Health Headlines

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Mechanism of Cocaine 'High' Pinpointed in Mice

By Keith Mulvihill

NEW YORK (Reuters Health) - Scientists have figured out how to block the pleasurable effects of cocaine in mice.

The findings could point the way toward treatments for cocaine addiction, according to researchers led by Dr. George Uhl of the National Institute on Drug Abuse in Baltimore, Maryland. As yet, there are no medications that effectively treat cocaine dependence.

``We now know how cocaine produces its euphoria, something that previous studies had not been able to identify correctly,'' Uhl said in an interview with Reuters Health. His team's findings are published in the April 24th issue of the Proceedings of the National Academy of Sciences.

Working with genetically engineered rats, Uhl and colleagues examined how cocaine causes euphoria--and leads to addiction--by blocking certain types of ``transporters'' in the brain. Transporters are pump-like structures that clear chemical messengers--called neurotransmitters--from the synapses, or gaps, between brain cells. In effect, transporters limit the cells' exposure to these chemical messengers, and, consequently, limit the neurotransmitters' effects, Uhl explained. Cocaine is known to block transporters for the neurotransmitters dopamine, serotonin and norepinephrine, increasing the brain's exposure to these chemicals.

``Cocaine...acts as transporters for dopamine and for serotonin to produce its euphoric high. There is redundancy. If you eliminate one site, the other is able to still produce a rewarding signal,'' Uhl added.

When Uhl and colleagues gave cocaine to genetically engineered mice that had no dopamine transporters, the mice still appeared to become addicted to the cocaine. The mice "sought" the drug, spending most of their time in a part of their cage where they were accustomed to receiving cocaine. The same thing happened in mice that lacked only serotonin transporters.

However, when the mice lacked dopamine transporters and at least 50% or more of the serotonin transporters, they did not choose to be in the part of the cage where they received cocaine.

``Many brain processes have redundancy, so that they can continue even if part of the brain is damaged or blocked by drugs. Cocaine may be so rewarding because it works on two circuits that both can provide drug reward signals,'' Uhl told Reuters Health.

``These findings are important for treatment since no effective cocaine treatment now exists,'' Uhl stated.

``Many of the attempts at cocaine treatments to date have focused on the dopamine transporter alone. We now know that this is not enough. Effective treatments are likely to have to influence both the dopamine and serotonin reward circuits in the brain in order to be effective,'' he said.

``Making drugs that could do this is feasible, but a bit tricky,'' Uhl concluded.

SOURCE: Proceedings of the National Academy of Sciences 2001;98:5300-

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