Elucidating the Expression Profile of EZH2 Isoforms in Endometriosis: An immunohistochemical study

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Elucidating the Expression Profile of EZH2 Isoforms in Endometriosis: An immunohistochemical study

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Abstract

Endometriosis is an estrogen-dependent gynecological disease that affects 1 out of 10 women of reproductive age causing severe pelvic pain and infertility. Factors including genetics, environment, inflammation, and recently epigenetics have been shown to play roles in the pathophysiology of this disease. Histone methylation is an epigenetic modification that modulates gene expression by causing changes in the chromatin structure. Trimethylation of histone 3 at lysine residue 27 (H3K27me3) is a histone mark related to gene repression. EZH2 is the histone methyltransferase (HMT) responsible of catalyzing H3K27me3. It has been shown that the EZH2 is involved in carcinogenesis; however, the specific role of EZH2 in endometriosis is unknown. This is important because there are drugs available that block this enzyme's functioning, and could serve as a potential new treatment.

We have previously shown that endometriotic lesions are characterized by high H3K27me3 nuclear immunostaining. Therefore, we hypothesize that EZH2 will be highly expressed in lesions compared to endometrium of patients and controls. Using immunohistochemistry (IHC) of an endometriosis Tissue Micro Array (TMA), EZH2α and β nuclear intensity were assessed using specific antibodies. We observed that pelvic endometriotic lesions (peritoneal and fallopian tube) have higher EZH2α intensity scores compared to control tissues. EZH2β nuclear immunostaining analysis is ongoing. We expect to observe a higher nuclear intensity score in endometriotic lesions compared to endometriosis free endometria.

This study is the first to analyze the expression profile of EZH2 isoforms in endometriosis. These studies will help better understand the role of EZH2 in this disease.

Keywords

EZH2; H3K27me3; endometriosis; epigenetic

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ABSTRACT

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