# Epithelial to Mesenchymal Transition (EMT) in Pathogenesis of Endometriosis

**Mecha, E.[1,2], Omwandho, C. O.A.[2,3], Cong Sui[1], Hans-R. T[1], Konrad, L[1].**
**[1] Justus-Liebig University, Germany; [2]University of Nairobi, [3]Kirinyaga University, Kenya.**
***Correspondence:*** *ezekiel_mecha@yahoo.com*

**Abstract**

Epithelial-mesenchymal transition (EMT) is characterized by loss of epithelial and acquisition of mesenchymal cell phenotype. The aim of this study was to assess the epithelial phenotype in pathogenesis of endometriosis by performing IHC studies with epithelial and mesenchymal markers. We compared endometrium with and without endometriosis to peritoneal, ovarian and deep infiltrating endometriosis (DIE) with two structural (keratin-18, -19), one membrane-associated (mucin-1) and one mesenchymal protein (vimentin) to analyze the epithelial and mesenchymal phenotype of endometrial glands and endometriotic lesions. Quantitation with the HSCORE showed no differences for keratin-18 (K18), keratin- 19 (K19) and mucin-1 (MUC1) between endometrium with and without endometriosis. Also, K18 expression was not different between endometrium and endometriotic lesions. In contrast, K19 and MUC1 were significantly decreased in the endometriotic lesions compared to endometrium. However, all three proteins were found in almost every endometrial and endometriotic gland or cyst and in nearly all epithelial cells. Protein expression of vimentin was lower in the endometriotic lesions compared to endometrium, especially in the ovary. Expression of the epithelial markers in nearly all glands and epithelial cells in endometrium endometriotic entities clearly indicates no loss of epithelial cell phenotype. Additionally, the reduced expression of vimentin in the endometriotic lesions, suggests no shift of the epithelial phenotype to amesenchymal one. We propose that EMT is not a main factor in the pathogenesis of endometriosis.

**Keywords:** *Epithelial, Mesenchymal Transition (EMT), Pathogenesis, Endometriosis*