



INTRODUCTION

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The relationship between cancer cervix and human papillomavirus (HPV) is unquestionably one of the hot topics in gynecological research. Over 60 different types of HPV have been identified, many of them can infect anogenital epithelium (*De Villiers, 1989*).

The genital HPV types were classified into 2 groups : low risk group, which are predominantly associated with benign lesions and appear to carry a low risk for malignant progression, and those frequently found associated with anogenital carcinoma which are considered as high risk group (*Vousden, 1989*).

Different studies demonstrated that, the prevalence of HPV in cervical carcinoma varies from 18.2 to 95% (*Munoz et al., 1988, Zur Hausen., 1991, Helland et al., 1993*).

It was found that, early proteins 6 and 7 of the high risk HPVs formed complexes with retinoblastoma and P53 proteins respectively. This may lead to rapid proteolytic degradation and loss of function of these proteins (*Crook et al., 1992*).

P53 is a tumor suppressor gene present on chromosome 17P and control entry into the S phase of the cell cycle. It encodes a 53 Kilo Dalton (KD) nuclear phosphoprotein which is involved in the negative regulation of cell growth (*Holm et al., 1993*).

P53 gene mutation may lead to the development of inactive P53 protein which losses its tumor suppressor function and this may

play an important role in the development and progression of many tumors (*Oka et al., 1993. and Akasofu and Oda, 1995*).

Wild types of P53 protein have a very short half life and thus can not be detected immunohistochemically *Iggo et al., 1991*, however, the mutant forms are more stable and have an extended half life and can be easily detected by immunohistochemical methods. Thus P53 protein detected by immunohistochemical methods is in the mutant form (*Oka et al., 1993.*)

P53 protein has been identified immunohistochemically in a variety of tumors such as melanomas, soft tissue sarcomas, and in carcinomas of the breast, colon, lung, stomach, ovary and pancreas (*Holm et al., 1993.*)

Cytophotometric analysis of DNA had shed some light on the correlation between DNA ploidy pattern and the biological characteristics of individual tumors (*Baba et al., 1989*). It was concluded that there is a significant correlation between DNA ploidy pattern and tumor prognosis in cases of colon, prostatic and gastric carcinoma. Aneuploid tumors tend to manifest rapid growth, high incidence of nodal involvement and distant metastasis (*Banner et al., 1985*). Moreover the correlation between DNA ploidy pattern and cervical carcinoma is still contradictory. Although some researches studied the relationship between HPV, P53 , and DNA in cancer cervix, data on their prevalence, and their correlation in cervical carcinomas are still lacking.