disorder. It affects 5~10% of women of reproductive age. Significant number of PCOS patients show impaired glucose tolerance and are in potential risk of developing type 2 diabetes. Approximately half of women with PCOS are known to be obese or overweight. Obesity is associated with gonadotropin dynamics, hyperandrogenism, insulin resistance, and disorders of spontaneous ovulation. Mitochondria have the function to support the cell energy. PCOS may be associated with the number of mitochondrial DNA (mtDNA) copies. We analyzed quantification of the mtDNA to investigate the relationship of mitochondria supporting the cell energy and pathogenesis of PCOS.

Method: Peripheral blood samples were collected from 28 patients with PCOS, who were under the inclusion criteria for PCOS, and from 28 healthy controls. Each sample was quantified 10 μg/ml for PCR by nano drop. Genomic DNA was used to analyze real-time PCR for mitochondrial DNA quantification. The PCR reaction was performed in duplicate using the MJ Research Opticon 2. The mitochondrial copy number was compared between the control and PCOS groups. All data was expressed as mean ± SD. Statistical analysis was assessed by t-test.

Results: In this study, the mtDNA CT was 11.67±0.422 in PCOS patients and 11.51±0.722 in control group, respectively. The mtDNA copy number was 1726410.71±407858.591 in PCOS patients and 2167887.5±1252459.28 in control group ($p=0.08$), respectively. Using t-test, no significant difference between PCOS patients and control group was found.

Conclusions: We imply that mtDNA copy number affect the development of PCOS caused by abnormalities in the mitochondrial mechanism, such as impaired apoptosis, insulin-stimulated pathways, insulin resistance and anovulation. Our results show that mtDNA copy number reduced slightly on patients with PCOS, but not significant. Further studies are warranted to elucidate the roles of apoptosis and mitochondrial function in the pathogenesis of PCOS.

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O-17(기초) Expression of Nur77 Family Genes During Follicle Development in Rat Ovary

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Background & Objectives: NGFI-B deficient mice have no phenotype, suggesting the possible existence of a protein with redundant function to NGFI-B. The present study was designed to determine the possibility that Nurr1 or Nor1 expression may perform a similar function to NGFI-B expression during ovulatory process in rat ovary.

Method: Northern blot analysis of ovaries obtained from PMSG/hCG-treated immature animals revealed the rapid and transient induction of Nurr1 and Nor1 transcripts by hCG, reaching a maximum at 1 h, and decline to control levels at 3 h.

Results: The major cell types expressing NGFI-B subfamily, such as NGFI-B, Nurr1 and Nor1, were granulosa cells of preovulatory follicles and theca/interstitial cells. However, unlike the expression of NGFI-B in theca cells, Nurr1 and Nor1 are not present in theca cells in the ovaries obtained 2 days after PMSG treatment. In preovulatory follicles cultured in serum-free medium, Nurr1 and Nor1 transcripts were transiently induced by LH, reaching a peak within 1 h.

Conclusions: These results demonstrate the cell type-specific expression and gonadotropin induction of Nurr1 and Nor1 in granulosa cells of preovulatory follicles, similar to that of NGFI-B. We suggest that Nurr1, Nor1 and NGFI-B share many roles during ovulatory process.

O-18(기초) Ubiquitin Specific Protease USP-t and its Putative Substrates in Mouse Testis

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Background & Objectives: Ubiquitination and deubiquitination are critical protein modification systems in cellular metabolism and homeostasis. Several deubiquitinating enzymes are known to function as important regulators in signal transduction. In this experiment, we analyzed characteristics of USP-t (Ubiquitin-Specific Protease-Testis specific) and candidate binding proteins.

Method: We searched several conserved domains of USP-t and identified expression pattern at RNA and protein level using northern blot and western blot analysis. To know whether USP-t has deubiquitinating activity or not, we performed Ub-β-gal assay and using Nedd8 construct, we identified deneddylating activity of USP-t. And then, using immunostaining analysis, we identified expression pattern of USP-t within the testis. For finding binding proteins of USP-t, we performed Co-IP. And from the result, we found several candidate binding proteins of USP-t including HSP90. The binding between USP-t and HSP90 were conformed by IP/western blot analysis and Co-localization analysis.

Results: USP-t has two CS domains, a putative Hsp90 binding domain, and the cellular localization of USP-t was merged with that of Hsp90 in the cytoplasm. The catalytic domain of USP-t contains a zinc finger domain, and Ub-β-galactosidase assay revealed that it deubiquitinates the monoubiquitin moiety of ubiquitin-β-galactosidase, but does not reduce the global pool of polyubiquitin chains. Transcripts of USP-t were highly expressed in the testis, heart, and muscle, but USP-t proteins were detected only in the testis. The immunostaining analysis revealed that USP-t is highly expressed in the meiotic germ cells in the testis. And using co-immunoprecipitation assay we identified several putative substrates including cadherin, leucine rich repeat neuronal protein, zinc finger protein, coronin and elongin.