Figure S1. Western blot analysis of DRP1 in the CON and PCOS groups (n=12 vs. n=12). DRP1, dynein-related protein 1; CON, normal ovarian reserve; PCOS, polycystic ovary syndrome.

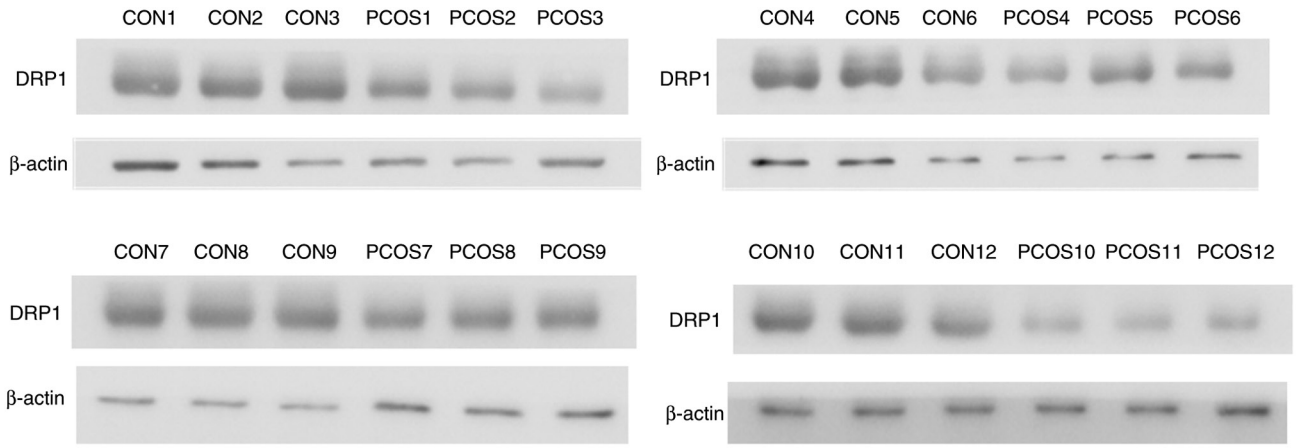

Figure S2. Western blot analysis of FISI in the CON and PCOS groups (n=18 vs. n=18). FIS1, mitochondrial fission 1; CON, normal ovarian reserve; PCOS, polycystic ovary syndrome.

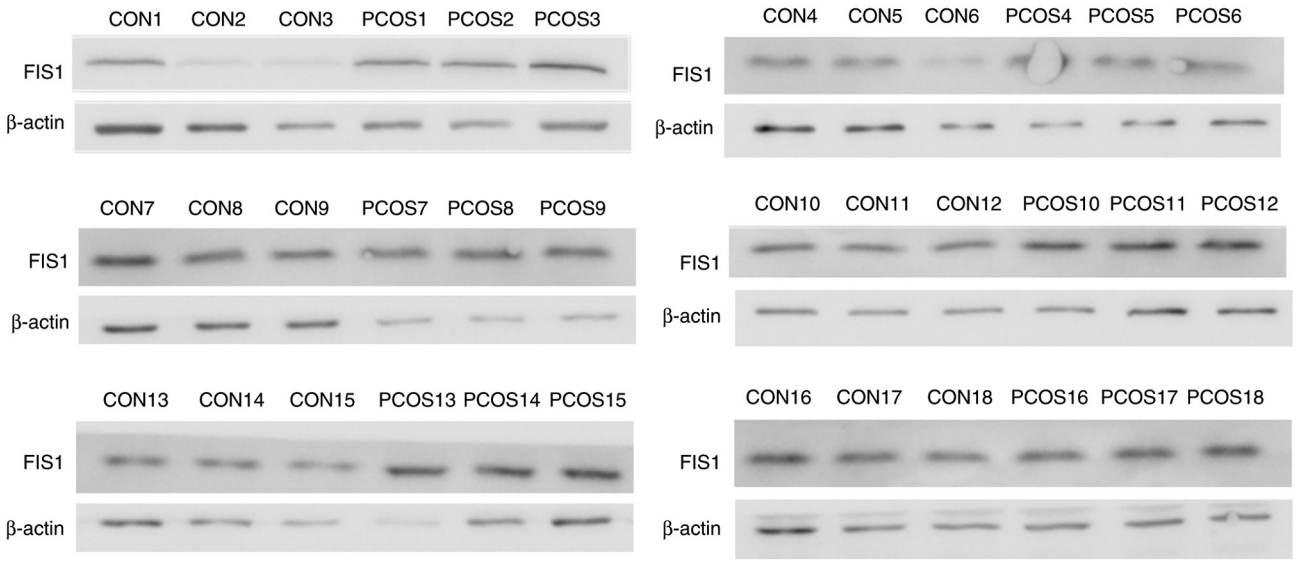

Figure S3. Western blot analysis of OPA1 in the CON and PCOS groups (n=21 vs. n=21). OPA1, optic nerve atrophy; CON, normal ovarian reserve; PCOS, polycystic ovary syndrome.

