Recovery of dopamine function emerges with recovery from smoking

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A new study in *Biological Psychiatry* reports that smoking-related deficits in brain dopamine, a chemical implicated in reward and addiction, return to normal three months after quitting. The normalization of dopamine systems suggests smoking-related deficits are a consequence of chronic smoking, rather than a risk factor. These findings raise the possibility that treatments might be developed that

normalize the dopamine system in smokers.

According to first author Dr. Lena Rademacher, postdoctoral fellow at the University of Lübeck in Germany, a major challenge in understanding substance-related disorders lies in uncovering why only some individuals become addicted.

Researchers think some people could possess a trait that predisposes them to addiction, and suspect that brain circuits involving dopamine may be involved. Drugs of abuse release dopamine, and addiction to nicotine is associated with abnormalities in the dopamine system. But researchers are uncertain if smoking induces those abnormalities or if they already exist and contribute to risk of nicotine addiction.

Senior author Dr. Ingo Vernaleken, Professor at RWTH Aachen University in Germany, led a team of researchers examining dopamine function in chronic smokers before and after long-term cessation. The researchers used a brain imaging technique called positron emission tomography to measure an index of the capacity for dopamine production in 30 men who were nicotine-dependent smokers and 15 nonsmokers. After performing an initial scan on all participants, 15 smokers who successfully quit were scanned again after three months of abstinence from smoking and nicotine replacement.

The initial scan revealed a 15-20% reduction in the capacity for dopamine production in smokers compared with nonsmokers. The researchers expected this impairment to persist even after quitting, which would suggest it could be a marker of vulnerability for nicotine addiction. "Surprisingly, the alterations in dopamine synthesis capacity normalized through abstinence," said Rademacher.

The role of dopamine in vulnerability toward nicotine addiction cannot be excluded, but the findings suggest that altered dopamine function of

smokers is a consequence of nicotine consumption rather than the cause.

Dr. John Krystal, Editor of Biological Psychiatry, noted the implications of these findings for developing better ways to help smokers trying to quit. "This study suggests that the first three months after one stops smoking may be a particularly vulnerable time for relapse, in part, because of persisting dopamine deficits. This observation raises the possibility that one might target these deficits with new treatments."

More information: Lena Rademacher et al. Effects of Smoking Cessation on Presynaptic Dopamine Function of Addicted Male Smokers, *Biological Psychiatry* (2016). DOI: 10.1016/j.biopsych.2015.11.009

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