LETTERS TO THE EDITORS

Cocaine use by older populations, sleep quality, and associated risks

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Reports of cocaine use by older populations have risen in recent years. The abuse of illicit substances by older adults was once considered unlikely, and aging was commonly seen as a protective factor against use of these substances. However, new cases of drug abuse by aging individuals are being reported through admissions for inpatient treatment.[1] In some cases, onset of cocaine consumption might occur after 50 years of age, contradicting previous assumptions. Yarnell[2] reported on this trend and highlighted that it is a growing health concern that has been underestimated and under-analyzed to date. Projections warn that approximately 5.7 million people over the age of 60 years will have substance-use disorder by 2020.[3]

Several demographic aspects should be considered when assessing this population. A great number of these older cocaine users are male and unmarried. The profile for these substance abusers seems to be developing into one of predominantly white people with a secondary or higher education. Possible factors involved in the seeking of illicit psychoactive substances by older adults include emotional problems related to loneliness and stressful situations in later life, loss of productivity or a social role, and chronic pain. A high frequency of depression is also observed in these individuals. It is noteworthy that alcohol consumption may be an associated factor.[2] Regarding age, higher rates of cocaine use were reported in adults aged 50 to 64 than in those over 65 years old.[3]

Elderly people frequently experience neurologic, cognitive, and behavioral impairments that can be worsened by cocaine use. Attention and working and visual memory are the cognitive functions most prominently impaired in cocaine users.[4] Andersen et al.[5] reported a decrease in birth of new cells in the hippocampal dentate gyrus in experiments involving acute administration of cocaine in adult rats.

Cocaine is also known to disrupt the sleep pattern of its users severely, affecting sleep maintenance, causing sleep fragmentation, and altering sleep architecture,[6,7] all effects that warrant special attention in the elderly.

It is safe to assume that cocaine use can have an amplifying impact on several health problems associated with advanced age. Illicit drug use is on the rise, while life expectancy has increased. Substance abuse by older adults is an observed and ongoing trend that may represent a social and health concern of considerable proportions in the future. Specifically, we highlight the relevance of qualitative and quantitative evaluations of this trend and its possible implications.

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Disclosure

The authors report no conflicts of interest.

References

1 Fahmy V, Hatch SL, Hotopf M, Stewart R. Prevalences of illicit drug use in people aged 50 years and over from two surveys. Age Ageing. 2012;41:553-6.
2 Yarnell SC. Cocaine abuse in later life: a case series and review of the literature. Prim Care Companion CNS Disord. 2015;17. doi: 10.4088/PCC.14r01727.
3 Wu LT, Blazer DG. Illicit and nonmedical drug use among older adults: a review. J Aging Health. 2011;23:481-504.
4 Jovanovski D, Erb S, Zakzanis KK. Neurocognitive deficits in cocaine users: a quantitative review of the evidence. J Clin Exp Neuropsychol. 2005;27:189-204.
5 Andersen ML, Perry JC, Bignotto M, Perez-Mendes P, Cinini SM, Mello LE, et al. Influence of chronic cocaine treatment and sleep deprivation on sexual behavior and neurogenesis of the male rat. Prog Neuropsychopharmacol Biol Psychiatry. 2007;31:1224-9.
6 Irwin MR, Bjurstrom MF, Olmstead R. Polysomnographic measures of sleep in cocaine dependence and alcohol dependence: implications for age-related loss of slow wave, stage 3 sleep. Addiction. 2016;111:1084-92.
7 Angarita GA, Canavan SV, Forselius E, Bessette A, Morgan PT. Correlates of polysomnographic sleep changes in cocaine dependence: self-administration and clinical outcomes. Drug Alcohol Depend. 2014;143:173-80.

Coronary calcium score as an expression of multisystemic progression of bipolar disorder

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We read with interest the study by Wageck et al.[1] recently published in *Revista Brasileira de Psiquiatria*. In their

analysis, the authors describe a positive association between number of hospitalizations and coronary calcium score (CCS), a well-defined predictor of cardiovascular mortality in the general population, in individuals with bipolar disorder (BD). In the Discussion, the authors hypothesize that the activation of inflammatory response which is associated with mood episodes, especially more severe episodes (i.e., those requiring hospitalization), may be a causal explanation for the premature onset or progression of atherosclerotic pathology, although inflammatory status was not assessed in the study. While the results of this study still need to be replicated, they provide novel insights into the systemic pathophysiology associated with severe and recurrent mood disorders. In addition to being neuroprogressive illnesses,[2] mood disorders are perhaps more comprehensively conceptualized as conditions of multisystemic progression.[3] In accordance with this view, multisystemic progression would be mediated by persistent inflammatory activation, insulin resistance, and oxidative stress.[4,5] Within this framework, cardiovascular disease seems to be a final common endpoint of the alterations and effector systems mentioned earlier.

Alternatively, it could be hypothesized that the marked systemic alterations which lead to a high CCS score are causally associated with a more severe neuroprogression. This comports with a robust body of evidence from both animal studies and clinical trials which provides the basis for repurposing pharmacological agents that primarily target the metabolic system, such as anti-inflammatory or antidiabetic agents (e.g., intranasal insulin, GLP-1 agonists), to reduce symptoms or modify the course of mood disorders. Indeed, our group has proposed the term "metaboptosis" to refer to the myriad changes in energy balance of peripheral origin with central nervous system (CNS) penetration, causing apoptosis and changes in the substrates subserving neuroprogression and a less favorable clinical course in BD.

From a practical point of view, the findings of Wageck et al.[1] also highlight the need to reinforce cardiovascular assessment and care in individuals with mood disorders, especially in long-term and severe presentations of these psychiatric conditions. In addition, they shed light on the need for a holistic approach to individuals with mood disorders, blurring the limits between psychiatry and general medicine to provide evidence-based and integrated care.

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References

1 Wageck AR, Torres FS, Gama CS, Martins DS, Scotton E, Reckziegel R, et al. Cardiovascular risk and bipolar disorder: factors associated with a positive coronary calcium score in patients with bipolar disorder type 1. Rev Bras Psiquiatr. 2018;40:163-8.
2 Passos IC, Mwangi B, Vieta E, Berk M, Kapczinski F. Areas of controversy in neuroprogression in bipolar disorder. Acta Psychiatr Scand. 2016;134:91-103.
3 Czepielewski L, Daruy Filho L, Brietzke E, Grassi-Oliveira R. Bipolar disorder and metabolic syndrome: a systematic review. Rev Bras Psiquiatr. 2013;35:88-93.
4 Rosenblat JD, Brietzke E, Mansur RB, Maruschak NA, Lee Y, McIntyre RS. Inflammation as a neurobiological substrate of cognitive impairment in bipolar disorder: Evidence, pathophysiology and treatment implications. J Affect Disord. 2015;188:149-59.
5 Brietzke E, Kapczinski F, Grassi-Oliveira R, Grande I, Vieta E, McIntyre RS. Insulin dysfunction and allostatic load in bipolar disorder. Expert Rev Neurother. 2011;11:1017-28.