



## FENS Forum 2008

For posters, morning sessions: 9:30-13:30; afternoon sessions 13:30-17:30.  
Authors are expected to be in attendance at their posters at the time indicated.  
For other sessions, time indicates the beginning and end of the sessions.

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**First author** Mora Lee, Silvia (poster)

Poster board C69 - Mon 14/07/2008, 16:45 - Hall 1

Session 120 - Ischemia 2

Abstract n° 120.29

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**Authors** Mora Lee S., Pastor M. D., Fradejas N. & Calvo S.

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**Title** Ribosomal S6 kinase protects cerebellar granule cells from ischemic damage.

**Text** Cerebral ischemia triggers suppression of protein synthesis that is prominently correlated with neuronal vulnerability, suggesting that this cellular process plays a crucial role in the cascade of events leading to ischemic cell death.

The ribosomal S6 protein kinases (S6K) are a family of serine/threonine kinases involved in cell-cycle progression, cells size regulation and viability. S6K activation up-regulates ribosomal biosynthesis and enhances the translational capacity of the cell. In different models of stroke has been observed that downregulation of protein synthesis is linked to a decrease of total and phosphorylation S6K forms, indicating that this kinase could be involved in maintaining neuronal viability in the ischemic brain. We have tested this hypothesis in primary cultures of cerebellar granule cells (CGC) using the oxygen and glucose deprivation (OGD) as in vitro ischemia model. First, we analyzed the effect of OGD on S6K mRNA and peptides, finding that OGD induced a prominent downregulation of S6K1 transcript and both phosphorylated and total S6K protein. Secondly, we analyzed the effects of S6K suppression on CGC response to ischemia. CGC obtained from either wild type or S6K1; S6K2 knockout mice were subjected to OGD by different times and viability was analyzed afterwards. Cells lacking S6K showed a higher sensitivity to OGD at all the times tested, revealing S6K as an important protective factor against ischemia-induced neuronal death.

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**Theme** C - Disorders of the nervous system  
Ischemia - Inflammation, neuroprotection and tolerance

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