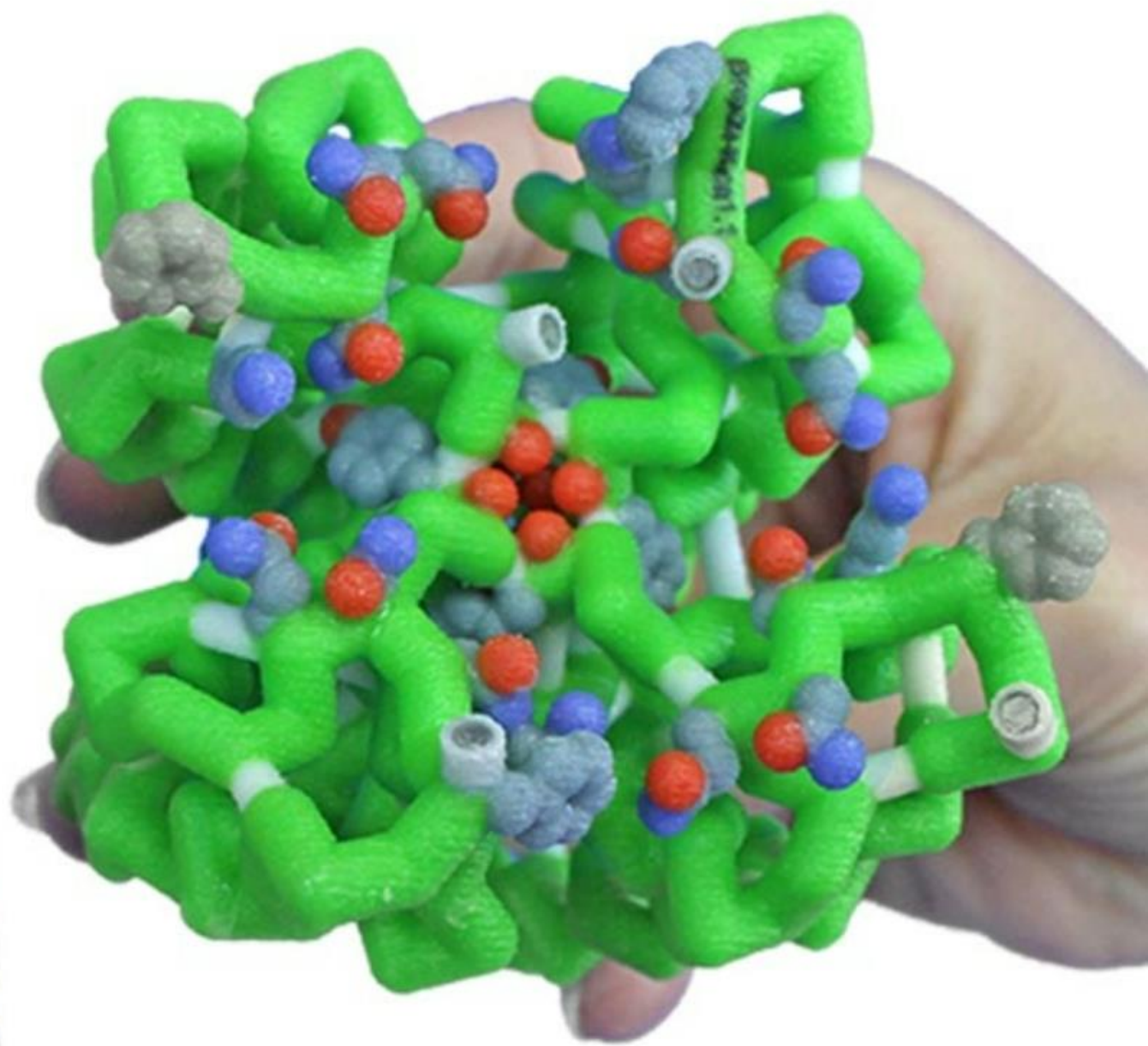


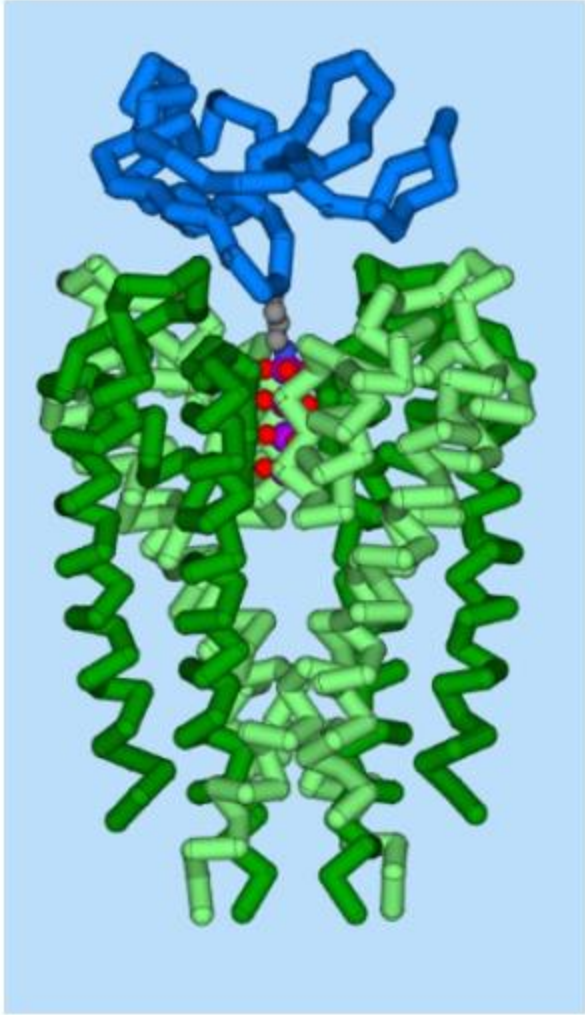
The aquaporin shows us **passive transport** but **active transport** can also be shown.

