

Can a mother's stress impact children's disease development?

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An expectant mother's psychosocial well-being may affect disease development in her child. Credit: Unsplash.

Stress on an expectant mother could affect her baby's chance of developing disease—perhaps even over the course of the child's life, UC researchers have found.

Psychosocial factors creating stress—such as lack of social support, loneliness, marriage status or bereavement—may be mutating their child's mitochondrial DNA and could be a precursor to a host of diseases, according to a University of Cincinnati study.

"There are a lot of conditions that start in childhood that have ties to mitochondrial dysfunction including asthma, obesity, attention deficit hyperactivity disorder and autism," says Kelly Brunst, Ph.D., assistant professor of environmental and public health sciences in the UC College of Medicine and lead author of the study.

"The fetal and infant period is a vulnerable time for environmental exposure due to heightened development during these periods," says Brunst.

"We don't just wake up one day and have asthma or attention deficit hyperactivity disorder. The programming effects resulting from environmentally induced shifts occur over time and likely start during gestation at the molecular and cellular level. These shifts alter physiological states that likely play a role in who is going to go on and develop adverse health outcomes."

As part of the study, researchers sequenced the mitochondrial genome and identified mutations in 365 placenta samples from birth mothers in Boston and New York City from 2013-18. A multivariable regression model was used to look at maternal lifetime stress in relation to the number of gene mutations in the placenta mitochondrial genome.

Women experiencing increased psychosocial stress—that can range from sexual assault, domestic violence or serious injury to incarceration, physical or mental illness and family hardship—over their lifetime exhibited a higher number of placental mitochondrial mutations. The strongest associations were observed among Black women. Higher stress-related DNA mutations in the placenta were seen in Black and white women, but not in Hispanic women.

The study's findings were published in the scholarly journal *Biological Psychiatry*.

"The idea behind this work is about understanding how our environment, in this case maternal stress and trauma, impact mitochondrial function and ultimately neurobehavioral development," says Brunst. "The hope is to gain insight as to why certain children are vulnerable to developing a range of complex conditions previously linked to environmental exposures such as chronic stress or air pollution."

"We ask about events that might have occurred prior to their pregnancy even during the mother's own childhood as part of our study," says Brunst.



"So what this is telling us is that the stress that a woman has experienced even before she is pregnant might have an impact on the fetal mitochondrial genome."

Brunst said there are some diseases for which Black women are more at risk—obesity, diabetes and certain cancers—so they might be more affected by stress and subsequently develop these diseases which have also been linked to stress."

"What was interesting about the study was that Hispanics exposed to stress had fewer placental mitochondrial DNA mutations," says Brunst.

She says one explanation could be what researchers call the "Hispanic paradox." It is the epidemiological phenomenon documenting better health and lower mortality relative to non-Hispanic whites despite greater risk and lower socioeconomic status for Hispanics."

"Despite exposure to more stress and trauma, sociocultural dynamics specific to Hispanics may attenuate experiences of <u>stress</u> which in turn has downstream effects on psychophysiological mechanisms and better outcomes," says Brunst. "This is just one possible explanation."

More information: Kelly J. Brunst et al. Associations Between Maternal Lifetime Stress and Placental Mitochondrial DNA Mutations in an Urban Multiethnic Cohort, *Biological Psychiatry* (2020). DOI: 10.1016/j.biopsych.2020.09.013

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