FOETAL TO NEONATAL TRANSITION IN A LOW OXYGEN ATMOSPHERE (14%) ENHANCES LUNG NON-ENZYMATIC ANTIOXIDANT DEFENCES IN NEWBORN MICE

J. Escobar¹, E. Cubells², M. Enomoto³, J. Belik³, M. Vento⁴

¹Health Research Institute, ²Division of Neonatology, University and Polytechnic Hospital La Fe, Valencia, Spain, ³Pediatrics, The Hospital for Sick Children, Toronto, ON, Canada, ⁴Division of Neonatology and Health Research Institute, University and Polytechnic Hospital La Fe, Valencia, Spain

Background and aims: Foetal-to-neonatal transition exposes lung to a substantial increase in oxygen which may cause oxidative stress, and functional and structural damage. We hypothesized that foetal-to-neonatal transition in a lower oxygen atmosphere (14% instead of 21%) would enhance postnatal adaptation by preventing oxidative stress.

Methods: Pregnant mice were put in 14% FIO₂ for 24hrs before delivery. The pups delivered in 14% FIO₂ and one hour thereafter switched to 21% and sacrificed (Lowox group). Lungs, brain and intestine were retrieved for reduced (GSH) by GSH-transferase assay (Brigelius 1983) and oxidized (GSSG) by mass spectrometry. Pups born to mice delivering in 21% FIO₂ served as controls (Normox group).

Results: Lung GSH content was significantly higher in the Lowox than in the Normox group at birth (2.88 ± 0.24 vs. 2.28 ± 0.23 mmol/g tissue; $p < 0.05$). Conversely, GSSG was lower in the Lowox group (Lowox vs. Normox: 0.015 ± 0.004 vs. 0.116 ± 0.004 mmol/ g tissue; $p < 0.01$). Hence, GSH/GSSG ratio the most relevant marker of intracellular redox status was significantly greater in the Lowox group. No differences were found for brain and gut tissue.

[GSH/GSSG RATIO]

Conclusions: Foetal to neonatal transition in a low oxygen atmosphere enhances non-enzymatic antioxidant reserves thus helping postnatal adaptation to a higher oxygen exposure. This experimental model may be useful to study extremely preterm infants' adaptation to extrauterine life.