**Abstract**

Endometriosis (EM) is a common gynecological disease, and its pathological process is accompanied by the migration and proliferation of uterine cells. Berberine (BBR) has been shown to exhibit antitumor activity; however, the effects of BBR on EM have seldom been reported to date. The expression of microRNA (miR)‑429 is upregulated in EM and miR‑429 can be used as a target for drug regulation of cancer cells. Whether BBR plays a regulatory role in EM by targeting miR‑429 has not been reported. Thus, the aim of the present study was to determine the effects of BBR on EM cells. The survival rate of immortalized human endometrial stromal cells (HESCs) was determined using a Cell Counting Kit‑8 assay. A colony formation assay was used to detect the rate of cell proliferation. The expression levels of proliferation‑related proteins, including proliferation marker protein Ki‑67 (Ki‑67) and proliferating cell nuclear antigen (PCNA), were detected by reverse transcription‑quantitative PCR (RT‑qPCR) and western blotting. Wound healing and Transwell assays were performed to detect cell migration and invasion, and western blotting was used to detect the expression of the migration‑ and invasion‑related proteins, including matrix metalloproteinase (MMP)2, MMP4 and MMP9. The expression of miR‑429 was detected by RT‑qPCR following its overexpression via cell transfection. The results revealed that treatment with 80 µM BBR significantly inhibited cell proliferation and colony formation, and inhibited the expression of Ki‑67 and PCNA proteins in HESCs. BBR inhibited cell invasion and migration, as well as the expression of MMP2, MMP4 and MMP9. In this process, it was found that the expression of miR‑429 decreased following treatment of the cells with BBR, whereas the inhibitory effects of BBR on cell proliferation, invasion and migration were suppressed following the overexpression of miR‑429. Overall, the findings of the present study indicated that BBR inhibited the proliferation, invasion and migration of HESCs by downregulating the expression of miR‑429.