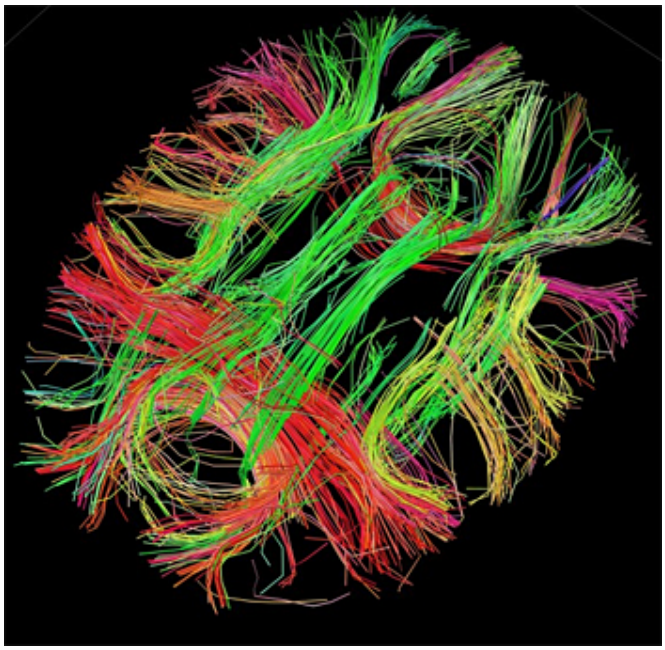
Your brain on drugs: Functional differences in brain communication in cocaine users

29 April 2015



White matter fiber architecture of the brain. Credit: Human Connectome Project.

The brain function of people addicted to cocaine is different from that of people who are not addicted and often linked to highly impulsive behavior, according to a new scientific study.

The variation in the way that different regions of the brain connect, communicate and function in people addicted to cocaine is an observation uncovered for the first time by a collaborative research team led by The University of Texas Medical Branch at Galveston and Virginia Commonwealth University. These findings were recently published in *NeuroImage: Clinical*.

Cocaine addiction, also called cocaine use disorder, afflicts an estimated 800,000 people in the U.S. alone, but despite decades of attempts, FDA-approved medications for cocaine use disorder remain to be discovered.

People who are addicted to cocaine are often highly impulsive and are prone to acting quickly, without regard to negative consequences. Impulsivity is associated with increased relapse to cocaine abuse and, thus, impulsivity may serve as an important behavioral target for the development of relapse prevention medications.

To measure impulsivity in humans, scientists often use the Go/NoGo task, which monitors a person's ability to thwart an impulsive response. In this task, participants are instructed to make a certain response, or "Go" when presented with a particular image and withhold their responses or "NoGo" when presented with different images. The present study sought to determine whether people with cocaine use disorder display impaired task performance and altered patterns of brain activity compared to non-cocaine users.

Researchers traditionally study differences in regional brain activity using functional magnetic resonance imaging. The researchers in this study took fMRI analysis one step further to decipher the connections and direction of information flow between brain regions in both cocaine and non-cocaine users, using a fMRI-based technique called Dynamic Causal Modeling. The DCM-based imaging provides a new tool to study brain connectivity and strategize the design and development of medications that can boost and/or restore such impairment in cocaine use disorder.

The study enrolled 13 cocaine users and 10 non-cocaine users to evaluate brain connectivity during performance of the Go/NoGo task within an fMRI scanner. Both cocaine users and non-cocaine users performed the task equally well, suggesting that the average ability to inhibit a response was the same in the two groups. However, there were intriguing differences between the cocaine users and non-users in the strength of communication

between key brain structures.

The left caudate, a brain structure known to control motor function, was itself activated in both groups of subjects during NoGo response inhibition. However, the cortical brain structures that regulate left caudate activity differed between cocaine users and non-cocaine users during harder questions of the Go/NoGo task.

"These findings suggest that, while some cortical brain regions show altered activity in cocaine users, other regions may compensate for cocaine-associated deficits in function," said UTMB lead author Kathryn A. Cunningham, Chauncey Leake Distinguished Professor of Pharmacology and Director of the Center for Addiction Research. "Targeting altered brain connections in cocaine use disorder for therapeutic development is a fresh idea, offering a whole new arena for research and the potential to promote abstinence and prevent relapse in these vulnerable individuals."

More information: *NeuroImage: Clinical*, www.sciencedirect.com/science/ … ii/S2213158215000571

Provided by University of Texas Medical Branch at Galveston