Abstract

Human papillomavirus is connected with induction of cervical carcinoma as well as for some other anogenital carcinomas and subset of carcinomas of head and neck. Presence of viral E6 and E7 oncoproteins may induce cell transformation, higher load of oncoproteins is caused by the regulatory E2 protein inactivation. Aims of recent study are mechanisms of E2 protein inactivation. One option is integration of viral DNA into the host genome, which is located into the *E2* gene region. Some carcinomas, where virus with extrachromosomal form was presented, were found. It appears that epigenetic changes can play the role in the development of this type of tumors, especially DNA methylation or mutation in the regulatory region of the virus. The methylation degree analysis on samples of tonsillar carcinomas with extrachromosomal and integrated form of the virus was conduced, as well as viral load of both groups was compared and the expression of E6 and E7 gene was confirmed. The results of methylation analysis showed increased methylation of the virus with integrated DNA. Mutations in the E2 protein binding sites are not revealed. The expression of the viral oncogenes were confirmed in all tumors regardless of the form of the viral genome. The mechanism of tumors induction, especially for virus with extrachromosomal form of the virus, remains a subject for further research.

Key words

Head and neck cancer, tonsillar carcinoma, HPV, human papillomavirus, DNA methylation, viral load, carcinogenesis, E2 binding sites.