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PII: S0022-3956(16)30333-8

DOI: [10.1016/j.jpsychires.2017.03.005](https://doi.org/10.1016/j.jpsychires.2017.03.005)

Reference: PIAT 3087

To appear in: *Journal of Psychiatric Research*

Received Date: 25 October 2016

Revised Date: 15 February 2017

Accepted Date: 2 March 2017

Please cite this article as: Areal LB, Herlinger AL, Pelicão FS, Martins e Silva C, Pires RGW, Crack cocaine inhalation induces schizophrenia-like symptoms and molecular alterations in mice prefrontal cortex, *Journal of Psychiatric Research* (2017), doi: 10.1016/j.jpsychires.2017.03.005.

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# Crack cocaine inhalation induces schizophrenia-like symptoms and molecular alterations in mice prefrontal cortex

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## Abstract

Crack cocaine (crack) addiction represents a major social and health burden, especially seeing as users are more prone to engage in criminal and violent acts. Crack users show a higher prevalence of psychiatric comorbidities – particularly antisocial personality disorders – when compared to powder cocaine users. They also develop cognitive deficits related mainly to executive functions, including working memory. It is noteworthy that stimulant drugs can induce psychotic states, which appear to mimic some symptoms of schizophrenia among users. Social withdraw and executive function deficits are, respectively, negative and cognitive symptoms of schizophrenia mediated by reduced dopamine (DA) tone in the prefrontal cortex (PFC) of patients. That could be explained by an increased expression of D2R short isoform (D2S) in the PFC of such patients and/or by hypofunctioning NMDA receptors in this region. Reduced DA tone has already been described in the PFC of mice exposed to crack smoke.

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