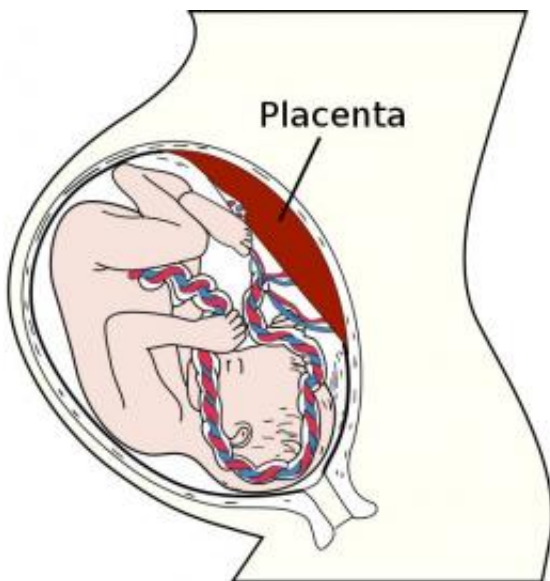


Air pollution can harm pregnancy by affecting gene expression in the placenta

March 11 2022, by Rae Lynn Mitchell



Fetus in utero, between fifth and sixth months. Image credit: Wikipedia.

Polluted air contributed to the deaths of more than 6.6 million people worldwide in 2019. It accounts for around [20 percent of newborn deaths](#) globally, mostly due to preterm birth and low birth weight. Because of this, it is crucial that researchers and policymakers working to improve public health understand how different aspects of various types of air pollution and their associated biological responses affect fetal development.

To add to this understanding, researchers at Texas A&M University and

other institutions conducted a study of air pollution's effects on [fetal development](#) in an [experimental model](#). In the study, published in the journal *Toxicological Sciences*, recent Texas A&M School of Public Health doctoral candidate Jonathan Behlen, associate professor Natalie Johnson, and colleagues analyzed the effects of exposure to different concentrations of ultrafine particles (UFPs) on fetal development and how exposure affected expression of certain genes in the placenta.

Particulate matter is a major type of air pollution that consists of particles suspended in the air. The smallest of these particles, UFPs, are smaller than 100 nanometers. A growing body of evidence is pointing to UFPs having [harmful effects](#) during pregnancy; however, there is less certainty about what mechanisms are involved in these adverse outcomes.

The researchers analyzed the effects of UFPs in an experimental animal model. Pregnant models were placed in three groups: one with filtered air, one with low dose UFPs and one with high dose UFPs. The models were exposed to a mixture of UFPs typically found in cities, the low dose below regulatory standards for PM_{2.5}. The researchers then measured placentas and fetuses of pregnant female models and analyzed changes in gene expression from placental tissues. Specifically, the researchers found altered expression of genes responsible for antioxidant, inflammatory and lipid transport activities.

Behlen, Johnson and colleagues found that the low dose group had notable reductions in fetal and placental length and decreasing trends in fetal weight for both male and female fetuses. They also found changes in expression for multiple genes. The changes in gene expression were higher in the low dose group than the high dose group and were greater in female fetuses than male ones.

Their analysis also found increased expression of antioxidant and

inflammatory genes in fetuses of both sexes in both dose groups as well as disruptions in genes responsible for lipid metabolism. Changes to how these [genes](#) are expressed could affect bodily processes in a way that increases the risks of adverse pregnancy outcomes like preeclampsia.

These findings indicate some of the biological underpinnings of how UFP exposure affects pregnancy; however, the researchers note that their study relied on analysis of many different cell types together. Future studies that focus on single cell types could further clarify the effects of UFPs on placental and fetal development. Additionally, the researchers identify a need for more in-depth analyses of possible mechanisms behind the differences they found between male and female fetuses.

The findings of this study align with other studies showing UFPs adversely affecting pregnancies. With more information on [gene expression](#) changes and other effects, scientists will have a better starting point for further study of exactly how air pollution impacts fetal development. Such an understanding could help inform efforts to mitigate the harmful effects of air pollution worldwide.

More information: Jonathan C Behlen et al, Gestational Exposure to Ultrafine Particles Reveals Sex- and Dose-Specific Changes in Offspring Birth Outcomes, Placental Morphology, and Gene Networks, *Toxicological Sciences* (2021). [DOI: 10.1093/toxsci/kfab118](https://doi.org/10.1093/toxsci/kfab118)

Provided by Texas A&M University

Citation: Air pollution can harm pregnancy by affecting gene expression in the placenta (2022, March 11) retrieved 19 April 2024 from <https://medicalxpress.com/news/2022-03-air-pollution-pregnancy-affecting-gene.html>

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