



# Exploring Viral Particle Formation and Spread in HPV-Infected Keratinocytes

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## ABOUT THE STUDY

Across the world, millions of people are affected by Human Papillomavirus (HPV), a commonly transmitted infection. While most HPV infections are benign and resolve spontaneously, persistent infection with high-risk HPV types can lead to the development of cervical, anal, and other anogenital cancers. Understanding the mechanisms of HPV production and infection is crucial for developing effective preventive strategies and therapeutic interventions. This study focuses on the production of HPV Quasiviruses and their infection of primary human keratinocytes, shedding light on the intricacies of HPV biology.

### HPV Quasivirus production

**Viral capsid assembly:** HPV Quasiviruses are produced by the assembly of viral capsid proteins around the viral genome. The major capsid protein, L1, and the minor capsid protein, L2, form a complex that interacts with the viral DNA to create the viral particle. This assembly occurs in the nucleus of infected keratinocytes and is regulated by various viral and host factors.

**Viral genome replication:** The HPV genome consists of a circular double-stranded DNA that replicates in the nucleus of infected cells. The viral replication machinery utilizes host cellular factors to synthesize viral DNA and produce new copies of the viral genome. The viral replication process is tightly regulated to ensure efficient production of viral particles.

**Epithelial differentiation and viral release:** As infected keratinocytes undergo differentiation, viral gene expression is tightly regulated to ensure proper viral particle formation. HPV Quasiviruses are released from the upper layers of the stratified epithelium during normal epithelial turnover or following tissue damage. The release of viral particles allows for the spread of infection to neighboring cells and the establishment of persistent infection.

### Infection of primary human keratinocytes

**Receptor-mediated attachment:** HPV infection begins with the attachment of viral particles to the surface of primary human keratinocytes. The initial attachment is mediated by interactions between viral capsid proteins, particularly L1, and cell surface receptors. Heparan Sulfate Proteoglycans (HSPGs) serve as initial attachment receptors, facilitating the subsequent interaction with specific entry receptors.

**Internalization and intracellular trafficking:** Following attachment, HPV particles are internalized into the keratinocyte through endocytosis. The exact mechanisms of HPV internalization and intracellular trafficking are still under investigation. It is believed that the interaction between the viral particle and entry receptors triggers cellular signaling events that lead to the uptake of the virus into endosomal compartments.

**Viral uncoating and nuclear entry:** Once inside the endosomal compartments, HPV particles undergo a series of conformational changes, resulting in the exposure of the minor capsid protein, L2. L2 plays a crucial role in mediating the escape of viral DNA from the endosomes and its transport to the nucleus. The viral genome is released into the nucleus, where it establishes infection by utilizing the host cellular machinery.

**Viral gene expression and replication:** Upon entering the nucleus, the viral genome undergoes transcription and replication. Early viral genes are expressed to facilitate the replication of the viral genome, while late viral genes are expressed to produce viral capsid proteins. The expression of viral genes is tightly regulated and coordinated to ensure proper viral replication and assembly.

**Viral particle assembly and release:** In infected keratinocytes, viral capsid proteins are synthesized and assembled around the viral genome. The newly formed viral particles are released from the cells, either through cell lysis or *via* exocytosis, leading to the spread of infection to neighboring cells.

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