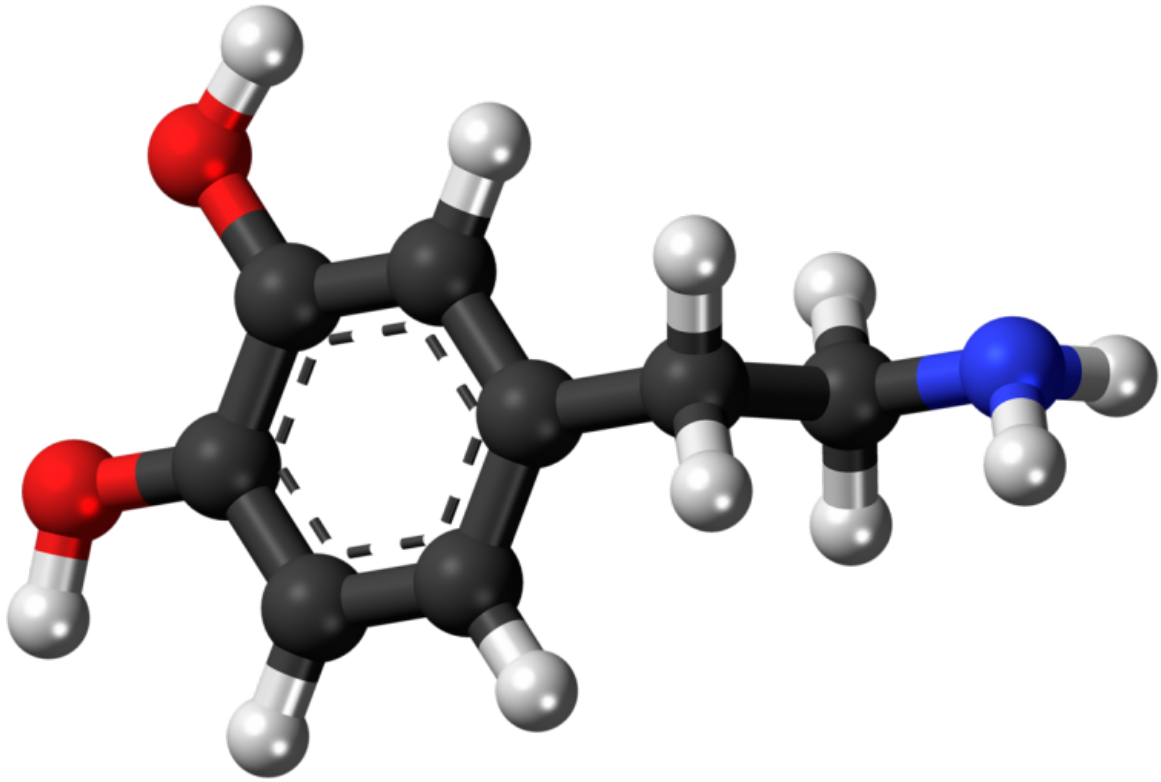
Brain chemical may offer new clues in treating chronic pain

May 6 2015, by Katherine Morales



Ball-and-stick model of the dopamine molecule, a neurotransmitter that affects the brain's reward and pleasure centers. Credit: Jynto/Wikipedia

A chemical in the brain typically associated with cognition, movement and reward-motivation behavior—among others—may also play a role in

promoting chronic pain, according to new research at The University of Texas at Dallas.

The chemical, dopamine, sets the stage for many important brain functions, but the mechanisms that cause it to contribute to chronic pain are less well understood.

In a recent paper published in The *Journal of Neuroscience*, UT Dallas researchers followed the sequence of pain impulses traveling from the brain to the spinal cord in mice. They found that by removing a collection of neurons called A11 that contain dopamine, chronic pain was selectively diminished.

"These findings demonstrate a novel role for how dopamine contributes to maintaining chronic pain states," said Dr. Ted Price, associate professor in the School of Behavioral and Brain Sciences at UT Dallas. "This may open up new opportunities to target medicines that could reverse chronic pain."

Pain signals travel like electricity from an injury to the spinal cord where they pass on electrical or chemical pain signals to other cells. Those pain signals then travel upward and relay that information to neurons in the brain where they can be distributed throughout. There is no single pain center in the brain, but there is substantial evidence that chronic pain changes how these pain centers are activated.

In people with chronic pain, neurons continue to send pain signals to the brain, even in the absence of injury, but the causes of this are not known.

A potential explanation comes from A11. These neurons didn't affect acute pain, but they did have a profound effect on chronic pain, researchers found. By targeting these neurons in mice with chronic pain, the researchers permanently reversed a chronic pain state.

Price said that previous studies have examined the role of other neurotransmitters in chronic pain including norepinephrine and serotonin.

"By process of elimination, we decided to look more closely at dopamine. We used a toxin that affected A11 neurons, and that's when we found that acute pain signals were still normal, but chronic pain was absent," he said.

In 2011, the Institute of Medicine estimated that more than 100 million Americans suffer from chronic pain, a condition that costs more than $600 billion each year in medical care and lost productivity.

Understanding the basis of chronic pain and all of its contributing factors could help point to more effective treatments.

"In future studies, we would like to gain a better understanding of how stress interacts with A11," Price said. "And we'd like to know more about the interaction between molecular mechanisms that promote chronic pain and dopamine."

Provided by University of Texas at Dallas