

Publication

Assessment of EGFR and TGF-alpha expression in relationship to HPV status and Ki-67 distribution in cervical intraepithelial neoplasms

JournalArticle (Originalarbeit in einer wissenschaftlichen Zeitschrift)**ID** 56698**Author(s)** Dellas, A; Schultheiss, E; Almendral, A C; Torhorst, J; Gudat, F**Author(s) at UniBasel** [Dellas, Athanassios](#) ;**Year** 1996**Title** Assessment of EGFR and TGF-alpha expression in relationship to HPV status and Ki-67 distribution in cervical intraepithelial neoplasms**Journal** International journal of cancer**Volume** 69**Number** 3**Pages / Article-Number** 165-9

Expression of epidermal-growth-factor receptor (EGFR), transforming growth factor alpha (TGF-alpha) and Ki-67 proliferation antigen in cervical intra-epithelial neoplasms were analyzed. To examine the interrelationship of TGF-alpha, EGFR, Ki-67 and HPV status in dysplasia and carcinoma in situ, formalin-fixed tissue sections of 92 women were immunostained with monoclonal antibodies to EGFR, TGF-alpha and Ki-67. The presence of HPV was assessed by in situ DNA hybridization. The highest positive TGF-alpha expression was seen in the group of mild dysplasia. The difference was significant between the relatively high expression in mild dysplasia and the low occurrence in severe dysplasia and carcinoma in situ as well. The same relation could be found between TGF-alpha expression in papilloma-virus-negative dysplasia and those with the presence of HPV 16/18. In contrast to these findings, the Ki-67 proliferation marker was intensely detectable in severe dysplasia and carcinoma in situ. Ki-67-stained neoplastic cell nuclei were found in a significantly higher percentage of HPV-positive than in HPV-negative lesions. TGF-alpha over-expression is obviously combined with low proliferating activity and vice versa. Irrespective of the grade of dysplasia or HPV status, EGFR was expressed abnormally as compared with normal squamous epithelium. Over-expression of TGF-alpha in mild dysplasia could be associated with the autocrine pathway of cell-growth regulation. In the presence of HPV 16/18 the EGFR/TGF-alpha pathway for growth stimulation is probably not involved.

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