Yale scientists revisit biochemical basis for depression

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Symptoms of depression and anxiety can be induced in mice by increasing levels of acetylcholine, suggesting that depression may have different biochemical roots than previously believed, Yale School of Medicine researchers report the week of Feb. 11 in the journal Proceedings of the National Academy of Sciences.

A quarter of a century ago, the introduction of Prozac changed how depression was treated. The drug targets brain cell receptors that respond to the neurotransmitter serotonin and is called a SSRI, or serotonin reuptake inhibitor. Today, the most commonly prescribed antidepressants are SSRIs.

However, the new study suggests depression could arise from disruption of a different neurotransmitter system.

“We have actually seen depression-like behavior in mice when there is a breakdown in a acetylcholine regulation,” said Marina Picciotto [1], the Charles B. G. Murphy Professor of Psychiatry and professor of neurobiology and of pharmacology and senior author of the paper. “We have also seen altered acetylcholine levels in the brain of people with depression, which shows that this is a good model for the human illness.”

The team also found that they could reduce those same symptoms in mice by introduction of an SSRI. Picciotto said the findings suggest that it is possible that depression is not caused by disruption of serotonin signaling at all.

“Serotonin may be treating the problem, but acetylcholine disruption may be a primary cause,” said Picciotto. “If we can treat the root cause, perhaps we can get a better response from the patient.”

First author of the paper is Yann S. Mineur.

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[1] http://bbs.yale.edu/people/marina_picciotto.profile
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