CASE REPORT

UDC: 616.24-02::616.89-008.441.3
DOI: https://doi.org/10.2298/VSP240606011V

Crack lung – a case report

Kokainska pluća

Mira Vasiljević*, Sanja Šarac*†, Rade Milić*†, Biljana Lazović-Popović‡

***Military Medical Academy, Clinic for Pulmonology, Belgrade, Serbia; ‡University Clinical Hospital Center "Zemun", Belgrade, Serbia; †University of Defence, Faculty of Medicine of the Military Medical Academy, Belgrade, Serbia**

Abstract

Introduction. Crack cocaine (CC), a potent form of cocaine, is well-known for its rapid and intense effects on the central nervous system. Its detrimental impact on the respiratory system is often disregarded. This type of cocaine originates from cocaine hydrochloride, a compound extracted from the leaves of the coca plant (*Erythroxylum coca*). Through various pathophysiological mechanisms, its pharmacokinetics and interaction with the respiratory system contribute to acute and chronic lung damage. **Case report.** We present a 35-year-old male with progressive dyspnea and chronic lung damage caused by long-term abuse of CC. Morphological changes in the lungs along with abnormalities in pulmonary function tests were also observed. The patient was treated with a combination of medication therapy and enrolled in a detoxification support program. The applied therapeutic measures led to a gradual reduction in symptoms and significant improvement in pulmonary function tests. **Conclusion.** CC-induced lung damage represents a clinical challenge with profound implications for patient health and well-being. Substance abuse counseling, relapse prevention strategies, and social support services are key components of comprehensive treatment to support patient recovery and prevent relapse.

Key words:
cocaine; diagnosis; lung diseases; substance-related disorders; therapeutics.

Apstrakt

Uvod. Krek kokain (KK), potentna forma kokaina, poznata je po svom brzom i intenzivnom uticaju na centralni nervni sistem. Njegov štetan uticaj na respiratorni sistem obično se zanemaruje. Ova vrsta kokaina potiče od kokain-hidrohlorida, jedinjenja koje se ekstrahuje iz listova biljke koke (*Erythroxylum coca*). Različitim patofiziološkim mehanizmima, njegova farmakokinetika i interakcija sa respiratornim sistemom doprinose akutnom i hroničnom oštećenju pluća. **Prikaz bolesnika.** Prikazan je 35-godišnji muškarac sa progresivnom dispnejom i hroničnim oštećenjem pluća izazvanim dugogodišnjom zloupotrebom KK. Uočene su i morfološke promene u plućima, kao i abnormalnosti u testovima plućne funkcije. Bolesnik je lečen kombinovanom medikamentnom terapijom i uključen je u program podrške za odvikavanje od psihoaktivnih supstanci. Primenjene terapijske mere dovele su do postepene redukcije simptoma i značajnog poboljšanja parametara testova plućne funkcije. **Zaključak.** Oštećenje pluća izazvano KK predstavlja klinički izazov sa dubokim posledicama za zdravlje i dobrobit bolesnika. Savetovanje o zloupotrebi supstanci, strategije prevencije recidiva i socijalne usluge podrške su ključne komponente sveobuhvatnog lečenja, kako bi se podržao oporavak bolesnika i sprečio recidiv.

Ključne reči:
kokain; dijagnoza; pluća, bolesti; poremećaji izazvani supstancama; lečenje.

Introduction

Crack cocaine (CC), a potent form of cocaine, is notorious for its rapid and intense effects on the central nervous system. Its detrimental impact on the respiratory system is often overlooked. CC is derived from cocaine hydrochloride (HCl), a compound extracted from the leaves of the coca plant (*Erythroxylum coca*) [1]. Unlike HCl, which is typically snorted or injected, CC is smoked, resulting in rapid absorption into the bloodstream through the lungs [2]. CC exerts its effects primarily by blocking the reuptake of neurotransmitters, such as dopamine, norepinephrine, and serotonin, leading to increased neurotransmitter levels in the brain's synapses [3]. Smoking CC produces a rapid onset of effects, with peak blood concentrations reached within minutes [4]. The short duration of action (approximately

Correspondence to: Mira Vasiljević, Military Medical Academy, Clinic for Pulmonology, Crnotravska 17, 11 000 Belgrade, Serbia.
E-mail: mirasmc@gmail.com

5–10 min) contributes to its high addictive potential and frequent binge use patterns [5]. CC use has been associated with urban, low-income communities, although its use is not limited to any particular demographic group. Furthermore, this form of cocaine is not commonly consumed in our region, i.e., Southeastern Europe [6]. Many CC users also use other substances concurrently, such as alcohol, marijuana, or opioids, which can compound the risks and health consequences. CC's pharmacokinetics and interaction with the respiratory system contribute to acute and chronic pulmonary injury through various mechanisms, including direct toxicity, vasoconstriction, oxidative stress, inflammation, and impaired mucociliary clearance [7, 8].