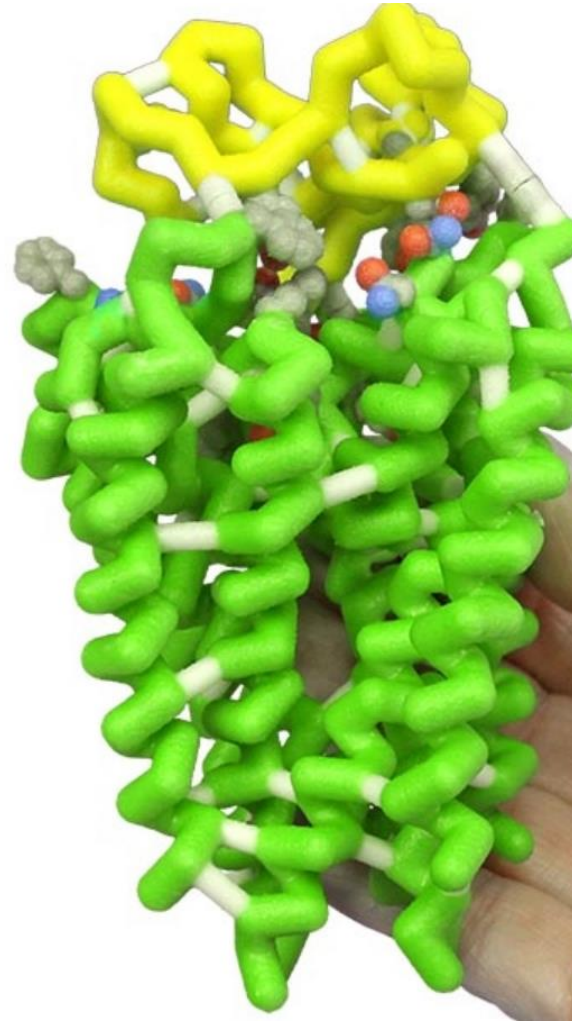


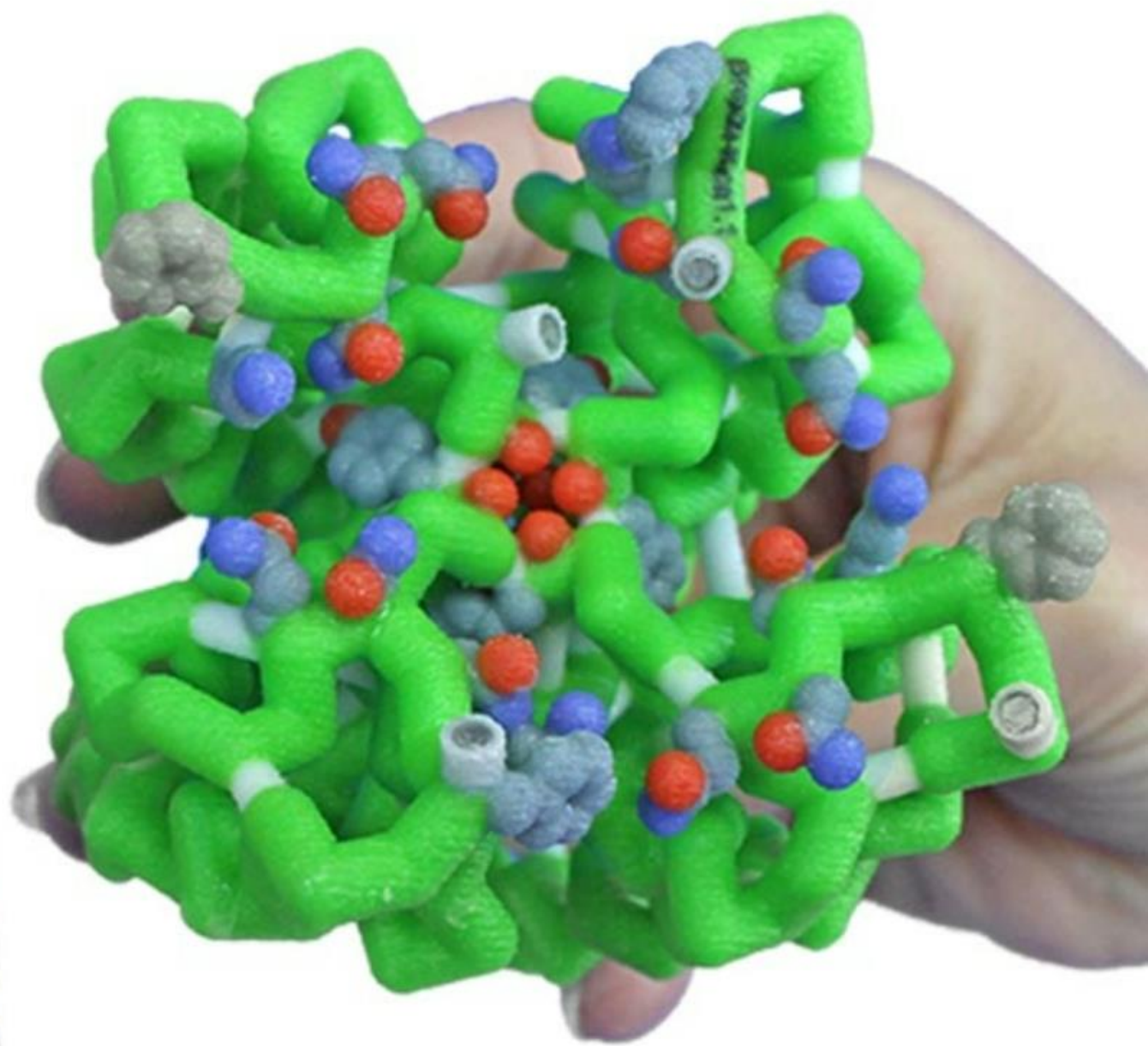
















3D Molecular Designs

1

Hydrophobic Non-Polar

- Valine **Val** V
- Isoleucine **Ile** I
- Leucine **Leu** L
- Phenylalanine **Phe** F
- Proline **Pro** P
- Alanine **Ala** A
- Glycine **Gly** G
- Tryptophan **Trp** W

