리시 3β-HSD, 17β-HSD, StAR mRNA 발현이 감소하였다. Western blot에서는 성장호르몬의 경우 BPA가 Leydig cell의 p-ERK, JAK2, p-JAK2의 발현에 있어서 GH나 IGF-1의 작용을 억제하거나 감소시키는 것을 확인하였고 특히 STAT3의 경우 BPA의 영향 하에서 성장호르몬의 작용을 확연하게 감소시켰다.

Conclusion: 실험 결과에서 확인할 수 있듯이 GH나 IGF-1 등의 성장인자는 Leydig cell내의 성장조절인자들의 발현을 증가시키지만 BPA에 의해서 그 효과가 감소하거나 억제되는 현상을 확인하였다. 또한 GH 처리에 의해 Leydig cell의 steroidogenesis에 관여하는 효소들의 발현이 증가하는 것을 통해 GH에 의한 생식소 기능이 직접적으로 조절됨을 알 수 있다. 이러한 BPA의 영향으로 인해 Leydig cell 자체의 활성이나 성장에 영향이 있을 것으로 예상되며 그러한 변화가 정자형성 과정으로의 negative 효과로 이어질 것으로 사료된다.

P-16 Genes Stimulated by the Activation of PKCζ after LH/hCG Treatment in the Rat Ovary

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Objectives: The present study was aimed to identify PKCζ-regulated genes in the preovulatory granulosa cells following LH stimulation by using annealing control primer-based PCR method.

Methods: 1. Granulosa cell isolation and culture 2. Annealing control primer (ACP) RT-PCR analysis and cloning of cDNAs 3. Northern blot analysis 4. In situ hybridization 5. Western analysis 6. MTT assay.

Results: Among 16 genes identified, six (testin, glypican-4, retrovirus SC1, connective tissue growth factor, aminolevulinic acid synthase 1 and serum inducible kinase) showed rapid and transient stimulation of mRNA levels by LH/hCG in the ovary of PMSG-primed immature rat. In situ hybridization analysis revealed that LH/hCG administration induced the expression of these six genes in granulosa cells of preovulatory follicles. The Western analysis showed that protein levels of testin and serum-inducible kinase were also increased by LH/hCG. The expression of remaining 10 identified genes in the ovary was stimulated during 24~72 h following LH/hCG treatment. MTT assay revealed that treatment of preovulatory granulosa cells with high dose of protein kinase C inhibitor RO 31-8220 (10 μM) or PKCζ-specific inhibitor pseudosubstrate peptide markedly suppressed cell survival, indicating anti-apoptotic function of PKCζ pathway.

Conclusion: The present data demonstrate the identification of PKCζ-regulated genes during ovulation which implicates the possible role of PKCζ pathway in the process of luteinization.