**Abstract**

Adenomyosis (ADS) is an estrogen-dependent gynecological disease with unspecified etiopathogenesis. Local hyperestrogenism may serve a key role in contributing to the origin of ADS. Talin1 is mostly identified to be overexpressed and involved in the progression of numerous human carcinomas through mediating cell proliferation, adhesion and motility. Whether Talin1 exerts an oncogenic role in the pathogenesis of ADS and puts an extra impact on the efficacy of estrogen, no relevant data are available yet. Here we demonstrated that the adenomyotic eutopic and ectopic endometrial stromal cells (ADS\_Eu\_ESC and ADS\_Ec\_ESC) treated with β-estradiol (β-E2) presented stronger proliferative and pro-angiogenetic capacities, accompanied by increased expression of PCNA, Ki67, VEGFB and ANGPTL4 proteins. Meanwhile, these promoting effects were partially abrogated by Fulvestrant (ICI 182780, an estrogen-receptor antagonist). Aberrantly upregulation of Talin1 mRNA and protein level was observed in ADS endometrial specimens and stromal cells. Through performing functional experiments in vitro, we further determined that merely overexpression of Talin1 (OV-Talin1) also enhanced ADS stromal cell proliferation and pro-angiogenesis, while the most pronounced facilitating effects were found in the co-intervention group of OV-Talin1 plus β-E2 treatment. Results from the xenograft nude mice model showed that the hypodermic endometrial lesions from co-intervention group had the highest mean weight and volume, compared with that of individual OV-Talin1 or β-E2 treatment. The expression levels of PCNA, Ki67, VEGFB and ANGPTL4 in the lesions were correspondingly elevated the most in the co-intervention group. Our findings unveiled that overexpressed Talin1 might cooperate withβ-E2 in stimulating ADS endometrial stromal cell proliferation and neovascularization, synergistically promoting the growth and survival of ectopic lesions. These results may be beneficial to provide a new insight for clarifying the pathogenesis of ADS.