

**G101****Expression and Localization of Sulfated Glycoprotein-2 in Rat Brain Treated with Ethanol****Sung-Kyu Ju<sup>1</sup>, Kyoung-Cheol Sohn<sup>1</sup> and Kwan-Hee You<sup>2</sup>**

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SGP-2 (sulfated glycoprotein-2) has been implicated in physiological processes including sperm maturation, control of complement-mediated cell lysis, transport of lipid, membrane remodeling, cell aggregation, and so on. SGP-2 is also associated in apoptosis, synaptic remodeling, neuron sprouting, and neurodegenerative diseases. Recently, over-expression of SGP-2 was observed in central nerve system after neurotoxin treatment. However, so far SGP-2 expression and localization on the brain injury is not established. To address this issue, in the present study, SGP-2 expression and localization in brain from rats treated with ethanol was investigated. Northern blot and RT-PCR analysis of the effects of ethanol administration on rat brain SGP-2 expression showed that no significant difference was observable in the level of SGP-2 mRNA compared with control rat brain. On the other hand, SGP-2 protein expression showed about 3-fold increase during ethanol treatments. Thus, to resolve this problem, we presume that rat brain SGP-2 expression is regulated on post-translational modification level during the rat brain injury, as it is the case of SGP-2 biogenesis during apoptosis in the regressing rat ventral prostate. Further studies are required to determine this possibility in more detail.

**G102****Determination of clusterin mRNA expression of apoptosis induced rat thymocytes in vivo and in vitro.****Ji-Sun Park<sup>1</sup>, Jung-Hyun Park<sup>1</sup>, Sung-Kyu Ju and Kwan-Hee You**

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Clusterin is a 75-80 kDa secreted glycoprotein, which exact biological role is still veiled. Detection of increased clusterin expression in apoptotic rat prostate cells had suggested a putative role in regulating apoptosis, however, whether it protects or promotes programmed cell death is not known. Prior studies on apoptotic thymocytes also couldn't clarify this issue, since in these cases, contradictory results on clusterin mRNA expression were observed in Northern blot assays, which raised questions about the involvement of clusterin in apoptosis itself. Therefore to examine and determine whether the regulation of clusterin expression is indeed subjected to induction of apoptosis, in the present study, rat thymocytes were driven to programmed cell death by dexamethsone treatment, and mRNA expression was analyzed by semi-quantitative RT-PCR before and after induction of apoptosis. However, neither the treatment of isolated thymocytes with dexamethasone in vitro nor a massive induction of apoptosis of immature thymocytes in vivo showed modulation of clusterin mRNA expression. On the other hand, the activation with concanvalin A and interleukin-2 promoted clusterin expression in thymocytes, so that it is rather thinkable that clusterin expression is under the control of some activation-dependent that apoptosis-induced signals.