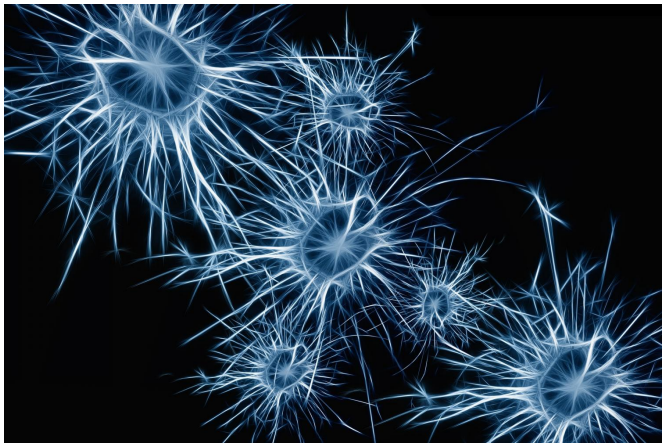
Inflammation inhibitor blocks neurodevelopmental disorders in mouse model

19 March 2019, by Andy Fell



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Work published this week in the journal *Proceedings of the National Academy of Sciences* shows that an enzyme inhibitor developed by Professor Bruce Hammock and colleagues at UC Davis reduced inflammation in the brains of mice born to mothers with maternal immune activation. Inflammation triggered by the enzyme, soluble epoxide hydrolase, is linked to neurodevelopmental disorders in these mice.

"Inhibiting that enzyme stops the inflammation and the development of autism-like and schizophrenia-like symptoms in animal models," said co-author Kenji Hashimoto, a professor with the Chiba University Center for Forensic Mental Health, Japan.

The work flows from the idea that development of disorders such as autism or schizophrenia can be influenced by infections during pregnancy that expose the developing fetus to inflammatory chemicals. These disorders also have a strong genetic component.

By inhibiting soluble epoxide hydrolase, the researchers reversed cognitive and social interaction deficiencies in the mice pups. This might be due to an increase in natural brain chemicals that prevent brain inflammation.

Maternal immune response and autism

"There is mounting evidence that inappropriate maternal immune responses during pregnancy to infection contributes elevated risk to autism spectrum disorder, at least in a fraction of cases," said Isaac Pessah, distinguished professor of molecular biosciences and associate dean of research and graduate education at the UC Davis School of Veterinary Medicine. Pessah was not involved in the study.

The findings show that a mouse model of some of the symptoms in autistic children can be suppressed by inhibiting soluble epoxide hydrolase, a target not previously explored, Pessah said.

More information: Key role of soluble epoxide hydrolase in the neurodevelopmental disorders of offspring after maternal immune activation, *Proceedings of the National Academy of Sciences* (2019). DOI: 10.1073/pnas.1819234116 , www.pnas.org/content/early/2019/03/18/1819234116

Provided by UC Davis

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