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# HUMAN PAPILLOMA VIRUS AND ITS REPLICATION CYCLE

Medical Microbiology

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## ABSTRACT

In this paper aim to describe the role of viral protein in human papilloma virus productivity and replication of the life Cycle. Human papilloma virus have involved over millions of years to propagate themselves in a range of different animal Species including man.they are small double standard DNA that replicate in squamous epithelium and cause hyper Proliferative lesion, some of which are at risk of malignant transformation .the infectious human papilloma virus Replication cycle is intimately linked to the differentiation program of the tissue. Most individuals will experiencetransient HR HPV infection during their lifetime but these infection will not progress to clinically significant cervical disease or Cancer because the immune system eventually recognize and clear the virus .These viruses activate the cell cycle as the Infected epithelial cell differentiates in order to create a replication competent environment that allows viral genome Amplification and packaging into infection.

## KEYWORDS

human papillomavirus, infection cell cycle, growth, cancer progression.

## INTRODUCTION-

Human papilloma virus an Infected cell division and basal cell start the procedure of keratinocyte differentiation that the triggers a strongly orchestrated pattern to the productive infection of the viral gene infection (1). Ds DNA genome present in to the nuclei where in the epithelial cell dividing Cervical disease divided in to the high and low reason of the subset mucosa infective(2). Mostly high risk HPV infection during lifetime but cervical disease and cancer immune system recognizes and clear the virus (3). In men high risk human papilloma virus predomenentially a subset of or pharyngeal cancer and the women more than 99.7% in cervical cancer (4) .cancer progression is due to the persistent infection with the high risk human papilloma virus.one of the most common part cancer at the molecular level, cancer progression is due to the high expression of the viral oncoproteins E6 and E7, inhibit apoptosis and also accumulation of DNA damage and activate the cell cycle (5, 6).

### Human Papillomavirus Life Cycle Of The Replication Divided Three Phases.

**Stages**-first RNA species to be detected upon nuclear infection on HPV 31 revealed that RNA encoding the viral replication factor E1 and E2 (7). Early expression of the viral transcription factor E2 would allow to the regulation of the viral early promoter to direct expression of the E6 and E7 regulatory proteins (8). The E2 possesses one protein binding domain and DNA binding they linked by a flexible hinge region they create homodimer they bind four palindromic sites. They are required E1 activated viral replication and these site located three part (9) .three of sites required for E1 activated viral replication and located to the viral replication (10).

E2 bind E1 they bind as a dimer of hexamers initial replication of HPV genome they generate many copies per nucleus (11) These amplification has been shown to be controlled by the E8^ E2 Protein they make the complex of NCoR/SMRT.The first phase of viral DNA replication and Contrast, the low risk HPV11 E4 has been found not to be essential genome maintenance. Infected cell circular viral replication are replicated in concert with replication of cellular DNA and equally These amplification has been shown to be controlled by the E8^ E2 Protein they make the complex of NCoR/SMRT.The first phase of viral DNA replication and genome maintenance. Infected cell circular viral replication are replicated in concert with replication of cellular DNA and equally divided into daughter cell through tethering of virus genome to host cell chromosome E2 bound to the chromatin binding protein (12). The E1-E2 complex itself may be sufficient for some HPVs to locate to cellular chromosome.HPV is capable of maintaining infection of epithelial cells over a significant time. Division of an infected basal epithelial cell can produce a transit amplifying cell that is capable of differentiating and moving into the upper epithelial layer. Second stage of the replication host cell in concert .viral genome is again amplified (13)

In this last way they are divided two distinct replication cycle of human papilloma virus (14). First mechanisms utilized the recombination dependent replication by involves the hpv 18 and second mechanism is bidirectional replication (15).

## CONCLUSION

HPV infection and its replication cycle has been the subject Of intense research a ctivity for over 50 years. There are Still significant gaps in our understanding of both Strands of HPV pathogenesis. Tight regulation by E2 of Expression levels of these oncoproteins in the dividing Cells of an infected epithelium cell. Dampening downAmplification levels of viral genomes during a normal Infectious cycle via the E8^ E2 protein may also help to Reduce the chances of elevated levels of E6 and E7 in Differentiated keratinocytes. The fine balance between The activities of E2, E4, and E5 on the cell cycle and Apoptosis to allow viral genome amplification, while Avoiding viral genome integration into the host genome, Is crucial for the virus to complete its infectious life Cycle.

## REFERENCES

1. Van Doorslaer K., Liz., Xirasagar S., Maes P., Kaminsky D , Liou D.et al (2012) Papillomavirus episteme: a comprehensive Papillomaviridae database and analysis resource.
2. Van Doorslaer K., Liz, XirasagarS, Maes P., Kaminski D., LiouD.et al (2011) The Papillomavirus Episteme: a major update to the papillomavirus sequence database. *Nucleic Acids*
3. Stanley M. (2010) Pathology and epidemiology of HPV infection in females. *Gynecol.Oncol.*
4. GiulianoA.R. NyitrayA.G., KreimerA.R., CampbellC.M, Goodman M.T., SudengS.L et al (2015) EUROGIN 2014 roadmap: differences in human papillomavirus infection natural history, transmission and human papillomavirus-related cancer incidence by gender and anatomic site of infection.
5. AnicG.M. and GiulianoA.R. (2011) GenitalHPV infection and related lesions in men. *Prev. Med.*53, S36–S41
6. Heidegger I., BorenaW. And Pichler R. (2015) the role of human papillomavirus in urological malignancies.
7. Oz bun M.A. (2002) Human papillomavirus type 31b infection of human keratinocytes and the onset of early transcription..*J. Virol.*76, 11291–11300.
8. Arginine (SR) protein kinase SRPK1 and inhibits phosphorylation of host SR proteins and of the viral transcription and replication regulator E2..*J. Virol.* 88, 12599–12611.
9. Moody C.A and Laimins L.A (2010) Human papillomavirus oncoproteins: pathways to transformation. *Nat. Rev. Cancer* 10, 550–560.
10. McBride A.A. (2013) the papillomavirus E2 protein. *Virology* 445, 57–79.
11. Sanders C.M. and Stenlund A. (1998) Recruitment and loading of the E1 initiator protein: an ATP-dependent process catalyzed by a transcription factor. *EMBO J.*17, 7044–7055
12. Dreer M., van de Poel S. and Stubenrauch F. (2017) Control of viral replication and transcription by the papillomavirus E8^E2 protein. *Virus Res.* 231.
13. McBride A.A. (2013) the papillomavirus E2 proteins. *Virology* 445, 57–79.
14. Olden M., Smola H., Aumailley M., Rivero F, Pfister H.J.and Smola-Hess. (2004) the human papillomavirus type 8 E2 protein oppresses beta4-integrin expression in primary human keratinocytes..*J. Virol.*78, 10738–10746.
15. U. Bernard, R.D. Burk, Z. Chen, K. van Doorslaer, H. Hauser, E.M. de Villiers (2010) Classification of papillomaviruses (PVs) based on 189 PV types and proposal of taxonomic amendments Virology, 401 pp. 70-79.