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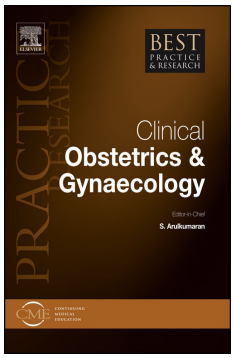

Host Control of Human Papillomavirus Infection and Disease

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Host Control of Human Papillomavirus Infection and Disease

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Abstract

Most human papillomaviruses cause inapparent infections, subtly affecting epithelial homeostasis to ensure genome persistence in the epithelial basal layer. As with conspicuous papillomas, these self-limiting lesions shed viral particles to ensure population-level maintenance, and depend on a balance between viral gene expression, immune cell stimulation, and immune surveillance for persistence. The complex immune evasion strategies, characteristic of high-risk HPV types, also allow the deregulated viral gene expression that underlies neoplasia. Neoplasia occurs at particular epithelial sites where vulnerable cells such as the reserve or cuboidal cells of the cervical transformation zone are found. Beta papillomavirus infection can also predispose to the development of cancers in individuals with immune deficiencies. The host control of HPV infections thus involves local interactions between keratinocytes, as well as the adaptive immune response. Effective immune detection and surveillance limits overt disease, leading to HPV persistence as productive microlesions, or in a true latent state.

keywords papillomavirus, epithelial homeostasis, wart, CIN, HPV, infection