Traumatic response to bad memories can be minimized

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(PhysOrg.com) -- UC Irvine researchers have identified the brain mechanism that switches off traumatic feelings associated with bad memories, a finding that could lead to the development of drugs to treat panic disorders.

Scientists from UCI and the University of Muenster in Germany found that a small brain protein called neuropeptide S is involved in erasing traumatic responses to adverse memories by working on a tiny group of neurons inside the amygdala where those memories are stored.

“The exciting part of this study is that we have discovered a completely new process that regulates the adverse responses to bad memories,” said Rainer Reinscheid, pharmacology and pharmaceutical sciences associate professor at UCI. “These findings can help the development of new drugs to treat conditions in which people are haunted by persistent fears, such as posttraumatic stress disorder or other panic disorders.” The study appears in the July 31 issue of *Neuron.*

In tests, scientists exposed mice to situations that caused adverse memories. The scientists saw that when NPS receptors in amygdala neurons are blocked, the traumatic responses to bad memories persisted longer. In turn, when scientists treated the mice with compounds activating these receptors, traumatic responses disappeared faster.

After a traumatic experience, environmental cues often become associated with the bad experience and re-exposure to the same

environment can trigger fearful emotions or even panic attacks, according to Reinscheid.

Other research has shown that forgetting such negative experiences may require "new learning," such as re-exposure to the place where the original experience occurred but this time without any harmful consequences. Reinscheid said this process, called the extinction of memories, occurs in both humans and laboratory animals such as mice. Until this study, scientists did not know about the specific neurons and molecules involved with extinction learning of fear memories in the brain.

Previous work by Reinscheid's group has shown that NPS is involved in regulating wakefulness and anxiety. Last year, they found evidence that a particular genetic variant of the NPS receptor may increase vulnerability to panic disorder.

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Provided by UC Irvine