

Atlas of Exfoliative Cytology (Commonwealth Fund Publications)



Review

Deciphering the Multifactorial Susceptibility of Mucosal Junction Cells to HPV Infection and Related Carcinogenesis

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Abstract: Human papillomavirus (HPV)-induced neoplasms have long been considered to originate from viral infection of the basal cell layer of the squamous mucosa. However, this paradigm has been recently undermined by accumulating data supporting the critical role of a discrete population of squamo-columnar (SC) junction cells in the pathogenesis of cervical (pre)cancers. The present review summarizes the current knowledge on junctional cells, discusses their high vulnerability to HPV infection, and stresses the potential clinical/translational value of the novel dualistic model of HPV-related carcinogenesis.

Keywords: human papillomavirus; squamo-columnar junctions; (pre)neoplastic lesions

1. Introduction

Cancer of the uterine cervix is generally observed near the squamo-columnar (SC) junction and is almost always caused by carcinogenic human papillomaviruses (HPV). However, the nature of cervical epithelial cells initially infected by HPV has been a matter of debate/speculation for a long time. Classically, the HPV-target cells are presumed to be the basal keratinocytes of the pluristratified mucosa lining the outer part of the cervix (ectocervix) and the region where the endocervical columnar epithelium, sensitive to the acidic vaginal pH, has undergone a squamous metaplastic transformation during puberty (transformation zone (TZ)). According to this theory, microtrauma, ulcerative lesions, or abrasions into the squamous epithelium would render the proliferating basal cell layer uniquely vulnerable to viral infection [1]. This hypothesis was emphasized by (1) the detection of HPV transcripts in basal cells; (2) the basal location of dysplastic cells in the initial phase of malignancy (low-grade cervical intraepithelial neoplasia (CIN1)); and (3) the observation that chemical disruption of the integrity of the stratified epithelium is required for pseudovirion infection of the murine genital tract [2]. Obviously, it is indisputable that transcriptionally-active HPV infection can occur in basal keratinocytes and result in the subsequent development of (pre)neoplastic lesions in both the ectocervix and the lower genital tract (vagina/vulva). This mechanism is also likely to explain the HPV infections occurring in both the oral cavity and the anal margin/perianal skin (microtrauma resulting from mastication and anal intercourse, respectively) because these two sites are entirely lined by a pluristratified epithelium. However, this hypothesis is incompatible with the historical observation that HPV infection causes cervical cancer and its precursor lesions mainly within the SC junction microenvironment [3,4]. In addition, this theory does not explain the discrepancy in terms of cancer risk between the uterine cervix and other HPV-infected

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