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P51. Potential role of MicroRNA-32 on the proliferation of granulosa cells in polycystic ovarian syndrome women

Y li (CN) [1], y li (CN) [2], s wang (CN) [3], x yang (CN) [4]

Context: Polycystic ovarian syndrome (PCOS) is a common endocrine disorder accompanied with hyperandrogenism, chronic anovulation and polycystic ovary. The elevated androgen levels are generally considered the key pathological factor, which affects the growth, development, selection and atresia of follicles in ovary. Though differential microRNA (miRNA) expression patterns have been identified in several ovarian disorders, little is known about the role of miRNAs in the pathogenesis of PCOS. Objective: Here we focused on studying miR-32 expression patterns in PCOS women and explore the influence both of miR-32 on the proliferation in ovarian granulosa cells. Methods: MiRNA microarray and RT-qPCR were used to examine miR-32 expression in serum and granulosa cells from normal cycling and PCOS women. Bioinformatics analysis was used to analyze the target gene of miR-32. Patient(s): The normal cycling and PCOS women. Intervention(s): Primary human granulosa cells were treated with miR-32 up and down lentivirus vectors. Main Outcome Measure(s): The proliferation of granulosa cells was examined by MTT assay and the expression of Smad2 were determined by Western blotting (WB) and real time polymerase chain reaction (PCR). Results: MiR-32 was up-regulated in both serum and granulosa cells of PCOS women. Bioinformatics analysis demonstrated that Smad2 was the target gene of miR-32. Cell proliferation of miR-32 up lentivirus vector group was decreased significantly compared to the miR-32 down and the negative control lentivirus vector groups. Smad2 expression was decreased after miR-32 up lentivirus treatment. Conclusions: Up-regulation of miR-32 may contribute to the pathogenesis of PCOS. MiR-32 suppresses the proliferation of human granulosa cells by down-regulating Smad2.

[1] Beijing Obstetrics and Gynecology Hospital, Capital Medical University, Beijing, [2] Beijing Obstetrics and Gynecology Hospital, Capital Medical University, Beijing, [3] Beijing Obstetrics and Gynecology Hospital, Capital Medical University, Beijing, [4] Beijing Obstetrics and Gynecology Hospital, Capital Medical University, Beijing