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The evading mechanisms of HPV with E7 protein

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Cutaneous warts are hyperkeratotic spiny papules or nodules which often occur on the hands, fingers and soles of children. They are very common infection of skin by human papillomaviruses (HPV). HPV have evolved strategies to evade the effects of the innate immune system. For this reason, most of wart lesions have little inflammation clinically. Thus, we used to adopt a kind of immunotherapeutic modalities including diphenylcyclopropenone and imiquimod, which can provoke inflammation for the treatment of recalcitrant warts. The *E7* gene of high risk HPV has been known to play a role in evading host immune response. However, the role of *E7* in low risk HPV like cutaneous wart is not clearly understood. Toll-like receptors (TLRs) are the first line of defense in host protection against invasion of microbial pathogens. Since TLR signaling pathways induce NF-κB, we investigated whether *E7* can attenuate NF-κB signaling in normal human keratinocytes (NHKs). The NF-κB activity was significantly decreased in the NHKs transfected with HPV2 *E7*, compared to the NHKs transfected with the empty vector. Furthermore, it is reported that HPV has a mechanism to evade the effects of interferons and cytokines through induction of interferon-stimulated genes (ISGs). Viperin is one of ISGs, which can inhibit many DNA and RNA viruses. According to our investigation, the mRNA and protein expression of viperin in the E7 transfected cells was significantly decreased, in comparison to that in the empty vector transfected cells. Suppressed E7 might be a potential therapeutic target for the clearance of cutaneous warts.

Biography

Chul Hwan Bang is pursuing his PhD at the Catholic University of Korea. He is a Clinical Fellow of Seoul St. Mary's Hospital.

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