Hydrophilic Polar

- Acidic -

- Aspartic Acid **Asp** D
- Glutamic Acid **Glu** E

+ Basic +

- Arginine **Arg** R
- Lysine **Lys** K
- Histidine **His** H
- Tyrosine **Tyr** Y
- Cysteine **Cys** C
- Methionine **Met** M
- Threonine **Thr** T
- Asparagine **Asn** N
- Glutamine **Gln** Q
- Serine **Ser** S

2

Amino Acid Sidechain List

Name	Amino Acid	Sidechain	Name	Amino Acid	Sidechain	Name	Amino Acid	Sidechain	Name	Amino Acid	Sidechain
Alanine	Ala	CH ₃	Asparagine	Asn	CH ₂ CONH ₂	Glutamine	Gln	CH ₂ CH ₂ CONH ₂	Proline	Pro	5-membered ring
Arginine	Arg	(CH ₂) ₃ NH ₂ ⁺	Aspartic Acid	Asp	CH ₂ COO ⁻	Cysteine	Cys	CH ₂ SH	Threonine	Thr	CH(OH)CH ₃
Asparagine	Asn	CH ₂ CONH ₂	Glutamic Acid	Glu	CH ₂ CH ₂ COO ⁻	Glutamine	Gln	CH ₂ CH ₂ CONH ₂	Tryptophan	Trp	CH ₂ indole
Aspartic Acid	Asp	CH ₂ COO ⁻	Histidine	His	CH ₂ imidazole	Isoleucine	Ile	CH(CH ₃)CH ₂ CH ₃	Valine	Val	CH(CH ₃) ₂
Cysteine	Cys	CH ₂ SH	Lysine	Lys	(CH ₂) ₄ NH ₂ ⁺	Methionine	Met	CH ₂ CH ₂ CH ₂ SCH ₃	Phenylalanine	Phe	CH ₂ benzene
Glutamine	Gln	CH ₂ CH ₂ CONH ₂	Serine	Ser	CH(OH)CH ₃	Threonine	Thr	CH(OH)CH ₃			
Glutamic Acid	Glu	CH ₂ CH ₂ COO ⁻									
Isoleucine	Ile	CH(CH ₃)CH ₂ CH ₃									
Leucine	Leu	CH(CH ₃) ₂ CH ₂ CH ₃									
Methionine	Met	CH ₂ CH ₂ CH ₂ SCH ₃									
Phenylalanine	Phe	CH ₂ benzene									
Proline	Pro	5-membered ring									
Threonine	Thr	CH(OH)CH ₃									
Tryptophan	Trp	CH ₂ indole									
Valine	Val	CH(CH ₃) ₂									

3

4

5

6 & 7
(also shown on mini toober)

8



[Voltage-Gated Sodium Channel in Grasshopper Mice Defends Against Bark Scorpion Toxin | Science](#)

[The scorpion toxin and the potassium channel - PMC \(nih.gov\)](#)

[Toxins | Free Full-Text | Scorpion Toxins Specific for Potassium \(K⁺\) Channels: A Historical Overview of Peptide Bioengineering | HTML \(mdpi.com\)](#)



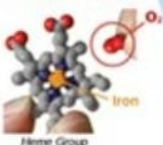
What stories could your students investigate with these models?

Hemoglobin

The Oxygen Transporter

Hemoglobin Protein Structure

Hemoglobin is a four-subunit protein complex composed of four separate proteins: two α -globin proteins and two β -globin proteins. Each protein subunit has a tightly bound **heme group**, which in turn binds one O_2 molecule to an iron atom in the center of the heme group. It is this heme group that gives blood its red color.



The β -globin protein is composed of 146 amino acids. This protein spontaneously folds up into its compact globular shape, following basic principles of chemistry and physics.

- The hydrophobic amino acids are buried on the inside of the protein, where they are protected from water.
- The polar and charged amino acids are positioned on the surface of the protein, where they interact with water.
- Positively-charged, basic amino acids often interact with negatively-charged, acidic amino acids to further stabilize the folded structure.

