D113 Role of Heat Shock Protein 25 (hsp25) in Response to Heat Shock in HiB5 Hippocampal Progenitor Cells

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The present study examined the roles of heat shock protein 25 (hsp25) against heat shock in a hippocampal cell line, HiB5. Expression of hsp25 was induced by heat shock at the mRNA and protein levels, and the binding activity of heat shock factor (HSF) to a conserved heat shock element (HSE) was increased remarkably. In normal conditions, hsp25 was located in the cytoplasm but it redistributed into the nucleus by heat shock. The phosphorylation of hsp25 was accompanied during this process. Transfection experiment with hsp27 mutants in which specific serine residues were substituted with alanines or aspartic acids showed that phosphorylation of hsp25 is essential for the translocation of hsp25 to the nucleus. Hsp25 was phosphorylated by p38 MAPK and ERK1/2 in response to heat shock. SB203580 (p38 inhibitor) and PD098059 (MEK inhibitor) had no effect on the actin filament stability in the absence of heat shock. However, actin filament was destroyed by heat shock when phosphorylation of hsp25 was inhibited by SB203580 and PD098059. The death rate of HiB5 cells by heat shock was also increased when the cells were pretreated with SB203580 and PD098059. Cytochalasin D, an actin filament disrupter, disrupted actin filament in normal HiB5 cells and Ala-hsp27 transfected cells. However, actin filament was not disrupted by cytochalasin D in the Asp-hsp27 transfected cells. These results support the idea that phosphorylation of hsp25 is critical for maintaining structure of actin filament and thus enhances thermo-resistance. During the recovery period, the hsp25 protein level was reduced in a time-dependent manner, and the translocated hsp25 returned to the cytoplasm. The dead cell population also decreased in a recovery time-dependent manner, however, the proportion of apoptotic cell death was increased during the recovery period: HiB5 cells were stained with dichlorofluorescein (DCF) by 3 hr heat shock, suggesting that heat shock may damage mitochondria and thus induce oxidative stress.

D114 A Role of Kinesin-Associated Peptide 3 in the Process of Female Puberty Onset

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We have cloned many estrogen-responsive genes in the rat hypothalamus using differential display PCR technology and estrogen-sterilized rat model, and determined their mRNA levels in the hypothalamus during the postnatal development. One of the genes showing a dramatic change in gene expression during the development was KAP3, an associated peptide to the axonal motor protein kinesin 3A and 3B. Therefore we presumed KAP3 may play an important role in the process proceding the female puberty onset. To test this hypothesis, we determined mRNA level of KAP3 during the pre- and peripubertal period. Level of KAP3 mRNA was greatly changed before pubertal process. KAP3 mRNA was increased after 22-day of age, reached peak level at the anestrous phase and kept high level during the rest of pubertal process. In the next step, an antisense KAP3 oligodeoxynucleotide (ODN) was stereotaxically administered into the ventral third ventricle, and the day at puberty onset was observed. Puberty was clearly delayed, and the mRNA level of synaptophysin, a synapse-specific protein, was suppressed by the administration of ODN. Taken together, the present results strongly suggests that KAP3 play a role in the regulation of pubertal process such as synaptic remodeling.