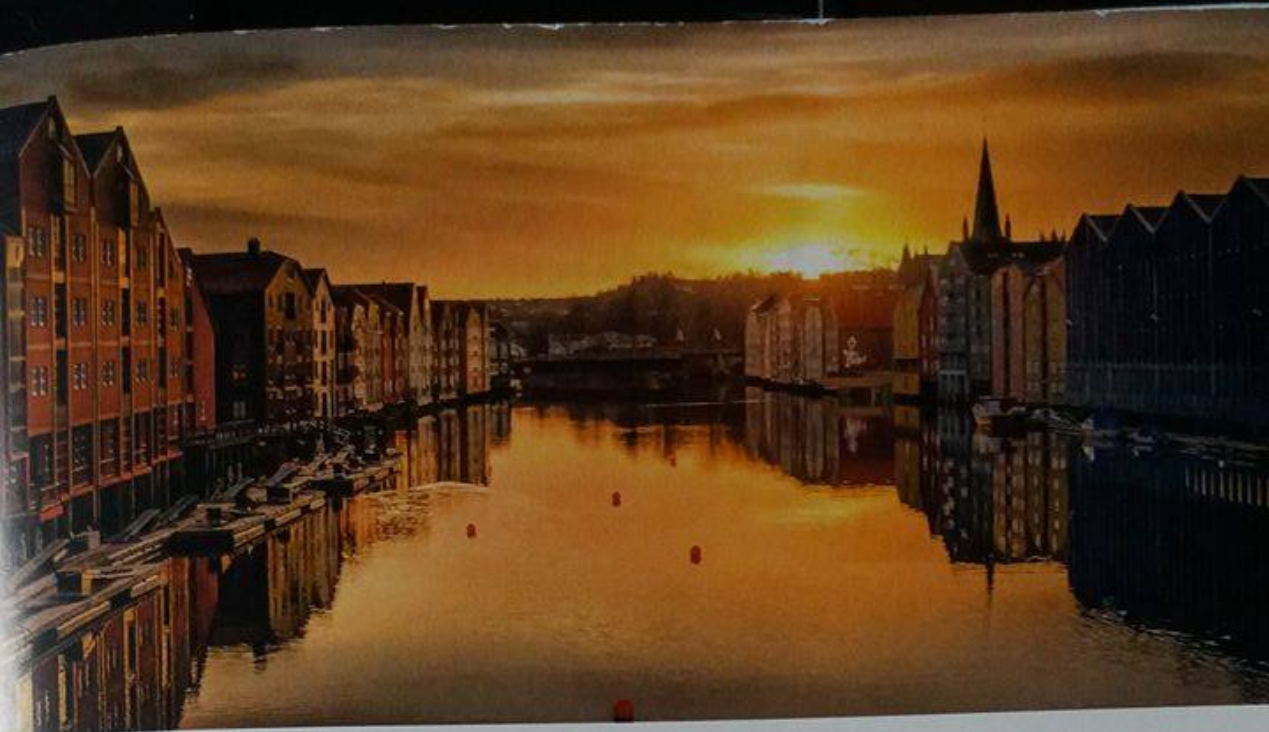




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Investigating neuroinflammation and neural apoptosis in fetal alcohol syndrome (FAS); the role of Hsp27 in the regulation of inflammation and reactive astrogliosis.

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Heat shock proteins (Hsps) are rapidly induced during different stress conditions or diseases, and it seems that besides their molecular chaperone activities they are involved in the regulation of inflammatory processes. In this study we investigated the role of Hsp27 in inflammation in a neurodegenerative condition, termed fetal alcohol syndrome. 7 day-old Hsp27 overexpressing transgenic and wild type mice were treated with 5g/kg ethanol injection then brains were removed for RNA isolation or preparing frozen sections. Dramatically increased cell death was detected in the brain of the animals 24 hours after the treatment using TUNEL assay and Fluoro JadeC staining. The most damaged areas were found in the cortex, thalamus and striatum, while interestingly there was no significant cell death in the cerebellum. Although we detected a slightly decreased level of apoptosis in Hsp27 overexpressing animals compared to wild-types, the result was not statistically significant. Quantitative RT-PCR was used to study the expression level of marker genes for inflammation, reactive astrogliosis and microgliosis. 24 hours after the treatment the levels of TNF α , IL-1 β and GFAP were increased by twofold in the wild-type animals, while they showed a much higher (10-, 7- and 4.5 fold) increase in the transgenic mice. The Hsp27 overexpression alone did not change the level of these cytokines or GFAP, only in combination with ethanol treatment. The level of the microglia marker, Iba-1 was slightly increased in the brain of non-treated Hsp27 transgenic animals, however after ethanol treatment it did not show any further increase. These data suggest that Hsp27 can promote the production of cytokines in response to ethanol induced brain injury and it is probably involved in the regulation of astrocyte and microglia activations.