Case report

A 35-year-old male football coach presented to the emergency department complaining of worsening dyspnea, chronic cough, and episodes of minimal hemoptysis over the past six months. He also reported a history of CC abuse for the past ten years, smoking approximately 10 rocks *per* day. He denied any recent fever, chest pain, or wheezing but admitted to progressive fatigue and exercise intolerance. His symptoms had escalated over the past few months, prompting him to seek medical attention. No significant past medical history was reported. There was no history of asthma, chronic obstructive pulmonary disease (COPD), or other respiratory conditions. The patient was a heavy smoker (about 20 pack-years). He denied alcohol or drug use other than CC. Physical examination revealed mild dyspnea. Vital signs were notable for tachypnea, with a respiratory rate of 24 breaths *per* minute. Lung auscultation revealed decreased bilateral breath sounds with diffuse expiratory wheezing. Cardiac examination was unremarkable, and there were no signs of peripheral edema or cyanosis. Arterial blood pressure was 130/80 mmHg, heart rate was 90 beats *per* minute, and oxygen saturation was 82% on room air, measured with a pulse oximeter (normal range from 95% to 100%). Laboratory tests were within normal limits, including a complete blood count and comprehensive metabolic panel. However, urine toxicology screening was positive for cocaine metabolites.

Chest X-ray demonstrated discrete bilateral interstitial changes (Figure 1).

Multi-detector computed tomography (MDCT) of the chest revealed ground-glass opacities (GGO), interstitial thickening, and mild bronchial wall thickening, suggestive of interstitial lung disease (Figure 2).

Pulmonary function tests were performed. Spirometry showed a mixed ventilation disorder with reduced forced vital capacity (FVC) – 43.8% [normal range (NR): 80–120% predicted], forced expiratory volume in 1 second (FEV1) – 37.4% (NR: 80–120%), FEV1/FVC ratio 62.85% (NR: 70–80%), along with decreased diffusion capacity of the lungs for carbon monoxide (DLCO) – 66.8% (normal above 80%) (Figure 3).

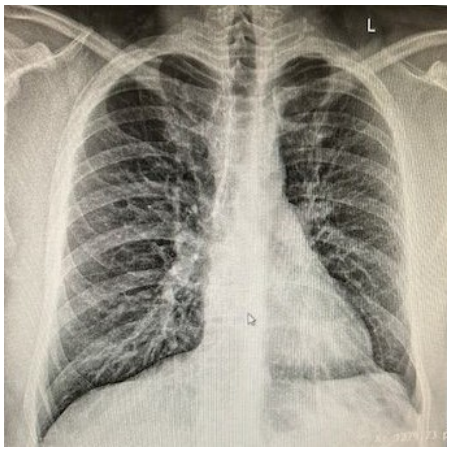
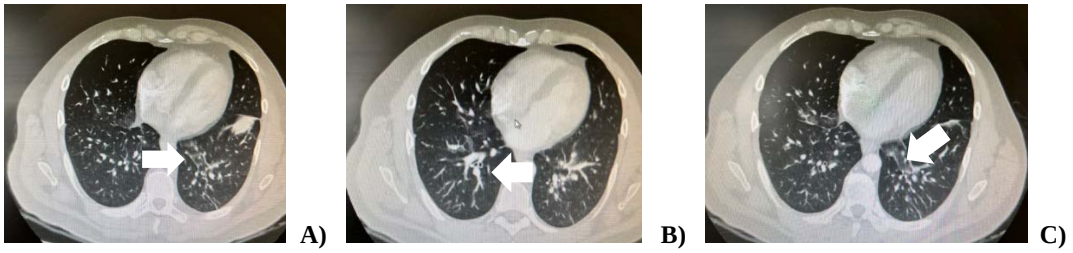


