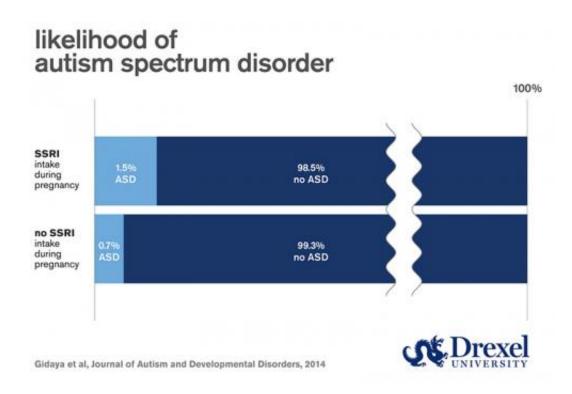


In utero exposure to antidepressants may influence autism risk

June 3 2014



Drexel University researchers found a two-fold increased risk for autism spectrum disorders associated with in utero exposure to selective serotonin reuptake inhibitors, compared to an unexposed reference group. However, the autism rate in both groups was very low. Credit: Drexel University

(Medical Xpress)—A new study from researchers at Drexel University adds evidence that using common antidepressant medications during pregnancy may contribute to a higher risk of autism spectrum disorders



(ASD) in children, although this risk is still very small.

Results from past studies of prenatal use of selective serotonin reuptake inhibitors (SSRIs) and ASD risk have not been consistent. An ongoing challenge in this line of research is trying to tease apart potential effects of the medication on risk from the effects associated with the condition for which the medication was prescribed (most commonly depression). Based on past studies, both SSRIs and genetic factors associated with depression are likely associated with greater risk of ASD.

This new study, published online ahead of print last month in the *Journal* of Autism and Developmental Disorders, suggests that under-reporting of maternal depression, if not properly considered in analyses, may influence results of studies trying to address this question.

In the study, the Drexel team analyzed large population based registers of nearly 750,000 births in Denmark from 1997 through 2006. They found that about 1.5 percent of children born to women who had taken an SSRI during pregnancy were diagnosed with ASD, compared to about 0.7 percent of children born to an otherwise similar group of women not taking the medication.

"We found a two-fold increased risk for ASD associated with in utero exposure to SSRIs compared to the unexposed reference group" said lead author Nicole Gidaya, PhD. "More importantly, in our analysis we accounted for under-reporting of maternal depression in the register. This suggests that under-reporting of the confounder, maternal depression, may be a limitation in approaches previously used in the other studies."

Gidaya, who performed this study while a doctoral student in the Drexel University School of Public Health, noted that "if the increased ASD risk we saw here is real, it is important to realize that the number of



ASD cases that could be prevented by reducing SSRI exposure in pregnancy still represents only a small fraction of overall cases of ASD."

The researchers further advised caution in interpreting the results in practice. Because of the challenges of distinguishing effects of medications from those of the condition indicating their use, more research in larger study populations will be needed to confirm the findings. In addition, the decision whether or not to use an SSRI in pregnancy is a complex one; pregnant women and their doctors need to consider women's physical and mental health needs as well as other pregnancy-associated risks, including risks associated with untreated depression both during and after pregnancy.

However, the research team believes that the greater value of this finding is to direct further attention on understanding the mechanisms by which in utero SSRI exposure might influence the developing brain. Serotonin is a neurotransmitter whose use by the brain is altered during depression and modified by SSRI use, and has been shown to play an important role in brain development.

The authors of the current study point out that there is still a need for more population studies of possible associations between maternal SSRI use and autism, in light of the limitations of the present study and the conflicting results within the field's previous studies of the question. They say future studies should use a large population sample where there is good quality data about exposure to medication, mental health diagnoses as well as ASD diagnoses.

"As we complete research in our attempts to understand autism's causes we continue to realize that there are likely many genetic and non-genetic contributors," said Craig Newschaffer, PhD, director of the A.J. Drexel Autism Institute and professor in Drexel's School of Public Health, and the study's senior author. "We must begin trying to map these multiple



risk factors on to common pathways, so that these pathways can be a focus in our effort to prevent the impairment associated with ASD. Pathways involving the brain's serotonin system are still one viable candidate."

More information: dx.doi.org/10.1007/s10803-014-2128-4

Provided by Drexel University

Citation: In utero exposure to antidepressants may influence autism risk (2014, June 3) retrieved 17 April 2024 from

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