PO-104 - (23926) - PRENATAL VITAMIN A MODULATES ENOS/INOS EXPRESSION IN MURINE LUNG DEVELOPMENT

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A decrease in pulmonary vascular pressure is pivotal for normal mammalian lung development, with nitric oxide as one of the most important regulators of this process. Several studies relate respiratory distress syndrome to eNOS deficiency and inhaled nitric oxide can be used in the treatment of premature newborns. Our previous studies showed that maternal administration of vitamin A, once the period of retinoid-induced teratogenesis is over, results in an enhancement of lung fetal organogenesis. To better understand how NOS expression can be regulated by vitamin A we conducted an in vivo study in fetal and neonatal mouse lungs.
Pregnant mice were subjected to subcutaneous administration of vitamin A on the 12th gestational day. Lungs from control and exposed animals were collected daily from the 15th gestational day till the 3th day of life and processed for routine immunohistochemistry.
Our results show a higher expression of eNOS and iNOS during late gestational stages followed by a post natal decrease. There is a marked increase of eNOS expression at 16thday of gestation, but an inversely decrease in iNOS expression at the same day. At this gestational day, lung undergoes marked vasculogenesis and angiogenesis, in which eNOS plays an essential role, and the fact that iNOS decreases suggests an interregulation between both enzymes.There were no significant diferences between control and exposed animals.

However, vitamin A seems to stimulate eNOS expression without altering iNOS, depicting an intricate balance important for lung's vascular development, and a beneficial role of vitamin A during lung maturation.

Palavras-chave : Lung development, vitamin A, nitric oxide