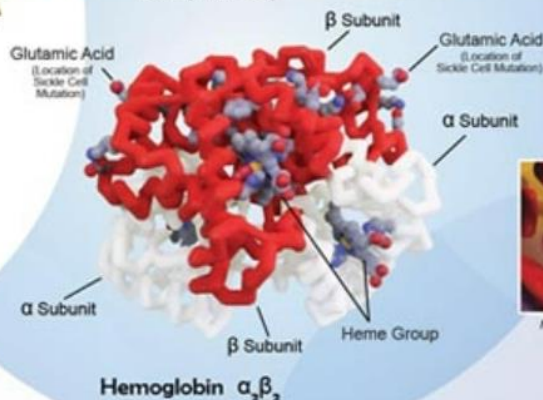
Hemoglobin Genes

If each β -globin protein consists of a unique sequence of 146 amino acids, how does the cell remember the correct sequence of amino acids to join together to make this protein 270 million times in each red blood cell? The answer is DNA. The sequence of amino acids in the β -globin protein is encoded in the sequence of nucleotides that make up the β -globin gene. Because DNA cannot leave the nucleus and move to the cytoplasm where ribosomes are ready to make protein, the gene is first copied into messenger RNA. The sequence of nucleotides in the messenger RNA is AUGGUGACACUGACUCCUGAG. In the cytoplasm, ribosomes then translate this messenger RNA three-nucleotides at a time (triplet genetic code) into the correct sequence of amino acids that make up the β -globin protein (Met-Val-His-Leu-Tyr-Pro-Glu...). The initial methionine is later removed.

There are separate genes for α -globin and β -globin proteins, located on separate chromosomes. In developing red blood cells, the expression of the globin genes is regulated so that equal numbers of α -globin and β -globin proteins are made, to allow the efficient assembly of the four-subunit hemoglobin.

Why Do We Breathe?

We all know that we must breathe to stay alive. But what does this really mean? We need oxygen to burn the fuel (food) that we eat -- to release the energy that powers our bodies. Every time we take a deep breath, our respiratory system and our circulatory system collaborate to deliver oxygen from our lungs to all of the tissues of our body. As our heart pumps red blood cells through our lungs, they pick up O_2 by binding it to a special protein called hemoglobin. Each red blood cell contains approximately 270 million copies of this hemoglobin protein. Hemoglobin is able to bind O_2 and prevent it from reacting inappropriately with other molecules while it is being delivered to distant parts of our body.



Sickle Cell Anemia – An Inherited Disease



A mutation in the β -globin gene is responsible for the genetic disease sickle cell anemia. When a codon is changed from GAG to GTG, the 7th amino acid of the protein is changed from glutamic acid (Glu) to valine (Val). The normal amino acid, Glu, is negatively charged and is exposed on the outside surface of the protein, where it interacts with water and contributes to the solubility of the protein inside red blood cells. When changed to Val – a hydrophobic amino acid – the solubility of the mutant protein is reduced, and the hemoglobin proteins begin to stick together. This aggregation of the mutant protein distorts the shape of the red blood cells. These sickle cells clog up the very narrow blood vessels in capillary beds, leading to the complications of sickle cell anemia.



Normal Red Blood Cells

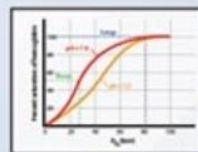


Sickled Red Blood Cells

The Physiology of O_2 Transport

The structure of hemoglobin has evolved over time to result in a protein that is exquisitely suited to transport oxygen. The four-subunit hemoglobin is designed to efficiently bind O_2 in the oxygen-rich environment of the lungs ($P_{O_2} \approx 80$ torr), and then efficiently release this O_2 in the oxygen-poor environment of peripheral tissues ($P_{O_2} \approx 26$ torr). Moreover, the more acidic environment of actively exercising muscle triggers oxygen release for use in generating energy.

