

## Model for Population Variation – Learning Segment Table

Seg	Model Move	Time (min)	Overview	What did we figure out?
1	M→P	55	We begin the unit with reframing our conversation about traits by “zooming out” to the population level. Though we’ve spent a lot of time understanding how traits are passed along and expressed in individual organisms, how does this help us to understand frequencies of traits in populations? We explore this question using frequency data we’ve collected from our classroom population.	In refocusing on traits in populations, we’ve asked the question, “What makes a trait more common or rare?” Since our ideas about dominance didn’t help us to understand frequencies in our classroom, we are still wondering about this question.
2	P→Q	35	We work in groups to make sense of maps showing the global distribution of 5 traits: Huntington’s disease, lactose intolerance, height, cystic fibrosis, and sickle cell anemia. After reporting out what we notice and what we wonder, we refine our driving question for the unit.	After looking at international frequency data for a number of human traits, we’ve settled on a driving question (a variant of “What makes traits more common or rare?” or “Why do trait frequencies differ across the world?”).
3	Q→M	25	We continue our work in groups—brainstorming our initial thoughts about the driving question and then compiling them into class list of initial model ideas.	We have an initial model but recognize that we need to explore further the ideas about how traits “behave” in populations.
4	M	65*	To help illuminate how variations in populations are related to the genetic makeup of its individual members, we use beans to simulate what happens to the gene pool of a population when all individuals survive and reproduce (i.e. there is no selection).  *The simulation takes 55 minutes and the debriefing an additional 10.	We’ve concretely explored the concept of the “gene pool” and how it relates to the frequency of traits in the moment and over time. In doing so, we have begun to formalize one model idea about the relationship between allele frequencies and genotype frequencies and a second idea about how those remain stable in populations where there is no selection at play. We have also begun to think about how selection might work in populations / gene pools.
5	P→M	40-75*	We return to the map of the HbS allele for Sickle Cell Anemia and learn a little about the disease and underlying traits so that we can explore ideas about how selection changes allele, genotype and trait frequencies.  *Time depends on a few different options in this learning segment.	We’ve extended our model to include differential survival and have tied the concepts of the gene pool and allele frequencies back to our Natural Selection model.
6	M→P	55-80*	We look yet again at the map for the HbS allele and wonder about its distribution, sharing out our ideas about selection or other processes. We then inspect a parallel map for the incidence of malarial infection and notice a relationship. This leads us to wonder about the link, to learn more through a video, and finally to work in groups to design a simulation (using the bean lab introduced earlier) that might explain the distribution of HbS.  *Time depends on a few different options in this learning segment.	We’ve essentially recognized (through our examination of sickle cell and malaria) that selection may act differently among populations, resulting in different patterns of allele and trait frequencies between different populations.

7	P→M	30	We revise our model and work toward a final version by asking which of our current model ideas are relevant to – or help explain – the case of sickle cell. We might also add ideas, for example, about mutation (imported from our genetics models about variation) and also migration. (If not already offered into the classroom, the contribution of migration can be generated through an exploration of North American sickle cell frequencies.)	We now have a relatively solid model that connects the environment to frequencies through selection. We also have an idea that states that alleles may be spread geographically by migration.
8	M→P	20-50	We return to the driving question and answer in writing as it applies to Sickle Cell Anemia. If time allows, we share and write consensus “best” explanations in our work groups.	We used the model to answer our driving question and explain the phenomenon of the distribution of frequencies in sickle cell anemia!