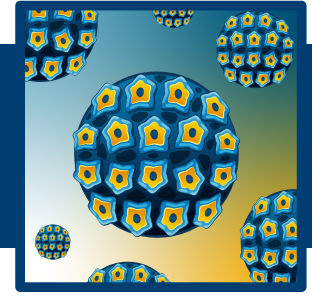


Student Processing Out

UNIT: GOING VIRAL! LESSON 1: SILENT SABOTAGE Activity 1B: FROM CODE TO CANCER



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HEALTH STARTS WITH SCIENCE

Directions: Consider Figure 2 and Table 1 from the article *Oncovirus Exposed: The HPV/Cancer Connection* to answer the following questions.

Figure 2: HPV 16 genome diagram

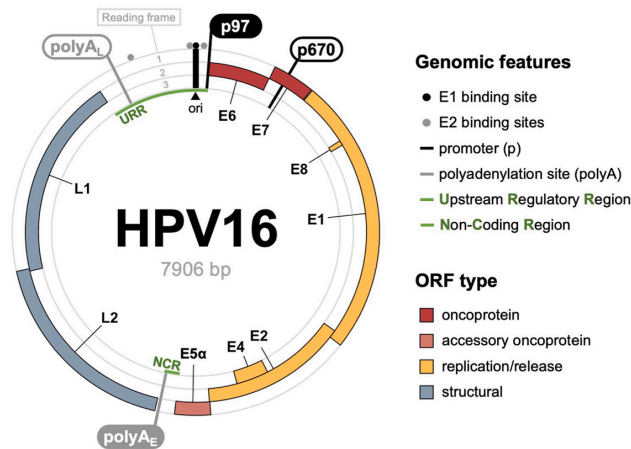


Table 1: Decoding HPV16 Genome

Expression	Genes	Functions
Early	E1	DNA helicase activity, DNA-dependent ATP binding, ATPase activity. Role in replication and replication repression.
	E2	Regulator of viral transcription and replication, control of the early expression region (early), necessary for efficient viral replication along E1.
	E4	Expressed primarily in differentiating epithelium, associated with the keratin cytoskeleton of epithelial cells in culture. Role in virus release.
	E5	HPV16 transformation activity in vitro. It possibly stimulates the initiation of cellular prevention in vivo but may have a role in the initiation of carcinogenesis.
	E6	Role in the transformation process together E7. Transcriptional activation properties. High-risk HPV's E6 inactivates p53 through rapid degradation through the ubiquitin pathway. Together with E7, it provides a cellular environment for viral replication.
	E7	Induces DNA synthesis in resting cells. E7 binds to the hypo-phosphorylated form of the pRb protein, resulting in its functional inactivation allowing functional progression to the S phase of the cell cycle. Low-risk HPV types 6 and 11's E7 protein binds less efficiently than the high-risk HPV types 16 and 18's E7 protein.
Late	L1	Larger capsid protein. Encodes main capsid protein.
	L2	Smaller capsid protein. Encodes secondary capsid protein.

MIDDLE & HIGH SCHOOL LEVEL

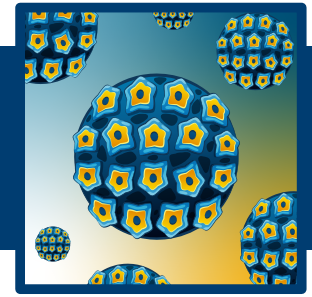
Teacher Enrichment Initiatives (TEI) | NIH SEPA | ©2026 | TxBiomed.org
NIH SEPA Project #1R25GM142021-01A1 | Some graphic elements courtesy of [Freepik](https://www.freepik.com)

Student Processing Out

UNIT: GOING VIRAL!

LESSON 1: SILENT SABOTAGE

Activity 1B: FROM CODE TO CANCER



1. Evolution occurs through genetic mutations. Using **Table 1** and **Figure 2**, analyze the HPV16 genome and propose a specific mutation to one of its genes (such as E1, E6, L1, etc.). Explain whether this mutation would pose a higher risk or a lower risk for cancer and justify your reasoning.



2. Based on your proposed mutation in Question 2, generate a claim as to whether this virus would most likely belong to sub-lineage A, B, C, or D (as shown in Figure 4B). Identify evidence, providing where the evidence is located in the article data and explain your reasoning as to how the evidence supports your claim.



Claim:

Evidence:

Reasoning: