**Abstract**

**Background:** Endometriosis is a chronic hormonal inflammatory disease characterized by the presence of endometrial tissue outside the uterus. Endometriosis often causes infertility, which brings physical and mental pain to patients and their families.

**Methods:** We examined the functions of heat shock factor 1 (HSF1) in endometriosis development through cell count assay, cell-scratch assay and clone formation experiments. We used quantitative real-time PCR (qRT-PCR) and Western blot (WB) to detect HSF1 expression. Glucose and lactate levels were determined using a glucose (GO) assay kit and a lactate assay kit. Furthermore, we used a HSF1 inhibitor-KRIBB11 to establish a mouse model of endometriosis.

**Results:** Our data demonstrated that HSF1 promoted endometriosis development. Interestingly, HSF1 enhanced glycolysis via up-regulating PFKFB3 expression in endometriosis cells, which was a key glycolysis enzyme. Consistently, the HSF1 inhibitor KRIBB11 could abrogate endometriosis progression in vivo and in vitro.

**Conclusions:** Findings indicate that HSF1 plays an important role in endometriosis development, which might become a new target for the treatment of endometriosis.