INFECTIOUS DISEASES (BACTERIAL, FUNGAL, VIRAL, PARASITIC, INFESTATIONS)

HPV E7 INHIBITED CELL PYROPTOSIS BY PROMOTING TRIM21 MEDIATED THE DEGRADATION AND K33 LINKED-UBIQUINATION OF IFI16 INFLAMMASOME

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Bachground: Human papillomavirus (HPV) is a DNA virus that causes sexually transmitted infections. HPV oncoprotein E7 is critical in regulating host immunity to promote self-escape, occurrence of cervical cancer and genital warts. Pyroptosis, which is a highly inflammatory form of programmed cell death, can be induced by the inflammasomes and acts as a defense against infection when the host is infected with pathogens.

Objective: This study focused on the role and mechanisms of HPV E7 in cell pyroptosis for promoting HPV immune escape and tumor development.

Materials and Methods: This study explored the affection of HPV E7 in regulating cell pyroptosis through flow cytometry analyses, microarray, mass spectrum, immunoprecipitation and western blot.

Results: We found HPV E7 could inhibit cell pyroptosis induced by transfection with dsDNA. Also, HPV E7 could inhibit the activation of inflammasomes and the production of IL-18 and IL-1β. Mass spectrum and immunoprecipitation showed that HPV E7 could interact with IFI16 and TRIM21. We also found HPV E7 promoted K33-linked ubiquitination and degradation mediated by E3 ligase TRIM21.

Conclusions: HPV E7 could recruit the E3 ligase TRIM21 to ubiquitinate and degradate IFI16 inflamasome, leading to inhibit the cell pyroptosis and promote HPV immune escape and tumor development.