

Review Article

Adolescent cocaine use, immature brain, and health consequences

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ABSTRACT

The use of psychoactive substances is a global public health problem that affects a great number of adolescents and young adults, also causing social, legal and economic consequences. Among the various psychoactive substances used in this age group, cocaine stands out, as it has a high potential for abuse and dependence and compromises the entire organism, mainly causing cognitive, emotional and behavioral changes. Adolescence is a phase of life in which physical, emotional, neurobiological and social changes are observed. It is a period of great plasticity of the brain and development of cognitive capacity, which makes adolescents vulnerable to the use/abuse of psychoactive substances and their harm. The use of cocaine during this period can cause interference in neural circuits, synaptic plasticity and neurotransmitter systems, causing lasting behavioral and cognitive consequences. This article presents the main characteristics and clinical effects of cocaine on the human organism, highlighting its role in the general impairment of health and the serious possible acute and chronic repercussions on the immature and vulnerable nervous system of adolescents.

Keywords: Adolescent, Adolescent health, Cocaine, Crack, Psychoactive drugs

INTRODUCTION

The use of psychoactive substances is a public health problem worldwide that affects a great number of adolescents and young adults. Nowadays, cocaine has occupied an increasingly larger space in this context, contributing to the increase in direct and indirect morbidity and mortality rates associated with its use and with many social, legal and economic consequences.¹⁻³ Cocaine is a substance with a high potential for abuse and dependence, and its use is facilitated by social environments and rituals and living in groups, associated with excessive alcohol consumption.^{4,5} Due to the expansion of production, distribution and the emergence, in the mid-1980s, of an alternative form of consumption (called crack) with lower costs, the number of users worldwide has been increasing.^{2,6}

ADOLESCENCE

Adolescence is a period of life in which major physical, emotional, neurobiological and social changes are

observed, comprising the transition from childhood to adulthood, characterized by the search for new activities and challenges, strong social influence, greater predisposition to risks and modification of relationships with groups of friends and family.^{3,7,8} It is a period in which the brain and cognitive capacity are developing rapidly to meet the new demands specific to this phase, including synaptic pruning, myelination and maturation of white and gray matter.⁹⁻¹¹ In addition, risk behavior increases motivated by emotionally strong situations and the search for experimentation with novelties tends to become a constant need.^{7,12,13}

As the period of brain development in adolescence is marked by great neuroplasticity, adolescents are very vulnerable to external influences, due to the disparity in maturation between the subcortical systems that are already developed and the cortical systems that have not yet undergone the maturation process.¹⁴ Factors such as dysfunctional family relationships, involvement with influential peer groups, personality traits with emotional instability and neuroticism increase the risk of contact with

psychoactive substances.¹⁵ Furthermore, it is important to highlight that some mental problems such as mood swings, depression and anxiety can appear in adolescents who do not use these substances, but which can be exacerbated by cocaine use.^{9,10} Since adolescence is marked by profound brain maturation and greater vulnerability to substance abuse - since the prefrontal cortex, responsible for executive functions such as decision-making, impulse control and risk assessment, is still developing - cocaine consumption becomes dramatically aggressive and has serious consequences.¹⁶ Cocaine use during this period can lead to interference in neural circuits, including changes in synaptic plasticity and neurotransmitter systems, which are associated with behavioral and cognitive consequences that can be long-lasting.¹⁶

COCAINE

Cocaine is a tropane alkaloid, called methyl-3-benzoyloxy-8-methyl-8-azabicyclo (3.2.1) octane-4-carboxylate, derived from the leaves of the *Erythroxylon coca* plant, very common in some South American countries, which was isolated and purified for the first time in 1859 and initially used for medicinal purposes, proving to be a highly addictive substance with an addictive effect.^{1,17} Cocaine is currently found in two forms: a) cocaine hydrochloride (fine, white powder, soluble in water) consumed mainly by the intranasal, oral and intravenous routes; b) free base (resulting from the reaction of cocaine hydrochloride with ammonium or sodium bicarbonate), commonly known as “crack”, and typically consumed by inhalation.^{2,18}

