

Name: \_\_\_\_\_ Period: \_\_\_\_\_ Date: \_\_\_\_\_

Open **peebedu.com** and navigate to **Variant Ventures**. Click the **Introduction** button in the header and read through all steps before beginning. The introduction explains how each virus carries three RNA codons that determine its spread radius, antigen shape, and infection duration, and how the simulation models mutation, receptor-antigen matching, and zoonotic transfer.

## Part 1 – Model Evaluation (MAPP Framework)

*Scientific models are simplified representations of complex biological phenomena. Use the MAPP framework below to evaluate Variant Ventures as a scientific model.*

### M – Mode

What type of model is Variant Ventures? Describe how this simulation represents viral evolution and host-pathogen interactions. In your answer, identify at least three specific simulation elements and explain what each one represents about how viruses evolve and spread.

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### A – Accuracy

(a) Identify two things this simulation represents **accurately** about viral evolution and host immunity. For each, name the specific simulation feature and explain what biological concept it demonstrates.

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(b) Identify two things this simulation **oversimplifies or omits** about how real viruses mutate and spread. Consider what cannot be observed in this simulation that would be important for a complete understanding of viral evolution.

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## **P – Purpose**

What is the learning goal of this simulation? Explain how the codon-to-phenotype system and the genealogy tree work together to help you understand a key concept in evolutionary biology. Connect at least one simulation feature to its biological importance in the real world.

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## **P – Permanency**

Could this model change with new scientific evidence? Describe one way that new discoveries about how viruses mutate or how host immune systems respond might require the simulation to be revised. Explain why scientific models are always subject to revision as new evidence accumulates.

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## **Small-Group Discussion**

With your group, discuss the following:

- What are the strengths of this simulation as a model for viral evolution?
- What are its most significant limitations?
- If you could add one feature to improve the biological accuracy of this simulation, what would it be and why?
- How does the simulation help connect molecular-level mutations to population-level changes in variant frequencies?

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## Part 2 – Free Response Questions

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### Conceptual Analysis

#### Question 1 – Mutation, Variation, and Natural Selection

*Simulation Task: Start a new simulation in the Free Range environment with 30 pigs and 10 humans. Click Start Outbreak and let the simulation run until at least three variants have appeared in the Variants counter. Click on an infected pig to open the Viral Sequence Analysis panel and record the RNA sequence of the original virus and at least two variants, noting which codon changed in each.*

**(A)** (1 pt) **Describe** how random mutations serve as a source of genetic variation in a population.

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**(B)** (1 pt) **Explain** how the simulation reveals the mechanism by which heritable variation in viral antigen sequences is subject to natural selection through host immunity.

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**(C)** (1 pt) **Predict** what would happen to the frequency of antigen variants in the pig population over multiple outbreak cycles if hosts that recovered from infection were unable to develop antibodies.

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**(D)** (1 pt) **Justify** why the mutations that increase viral spread or enable infection of new host types would be expected to increase in frequency in the population over time, while mutations that reduce spread would be expected to decrease.

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## Analyze Model / Visual Representation

### Question 2 — Codon-to-Phenotype Mapping and Zoonotic Transfer

*Simulation Task: Reset and select the Indoor Minimum environment (50 pigs, 10 humans). Start the outbreak and run until the ZONOTIC TRANSFER alert appears. Click the infected human to open the Viral Sequence Analysis panel. Compare the antigen codon of the zoonotic variant to the original pig virus and to the starting human receptor displayed in the panel. Then open View Genealogy to examine the evolutionary path from the original virus to the zoonotic variant.*

**(A)** (1 pt) **Describe** how a change in an RNA nucleotide sequence can produce a change in the phenotype of an organism.

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**(B)** (1 pt) **Explain** how the relationship between viral antigen shape and host receptor type in the simulation determines whether a variant can infect a pig, a human, or neither, and how this represents a difference in fitness across environments.

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**(C)** (1 pt) **Represent** the receptor-antigen binding mechanism at the molecular level. In the box below, draw a diagram showing at least two scenarios: one in which a viral antigen successfully binds a matching host receptor to initiate infection, and one in which a host antibody blocks the antigen and prevents binding. Label the antigen, receptor, and antibody in each scenario.

*Draw your molecular diagram here. Label all structures.*

**(D)** (1 pt) **Explain** how the zoonotic transfer of a pathogen from pigs to humans, as modeled in this simulation, represents an example of a human-influenced disruption to ecosystem dynamics. Connect your answer to a principle from Unit 8 ecology, and do not repeat the molecular mechanism described in Part B.

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