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REVIEW ARTICLE

Diagnosis of Transient/Latent HPV Infections - A Point of View!

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Diagnosis of transient/latent HPV infections requires a rethinking of ideas concerning the host virus relationship. With this in mind, we address several concepts, such as mutualism and commensalism, to understand better the different stages of development, in addition to briefly covering current methods of detection. We suggest analyzing molecules related to the innate immune response for earlier diagnosis. © 2018 IMSS. Published by Elsevier Inc.

One of the main problems in the detection of cervical cancer is a lack of distinguishing markers of a transient/latent infection of human papillomavirus (HPV). Patients with cervical intraepithelial neoplasia, grades 2 or 3, are at particularly high risk (1). Although HPV DNA is found in almost all cases of cervical cancer, genotyping alone cannot distinguish between latent/transient infections (2).

We begin with some background information and a few definitions. Symbiosis is an association between organisms of different species that implies a unilateral or bilateral exchange of material or energy. Regardless of whether this exchange is caused by mutualism, commensalism or parasitism, there are notable differences in the life cycle of a virus. When one or more associated species benefits and the others do not show any apparent beneficial or detrimental effects, it is referred to as commensalism. In mutualism, the two species have reciprocal benefits, unlike parasitism, in which the symbiont survives by damaging part or all of the host (3). The definitions clarify the differences between a precancerous diagnosis, requiring surgical intervention, and the commensalism stage, which resolves HPV infections spontaneously.

Tumour viruses are divided into direct carcinogens, in which a viral oncogene induces cell proliferation, and indirect carcinogens, where the virus-induced chronic inflammation initiates the growth of cancer cells (4). Cancer induction by HPV occurs after several years of infection.

HPV colonizes the skin and mucosa of practically all humans. Its prevalence among children of 1 month to 4 years varies from 50–70% (5). Viral particle up-taking and internalization in the epithelium takes approximately 2 h (6). HPV transmission and continuous re-infection is observed in families, particularly from mother to child, replicating at very low levels without any apparent clinical or cellular damage (7).

Most HPV infections are ‘‘acute’’, i.e., not persistent or transient (8) or at the commensalism stage. In an acute HPV infection, a loss of equilibrium results in replication. This involves the production or elimination of the viral genome,

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