

Natureza do trabalho: Resumo Revisão de Literatura

TÍTULO

DEPENDENCE ON CRACK/COCAINE: A NEUROBIOLOGICAL AND SOCIAL ANALYSIS

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ABSTRACT

Introduction: Chemical addiction is a chronic, relapsing disease in which the use of psychoactive substances causes structural and functional changes in the brain leading to behavioral and social repercussions. In the 1990s, crack was introduced in Brazil, making the country its largest market currently¹. This is the smoked form of cocaine, seeking for faster and more intense effects, with higher addictive potential^{2,3}. They differ in the pharmacokinetics, as physical/chemical form, route of administration, genetics and effects on body⁴. Cocaine/crack are seen as a public health problem due to the growth of consumption³, but for its higher compulsivity, crack has brought major social consequences such as violence and sexual risk behaviors^{5,1}. **Objective:** To execute a neurobiological and social analysis of drugs, focusing on crack/cocaine. **Methods:** A literature review was conducted during the month of August 2015 in the databases of PubMed and BVS, with the following keywords: *crack/cocaine*, *addiction* and *treatment*. Articles published prior to 2010 were excluded. **Discussion:** Drugs operate on mesocorticolimbic pathway of the dopaminergic neurotransmission, mainly in the *nucleus accumbens* (feeling of pleasure and reinforcement in search behavior) and prefrontal cortex (inhibitory control, hypofunctioning in dependents), composing the brain reward system³. There are three interacting stages that are gradually intensified leading to addiction, they are also involved in the neuroplasticity of neural circuits: excess/intoxication, abstinence/negative affect, preoccupation/anticipation; impulse control disorders appear in the early stages and compulsive disorders in the later ones⁶. Cocaine inhibits the presynaptic dopamine transporter, increasing its quantity in the synaptic cell and unchaining the repetitive behavior behind the drug, reducing this neurotransmitters postsynaptic receptors; simultaneously, glutamatergic neurons are activated in the prefrontal cortex and glutamate is liberated, responsible for regulating the locomotor sensitization, drug seeking and conditioned responses to drug-associated stimuli^{3,7}. Crack conduces to addiction faster, to withdrawal stronger and to prognosis worse than cocaine⁸: a five-years follow-up study developed with 131 Brazilian crack users revealed that 18,5% died during this time, 56,6% of these related to homicides^{3,2}. The tendency toward compulsiveness generated by the use of crack is one of the responsible for those higher rates of violence, beyond others social conflicts, such as sexual promiscuity that increases the STDs, and also the transmission of respiratory diseases, like tuberculosis^{5,2}. Chronical use of crack disturbs the general cognitive functioning, verbal memory and attention⁸, such cognitive sequelae may prove a hindrance to the effectiveness and adherence to treatment. **Conclusion:** The crack/cocaine consumption rebounded in all social classes, although the predominance of users still remains on marginalized groups³, overlapping the profile of young man, low socioeconomic and educational levels and with larger family problems^{3,1}. In Brazil, the stigma over the dependents has been hampered the providence of effective and humane treatment⁹. This must be multidisciplinary and interdisciplinary, in need of support in social, physical, mental and legal areas, such as in the quality of life and relapse prevention strategies³. Treatments also must contain social reintegration strategies supported by health professionals, family and community¹⁰, seeking approaches that improve neurocognitive functioning of these patients.

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