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Maturational Changes in the Phosphotyrosine Proteins under Capacitation Condition of Mouse Spermatozoa

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The difference and changes in phosphotyrosine proteins (PTP) under capacitation condition was examined in mouse spermatozoa from different region of epididymis. Total content of PTP of sperm was different according to regions of epididymis. PTP and spontaneous acrosome reaction increased under incubation in Tyrode solution. Phosphorylation of M_r 95K and 56K PTPs increased in caput sperm but slightly decreased in the corpus and cauda epididymal sperm. It suggested that these two protein might be present in plasma membrane overlaying the acrosome. Phosphorylation of M_r 85K and 66K PTPs increased in corpus and cauda epididymal sperm. PTP with M_r 29K which was constitutively phosphorylated on its tyrosine residue in corpus and cauda epididymal sperm was not changed under capacitation condition and supposed to a not an acrosomal antigen. These results suggested that content of PTP and sperm's ability to undergo protein phosphorylation are different according to regions of epididymis.

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In vivo regulation of gonadotropin-releasing hormone (GnRH) gene expression by GnRH analogs in ovariectomized, estradiol-primed rats

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Although recent evidence demonstrates the existence of ultrashort loop feedback circuits for GnRH secretion in the hypothalamus, it is uncertain whether the same or similar mechanism may be involved in GnRH gene expression. In the present study we examined an autocrine regulation of GnRH gene expression *in vivo*. GnRH agonist, (des-Gly_{10}, D-Ala_6-*proethylamide*)-GnRH, was microinjected into the lateral ventricle of ovariectomized and estradiol-primed rats. The amounts of GnRH mRNA in the preoptic area micropunched from brain slices were evaluated by a competitive reverse transcription-polymerase chain reaction. Administration of GnRH agonist significantly decreased GnRH mRNA levels in a time- and dose-related manner: GnRH agonist (6 ng) completely suppressed GnRH mRNA levels within 2h, while it biphasically regulated serum LH levels. Serum LH levels were increased until 2h and gradually decreased to basal level at 8h. Agonist-induced suppression of GnRH mRNA level was restored by treatment of antide (10 ng), a potent GnRH antagonist. Furthermore a blockade of the GnRH receptor with GnRH receptor antisense oligonucleotides (22mer) resulted in a partial recovery of GnRH mRNA levels which was suppressed by GnRH agonist. These results suggest that GnRH may play an autocrine role in the regulation of GnRH gene expression *in vivo* presumably through its receptor in the hypothalamus, but there is temporal discordance between GnRH gene expression and pituitary LH secretion.