

Molecular Bases of Human Papillomavirus Pathogenesis in the Development of Cervical Cancer

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1. Introduction

Human papillomavirus (HPV) is a DNA virus which belongs to the newly named *Papillomaviridae* family (de Villiers et al., 2004). The papillomaviruses induce a variety of proliferative lesions in the skin and internal mucosa. Nearly half of the over 200 types of HPVs can infect the genital mucosa, producing benign epithelial lesions. However, a subset of these viruses is found in over 90% of malignant carcinomas of the genital tract, the majority of which contain the HPV16 and 18 types considered to be of "High Risk" (HR), as they help the progression into cervical cancer (Lorincz et al., 1992; Shah & Buscema, 1988).

The mechanisms through which HPVs induce cell transformation have been intensively investigated in the last few years. The most abundant viral transcripts in tumor and tumor cell lines are produced from the E6 and E7 Open Reading Frames (ORFs) that are known to be oncogenic. These two genes from HPV are necessary and sufficient to induce HPV-mediated transformation of murine cells (Yasumoto et al., 1986), transform human fibroblasts (Pirisi et al., 1987), and in cooperation with *ras* are capable of transforming baby rat kidney cells (Phelps et al., 1988) and primary human keratinocytes (Yoshida et al., 2008). The best evidence for the role of E6 and E7 proteins in malignancy comes from biochemical studies. E6 and E7 oncoproteins from the HR-HPV types have the ability to alter pathways involved in cell cycle control, interacting with and neutralizing the regulatory function of primarily two suppressor proteins, p53 and pRb respectively (Dyson et al., 1992; Werness et al., 1990). These effects produce genetic instability and accumulation of mutations that might contribute to the oncogenic process. Besides, several other target proteins for E6 and E7 oncoproteins have been identified and probably those interactions also promote cellular transformation. The latest oncogene identified from HPV was the E5 from HPV16. The E5 oncoprotein showed to be capable of increasing the half-life of Epidermal Growth Factor

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