**Abstract**

**Background:** Patients with endometriosis often experience infertility and have poor IVF outcomes, with low fertilization and pregnancy rates. Although many theories have tried to explain the mechanisms underlying infertility in these patients, none of them is conclusive.

**Objective and rationale:** In this review, we discuss the pathologic mechanisms through which endometriosis likely leads to infertility along with the therapeutic options used to date to treat endometriosis-related infertility and, thereby, to improve IVF outcomes in patients with endometriosis.

**Search methods:** We performed a comprehensive literature search of clinical outcomes in endometriosis and the molecular mechanisms contributing to oocyte quality using the PubMed database to identify human and animal studies published from 1992 until September 2020. In total, 123 manuscripts were included.

**Outcomes:** While some theories propose that endometriosis patients may have fertility problems as a result of decreased endometrial receptivity, others reinforce the idea that infertility could be associated with oocyte alterations and lower implantation rates. Single-cell RNA sequencing of oocytes from patients with endometriosis has identified dysregulated mechanisms involved in steroid metabolism and biosynthesis, response to oxidative stress and cell cycle regulation. Dysregulation of these mechanisms could result in the poor IVF outcomes observed in patients with endometriosis. Further, impaired steroidogenesis may directly affect oocyte and embryo quality. Increased oxidative stress in patients with endometriosis also has a detrimental effect on the follicular microenvironment, inducing cell cycle dysregulation in oocytes, poor oocyte quality, and infertility. Moreover, granulosa cells in the context of endometriosis undergo increased apoptosis and have an altered cell cycle that could adversely affect folliculogenesis, oocyte and embryo quality, and IVF outcomes. Endometriosis is also associated with inflammatory damage and impaired angiogenesis, which could be directly correlated with poor IVF outcomes. While therapeutic options using GnRH analogues, progestins and aromatase inhibitors do not improve endometriosis-related infertility, anti-inflammatory agents and antioxidant supplementation could improve oocyte quality as well as implantation and clinical pregnancy rates in patients with endometriosis.

**Wider implications:** Endometriosis is a heterogeneous disease whose pathogenesis is complex and could affect fertility by altering a collection of molecular mechanisms in oocytes. Thus, a single model is not sufficient to describe endometriosis-related infertility. Dysregulation of steroidogenesis, oxidative stress, cell cycle progression, inflammation and angiogenesis in the follicular environment and oocytes in individuals with endometriosis are all possible contributors to endometriosis-related infertility. Therefore, treatments targeting these mechanisms could be therapeutic alternatives to improve IVF outcomes for these patients.