

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

Open [peebedu.com](http://peebedu.com) and navigate to **CFTR Channel Simulation**. Read through the **Introduction** slides, which cover the CFTR protein, the central dogma, the deltaF508 mutation, membrane transport, and the cholera connection. Click **Get Started** to begin.

## Part 1 – Model Evaluation (MAPP Framework)

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

### M – Mode

What type of model is the CFTR Channel Simulation? Describe how this computational simulation represents the relationship between genotype, protein structure, and membrane transport.

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### A – Accuracy

**(a)** Identify two things this simulation represents accurately about how mutations affect protein function and membrane transport.

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**(b)** Identify two things this simulation oversimplifies or leaves out about the real biological process from gene to functional protein at the cell membrane.

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## **P – Purpose**

What is the learning goal of this simulation? Explain how the CFTR Channel Simulation is designed to help you understand how a DNA mutation can alter protein structure and disrupt cellular function.

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## **P – Permanency**

Could this model change with new scientific evidence? Describe one way that new discoveries about CFTR protein trafficking or gene therapy might change or improve a simulation like this one.

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## **Small-Group Discussion**

With your group, discuss the following:

- What are the strengths of this simulation as a model for understanding the connection between genotype and phenotype?
- What are its limitations?
- If you could add one feature to improve this simulation, what would it be and why?
- How does the cholera toggle help you understand heterozygote advantage?

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## Part 2 – Free Response Questions

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### Conceptual Analysis

#### Question 1 – From Gene to Protein: The Central Dogma and CFTR

*Simulation Task: Set both the **Maternal Allele** and **Paternal Allele** to **Wild-type** and click **Run Simulation**. Observe the central dogma steps displayed on the left panel (DNA, mRNA, protein sequence) and the chloride ion movement on the membrane canvas. Then change one allele to **deltaF508** and run again. Compare the DNA sequences, mRNA codons, and protein products for both alleles.*

**(A)** (1 pt) **Describe** the process by which genetic information encoded in DNA is used to produce a functional protein.

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**(B)** (1 pt) **Explain** how the deletion of three nucleotides in the deltaF508 mutation leads to a misfolded CFTR protein.

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**(C)** (1 pt) **Predict** what would happen to chloride ion transport and mucus production in the lungs of an individual homozygous for the deltaF508 allele.

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**(D)** (1 pt) **Justify** your prediction.

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## Analyze Model / Visual Representation

### Question 2 – Heterozygote Advantage and Selective Pressure

*Simulation Task: Set one allele to **Wild-type** and the other to **deltaF508** (heterozygous). Run the simulation and observe chloride ion flow. Then activate the **Add Cholera** button and observe the change in ion movement and water flow. Compare this to the homozygous wild-type (both wild-type alleles) with cholera active.*

**(A)** (1 pt) **Describe** the process by which a change in amino acid sequence affects the three-dimensional structure and function of a protein.

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**(B)** (1 pt) **Explain** why heterozygous carriers of the deltaF508 mutation have reduced vulnerability to cholera compared to homozygous wild-type individuals.

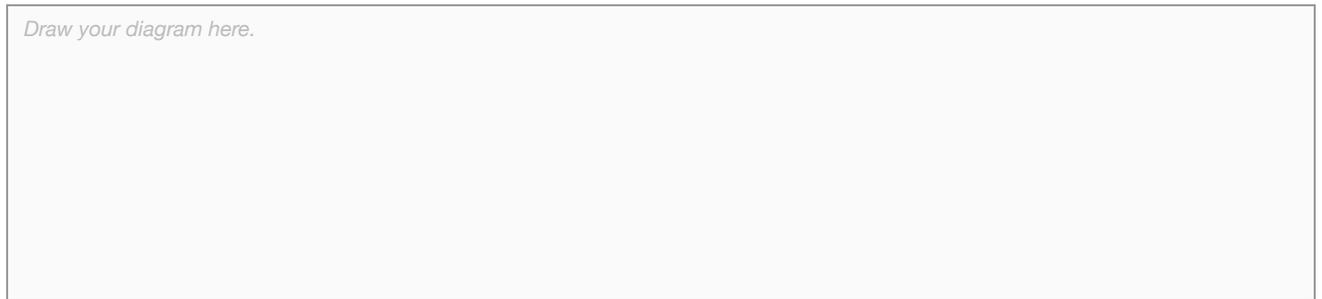
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**(C)** (1 pt) **Represent** the structure of a cell membrane with an embedded CFTR channel protein. Label the phospholipid bilayer, the CFTR channel, and the direction of chloride ion movement under normal conditions.

*Draw your diagram here.*



**(D)** (1 pt) **Explain** how the persistence of the deltaF508 allele in human populations can be understood as a result of natural selection in environments where cholera is prevalent.

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EK 1.7.A.3, 1.7.A.5, 2.3.A.1, 7.2.A.2