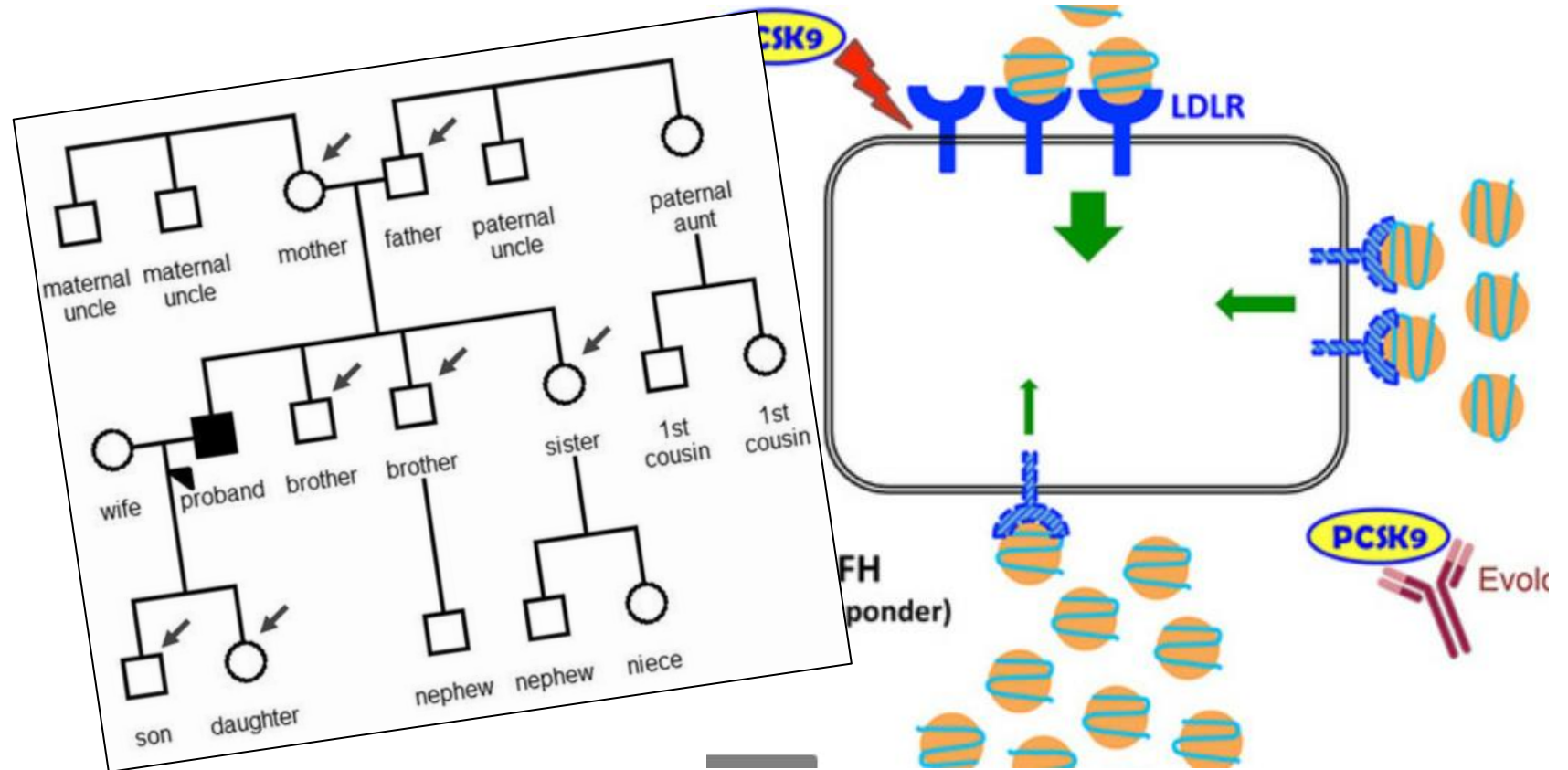
There are several different versions of the β -globin gene, used at different developmental stages, to produce versions of the hemoglobin protein that will most efficiently deliver O_2 to the developing fetus. Fetal hemoglobin binds oxygen more readily than adult (maternal) hemoglobin.



Oxygen Affinity

Sickle Cell Anemia

Heart Disease





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- Students make sense by investigating phenomena that reveals student understanding of all three dimensions of the NGSS.
- Students explicitly use the practices (SEPs) and cross cutting concepts (CCCs) to figure out the disciplinary core ideas (DCIs) to make their own sense of the phenomenon.
- Teachers use tasks that invite students to explain the phenomenon and reveal the students understanding or proficiency in all three dimensions.

(adapted from BSCS & NGSS Framework)