

Insights into depression could aid development of new treatments

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Fresh insights into changes in the brain linked to depression could pave the way for new therapies.

The study also sheds light on why a certain category of [antidepressant drugs](#) stop working in some people.

Researchers at the University of Edinburgh pinpointed a key molecule that may protect the brain from depression.

The team studied mice that were bred to have defects in their ability to activate this molecule, called eIF4E.

These animals showed signs of depression, including reduced levels of the hormone serotonin, which is a hallmark of the condition.

The mice also showed behavioural changes linked to depression, such as disinterest in food.

Treatment with a commonly prescribed antidepressant called fluoxetine failed to produce a response in the mice.

This suggests that activation of eIF4E is required for the beneficial antidepressant effects of fluoxetine, which belongs to a category of medicines called selective serotonin re-uptake inhibitors (SSRIs).

Researchers say this could help explain why some patients stop responding to SSRIs.

Previous studies have shown that eIF4E plays a key role in regulating protein synthesis in the [brain](#). Defects in eIF4E have been linked with other neurological conditions, including autism and Fragile X syndrome. This is the first time the molecule has been implicated in depression.

Experts say the latest findings could help develop new medications for [depression](#), which affects about one in four people in the UK each year.

More information: Inês Silva Amorim et al, Loss of eIF4E phosphorylation engenders depression-like behaviors via selective mRNA translation, *The Journal of Neuroscience* (2018). [DOI: 10.1523/JNEUROSCI.2673-17.2018](#)

Provided by University of Edinburgh

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