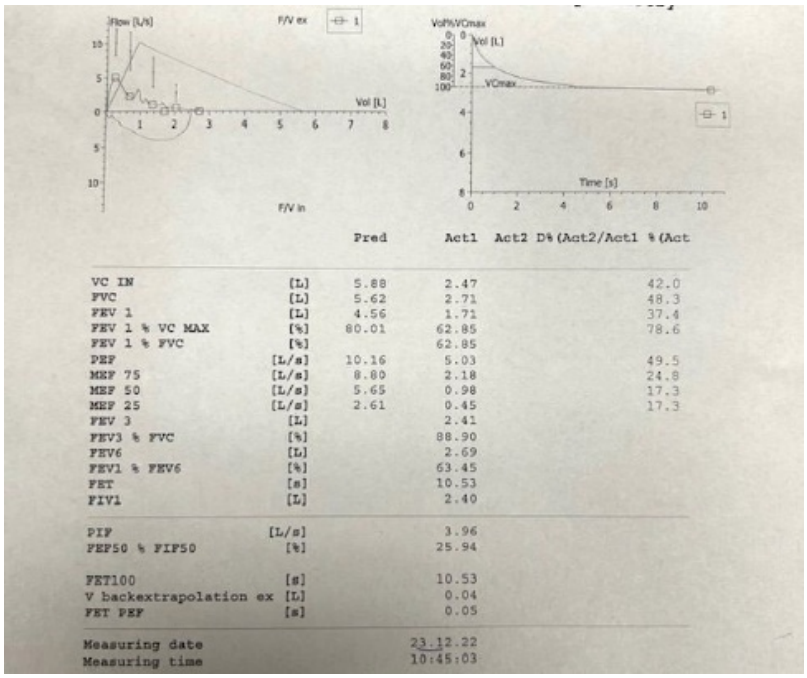
Fig. 1 – The chest radiograph shows bilateral interstitial changes.



A) B) C)

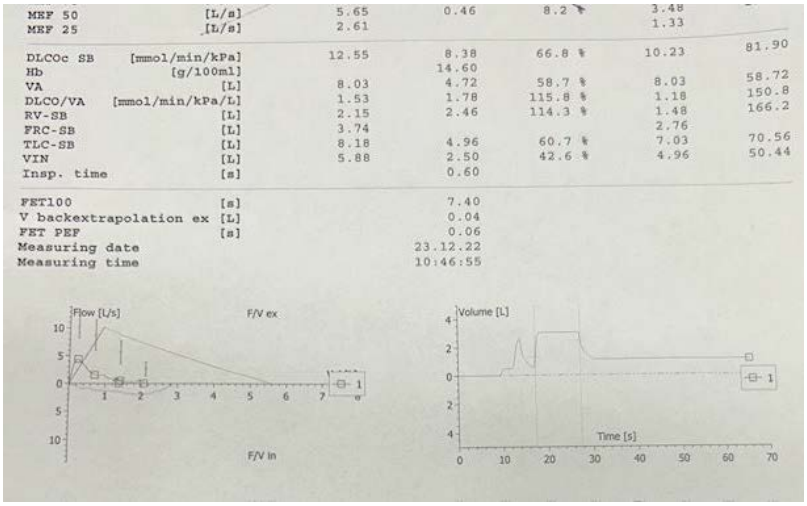
Fig. 2 – Multi-detector computed tomography shows: ground-glass opacities (A), mild bronchial wall thickening (B), and interstitial thickening (C).

A)



		Pred	Act1	Act2	D% (Act2/Act1	% (Act
VC IN	[L]	5.88	2.47			42.0
FVC	[L]	5.62	2.71			48.3
FEV 1	[L]	4.56	1.71			37.4
FEV 1 % VC MAX	[%]	80.01	62.85			78.6
FEV 1 % FVC	[%]		62.85			
PEF	[L/s]	10.16	5.03			49.5
MEF 75	[L/s]	8.80	2.18			24.8
MEF 50	[L/s]	5.65	0.98			17.3
MEF 25	[L/s]	2.61	0.45			17.3
FEV 3	[L]		2.41			
FEV3 % FVC	[%]		88.90			
FEV6	[L]		2.69			
FEV1 % FEV6	[%]		63.45			
FET	[s]		10.53			
FIV1	[L]		2.40			
PIF	[L/s]		3.96			
FEF50 % FIF50	[%]		25.94			
FET100	[s]		10.53			
V backextrapolation ex	[L]		0.04			
FET PEF	[s]		0.05			
Measuring date			23.12.22			
Measuring time			10:45:03			

