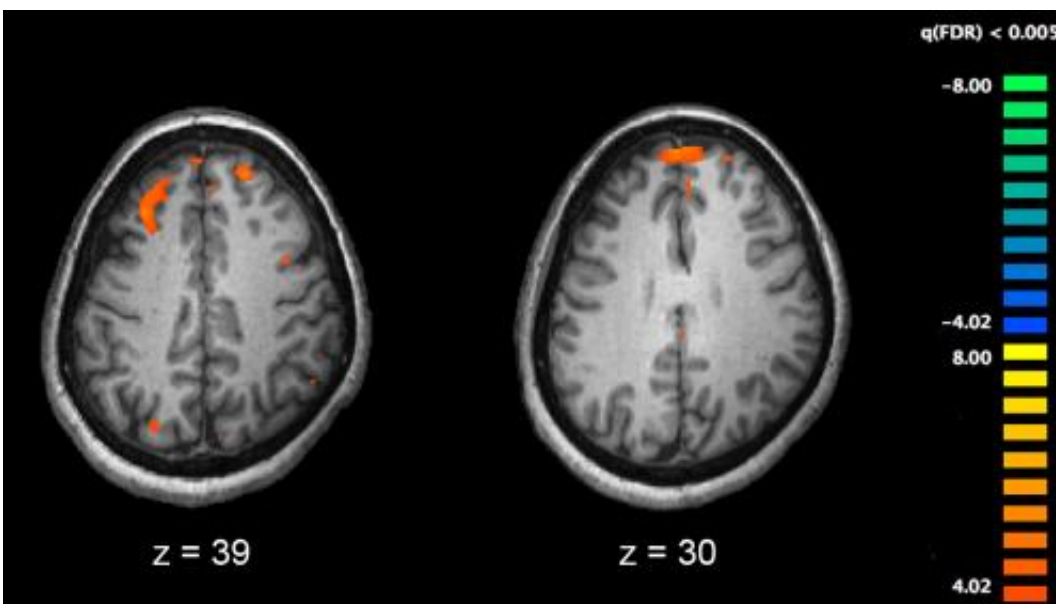


Schizophrenia: Nurture cannot overcome nature

September 3 2020, by Nicole Feldman



Functional magnetic resonance imaging (fMRI) and other brain imaging technologies allow for the study of differences in brain activity in people diagnosed with schizophrenia. The image shows two levels of the brain, with areas that were more active in healthy controls than in schizophrenia patients shown in orange, during an fMRI study of working memory. Credit: Kim J, Matthews NL, Park S./PLoS One.

In the great nature vs. nurture debate, nature just got a win.

UCI research led by Amal Alachkar found that too much of a certain amino acid in utero caused [schizophrenia](#) in mice despite the quality of

postpartum caregiving—and the study suggests that the same would be true with humans.

The team plans to use the findings to develop better treatments for—and possibly avert—schizophrenia and other psychiatric disorders.

"My principle is to not wait until you see the symptoms," says Alachkar, associate professor of teaching in pharmaceutical sciences. "The roots of the symptoms are very, very early, so why don't we do something to prevent rather than to intervene?"

The nature of schizophrenia

The study, published in *Communications Biology*, is the most recent of three showing how mice that receive extra methionine, an amino acid essential to metabolism, experience genetic and [behavioral changes](#) that mark schizophrenia in humans.

The [first](#) found that giving extra methionine to adult mice caused them to exhibit symptoms of schizophrenia, such as social withdrawal and impaired communication, memory and reasoning skills. The [second](#) demonstrated that mice that received excess methionine during the brain development stage of pregnancy had offspring whose genes and behavior indicate schizophrenia in people.

These studies led Alachkar to ask: Were schizophrenic mother mice inducing symptoms in their children, or were these symptoms purely natural?

"We know that caregiving in the early stage of life can be very important for psychiatric disorders," she says.

The third study answers the question. The researchers switched the

offspring at birth so that the pups with extra methionine were fostered by mothers without it and vice versa.

It made no difference: The pups with excess methionine showed signs of schizophrenia, and those without it did not. Nurture was unable to rescue the former from developing schizophrenic behaviors. Nor did it engender the disorder in offspring without the extra amino acid.

"That means there are changes that occur very early in life, before nurture can have any effect," Alachkar says.

Working with UCI's Geoffrey Abbott, professor of physiology & biophysics, and Pierre Baldi, Distinguished Professor of computer science, the team analyzed the pups' brain chemistry, genes and activity. They found that about 800 genes were affected by the excess methionine and that within 24 hours of birth, these genes had altered the pups' brains. The results match schizophrenia in humans, and the researchers are confident that the changes the mice experienced would occur in people as well.

Because they were able to detect the differences so early in life, the scientists believe that a drug could be developed to stop these changes from happening.

"This could point the way to biomarkers or even potential therapies for schizophrenia," Abbott says.

Other applications

The goal is to create pharmaceuticals to treat, cure or—ideally—prevent schizophrenia. Currently, medications can only address a few symptoms, such as hallucinations.

"But what's disabling in schizophrenia patients are the cognitive dysfunctions that make them really withdrawn from life, unable to even have employment," Alachkar says. "If we were able to develop a better treatment, those patients would be able to work, to communicate with others, to live a normal life."

And [schizophrenia patients](#) may not be the only ones to benefit.

One gene that showed a particularly large change is already associated with epilepsy, autism and Alzheimer's, "suggesting at least one common thread between these diseases and schizophrenia," Abbott notes.

"There are a lot of overlapping symptoms between schizophrenia and autism and between schizophrenia and Alzheimer's," Alachkar says. "So I started to think, 'Are we seeing the same mechanisms that start very, very early in life?'"

She wonders whether autism, schizophrenia and Alzheimer's are actually the same disease presenting itself differently in childhood, adolescence and old age, respectively.

Says Alachkar: "I'm very interested now in collaborating to see if we can use these pathways as targets for therapies not only for schizophrenia, but also for autism and Alzheimer's."

More information: Siwei Chen et al. Metabolomic and transcriptomic signatures of prenatal excessive methionine support nature rather than nurture in schizophrenia pathogenesis, *Communications Biology* (2020). [DOI: 10.1038/s42003-020-01124-8](https://doi.org/10.1038/s42003-020-01124-8)

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