# Neurological Complications Due to the Use of Illicit Drugs: A Mini-Review

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# Abstract

In this paper, it is approached the neurological complications caused by the use of illicit drugs, such as LSD, Crack, Cocaine and Marijuana. These illicit drugs are used recreationally, but they are closely related to signals and symptoms many times unexplained. Many of these reactions involve the neurological system, causing common and known complications, which are present in the use of all the drugs discussed here, like visual illusions, paranoia, psychosis, hallucination, anxiety and euphoria, for instance. However, more uncommon and serious complications need special treatment for quickly actions or patient instructions, such as seizures, strokes, encephalopathies, cognitive deficits, worsening of verbal fluency and attention. In the specific case of the LSD use, the patient may present flashbacks even after the end of the drug effect.

Keywords: Cocaine; Crack; LSD; Cannabis; Neurological Disorders

# Abbreviation

LSD: Lysergic Acid Diethylamide

# Introduction

The use of illicit drugs has been increasing. However, the consumption is closely linked to physical and psychic reactions [1,2] which may trigger complications of different categories, especially neurological. Due to that, doctors should be alert to neurological conditions their patients may present and be able to clinically reason in order to link the patient's clinic and an illicit drug use in order to establish a better therapy [1]. Among the most commonly used illicit drugs, which may have complications commonly encountered in the medical routine, are: Cannabis, Cocaine, Crack and Lysergic Acid Diethylamide (L.S.D). Thus, the present paper is going to study the most frequent complications related to the consumption of these substances.

# **Materials and Methods**

Papers were searched using the following keywords "illicit drugs", "cocaine", "LSD", "marijuana" and the databases Cochrane Library, Science direct, MEDLINE, Embase, and CINAHL databases, and Science Direct. By result, papers in Portuguese and English from 1991 to 2017 were used. Some papers were not considered because the complete text wasn't available.

## **Results and Discussion**

## Cannabis

The use of cannabis is associated with social, intellectual and coordination injuries; besides the possible development of addiction. According to epidemiological studies, about 9% of adult marijuana users will develop an addiction [3,4], with an increase to 17% when the use starts in adolescence [3,5]. In this case, a maturing adolescent brain appears to be more vulnerable to long-term neurologic complications due to marijuana use [3,6]. The acute effect of the first cannabis usage typically involves euphoria and relaxation, but it may also be seen occasionally dysphoria, anxiety, and hallucinations [3,7]. Meanwhile, the chronic usage is associated with memory loss and cognitive deficits [3,6,8,9]. Verbal memory difficulties are the most widely reported and persistent cognitive deficit associated with early marijuana use [10].

Starting the drug use in adolescence and the higher consumption of cannabis increase the risk of psychosis development [3,11] and also anticipate the onset of this psychiatric disorder. Shakoor., et al. [12], using a genetically informative study twin design, demonstrated that environmental risk factors contribute to the association between marijuana use and psychotic experiences, such as paranoia, hallucinations, cognitive disorganization, grandiosity, anhedonia and parent-rated negative symptoms. However, genetic contributions didn't appear to influence the association between the drug and psychotic experiences in teenagers, in comparison to adults where it is more heritable [12]. It is also known the correlation of cannabis use disorder with alterations in the hippocampal morphology, along with impaired episodic memory performance (the type of memory associated with biographical events) [13].

# Cocaine

Cocaine is a psychostimulant drug extracted from the Erythroxylum coca plant [2]. The pleasant subjective effects that characterize cocaine include euphoria, mood elevation, enhanced feelings of well-being, and mental stimulation. With escalating doses and patterns of repeated use, the nature of the stimulant experience changes with these effects transitioning to negative or aversive effects for some users [14]. It is used in two ways: cocaine hydrochloride and alkaloidal cocaine (crack):

#### **Cocaine hydrochloride**

Cocaine hydrochloride was initially developed as a local anesthetic for surgical interventions in the eyes, ears and throat. This drug when commercialized is mixed with several non-psychoactive substances, among them one may list amide, talcum powder and flour. Some common administration methods, when used recreationally, are intranasal and intravenous and it could cause, due to its addictive properties, brain circuits and functioning alteration [15,16]. By binding to dopamine, cocaine prevents this neurotransmitter, responsible for feeling pleasure, to be recaptured by neurons. Therefore, it would stay a longer time in the synaptic cleft, resulting in the characteristic sensation of euphoria [17].

The neurological complications resulted from the use of this substance are related to blood pressure increase and vascular lesions. There is a vasoconstrictive activity (including cerebral), it promotes arrhythmia and an increased heart rate, hypertensive peak and coronary contraction [18-21]. Thus, patients presenting aneurism have a higher rate of preexisting lesion rupture. In case of ischemic lesion, it could increase the occurrence of vascular inflammatory development, similar to atherosclerosis, in chronic young addicts. Other disorders, like psychosis and paranoia, could be presented, besides the euphoria [19]. The following could also be recurrent: dystonia, dysarthria, dyskinesia, shaking, rigidity and postural instability [19,20]. It's possible the occurrence of encephalopathies due to brain atrophy, consequence of ischemia resulting from continuous use [18]. The drug abstinence leads to fatigue, depression and increased sense of hunger [17].

