Lacking hypoxia-mediated downregulation of E-cadherin in cancers of the uterine cervix.

Experimental studies have established a causal connection between tumour hypoxia, hypoxia-associated proteome changes and downregulation of E-cadherin, the final common pathway of epithelial-to-mesenchymal transition (EMT). Our study aimed at elucidating the interrelationship of these processes in cancers of the uterine cervix in vivo. Tumour oxygenation was assessed in 48 squamous cell carcinomas (SCC) of the uterine cervix using polarographic needle electrodes. The expression pattern of E-cadherin was investigated by immunohistochemistry and western blotting, and was compared with that of the hypoxia-inducible proteins glucose transporter (GLUT)-1 and carbonic anhydrase (CA) IX in biopsy specimens of the oxygenation measurement tracks. The majority of cervical cancers (52%) were E-cadherin positive, with a complete absence of the antigen in only 10% of the tumours. No correlation was found between the level of E-cadherin expression and the oxygenation status (mean pO(2), median pO(2) and hypoxic fractions). In patients showing partial expression of E-cadherin (38%), staining was not preferentially diminished in GLUT-1- or CA IX-positive areas, and loss of E-cadherin occurred independently of tumour cell scattering. Our data provide no evidence in favour of a hypoxia-induced EMT as a mechanistic basis of cervical cancer invasiveness.