

Peripheral immune system may regulate vulnerability to depression

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A new study shows that immune cells outside the brain may regulate propensity to develop depression. The data were presented today at the American College of Neuropsychopharmacology (ACNP) Annual Meeting.

Depression is a [chronic disorder](#) with a devastating impact on the quality of life, health and [life expectancy](#) of those who suffer from the disorder. The underlying causes of the disorder remain something of a mystery.

In a study, led by Georgia Hodes at the Icahn School of Medicine at Mount Sinai, the effects of the circulating pro-inflammatory immune chemical called interleukin-6 on depression-like behaviors was investigated in rodents.

The investigators found that rodents with increased propensity to show depression-like behaviors had elevated levels of circulating interleukin-6, suggesting that individual differences in the peripheral immune system contributes to vulnerability to developing depression.

To more directly investigate the role for immune responses in depression-like behaviors, the investigators used irradiation to lesion the immune system of mice. They then carried out [bone marrow transplants](#) to replace the immune system with one from mice that showed either high or low levels of interleukin-6 levels in response to stress. It was found that mice that received transplants from high-responding donors had increased expression of depression-like behaviors compared to those

who received transplants from low-responding donors.

The findings suggest that circulating immune chemicals that can act in the brain may influence vulnerability to depression. As noted by Dr. Hodes, "These studies represent a new way of thinking about diagnosing and treating depression as an inflammatory illness in the body rather than the brain."

Future studies will be required in humans to determine if a similar role for the peripheral immune system in depression can be established. If so, this may lead to novel treatment approaches for the disorder.

Provided by American College of Neuropsychopharmacology

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