

OXIDATIVE STRESS IN PERINATAL ASPHYXIA AND FETAL ASPHYCTIC PRECONDITIONING

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Background: Asphyctic preconditioning is thought to attenuate the cellular stress response which reduces brain damage.

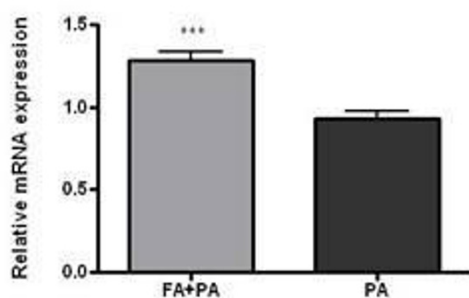
Objective: We evaluated the expression of stress related genes in pre- and neonatal pups after fetal preconditioning (FA) and perinatal asphyxia (PA).

Methods: FA was induced on E17 by clamping the uterine circulation for 30'. On P0 PA was induced by placing the fetuses-containing uterine horns in water for 19'. Control and FA pups were sacrificed 96h after the FA insult. Control, FA, PA and FA+PA pups were sacrificed 6h (P0) and 96h (P4) after birth. mRNA expression of iNos, nNos and Sod 1 was evaluated in the right hemisphere with qRT-PCR.

Results: Sod1 was significant down-regulated ($p=0.0057$) on E21 for the FA group compared to controls. On P4 both the PA and the FA+PA group were down-regulated when compared to controls ($p=0.0021$, $p=0.0132$) and compared to the FA group ($p=0.0007$, $p=0.0297$). On P4 there was a significant difference between the PA and the FA+PA groups ($p=0.0005$) (fig.1). nNos expression revealed a significant increase on P0 for the PA and FA+PA groups compared to the FA group ($p=0.0012$, $p=0.0145$). iNos showed no significant differences.

Conclusions: Modulation of the expression of antioxidants like Sod1 by FA could contribute to neuroprotection at birth. Additional whole-genome expression will provide a complete overview of the preconditioned phenotype.

Sod1 expression on P4



[fig. 1]