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FSH Receptor Binding Inhibitor Impacts K-Ras and c-Myc of Ovarian Cancer and Signal Pathway

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The present study aimed to investigate FSH receptor binding inhibitor (FRBI) effects on relative factors (K-Ras, c-Myc and Vascular endothelial growth factor (VEGF)) to ovarian cancer and expression levels of FSH receptor (FSHR) mRNAs and proteins in the cumulus-oocyte complex (COCs), to determine changes of protein kinase A (PKA) in sheep granulosa cells, further to elucidate signaling pathway of FRBI action. COCs were cultured in vitro for 24h under supplementation of varying concentrations of FRBI (0, 10, 20, 30 and 40µg/mL) or FSH (10IU/mL). Concentrations of K-Ras, c-Myc, VEGF, cAMP and FSH were detected in IVM media fluids, respectively. The results showed that the concentrations of c-Myc, K-Ras and FSH of FRBI groups were gradually reduced with the increase of FRBI doses. VEGF level of the FRBI-4 group was significantly greater than control group (CG). Expression levels FSHR mRNA and protein and PKA of FRBI-3 and FRBI-4 groups were less than that of CG or FSH group ($P<0.05$ or $P<0.01$). Inositol trisphosphate (IP3) concentrations of FRBI-3 and FRBI-4 groups were less than FSH group ($P<0.05$). FRBI administration doses had significant negative correlations to levels or concentrations of K-Ras, c-Myc, VEGF, FSHR mRNA and protein and PKA protein. K-Ras had significant positive correlations with FSHR mRNA and protein and PKA protein. In conclusion, FRBI could promote the production of VEGF of sheep COCs. Higher doses of FRBI (30 and 40µg/mL) suppressed the production of c-Myc and K-Ras and declined FSH concentrations in the IVM medium fluid and decreased the expressions of FSHR at the gene and protein levels, additionally attenuated expression of PKA protein in the granulosa cells.

Keywords: FSH receptor binding inhibitor; Ovarian cancer; K-Ras; c-Myc; Protein expression; Signal pathway