LncRNA AFAP1-AS1 regulates proliferation and apoptosis of endometriosis through activating STAT3/TGF-β/Smad signaling via miR-424-5p

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Aim: Endometriosis is a common gynecological disorder characterized by chronic pelvic pain and infertility, which negatively affects women's health worldwide. AFAP1-AS1 has been implicated in endometriosis lesions recently, but its mechanism of endometriosis progression remains unclear.

Methods: Endometrial stromal cells (ESCs) were used to identify the role of AFAP1-AS1 in endometriosis. The migratory capability was determined by transwell. Gene and protein expressions were identified by quantitative real-time polymerase chain reaction (qRT-PCR) and Western blotting. Cell viability and apoptosis were detected by MTT assays and flow cytometry, respectively. Luciferase report assays were used to identify the interaction of AFAP1-AS1, miR-424-5p and signal transducer and activator of transcription 3 (STAT3).

Results: AFAP1-AS1 knockdown or miR-424-5p overexpression inhibited proliferation and migration, and promoted apoptosis in ESCs. In addition, knockdown of AFAP1-AS1 repressed the expression of ki-67 and Bcl-2, and promoted the levels of cleaved caspase-3 and Bax. Furthermore, knockdown of AFAP1-AS1 inhibited the conversion of E-cadherin to N-cadherin and the expression of Snail. Moreover, AFAP1-AS1 activated the STAT3/transforming growth factor-β1 (TGF-β1)/Smad2 axis via directly targeting miR-424-5p. The regulatory effect of AFAP1-AS1 silencing in ESC migration, proliferation, and apoptosis was reversed by miR-424-5p inhibition or STAT3 overexpression.

Conclusions: AFAP1-AS1 silencing could inhibit cell proliferation and promote apoptosis by regulating STAT3/TGF-β/Smad signaling pathway via targeting miR-424-5p in ESCs. AFAP1-AS1 may be a potential therapeutic target of controlling the progression of endometriosis.

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