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Human Papilloma Virus, Cellular Genetics and Susceptibility to Cervical Cancer

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ABSTRACT Research in relation to the etiology of cervical cancer has made substantial progress in the last two decades both in scientific and operational terms. In many countries, HPV is the most common sexually transmitted infection (STI) and cervical cancer remains the second most common cancer among women worldwide. Although high risk HPV infection has been identified as the primary etiological agent for cervical cancer, various co-factors such as tobacco derived carcinogens, inhalation of air cotaminated through the combustion of coal and kitchen smoke have also been reported to be associated with cervical cancer. Studies on HPV 16 E6 and E7 gene variations provide evidence for the association of specific E6 gene variants with the risk of cervical cancer. While E6 variants may be important in contributing to increased severity of cervical cancer, polymorphisms of other host cellular proteins, such as p53 and p73, may also play an as yet undefined role in modulating the E6-mediated carcinogenic process. Tobacco smoking and chewing has also been found to be associated with increased risk of cervical malignancy. Major classes of carcinogens present in tobacco and tobacco smoke are converted into DNA-reactive metabolites by cytochrome P450 (CYP)-related enzymes, several of which display genetic polymorphism. Individual susceptibility to cancer is likely to be modified by the genotype for enzymes involved in the activation or detoxification of carcinogens in tobacco and repair of DNA damage. Polymorphisms in the carcinogenmetabolizing enzymes are thought to play a role in cancer susceptibility in humans. Associations of polymorphisms with cancer risk will be especially important in cases where there are known exposures to chemical carcinogens such as with tobacco smoking, high intake of food mutagens and industrial exposures. HPV infection, through the modulation of cellular xenobiotic metabolizing enzymes, may play a role in the ability of cells to handle environmental carcinogens.

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