B)



MEF 50	[L/s]	5.65	0.46	8.2 %	3.48	
MEF 25	[L/s]	2.61			1.33	
DLCOc SB	[mmol/min/kPa]	12.55	8.38	66.8 %	10.23	81.90
Hb	[g/100ml]		14.60			
VA	[L]	8.03	4.72	58.7 %	8.03	58.72
DLCO/VA	[mmol/min/kPa/L]	1.53	1.78	115.8 %	1.18	150.8
RV-SB	[L]	2.15	2.46	114.3 %	1.48	166.2
FRC-SB	[L]	3.74			2.76	
TLC-SB	[L]	8.18	4.96	60.7 %	7.03	70.56
VIN	[L]	5.88	2.50	42.6 %	4.96	50.44
Insp. time	[s]		0.60			
FET100	[s]		7.40			
V backextrapolation ex	[L]		0.04			
FET PEF	[s]		0.06			
Measuring date			23.12.22			
Measuring time			10:46:55			

Fig. 3 – A) Forced spirometry: a mixed ventilatory disorder with reduced forced vital capacity (FVC), forced expiratory volume in 1 second (FEV1), and FEV1/FVC ratio; B) diffusing capacity of the lungs for carbon monoxide (DLCO): decreased DLCO – mild degree.

Arterial blood gas analysis revealed hypoxemia with a partial pressure of oxygen (PaO2) of 45 mmHg (NR: 75–100 mmHg) with normal partial pressure of carbon-monoxide (PaCO2) of 38 mmHg (NR: 35–45 mmHg) and respiratory alkalosis with a pH of 7.48 (NR: 7.35–7.45).

The patient was admitted for hospital treatment for further evaluation and treatment. He was started on supplemental oxygen therapy to maintain oxygen saturation above 90%. Bronchodilators (short-acting beta-agonists and anticholinergics) and inhaled corticosteroids were initiated to reduce bronchospasm and inflammation. Sputum culture was negative for bacterial and fungal pathogens. Laboratory tests for viruses, such as human immunodeficiency virus, Epstein-Barr virus, hepatitis A and hepatitis B, and cytomegalovirus, were negative.

The patient received smoking cessation counseling, and nicotine replacement therapy was offered to assist with cessation. He was referred to addiction support services for comprehensive substance abuse (SA) treatment, including behavioral therapy and pharmacotherapy.

During hospitalization, the patient showed gradual improvement of symptoms with bronchodilators, corticosteroids, and supplemental oxygen therapy. However, he remained tachypneic with persistent wheezing on auscultation. Arterial blood gases and oxygen saturation were normalized. After two weeks of hospitalization, the patient was discharged from the hospital and referred to outpatient counseling and a program for SA rehabilitation.

Due to the history of smoking and suspicion of COPD, dual bronchodilator therapy (tiotropium/olodaterol) was included.

Despite initial improvement, long-term follow-up revealed persistent respiratory symptoms, emphasizing the chronic nature of CC-induced lung injury. The patient's respiratory symptoms gradually improved with treatment, which included oral corticosteroid (prednisone) in tapering dose and inhalation bronchodilator, although he continued to experience dyspnea during physical activities. He has not reported hemoptysis since discharge from the hospital. Furthermore, he remained abstinent from CC and engaged in ongoing SA counseling. Pulmonary rehabilitation was initiated to improve his exercise tolerance and lung function.

Six months after he was discharged from the hospital, follow-up spirometry showed significant improvement – mild obstructive ventilation disorder (Figure 4).

There was no significant improvement in DLCO. The patient discontinued inhalation bronchodilator therapy after three months because he concluded that he no longer needed it.

Nine months after treatment and discontinuation of CC use, the patient reported a much-improved condition and tolerance to physical exertion. A follow-up MDCT of the chest was performed, which showed mild bronchial wall and interstitial thickening without GGO (Figure 5).

Long-term follow-up was planned due to the possibility of disease progression and to provide ongoing support for his SA recovery.