Other ways to consume cocaine include teas, chewing the leaves, and rectally and vaginally.^{2,19} The peak blood concentration ranges from 1 to 5 minutes (smoking or intravenous injection) to 60 to 90 minutes (oral administration). The duration of the effect ranges from 5 to 60 minutes after smoking or intravenous administration and up to 180 minutes after oral administration.¹⁹ Inhalation produces peak stimulation between 1 and 3 minutes after administration, lasting between 5 and 15 minutes. The intranasal route determines a longer effect, ranging from 15 to 30 minutes.^{1,2}

These effects generally last from 30 to 40 minutes after an intravenous dose or smoking and from 30 to 60 minutes after nasal or oral administration.¹⁷ The half-life of cocaine is between 40 and 90 minutes and may vary according to the route of administration.² Cocaine is rapidly absorbed through the oral and nasal mucosa, as well as through the respiratory, gastrointestinal and genitourinary tracts.¹⁷ It binds to albumin and α 1-acid glycoprotein and can be found mainly in the brain, heart, kidneys, spleen, lungs, liver, placenta and adrenal glands. It can be detected and measured in urine, blood, hair, sweat, saliva, meconium and breast milk.²⁰ Because it is unstable in the blood, cocaine is subject to the action of plasma and hepatic cholinesterase enzymes, being rapidly hydrolysed into benzoylecgonine, which also has vasoconstrictive action,

and ecgonine methyl ester.^{2,18} These water-soluble metabolites can be detected for up to 4 days after use and are excreted in the urine.^{17,19,20,21} Excretion rates depend on the amount consumed, and traces of cocaine can remain in the body for weeks and even months in the hair.¹⁸

ACTION AND EFFECTS OF COCAINE ON THE CENTRAL NERVOUS SYSTEM

Cocaine is one of the most powerful stimulants that affect the central nervous system, causing cognitive, behavioral, and emotional symptoms.¹ In moderate doses, it promotes a general feeling of well-being and reduced fatigue, causes euphoria, excitement, improved alertness and concentration, increased libido, and decreased appetite. However, these manifestations are usually accompanied by undesirable effects such as tremors, irritability, insomnia, anxiety, and impulsive behavior.²

The nervous system can undergo molecular, structural, and functional changes.²² Cocaine can compromise the brain through inflammation, neurotoxicity, and oxidative stress, increasing the production of reactive oxygen species and stimulating the release of neurotransmitters such as dopamine, serotonin, and norepinephrine which leads to exaggerated and prolonged activity of the sympathetic nervous system.^{1,2,17, 20-25} Associated with this, there is also inhibition of neurotransmitter reuptake resulting in accumulation in the synaptic cleft.²⁰ Cocaine-induced vasoconstriction can cause damage to the arteries, reducing cerebral blood flow.²⁷ Other mechanisms of pathophysiological impact caused by cocaine consumption may include increased platelet activation, increased production and activation of prostaglandins, increased sympathetic activity and endothelial dysfunction.²⁸

Chronic cocaine use also compromises brain structure. Studies have observed a smaller volume of gray matter in the frontal lobe, temporal lobe and some subcortical regions such as the amygdala and caudate nuclei.²⁴ Other affected regions are the prefrontal cortex, cingulate, insula, striatum and cerebellum.^{1,26,29,30} Emotional manifestations caused by cocaine use include impairment of mood, emotions, cognition, and behavior with deficits in attention, working memory, and visual memory, worsening of executive functions difficulty controlling impulses and desires, panic attacks, suspiciousness, impairment of verbal fluency and judgment, inhibition of responses, and impulsivity.^{20,23,25} Restlessness, euphoria, irritability, agitation, repetitive stereotyped behaviors, anxiety, loss of sleep, depression in addition to the possibility of inducing psychotic symptoms such as paranoia, delusions, tactile or visual hallucinations and schizophrenia have also been reported.^{1,20,23,24}

GENERAL EFFECTS ON THE BODY

Cocaine use can cause a wide variety of effects on different organs and systems, which are directly related to the route of administration, user characteristics and quantity,

occurring even after a single dose.²¹ Cocaine stimulates the sympathetic system, causing vasoconstriction, increased blood pressure, tachycardia and increased metabolic demand, which are risk factors for many diseases.^{19,23} The cardiovascular system can be severely compromised with arrhythmias, heart failure, myocarditis, aneurysms, acute myocardial infarction, constriction and reduction of coronary blood flow and sudden death.^{2,17,19,20,23}

