International Journal of
Health Systems and Medical Science

ISSN: 2833-7433
Volume 1 | No 5 | Nov-2022

# Assessment of Local Cervical Immunity in Women with Neoplastic Cervical Processes

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**Abstract:** Numerous data of the scientific literature indicate polyetiology of pathological processes of the ecto- and endocervix, but the key factor in the etiopathogenesis of cervical cancer (CC) and cervical intraepithelial neoplasia (CIN) is certainly the human papillomavirus (HPV) infection. Epidemiological studies confirm the role of early sexual initiation, early first childbirth, and frequent changes in sexual partners, smoking, and a number of sexually transmitted infections in the occurrence of CIN and CRC.

**Keywords:** cervical intraepithelial neoplasia, cervical cancer, human papillomavirus, etiopathogenesis.

**Introduction.** High-risk human papillomavirus (HR-HPV) infection is a necessary but not sufficient cause of cervical cancer. In sexually active women, the lifetime incidence of cervical HPV infection is estimated to be 80%. Approximately 90% of these infections go away spontaneously after about 2 years. In contrast, when the infection persists (about 10% of infected women), the risk of progression to severe lesions and cervical cancer increases, especially in HPV- and 18-positive women. In the natural course of HPV infection, cervical intraepithelial neoplasia (CIN) 1 has a higher regression rate than CIN3, which is mainly associated with persistent HR-HPV infection. Similarly, the rate of progression of invasive cancer is lower for CIN1 (1%) than for CIN3 (>12%).The immune response is considered to be a cofactor that may play a role in different stages of the natural course of cervical cancer. Although tumour infiltrate lymphocytes (TILs) have been observed in preneoplastic and tumour tissues, it has been difficult to determine the role that these immune cells may play in providing regression of different degrees of CIN. Studies have shown an equal proportion of CD4+ and CD8+ cells in the stroma of preneo plastic lesions and a low CD4+ cell density in the epithelium of preneoplastic lesions compared to normal tissues. Moreover, in cervical cancer, high CD8+ TIL counts are associated with absence of metastases and low numbers of immune cell types with recurrence. Others report that a large number of CD8+ cells are present in invasive cervical cancer**.** In this study, we performed a thorough statistical analysis of the reproducibility of the number of three important immune cell types (CD8+, CD4+) and regulatory T cells (CD25+ Foxp3+) in precancerous and malignant cervical cancer lesions. Some authors gave a descriptive density analysis (number of cells per mm2) in infiltrates of these lesions and determined whether the density of cell subtypes in the stroma or epithelium is associated with any histological grade.

Studies of the cervical secretion as well as the cells composing the endocervix provided evidence of a functional and potentially important immunological system in the mucosa of this organ. The availability of cell biology tools as well as three agents that can be used as probes to experimentally infect the cervical mucosa has enabled a detailed approach to determining the structural and functional characteristics of local cervical immunity. The long-term goal of these studies is to

determine how the cervical immune response can be regulated in order to reduce local viral replication and virus-associated disease. Using Langerhans cells for antigen presentation, the cervical immune response usually persists for more than 30 days, is predominantly of the IgA isotype, can be influenced by oestrogen or progesterone, and is best induced by local rather than systemic exposure to the antigen. Cervical immune responses to human papillomavirus (HPV) are of particular importance in this respect, as this virus is associated with cervical neoplasia. While serum responses to HPV-16 L1, E4 and E7 proteins have been detected in 78% of individuals with HPV-related cervical neoplasia, data showing that a local response with comparable frequency consistently occurs remains to be confirmed. The current status of local HPV-16-specific immunoglobulin as a potentially useful indicator of HPV-16 infection or precancer is controversial and confounded by several potentially important factors, including patient age, estrogen/progesterone levels, smoking status. , and sample mix with serum immunoglobulin. Cervical immune responses to human papillomavirus (HPV) are of particular importance in this regard, as this virus is associated with cervical neoplasia. While serum responses to the L1, E4 and E7 HPV-16 proteins have been found in 78% of those with HPV-related cervical neoplasia, data showing that a local response with comparable frequency consistently occurs remains to be confirmed. The current status of local HPV-16-specific immunoglobulin as a potentially useful indicator of HPV-16 infection or precancer is controversial and confounded by several potentially important factors, including patient age, estrogen/progesterone levels, smoking status. , and sample mix with serum immunoglobulin. Cervical immune responses to human papillomavirus (HPV) are of particular importance in this regard, as this virus is associated with cervical neoplasia. While serum responses to the L1, E4 and E7 HPV-16 proteins have been found in 78% of those with HPV-related cervical neoplasia, data showing that a local response with comparable frequency consistently occurs remains to be confirmed. The current status of local HPV-16-specific immunoglobulin as a potentially useful indicator of HPV-16 infection or precancer is controversial and confounded by several potentially important factors, including patient age, estrogen/progesterone levels, smoking status. , and sample mix with serum immunoglobulin.

**Conclusions**: The cellular immune response is one of the determinants of the persistence or elimination of HPV infection and its evolution into precancerous lesions. Although the extent to which this occurs is not entirely clear, it is known that the progression of cervical neoplastic lesions contributes to a change in cytokine secretion patterns with the active involvement of regulatory T lymphocytes. Thus, the anti-tumour immune response requires a balance in cytokine production, as tumour progression or regression depends on the type and amount of cytokines secreted. Activation of dendritic cells, Langerhans cells and natural killer cells is required in response to HPV infection. With a focus on preventing early HPV infection, the use of prophylactic vaccines is already well established in clinical practice.

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