* **CBD WORKS FOR SMOKING CESSATION**
* [**Format**: Abstract](https://www.ncbi.nlm.nih.gov/pubmed/23685330)

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[Addict Behav.](https://www.ncbi.nlm.nih.gov/pubmed/23685330) 2013 Sep;38(9):2433-6. doi: 10.1016/j.addbeh.2013.03.011. Epub 2013 Apr 1.

# Cannabidiol reduces cigarette consumption in tobacco smokers: preliminary findings.

[Morgan CJ](https://www.ncbi.nlm.nih.gov/pubmed/?term=Morgan%20CJ%5BAuthor%5D&cauthor=true&cauthor_uid=23685330)1, [Das RK](https://www.ncbi.nlm.nih.gov/pubmed/?term=Das%20RK%5BAuthor%5D&cauthor=true&cauthor_uid=23685330), [Joye A](https://www.ncbi.nlm.nih.gov/pubmed/?term=Joye%20A%5BAuthor%5D&cauthor=true&cauthor_uid=23685330), [Curran HV](https://www.ncbi.nlm.nih.gov/pubmed/?term=Curran%20HV%5BAuthor%5D&cauthor=true&cauthor_uid=23685330), [Kamboj SK](https://www.ncbi.nlm.nih.gov/pubmed/?term=Kamboj%20SK%5BAuthor%5D&cauthor=true&cauthor_uid=23685330).

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### Abstract

The role of the endocannabinoid system in nicotine addiction is being increasingly acknowledged. We conducted a pilot, randomised double blind placebo controlled study set out to assess the impact of the ad-hoc use of cannabidiol (CBD) in smokers who wished to stop smoking. 24 smokers were randomised to receive an inhaler of CBD (n=12) or placebo (n=12) for one week, they were instructed to use the inhaler when they felt the urge to smoke. Over the treatment week, placebo treated smokers showed no differences in number of cigarettes smoked. In contrast, those treated with CBD significantly reduced the number of cigarettes smoked by ~40% during treatment. Results also indicated some maintenance of this effect at follow-up. These preliminary data, combined with the strong preclinical rationale for use of this compound, suggest CBD to be a potential treatment for nicotine addiction that warrants further exploration.

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# Inhibition of monoacylglycerol lipase reduces nicotine withdrawal.

[Muldoon PP](https://www.ncbi.nlm.nih.gov/pubmed/?term=Muldoon%20PP%5BAuthor%5D&cauthor=true&cauthor_uid=25258021)1, [Chen J](https://www.ncbi.nlm.nih.gov/pubmed/?term=Chen%20J%5BAuthor%5D&cauthor=true&cauthor_uid=25258021), [Harenza JL](https://www.ncbi.nlm.nih.gov/pubmed/?term=Harenza%20JL%5BAuthor%5D&cauthor=true&cauthor_uid=25258021), [Abdullah RA](https://www.ncbi.nlm.nih.gov/pubmed/?term=Abdullah%20RA%5BAuthor%5D&cauthor=true&cauthor_uid=25258021), [Sim-Selley LJ](https://www.ncbi.nlm.nih.gov/pubmed/?term=Sim-Selley%20LJ%5BAuthor%5D&cauthor=true&cauthor_uid=25258021), [Cravatt BF](https://www.ncbi.nlm.nih.gov/pubmed/?term=Cravatt%20BF%5BAuthor%5D&cauthor=true&cauthor_uid=25258021), [Miles MF](https://www.ncbi.nlm.nih.gov/pubmed/?term=Miles%20MF%5BAuthor%5D&cauthor=true&cauthor_uid=25258021), [Chen X](https://www.ncbi.nlm.nih.gov/pubmed/?term=Chen%20X%5BAuthor%5D&cauthor=true&cauthor_uid=25258021), [Lichtman AH](https://www.ncbi.nlm.nih.gov/pubmed/?term=Lichtman%20AH%5BAuthor%5D&cauthor=true&cauthor_uid=25258021), [Damaj MI](https://www.ncbi.nlm.nih.gov/pubmed/?term=Damaj%20MI%5BAuthor%5D&cauthor=true&cauthor_uid=25258021).

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### Abstract

#### BACKGROUND AND PURPOSE:

Abrupt discontinuation of nicotine, the main psychoactive component in tobacco, induces a withdrawal syndrome in nicotine-dependent animals, consisting of somatic and affective signs, avoidance of which contributes to drug maintenance. While blockade of fatty acid amide hydrolase, the primary catabolic enzyme of the endocannabinoid arachidonoylethanolamine (anandamide), exacerbates withdrawal responses in nicotine-dependent mice, the role of monoacylglycerol lipase (MAGL), the main hydrolytic enzyme of a second endocannabinoid 2-arachidonylglycerol (2-AG), in nicotine withdrawal remains unexplored.

#### EXPERIMENTAL APPROACH:

To evaluate the role of MAGL enzyme inhibition in nicotine withdrawal, we initially performed a genetic correlation approach using the BXD recombinant inbred mouse panel. We then assessed nicotine withdrawal intensity in the mouse after treatment with the selective MAGL inhibitor, JZL184, and after genetic deletion of the enzyme. Lastly, we assessed the association between genotypes and smoking withdrawal phenotypes in two human data sets.

#### KEY RESULTS:

BXD mice displayed significant positive correlations between basal MAGL mRNA expression and nicotine withdrawal responses, consistent with the idea that increased 2-AG brain levels may attenuate withdrawal responses. Strikingly, the MAGL inhibitor, JZL184, dose-dependently reduced somatic and aversive withdrawal signs, which was blocked by rimonabant, indicating a CB1 receptor-dependent mechanism. MAGL-knockout mice also showed attenuated nicotine withdrawal. Lastly, genetic analyses in humans revealed associations of the MAGL gene with smoking withdrawal in humans.

#### CONCLUSIONS AND IMPLICATIONS:

Overall, our findings suggest that MAGL inhibition maybe a promising target for treatment of nicotine dependence.

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