As a potent neurotoxic substance, cocaine can cause ischemia in the central nervous system, headache, tremors, seizures, strokes and intracranial hemorrhages.^{1,2,20,27} Cocaine can also compromise the integrity of the blood-brain barrier, leaving the central nervous system susceptible to toxins, metals and attacks by microorganisms.^{2,27} Cocaine use is associated with hyperthermia, which is clinically relevant because high body temperature can cause disseminated intravascular coagulation and rhabdomyolysis.² Changes in other organs and systems can also be observed, such as ulceration and perforation of the palate and nasal septum, atypical dental caries, gingival lesions, abdominal pain, nausea, reduced motility and delayed gastric emptying, ulcers and colitis.³¹

Other changes include liver toxicity, periportal necrosis, inflammation, impairment of neuroendocrine regulation during pubertal development and changes in the hypothalamic-pituitary-adrenal axis.^{2,9,13,20} Renal failure, renal infarction and vasculitis have also been described.² The respiratory system may be compromised and cause bronchoconstriction, chest pain, pulmonary hemorrhage, pneumothorax and pneumomediastinum.^{2,31}

CONCLUSION

Cocaine use represents one of the major public health challenges that requires, in addition to specific health actions, the involvement of the entire society, as it is responsible for many physical and emotional damages, in addition to compromising family and social relationships, reserving a future without prospects for individuals. Factors such as the glamorization of the use of psychoactive substances, the expansion of markets and availability can generate a feeling of normalization, contributing to the perception of these substances as a status symbol and a normal social component. Social attitudes and policies have also been shown to impact the perception of drug use and influence whether it is seen as acceptable by society.

Despite all the efforts directed at educating about the serious consequences that can be caused by cocaine, its consumption is still high among adolescents. Knowledge of the physical and emotional characteristics that human beings present between childhood and adulthood, including great neuroplasticity, the risks to which they are subjected and the mechanisms of drug use by adolescents can contribute to the development of interventions and prevention and treatment/recovery strategies aimed at

mitigating the adverse effects of cocaine on the adolescent brain.

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REFERENCES

- Castillo SM. Memory, decisions and emotions after the administration of cocaine: role of different neuroanatomical structures. *Therapeia*. 2019;7:71-83.
- Bravo RR, Faria AC, Brito-da-Costa AM, Carmo H, Mladěnka P, Silva DD, et al. Cocaine: an updated overview on chemistry, detection, biokinetics, and pharmacotoxicological aspects including abuse pattern. *Toxins (Basel)*. 2022;14:1-25.
- Casey BJ, Cohen AO, Galvan A. The beautiful adolescent brains: an evolutionary developmental perspective. *Ann NY Acad Sci*. 2025;1546:58-74.
- Frøyland LR, Pedersen W, Enstad F, von Soest T. School party culture as a driver of cocaine use among Norwegian adolescents: a cross-classified multilevel analysis. *Drug Alcohol Dep*. 2025;271:1-7.
- Tokle R, Pedersen W. Cubs of Wall Street: cocaine use in top-boy culture. *British J Sociol*. 2025;3:1-10.
- Robet T, Santos-Cruz J, Zullino D, Semedo NC, Baldo B, Iuga R, et al. Clinical and public health challenges of crack consumption. *Rev Med Suisse*. 2025;21:226-30.
- Shau S, Zhou R. The relationship between drug addiction and adolescent cognitive development. *Adv Soc Sci Educ Human Res*. 2022;638:1-6.
- Galván A. Adolescent brain development and contextual influences: a decade in review. *J Res Adolesc*. 2021;31:843-69.
- Salmanzadeh H, S. Ahmadi-Soleimani M, Pachenari N, Azadi M, Halliwell RF, Rubino T, Azizi H. Adolescent drug exposure: A review of evidence for the development of persistent changes in brain function. *Brain Res Bull*. 2020;156:105-17.
- Ferschmann L, Bos MGN, Herting MM, Mills KL, Tamnes CK. Contextualizing adolescent structural brain development: environmental determinants and mental health outcomes. *Cur Op Psychol*. 2022;44:170-6.
- Green R, Wolf BJ, Chen A, Kirkland AE, Ferguson PL, Browning BD et al. Predictors of substance use initiation by early adolescence. *Am J Psych*. 2024;181:423-33.
- Fuhrmann D, Knoll LJ, Blakemore S. Adolescence as a sensitive period of brain development. *Trends Cogn*. 2015;19:558-66.
- Baker AE, Galvan A, Fuligni AJ. The connecting brain in context: how adolescent plasticity supports learning and development. *Dev Cogn Nuerosci*. 2025;71:1-15.
- Braams BR, Krabbendam L. Adolescent development: from neurobiology to psychopathology. *Cur Op Psychol*. 2022;48:1-4.

