12° CONGRESS of the EUROPEAN SOCIETY of Gynecology
BARCELONA 18/21 OCTOBER 2017

P43. HEPATOCYTE NUCLEAR FACTOR-1 BETA (HNF-1Î') INHIBITS OXIDATIVE STRESS-INDUCED APOPTOSIS IN HUMAN ENDOMETRIOTIC CELLS

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Endometriosis has been considered as a clonal precursor for ovarian clear-cell carcinoma. Recently, it has been demonstrated that HNF-1Î² is overexpressed in endometriosis as well as ovarian clear cell carcinoma. However, biological role of HNF-1? in endometriosis were poorly characterized. In the present study, we investigated the effect of HNF-1? on oxidative stress-induced apoptosis of endometriotic cells and its molecular mechanism of action. To assess cell viability and apoptosis, we performed MTT assays and FACS analysis using Annexin and PI staining. We also investigated transcriptional activation of NF-kB and expression of NF-kB-dependent anti-apoptotic genes using luciferase assay and Western blot analysis, respectively. HNF-1? siRNA was used to elucidate the molecular mechanism of action of the gene. We found that HNF-1? downregulation using siRNA significantly increase the reactive oxygen species (ROS) stress-induced apoptosis of endometriotic cells. In addition, knockdown of the HNF-1? significantly suppressed the transcriptional activation of NF-kB in endometriotic cells. Moreover, HNF-1? regulated the expression of NF-kB-dependent anti-apoptotic genes in endometriotic cells. In conclusion, these data suggest that HNF-1? confers ROS resistance in endometriotic cells by regulating the NF-kB pathway.

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