D115 Gonadotropes and Somatotropes express Growth Hormone Releasing Hormone (GHRH) Gene in Rat Pituitary

Sung-Ho Lee
Department of Biology, Sangmyung University, Seoul 110-743

Recent studies reveal that some neuropeptides classically associated hypothalamus have been found in pituitary gland, suggesting the existence of local regulation of pituitary function. The present study was carried out to investigate the expression of the GHRH gene in rat anterior pituitary and the pituitary-derived cell lines. Significant amounts of GHRH-like molecules were detected in both pituitary tissue and cell cultures in GHRH RIA. Immunoreactive GHRH was detected in large- and medium-sized pituitary cells by immunocytochemistry. In cell fractionation experiment, the highest level of GHRH content was found in gonadotrope-enriched fractions followed by somatotrope-, lactotrope- and thyrotrope fractions. Treatment with GnRH decreased the GHRH secretion from the anterior pituitary cells while GHRH secretion was increased by phorbol ester treatment. In RNase protection assay, the level of pituitary GHRH mRNA was increased by ovaryectomy. GHRH transcripts were detected from two pituitary-derived tumor cell types, αT3 (originated from gonadotrope) and GH3 (from somatolactotrope). Treatment of pituitary cells with GHRH resulted in a dose-dependent increase of [^{3}H]-thymidine incorporation. These results demonstrated that GHRH gene is expressed in the rat gonadotropes and somatotropes, and suggest that GHRH could be participated in the paracrine and autocrine regulation of cell proliferation, as well as promoting growth hormone secretion.

D116 Effects of All-*Trans* and 9-*cis* Retinoic Acid on the Proliferation and Death of HiB5 Hippocampal Progenitor Cells

Jeanju Chung, Sehyung Cho and Kyungjin Kim
Department of Molecular Biology and Research Center for Cell Differentiation, Seoul National University, Seoul, Korea

In the present study, we demonstrate that all-*trans* (*t*-RA) and 9-*cis* RA retinoic acid (9-*cis* RA) affect the proliferation and death of hippocampal progenitor cell line HiB5. At 32C, a permissive temperature for *tsA58*, treatment of HiB5 cells with *t*-RA or 9-*cis* RA significantly increased the number of floating cells as well as viable cells in the defined medium N2. At 39C, both retinoids also attenuated the decrease in cell number. Accordingly, *t*-RA and 9-*cis* RA increased DNA synthesis rate in N2 medium. Dying cells underwent apoptosis and required *de novo* synthesis. Actimomycin D blocked *t*-RA induced-DNA laddering, wheares cycloheximide blocked DNA laddering induced by both retinoids. Proliferating HiB5 cells expressed mRNAs for RARα, RARγ, RXRα, and RXRβ. The levels of RARγ, RXRα, and RXRβ mRNA were decreased, but *t*-RA induced RARγ transcript under this condition. Moreover, functional retinoid receptor contents correlated well with the changes in these transcript levels. Taken together, these data suggest that both retinoids are heavily involved in the proliferation and apoptotic death of HiB5 cells.