15. Mirnics Z, Kovi Z, Tanyi Z, Grezsa F. Adolescent drug use, relational variables and personality factors. *Psychiatr Danub.* 2012;33:656-65.
16. Zeng X. Neuroplasticity and cocaine exposure during adolescence. *Commun Human Res.* 2024;40:211-8.
17. Benowitz NL. Clinical pharmacology and toxicology of cocaine. *Pharmacol Toxicol.* 1993;72:3-12.
18. Karch SB. Substance misuse: cocaine and other stimulants. *Encycl Forensic Legal Med.* 2016;4:363-70.
19. Kim ST, Park T. Acute and chronic effects of cocaine on cardiovascular health. *Int J Mol Sci.* 2019;20:584-605.
20. Ryan SA. Cocaine use in adolescents and young adults. *Pediatr Clin N Am.* 2019;66:1135-47.
21. Wang J, Deng X, Wu Y, Huang Y, Hou S, Zhang Y, Qiu T, Tong J, Chen X. Sub-lethal toxicity and elimination of the cocaine metabolite, benzoylecgonine: a narrative review. *Ann Palliat Med.* 2021;10:6936-47.
22. Steinfeld MR, Torregrossa MM. Consequences of adolescent drug use. *Transl Psychiatr.* 2023;13:1-22.
23. Beheshti I. Cocaine destroys gray matter brain cells and accelerates brain aging. *Biology.* 2023;12:1-14.
24. Yang Z, Biswal BB, Klugah-Brown B, Ding G, Zhou W. Brain structural differences in cocaine use disorder: insights from multivariate and neurotransmitter analyses. *Prog Neuropsychopharmacol Biol Psych.* 2025;136:1-9.
25. Schwartz EKC, Wolkowicz NR, Aquino JP, MacLean RR, Sofuoglu M. Cocaine use disorder (CUD): current clinical perspectives. *Sub Abuse Rehab.* 2022;13:25-46.
26. Wang X. Brain structural consequences of chronic cocaine exposure and their effects on behavior. *Biol Psych.* 2021;89:11-2.
27. Clare K, Park K, Pan Y, Lejuez CW, Volkow ND, Du C. Neurovascular effects of cocaine: relevance to addiction. *Front Pharmacol.* 2024;22:1-17.
28. Farooque U, Okorie N, Kataria S, Bollampally VC, Shah SF. Cocaine-induced headache: a review of pathogenesis, presentation, diagnosis, and management. *Cureus.* 2020;12:1-5.
29. Li K, Gu L, Cai H, Lu H, Mackie K, Guo F. Human brain organoids for understanding substance use disorders. *Drug Metab Pharmacol.* 2025;60:1-13.
30. Poireau M, Segobind S, Maillarda A, Clergue-Duvala V, Icicka R, Azuara J, et al. Brain alterations in Cocaine Use Disorder: does the route of use matter and does it relate to the treatment outcome. *Psych Res: Neuroim.* 2024;342:1-8.
31. Melo CAA, Guimarães HRG, Medeiros RCF, Souza GCA, Santos PBD, Torres ACSP. Oral changes in cocaine abusers: an integrative review. *Br J Otorh.* 2022;88:633-41.

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