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Maternal smoking disrupts retinoid pathways in the developing fetal lung

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Maternal smoking can lead to lung disease in babies, including asthma. New research published in BioMed Central's open access journal *Respiratory Research* shows that maternal smoking-related defects within the alveoli inside the lungs of offspring are associated with a disruption in retinoic acid signaling.

It is known that the effects of smoking on the developing lung have long term consequences for the child's health. Children of mothers who smoke have an increased risk of asthma and lung infections and have a more rapid decline of lung function if they begin to smoke as adults. In order to understand the mechanism behind this, researchers from the Brigham and Women's Hospital looked at the effect of maternal smoking on retinoic acid signaling in mice. Retinoic acid (RA) is produced in the body from vitamin A and is known to be involved in alveolar development as well as lung maintenance throughout life.

Maternal smoking affected the number and birth weight of their pups. It also affected the development of the pups' lungs (measured by approximate size of the gas exchange elements of the lungs) and disrupted RA signaling in their lungs. Reduction of RA signaling was also seen in lung cells treated with cigarette smoke in the lab.

Dr Kathleen Haley, who led the research, explained, "The pups were protected from breathing smoke themselves but were still affected by 'second hand' smoke before birth and through their mother's milk. Smoking affected the regulation of genes, controlled by RA, necessary

for lung development, including surfactant apoprotein B. It is known that complete loss of surfactant apoprotein B is linked to severe respiratory failure in infants and down regulation of this, and other genes regulated by RA, can have potentially serious consequences."

The decreases in RA signaling and in the expression of RA controlled genes were most pronounced during the 3-5 days after birth when the lungs are undergoing rapid development. In humans this development stage occurs before birth when the developing fetus receives 'second hand' smoke via the umbilical cord so it seems very likely that the same damaging effects of cigarette smoke on RA signaling are present in humans too.

More information: Maternal smoking and the retinoid pathway in the developing lung, Sara E Manoli, Lacey A Smith, Carrie A Vyhlidal, Chang Hyeok An, Yolanda Porrata, Wellington V Cardoso, Rebecca M Baron and Kathleen J Haley, *Respiratory Research* (in press)

Provided by BioMed Central

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