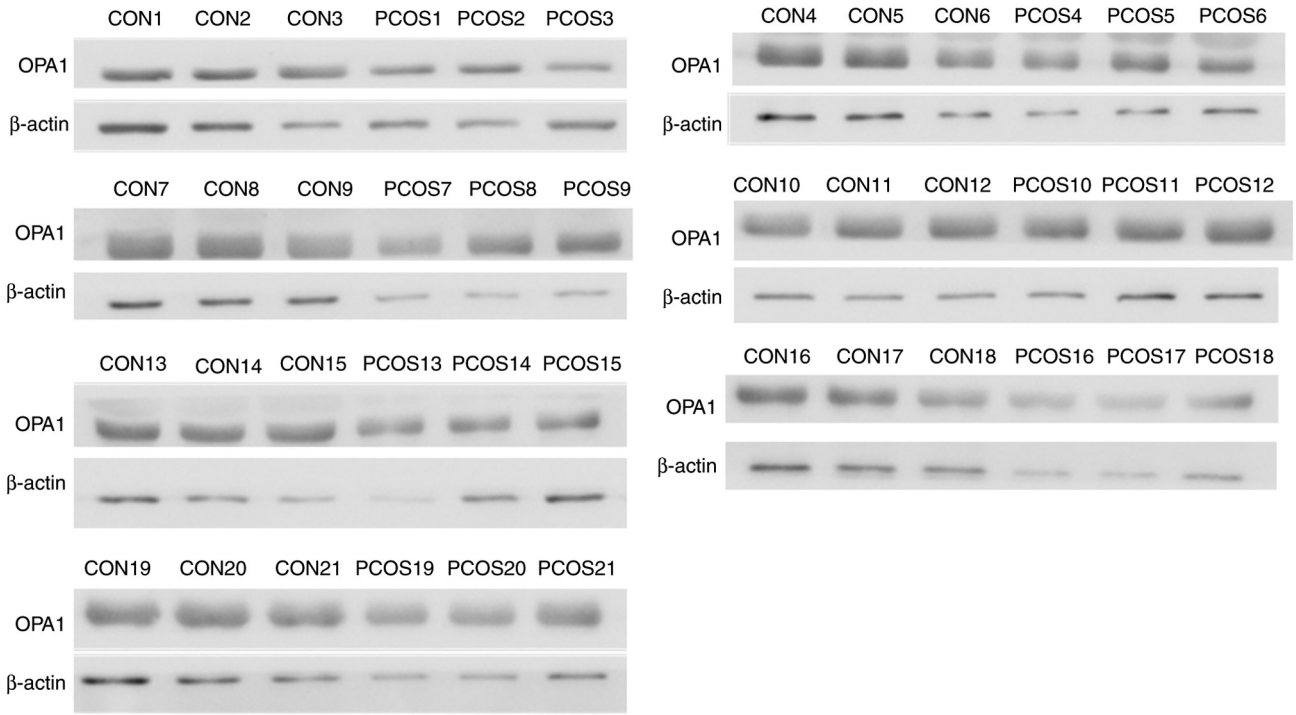

Figure S4. Western blot analysis of MFN2 in the CON and PCOS groups (n=27 vs. n=27). MFN2, mitofusin 2; CON, normal ovarian reserve; PCOS, polycystic ovary syndrome.

