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Ritalin may cause changes in the brain's reward areas

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(PhysOrg.com) -- A common treatment for attention deficit/hyperactivity disorder, prescribed millions of times a year, may change the brain in the same ways that cocaine does, a new study in mice suggests. Research from Rockefeller University shows that methylphenidate, commonly known as Ritalin, causes physical changes in neurons in reward regions of mouse brains. In some cases, the effects overlapped with those of cocaine.

The study highlights the need for more research into methylphenidate's long-term effects on the brain, the researchers say. The findings were published February 3 in the *Proceedings of the National Academy of Sciences*.

The researchers, led by Yong Kim, senior research associate, and Paul Greengard, Vincent Astor Professor and head of the Laboratory of Molecular and Cellular Neuroscience, exposed mice to two weeks of daily injections of cocaine or methylphenidate. They then examined reward areas of the brain for changes in dendritic spine formation — related to the formation of synapses and the communication between nerve cells — and the expression of a protein called delta Fos B, which has been implicated in the long-term actions of addictive drugs.

Both drugs increased dendritic spine formation and the expression of delta Fos B; however, the precise patterns of their effects were distinct. They differed in the types of spines affected, the cells that were affected and the brain regions. In some cases there was overlap between the two

drugs, and in some cases methylphenidate produced greater effects than cocaine, for example, on protein expression in certain regions. Both methylphenidate and cocaine are in the class of drugs known as psychostimulants.

“Methylphenidate, which is thought to be a fairly innocuous compound, can have structural and biochemical effects in some regions of the brain that can be even greater than those of cocaine,” says Kim. “Further studies are needed to determine the behavioral implications of these changes and to understand the mechanisms by which these drugs affect synapse formation.”

More information: *Proceedings of the National Academy of Sciences*: February 3, 2009

Provided by Rockefeller University