

Upregulation of CFTR in patients with endometriosis and its involvement in NF κ B-uPAR dependent cell migration.

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Abstract

Endometriotic tissues exhibit high migration ability with the underlying mechanisms remain elusive. Our previous studies have demonstrated that cystic fibrosis transmembrane conductance regulator (CFTR) acts as a tumor suppressor regulating cell migration. In the present study, we explored whether CFTR plays a role in the development of human endometriosis. We found that both mRNA and protein expression levels of CFTR and urokinase-type plasminogen activator receptor (uPAR) were significantly increased in ectopic endometrial tissues from patients with endometriosis compared to normal endometrial tissues from women without endometriosis and positively correlated. In human endometrial Ishikawa (ISK) cells, overexpression of CFTR stimulated cell migration with upregulated NF κ B p65 and uPAR. Knockdown of CFTR inhibited cell migration. Furthermore, inhibition of NF κ B with its inhibitors (curcumin or Bay) significantly reduced the expression of uPAR and cell migration in the CFTR-overexpressing ISK cells. Collectively, the present results suggest that the CFTR-NF κ B-uPAR signaling may contribute to the progression of human endometriosis, and indicate potential targets for diagnosis and treatment.