Discussion

Chronic CC abuse can lead to severe and often irreversible pulmonary damage, as exemplified in the presented case. CC-induced lung injury encompasses a complex array of pathological changes, including pulmonary edema, interstitial fibrosis, bronchiolitis obliterans, and alveolar hemorrhage [9]. The inhalation of CC exposes the lungs to various toxic compounds, resulting in acute and chronic pulmonary injury. Several mechanisms contribute to that damage. Direct

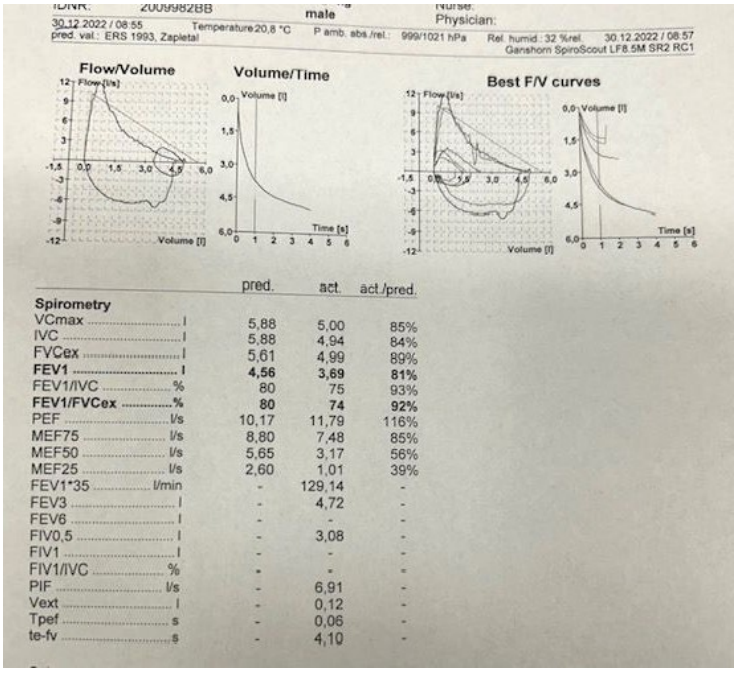


Fig. 4 – Forced spirometry after six months of discharge from hospital treatment – the maximal expiratory flow (MEF) was significantly reduced at 50% and 25% of the forced vital capacity – MEF 50 and MEF 25, respectively.

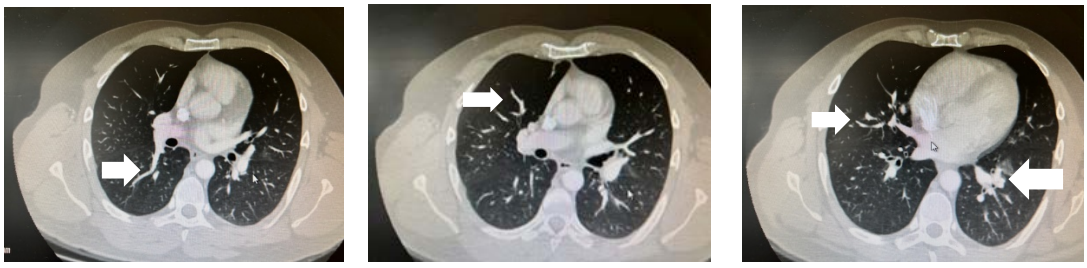


Fig. 5 – A follow-up multidetector computed tomography of the chest was performed after nine months of treatment and discontinuation of crack cocaine use – bronchial wall and interstitial thickening.

harmful effects are caused by a mixture of toxic substances, including particulate matter, volatile compounds, and pyrolysis byproducts [10]. These substances irritate the respiratory epithelium, leading to inflammation, bronchospasm, and tissue injury. The mentioned changes were present in the given patient. Vasoconstriction is also an effect of CC, achieved through its impacts on the sympathetic nervous system and endothelin release [3]. This can lead to pulmonary hypertension, increasing the workload on the right heart ventricle and predisposing to other cardiovascular complications. The metabolism of CC generates reactive oxygen species, leading to oxidative stress and cellular damage. Oxidative stress can impair pulmonary function, exacerbate inflammation, and contribute to the development of respiratory diseases. Chronic exposure to CC smoke triggers an inflammatory response in the lungs, characterized by the recruitment of inflammatory cells, the release of cytokines, and tissue remodeling [11]. That chronic inflammation can lead to airway remodeling, pulmonary fibrosis, and impaired lung function over time. CC smoke inhibits mucociliary clearance, impairing the lung's ability to remove inhaled particles and pathogens. This can increase the risk of respiratory infections and exacerbate existing lung diseases.

