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Lorena Bianchine Areal, Alice Laschuk Herlinger, Fabrício Souza Pelição, Cristina Martins e Silva, Rita Gomes Wanderley Pires

PII: S0022-3956(16)30333-8
DOI: 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


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

Lorena Blanchine Areal\textsuperscript{1,2,*}, Alice Laschuk Herlinger\textsuperscript{1,3,*}, Fabrício Souza Pelição\textsuperscript{4}, Cristina Martins e Silva\textsuperscript{1,3}, Rita Gomes Wanderley Pires\textsuperscript{1,2,3}

\textsuperscript{1}Laboratory of Molecular and Behavioral Neurobiology, Health Sciences Center, Federal University of Espirito Santo, Vitoria-ES, Brazil

\textsuperscript{2}Graduate Program in Neuroscience, Institute of Biological Sciences, Federal University of Minas Gerais, Belo Horizonte-MG, Brazil

\textsuperscript{3}Department of Physiological Sciences, Health Sciences Center, Federal University of Espirito Santo, Vitoria-ES, Brazil

\textsuperscript{4}Laboratory of Forensic Science Service, Espirito Santo State Police, Vitoria-ES, Brazil

* These authors equally contributed to this work and should be considered co-first authors.

Corresponding author: Alice L. Herlinger, Laboratory of Molecular and Behavioral Neurobiology, Health Sciences Centre, Federal University of Espirito Santo, 1468 Marechal Campos Ave., Vitoria, ES, 29043910, Brazil. E-mail: alaschuk@gmail.com.

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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