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Short communication

Association of *FCRL3* C-169T promoter single-nucleotide polymorphism with idiopathic infertility and infertility-related endometriosis

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ABSTRACT

An aberrant immunological mechanism is thought to be involved in the pathogenesis of endometriosis. The present study aimed to determine whether there is a relationship between endometriosis and/or infertility and the *FCRL3* C-169T polymorphism. This case-control study included 167 infertile women with endometriosis, 60 women with idiopathic infertility and 167 fertile women. Detection of the *FCRL3* C-169T polymorphism was performed using TaqMan PCR. A significant difference in the genotype and allele frequencies of the *FCRL3* C-169T polymorphism between endometriosis-related infertility ($p=0.003$ and $p=0.001$) and idiopathic infertility ($p=0.027$ and $p=0.0185$) versus controls was demonstrated. In conclusion, the results suggest that the *FCRL3* C-169T polymorphism may play an important role in the pathogenesis of endometriosis and/or infertility.

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1. 1. Introduction

Several studies suggest that aberrant immunological mechanisms are involved in the pathophysiology of endometriosis. The presence of specific anti-endometrial antibodies and generalized polyclonal B-cell autoimmune activation has been found in cases of endometriosis. Genetic factors are known to play a role in the pathogenesis of endometriosis, and therefore autoimmunity genes can reasonably be considered candidate genes (Nothnick, 2001).

Recently, the gene encoding Fc receptor-like 3 (*FCRL3*) was proposed as a novel autoimmune predisposing fac-

tor (Chistiakov and Chistiakov, 2007). The *FCRL3* protein is an orphan cell surface receptor with homology to the Fc immunoreceptors and is expressed predominantly in B lymphocytes of lymph nodes and germinal centers (Davis et al., 2002). A single nucleotide polymorphism (C-169T, rs7528684), located in the promoter region of this gene, was reported to be associated with rheumatoid arthritis, systemic lupus erythematosus, Graves' disease and Hashimoto thyroiditis (Kochi et al., 2005).

So far, there is no information in the literature about the *FCRL3* protein in the pathogenesis of endometriosis and/or infertility. It is well known that the *FCRL3* C169T polymorphism alters the expression of *FCRL3* by affecting the binding affinity of nuclear factor-kappa B (NF- κ B). Therefore, an increased level of *FCRL3* protein, which is correlated with the 169C susceptibility allele, may result in B-cell abnormalities and a higher level of autoantibodies. With regard to endometriosis, there is no doubt that B-lymphocyte abnormalities are responsible for

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