A multidisciplinary approach is essential in managing comprehensively CC-induced lung injury. Respiratory specialists, addiction counselors, social workers, and primary care providers collaborate to address the complex medical, psychological, and social aspects of the patient's condition. Pulmonary rehabilitation programs play a role in optimizing lung function, improving exercise tolerance, and enhancing the overall quality of life for individuals with chronic lung diseases, including those related to SA [12, 13].

The case underscores the importance of recognition and intervention in managing CC-induced pulmonary damage. Prompt diagnosis through thorough clinical evaluation, radiographic imaging, and pulmonary function testing enables healthcare providers to initiate appropriate treatment strategies tailored to the patient's needs.

Through early recognition and ongoing support, healthcare providers can diminish the adverse effects of CC abuse on the respiratory system and improve outcomes for affected individuals. However, addressing the broader societal factors contributing to SA remains essential in addressing the root causes of this public health issue.

Ongoing SA counseling, relapse prevention strategies, and social support services are essential components of comprehensive care to support the patient's recovery and prevent relapse [14].

Conclusion

Although the emphasis is on damage to the central nervous system, crack cocaine-induced pulmonary damage represents a significant clinical challenge with profound implications for patient health.

Conflicts of interest

The authors declare no conflict of interest.

REFERENCES

1. *Redman M.* Cocaine: What is the Crack? A Brief History of the Use of Cocaine as an Anesthetic. Anesth Pain Med 2011; (2): 95–7.
2. *Butler AJ, Rehm J, Fischer B.* Health outcomes associated with crack-cocaine use: Systematic review and meta-analyses. Drug Alcohol Depend 2017; 180: 401–16.
3. *Roque Bravo R, Faria AC, Brito-da-Costa AM, Carmo H, Mladěnka P, Dias da Silva D,* et al. On Behalf of The Oemonom Researchers. Cocaine: An Updated Overview on Chemistry, Detection, Biokinetics, and Pharmacotoxicological Aspects including Abuse Pattern. Toxins (Basel) 2022; 14(4): 278.
4. *Dinis-Oliveira RJ.* Metabolomics of cocaine: Implications in toxicity. Toxicol Mech Methods 2015; 25(6): 494–500.
5. *Docherty JR, Alsufyani HA.* Pharmacology of Drugs Used as Stimulants. J Clin Pharmacol 2021; 61 Suppl 2: S53–S69.
6. *EMCDDA.* European Drug Report 2021: Trends and Developments. Luxembourg: Publications Office of the European Union; 2021. p. 60.
7. *Drent M, Wijnen P, Bast A.* Interstitial lung damage due to cocaine abuse: pathogenesis, pharmacogenomics and therapy. Curr Med Chem 2012; 19(33): 5607–11.
8. *Underner M, Peiffer G, Perriot J, Jaafari N.* Pulmonary complications in cocaine users. Rev Mal Respir 2020; 37(1): 45–59. (French)
9. *Dolapsakis C, Katsandri A.* Crack lung: A case of acute pulmonary cocaine toxicity. Lung India 2019; 36(4): 370–1.
10. *Wireko F, Leys L, Donaldson S, Thomas A.* Crack lung with crack cocaine. Chest 2021; 160(4): A1725.
11. *Lison D, Arietti F, Segre E, Rossi S.* Crack-use-associated pneumonia: a case report. Ital J Emerg Med 2023; 12(1): 55–8.
12. *Fischer B, Blanken P, Da Silveira D, Gallassi A, Goldner EM, Rehm J,* et al. Effectiveness of secondary prevention and treatment interventions for crack-cocaine abuse: a comprehensive narrative overview of English-language studies. Int J Drug Policy 2015; 26(4): 352–63.
13. *Minozzi S, Saulle R, Amato L, Traccis F, Agabio R.* Psychosocial interventions for stimulant use disorder. Cochrane Database Syst Rev 2024; 2(2): CD011866.
14. *Junior MSC, Bezerra AG, Curado DF, Gregório RP, Galduróz JCF.* Preliminary investigation of the administration of biperiden to reduce relapses in individuals with cocaine/crack user disorder: A randomized controlled clinical trial. Pharmacol Biochem Behav 2024; 237: 173725.

Received on June 6, 2024
Revised on November 24, 2024
Accepted on December 11, 2024
Online First March 2025