

Gestational diabetes, low socioeconomic status linked with increased risk of ADHD in offspring

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Children exposed to maternal gestational diabetes mellitus and low socioeconomic status, particularly in combination, appear to be at an increased risk of developing childhood ADHD, according to a report published Online First by *Archives of Pediatrics & Adolescent Medicine*.

"[Gestational diabetes](#) mellitus (GDM) typically develops in the second and third trimesters and is defined as glucose intolerance with onset or first recognition during pregnancy," the authors write as background information in the article. "The prevalence of GDM has been rising for over 20 years, particularly among ethnic minorities and individuals with [low socioeconomic status](#) (SES), as have lifestyle changes that heighten [risk](#) including greater consumption of saturated fats, sugar, and processed foods, and sedentary working environments."

To examine the association of [gestational diabetes mellitus](#) (GDM) and low [socioeconomic status](#) with neurodevelopment and attention-deficit/hyperactivity disorder ([ADHD](#)) outcomes, Yoko Nomura, M.D., Ph.D., of Queens College, City University of New York, Flushing, and colleagues, compared offspring of mothers with and without GDM in an economically diverse sample. The authors distributed the ADHD Rating Scale-IV to parents of 3- and 4-year-old children in preschools surrounding Queens College, and recruited 212 participants at a 2:1 ratio of "at risk" to "typically developing" children. At-risk children had at least six inattention or six hyperactive and impulsive symptoms as rated

by parents, teachers, or both. "Typically developing" children had fewer than three symptoms in each domain.

The mean (average) inattention score at baseline for offspring exposed to mother's GDM was significantly higher than for offspring unexposed, but there was no difference in hyperactivity/impulsivity scores between the two groups. Children in low SES families, compared to high SES families, had greater inattention and hyperactivity/impulsivity scores. The results showed no difference in the risk for ADHD at baseline, but a two-fold increased risk at age 6 years among children exposed to GDM compared with children who were not exposed. There was also a two-fold increased risk for ADHD at baseline and at age 6 years among children in low SES families.

Children exposed to both GDM and low SES showed compromised neurobehavioral functioning, including lower IQ, poorer language abilities and diminished behavioral and emotional functioning. When examining the relationship of both GDM and SES exposure on outcomes, the authors found a 14-fold increased risk of developing ADHD among children exposed to both GDM and low SES. Conversely, children exposed to maternal GDM alone or low SES alone had no significant [increased risk](#) for ADHD.

"This study demonstrates that children of mothers with GDM raised in lower SES households are at far greater risk for developing ADHD and showing signs of suboptimal neurocognitive and behavioral development," the authors conclude. "Since ADHD is a disorder with high heritability, efforts to prevent exposure to environmental risks through patient education may help to reduce the nongenetic modifiable risk for ADHD and other developmental problems."

In an accompanying editorial, Joel Nigg, Ph.D., of Oregon Health and Science University, Portland, writes, "In the current issue of the

Archives we see additional evidence, in a retrospective design, that early developmental events are related to subsequent attention-deficit/hyperactivity disorder (ADHD) in children."

"Most of the relevant environmental risks are presumed to occur very early in development," continues Dr. Nigg. "If causal, and if able to be understood pathophysiologically, such environmental effects on ADHD are of 'game-changing' importance because they open the door to eventually preventing that portion of cases of ADHD caused by early insult to the nervous system."

"If a specific environmental causal influence can be demonstrated, even if effective in a subset of [children](#), and its biological mechanisms elucidated, then a powerful model will be created for how ADHD can develop," Dr. Nigg concludes. "That discovery will be a crucial stepping-stone toward parsing multiple causal routes to what may be a final common pathway of the ADHD phenotype."

More information: Arch Pediatr Adolesc Med. Published online January 2, 2012. [doi:10.1001/archpediatrics.2011.784](https://doi.org/10.1001/archpediatrics.2011.784)
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