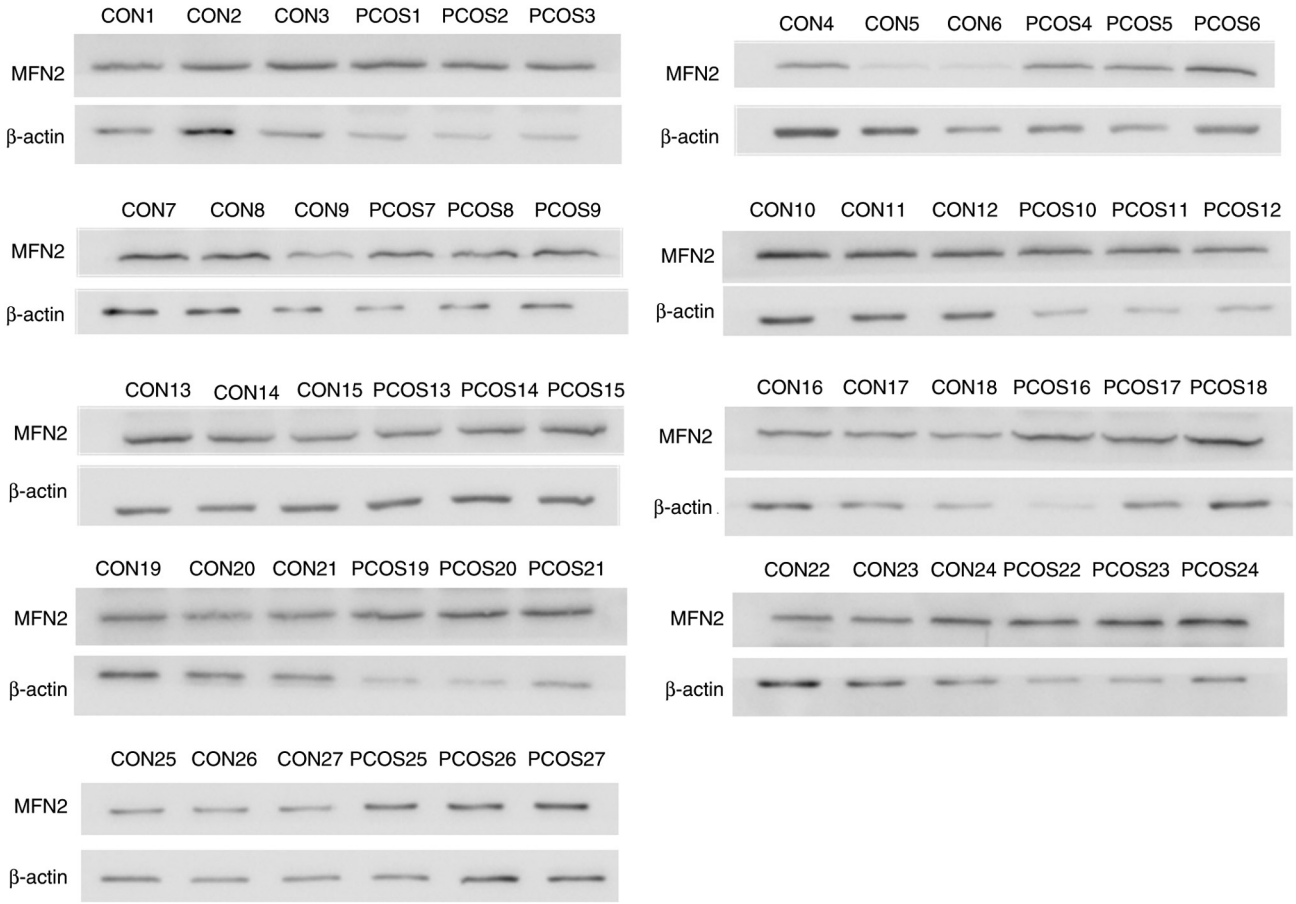

Figure S5. Western blot analysis of SIRT1 in the CON and PCOS groups (n=6 vs. n=6). SIRT1, silent information regulator 1; CON, normal ovarian reserve; PCOS, polycystic ovary syndrome.

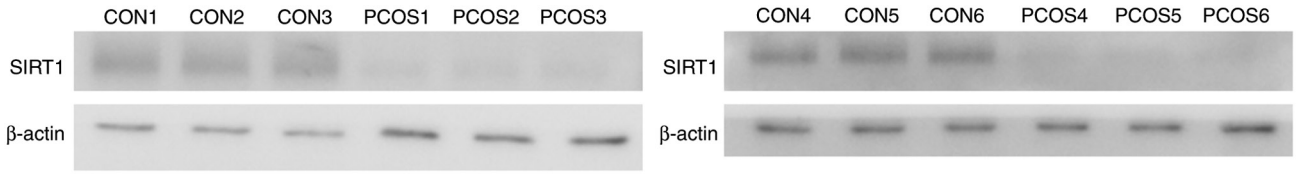

Figure S6. Western blot analysis of AMPK in the CON and PCOS groups (n=12 vs. n=12. AMPK, AMP-activated protein kinase; CON, normal ovarian reserve; PCOS, polycystic ovary syndrome.

