D-25 Mouse Oocyte Activation by Soluble Sperm Factors

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Mammalian oocyte activation following the fusion with sperm consists of a serial events accompanying the physiologically distinct changes such as activation of metabolism, reentry into cell cycle, and execution of developmental program.It is reported that soluble sperm protein evoked Ca^{2+}-oscillation and thus activated oocytes. The present study aimed to verify whether Ca^{2+}-calmodulin dependent cell signalling is involved in the activation of oocyte by soluble sperm factor (SSF). Oocyte activating abilities of the protein extracts from the sperm of mouse, pig and human sperm were examined. Parthenogenetic activation of the oocytes treated with ethanol were inhibited by trifluoperazine. W-7, calmodulin inhibitor, significantly inhibited the oocyte activation by SSF. There was a difference in oocyte activating ablity of SSF obtained from different species, and mouse SSF was the most active among species examined. These results suggest that Ca^{2+}-calmodulin dependent cell signalling is possibly involved in the activation of oocyte by soluble sperm factor.

D-26 Changes in the Phosphotyrosine Proteins and Possible Involvement of Tyrosine Kinase during Capacitation and Acrosome Reaction of Mouse Spermatozoa

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The changes in phosphorylation of tyrosine-containing proteins profile, and the possible involvement of tyrosine kinase in the capacitation and the acrosome reaction, were examined in mouse spermatozoa. Both the BSA and human follicular fluid (hFF) increased the contents of phosphotyrosine proteins during incubation for 90 min. Both BSA and HFF increased phosphorylation of tyrosine residues of M_r 106 K protein. hFF specifically increased tyrosine phosphorylation of the M_r 56 K protein (reduced form). Genistein inhibited the increase of PTP content during capacitation *in vitro*. Both spontaneous AR and hFF-induced AR were reduced by genistein but A23187-induced AR was not. These results suggest that phosphorylation of tyrosine-containing proteins and tyrosine kinase signaling mediated capacitation and AR of mouse spermatozoa in the upstream of Ca^{2+} influx.