

Early exposure to antidepressants affects adult anxiety and serotonin transmission

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About 15 percent of women in the United States suffer from anxiety disorders and depression during their pregnancies, and many are prescribed antidepressants. However little is known about how early exposure to these medications might affect their offspring as they mature into adults.

The answer to that question is vital, as 5 percent of all babies born in the U.S. - more than 200,000 a year - are exposed to [antidepressants](#) during gestation via transmission from their mothers.

Now, a UCLA team has studied early developmental exposure to two different antidepressants, Prozac and Lexapro, in a mouse model that mimics human third trimester medication exposure. They found that, although these serotonin-selective reuptake inhibiting antidepressants (SSRIs) were thought to work the same way, they did not produce the same long-term changes in anxiety behavior in the [adult mice](#).

The mice exposed to Lexapro had permanent changes in serotonin neurotransmission and were less anxious as adults than the mice exposed to Prozac, said study senior author Anne M. Andrews, professor of psychiatry and chemistry and biochemistry and the Richard Metzner Endowed Chair in Clinical Neuropharmacology at the Semel Institute for Neuroscience & Human Behavior and California NanoSystems Institute.

"This was quite surprising, since these medications belong to the same drug class and are believed to work by the same mechanism. The

implications of these findings are that with additional investigation, it may be possible to identify specific antidepressants that are safer for pregnant women," Andrews said. "It's important to recognize that major depressive disorders and [anxiety disorders](#) are serious medical conditions that often require therapeutic intervention. Prescribing the safest medication for mother and child is paramount."

The results of the six-year study appear early online Dec. 19, 2014 in the peer-reviewed journal *Neuropsychopharmacology*.

SSRIs like Prozac and Lexapro act by blocking the actions of a protein called the serotonin transporter, which removes the neurotransmitter serotonin from the signaling space between neurons. Andrews and her team also studied mice that had been genetically engineered to have a reduction or absence of serotonin transporters in the brain. They were able to compare early antidepressant exposure to permanent reductions in [serotonin transporter](#) function.

Genetic reductions in serotonin transporters are thought to be a risk factor, particularly when combined with stressful life experiences, for developing anxiety and mood disorders. And in fact, the genetically engineered mice Andrews studied showed more anxiety as adults.

"It might be possible that when mothers are treated for depression or anxiety during pregnancy that certain SSRIs may promote resilience to developing these disorders in children later in life," Andrews said.

"However, it will take much more research for us to understand whether this is true and whether certain SSRIs may be better at promoting these effects."

Going forward, Andrews and her team plan to investigate the effects of early exposure to antidepressants on the architectures of serotonin neurons. Based on the current findings, they suspect that early exposure

to Lexapro may alter the way [serotonin](#) neurons innervate brain regions involved in mood and anxiety behavior. They also plan to investigate other SSRIs such as Paxil and Zoloft.

"Current antidepressant therapies are ineffective in treating [anxiety](#) and depression in large numbers of patients, and advances in predicting individual responses are hindered by difficulties associated with characterizing complex influences of genetic and environmental factors on serotonergic transmission in humans," the study states. "Highly controlled animal models, such as those studied here, represent avenues by which to identify factors potentially influencing behavioral domains associated with emotion-related disorders."

Provided by University of California, Los Angeles

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