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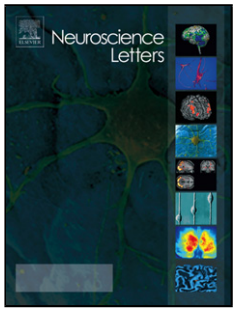

Title: Effects of antidyskinetic nicotine treatment on dopamine release in dorsal and ventral striatum

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Title: Effects of antidyskinetic nicotine treatment on dopamine release in dorsal and ventral striatum

Authors: Sakari Leino[a], Sini K. Koski[a], Saara Rannanpää[a], Outi Salminen[a]

[a]Division of Pharmacology and Pharmacotherapy, Faculty of Pharmacy, University of Helsinki, 00014 Helsinki, Finland

[*]**Corresponding Author:** Outi Salminen, Faculty of Pharmacy, P.O. Box 56, 00014 University of Helsinki, Finland; +358503199167; outi.salminen@helsinki.fi

Highlights

- Nicotine treatment inhibited development of levodopa-induced dyskinesia in mice
- Antidyskinetic nicotine treatment reduced α6β2*-mediated dopamine release
- α4β2*-mediated dopamine release was unaffected by antidyskinetic nicotine treatment
- Nicotine treatment restored basal dopamine release in lesioned ventral striatum

Abstract

The treatment of Parkinson's disease is often complicated by levodopa-induced dyskinesia (LID), and antidyskinetic treatment options are currently sparse. Nicotinic acetylcholine receptors have been suggested as potential targets for treatment of LID, as nicotinic agonists have been reported to alleviate LID in animal models. We aimed at the first independent replication of an antidyskinetic effect by nicotine using a mouse model of LID, and at investigation of its mechanisms by studying the release of [^{3}H]dopamine from synaptosomes prepared from the dorsal and ventral striatum. Chronic nicotine treatment in drinking water inhibited the development of LID in mice lesioned unilaterally with 6-hydroxydopamine and treated chronically with levodopa and benserazide. The antidyskinetic nicotine treatment had no effect on [^{3}H]dopamine release mediated by α4β2* nicotinic receptors, but decreased α6β2*-mediated [^{3}H]dopamine release in the lesioned dorsal striatum and the ventral striatum. In addition, nicotine treatment restored [^{3}H]dopamine release in the lesioned ventral striatum to intact levels. The results support a role for nicotinic receptors as drug targets for treatment of LID, and suggest that