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Brief Report

Exposure to tobacco smoke containing either high or low levels of nicotine during adolescence: Differential effects on choline uptake in the cerebral cortex and hippocampus

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Abstract

Introduction: There is a lack of experimental studies that investigate the effects of tobacco smoke exposure during adolescence. Here, we investigated the effects of tobacco smoke generated from cigarettes containing either high or low levels of nicotine on the cholinergic system.

Methods: From postnatal day (PN) 30 to 45, 18 C57BL/6 (inbred) and 16 Swiss (outbred) mice of both sexes were exposed to tobacco smoke (whole body exposure for 8 hr/day and 7 days/week) generated from one of two reference research cigarettes: type 3R4F (HighNIC group—nicotine = 0.73 mg/cigarette) or type 4A1 (LowNIC group—nicotine = 0.14 mg/cigarette). Control mice (CT) were exposed to air. On PN 45, cotinine (nicotine metabolite) serum levels and [^{3}H]choline uptake in the cerebral cortex and hippocampus were assessed.

Results: Cotinine serum levels were eight times higher in HighNIC mice (C57BL/6:142.0 ± 16.7 ng/ml and Swiss: 197.6 ± 11.1 ng/ml) when compared with LowNIC ones (C57BL/6:17.4 ± 7.4 ng/ml and Swiss: 24.6 ± 2.2 ng/ml). Only HighNIC mice presented a significant increase in [^{3}H]choline uptake in the hippocampus (C57BL/6: HighNIC > CT and HighNIC > LowNIC, $p < .001$ and Swiss: HighNIC > CT and HighNIC > LowNIC, $p < .001$), whereas in the cerebral cortex, both HighNIC and LowNIC mice presented increased [^{3}H]choline uptake (C57BL/6: HighNIC > CT and LowNIC > CT, $p < .05$ and Swiss: HighNIC > CT and LowNIC > CT, $p < .001$).

Discussion: Our results indicate that tobacco smoke exposure during adolescence increases [^{3}H]choline uptake. However, the effects are dependent on the type of cigarette and on the brain region.

Introduction

Most smokers begin their habit during adolescence, so that tobacco dependence is often considered to be a "pediatric disease." In fact, around the world, between 82,000 and 99,000 young people start smoking daily, and 4 million deaths each year are attributed to tobacco smoking (Prokhorov et al., 2006).

Nicotine is considered as the most important psychoactive substance present in tobacco and the one responsible for tobacco addiction (for review: Fowler, Arends, & Kenny, 2008). Nicotine indirectly affects a wide variety of neurotransmitter systems; however, as an acetylcholine analog, the ionotropic nicotinic acetylcholine receptors (nAChRs) are the primary cellular mediators of its effects. In fact, regarding the cholinergic system, there is evidence that indicates unique features of the response of the adolescent brain to nicotine: more profound and persistent upregulation of nAChRs as compared with adults and prolonged suppression of cholinergic synaptic activity upon withdrawal (for review: Slotkin, 2008), indicating, along with other observations, that adolescence is a period of vulnerability to nicotine.

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