Review paper

# THE ROLE OF HERPES SIMPLEX VIRUS AND HUMAN PAPILLOMAVIRUSES IN TRIGGERING MALIGNANCY

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## ABSTRACT

Certain DNA viruses are important as known causes of cancers in humans. Oncogenesis by DNA viruses is best understood for polyoma-, papilloma- and adenoviruses. All of them contain genes that behave as oncogenes. As for herpesviruses there is suggestion that a cellular oncogene might be involved. A number of HSV-specific proteins are produced and expressed by the transformed cells. Herpes simplex virus type 2 was seriously implicated in the etiology of cancer of the cervix. During the past years the theory of herpes simplex virus oncogenesis has lost favour in the face of mounting evidence to incriminate human papillomaviruses in the same disease.

KEY WORDS

*herpes simplex virus, human papillomaviruses, tumor induction*

When discussing tumor induction by DNA viruses we must have in mind that DNA tumor viruses interact with cells in one of the two ways:

- productive infection - virus completes its replication cycle resulting in cell lyses;
- nonproductive infection - virus transforms the cell without completing its replication cycle; the viral genome is integrated into the cellular DNA or the complete genome persists as an autonomously replicating episome. The genome continues to express early gene functions (1).

The molecular basis of oncogenesis by DNA viruses is best understood for polyomaviruses, papillomaviruses and adenoviruses; all of them contain genes which behave as oncogenes. These oncogenes act primarily in the nucleus, where they alter patterns of gene expression and regulation of cell growth. In every case the relevant genes encode early proteins having dual role: in viral replication and in cell transformation (2).

As for human herpesviruses no specific herpesvirus-transforming gene has been identified. It is possible that herpesvirus-induced oncogenesis might be fundamentally different from transformation mediated by viruses that encode transforming proteins (3). Human herpesvirus type 2 (HHV-2) has been associated with cervical squamous cell carcinoma. These observations have been predicted upon the site of cancer and the known natural tendency of HHV-2 to infect this site and upon the excess of specific antibodies determined. In support of this