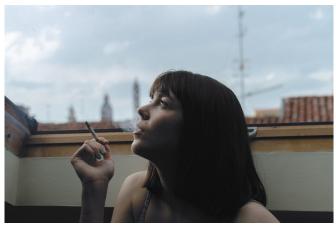


New research bolsters link between prenatal smoking and impaired lung development

9 October 2020



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adaptation to smoke exposure.

The Centers for Disease Control and Prevention estimates at least 5.5 million children and 19.2 million adults in the U.S. have asthma. There are 16 million adults in the U.S. with COPD. Both diseases make breathing difficult and are incurable.

More information: Khosbayar Lkhagvadorj et al. Prenatal smoke exposure dysregulates lung epithelial cell differentiation in mouse offspring: role for AREG-induced EGFR signaling, *American Journal of Physiology-Lung Cellular and Molecular Physiology* (2020). DOI: 10.1152/ajplung.00209.2020

Babies born to mothers who smoked during pregnancy are more likely to suffer from impaired lung development. They are also more susceptible to developing lung diseases such as asthma and chronic obstructive pulmonary disease (COPD), according to a new research article published in the *American Journal of Physiology*-Lung Cellular and Molecular Physiology. The article has been chosen as an <u>APSselect</u> article for October.

Another key takeaway from the research is that the protein amphiregulin—which enables tissue homeostasis—could be used to interrupt impaired lung development in babies of mothers who smoked during pregnancy. Evidence also suggests prenatal smoke exposure impairs lung development in babies in which increased amphiregulin/epidermal growth factor receptor signaling might have a role. In addition, researchers found that prenatal smoke exposure resulted in fewer ciliated cells—which help to protect and clear the respiratory airways—during bronchiolar development. The study was conducted in 50 one-day-old mice. The experiment lasted five weeks, including one week of

Provided by American Physiological Society



APA citation: New research bolsters link between prenatal smoking and impaired lung development (2020, October 9) retrieved 14 May 2021 from https://medicalxpress.com/news/2020-10-bolsters-link-prenatal-impaired-lung.html

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