Involvement of the nuclear factor-?B pathway in the pathogenesis of endometriosis

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Objective: To evaluate the role of nuclear factor-?B (NF-?B) in the pathogenesis of endometriosis. Design: A literature search was conducted in PubMed to identify all relevant citations. Result(s): Our findings highlight the important role of NF-?B in the pathophysiology of endometriosis. In vitro and in vivo studies show that NF-?B-mediated gene transcription promotes inflammation, invasion, angiogenesis, and cell proliferation and inhibits apoptosis of endometriotic cells. Constitutive activation of NF-?B has been demonstrated in endometriotic lesions and peritoneal macrophages of endometriosis patients. Agents blocking NF-?B are effective inhibitors of endometriosis development and some drugs with known NF-?B inhibitory properties have proved efficient at reducing endometriosis-associated symptoms in women. Iron overload activates NF-?B in macrophages. NF-?B activation in macrophages and ectopic endometrial cells stimulates synthesis of proinflammatory cytokines, generating a positi