

Faulty serotonin 1A receptor 'prevents' calm mental states

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Researchers at the MedUni Vienna have, for the first time, investigated the influence of the serotonin system on the default mode network (DMN) in the human brain and discovered that, in people with depression, the inhibitory effect of the serotonin 1A receptor is extremely small. This means, says Siegfried Kasper, Head of the University Department of Psychiatry and Psychotherapy, that affected patients “are virtually never able to be mentally calm”.

The regions of the brain in the default mode network, which is also called the default state network, are only active when we are not doing anything, or when our thoughts drift and we find ourselves in a state of complete calm. The serotonin 1A receptor is greatly involved with this.

Inner calm can only be achieved when it is adequately stimulated. In people with [depression](#), this mechanism does not function properly. Says Kasper: “As a result, the patients are constantly on edge.”

The study, carried out by Andreas Hahn at the University Department of [Psychiatry](#) and [Psychotherapy](#) has now been published in the highly respected journal *Proceedings of the National Academy of Sciences (PNAS)*. Alongside doctoral student Andreas Hahn, assisted by Rupert Lanzenberger, the study also involved other scientists at the MedUni Vienna such as Wolfgang Wadsak and Markus Mitterhauser from the University Department of Nuclear Medicine and Christian Windischberger from the Centre for Medical Physics and Biomedical Technology (MRI centre of excellence).

“This discovery opens up new opportunities for the research and treatment of psychiatric conditions such as depression, schizophrenia and anxiety states at molecular level,” says Kasper. This forms the basis for the development of effective drugs that will help to adequately influence the serotonin 1A receptor.

More information: [Differential modulation of the default mode network via serotonin-1A receptors](#). Hahn A, et al. *Proc Natl Acad Sci USA*, 2012 Feb 14; 109(7);2619-24.

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