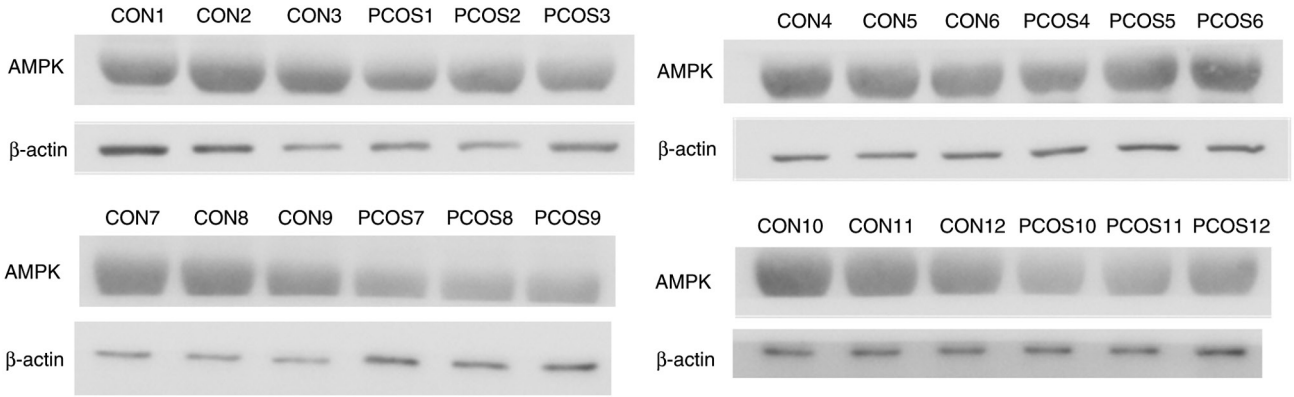

Figure S7. Western blot analysis of PGC-1α in the CON and PCOS groups (n=6 vs. n=6). PGC-1α, peroxisome proliferator-activated receptor-γ coactivator 1α; CON, normal ovarian reserve; PCOS, polycystic ovary syndrome.

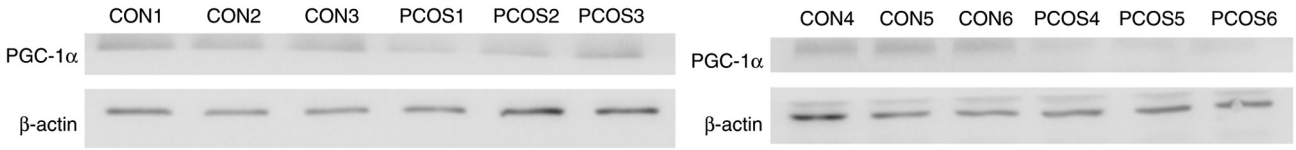

Figure S8. Western blot analysis of P-AMPK in the CON and PCOS groups (n=6 vs. n=6). P-AMPK, phosphorylated-AMP-activated protein kinase; CON, normal ovarian reserve; PCOS, polycystic ovary syndrome.

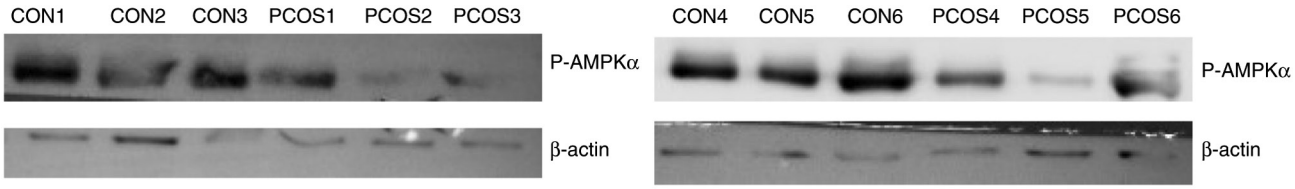

Figure S9. Schematic summary of the mitochondrial dysfunction in impaired GCs from patients with PCOS and the role of mitochondrial biogenesis underlying the process. Mitochondria in GCs from patients with PCOS could not sustain ATP synthesis but generated excessive ROS, promoting mitochondrial damage (mitochondrial membrane potential and mitochondrial number were decreased, and mitochondrial dynamics were disturbed) and eventually impairing the oocyte quality. In addition, the SIRT1/P-AMPK/PGC1α pathway expression was decreased. It was identified that the low expression of SIRT1/P-AMPK/PGC-1α pathway proteins may lead to decreased expression of mitochondrial transcription factors in GCs, which in turn affects mitochondrial function and ultimately affects the quality of oocytes. ROS, reactive oxygen species; SIRT1, silent information regulator 1; P-AMPK, phosphorylated-AMP-activated protein kinase; PGC1α, peroxisome proliferator-activated receptor-γ coactivator 1α; GC, granulosa cell; PCOS, polycystic ovary syndrome.