#### Alkaloidal cocaine (crack)

Alkaloidal cocaine, also called crack cocaine, arose in 1983 from the reuse of cocaine hydrochloride refining waste [19]. It is shaped like a stone which is smoked. As it is a different form of presentation, when used has effects similar to cocaine hydrochloride's, however,

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it deserves to be highlighted: laboratory tests showed the presence of foreign substances in crack (sodium bicarbonate, lidocaine, aspirin, sugars, benzocaine and procaine), classifying it as an impure drug [21], so it is possible to have unexpected effects; pharmacological studies indicate the absorption of crack in the pulmonary circulation occurs much faster than cocaine hydrochloride, so its initial effects are much faster and more intense [22]. The neurological complications and neuropsychological alterations of crack are similar to cocaine hydrochloride except for the occurrence of strokes, with similar frequency for both ischemic and hemorrhagic [17,19,20,22,23].

## Lysergic Acid Diethylamide

Lysergic Acid Diethylamide (LSD) is classified as a Serotonergic hallucinogen [24]. It is used as a recreational drug due to its effects on neurological perception and mood, which usually cause visual illusions, distortions in sensory input and synesthesia, which can last for 12 hours [1,25]. However, for some users, the somatic, psychological and perceptual effects of LSD may be experienced as repulsive sensations, causing paranoia, panic, agitation or dysphoria. These experiences are called "bad trips" [1,2]. There are also some users who experience the hallucinogen persisting perception disorder, popularly known as "flashbacks". This phenomenon consists of spontaneous recurrence of sensory, cognitive, or emotional symptoms of the hallucinogenic experience days to years after the ingestion of LSD [1,2,26]. Another long-term effect is the hallucinogen induced persistent psychosis, which consists of mental effects that persist longer than one month after LSD use. These effects are usually characterized by an important affective component, in a way mood fluctuates from mania to depression, religious thought contents, vivid visual disturbances and hallucinations [25,26].

LSD is also associated with other less common neurological complications. Some of them are: 1) Cerebral ischemia, as a consequence of vasospasm effect, which is a property of ergot agents [1,2,27]; 2) Permanent visual disturbances [1,28]; 3) Obtundation [2]; 4) Sero-toninergic syndrome, when combined with other serotonergic drugs [1]. LSD isnotassociated withanywithdrawalsymptoms [2].

#### Conclusion

The nervous system is susceptible to a considerable list of affections which range from hyper-acute to chronic. Drugs trigger direct effects in this system. In the acute context, it may be highlighted sensory and psychological changes, being some drugs as depressant (e.g. cannabis) and others as stimulating (e.g. cocaine). Analyzing the chronic effects, it may be highlighted permanent visual disorders caused by LSD and vascular malformations due to crack continuous use. It could also be emphasized all drugs listed, in this paper have neuropsychiatric impacts once it creates vice circuit which involves the reward system (stimulating the ventral tegumental area and accumbens nucleus). It's fundamental to analyze the neurological effects from illicit drugs and it's necessary to continue the research to update e detail those effects.

#### **Conflict of Interest**

The authors declare no conflict of interest.

# **Bibliography**

- Josephson SA. "Neurologic Complications of Recreational Drugs [Internet]". Aminoff's Neurology and General Medicine: Fifth Edition. Elsevier Inc (2014): 725-735.
- 2. Brust JCM. "Neurologic complications of illicit drug abuse". Continuum: Lifelong Learning in Neurology 20.3 (2014): 642-656.
- 3. Schrot RJ and Hubbard JR. "Cannabinoids: Medical implications". Annals of Medicine 48.3 (2016): 128-141.
- 4. Anthony J., *et al.* "Comparative epidemiology of dependence on tobacco, alcohol, controlled substances and inhalants: basic findings from the national comorbidity survey". *Experimental and Clinical Psychopharmacology* 2.3 (1994): 244-268.
- 5. "Marijuana: Facts for Teens". National Institute on Drug Abuse (2015).

*Citation:* Marco Orsini., *et al.* "Neurological Complications Due to the Use of Illicit Drugs: A Mini-Review". *EC Neurology* 8.6 (2017): 190-194.

- 6. Meier M., *et al.* "Persistent cannabis users show neuropsychological decline from childhood to midlife". *Proceedings of the National Academy of Sciences of the United States of America* 109.40 (2012): E2657-E2664.
- 7. Hubbard J. "Adverse effects of marijuana". Chapter 24. In: OnaiviEs, ed. The biology of marijuana: from gene to behavior, New York: Taylor and Francis Publisher (2002): 621-631.
- 8. Solowij N., *et al.* "Verbal learning and memory in adolescent cannabis users, alcohol users, and non-users". *Psychopharmacology* 216.1 (2011): 131-144.
- 9. Smith M., *et al.* "Cannabis-related working memory deficits and associated subcortical morphological differences in healthy individuals and schizophrenic subjects". *Schizophrenia Bulletin* 40.2 (2014): 287-299.
- 10. Schuster RM., et al. "Early Onset Marijuana Use Is Associated With Learning Inefficiencies". Neuropsychology 30.4 (2016): 405-415.
- Caspi A., *et al.* "Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-O-methyltransferase gene: longitudinal evidence of a gene X environment interaction". *Biological Psychiatry* 57.10 (2005): 1117-1127.
- 12. Shakoora S., *et al.* "Psychotic experiences are linked to cannabis use in adolescents in the community because of common underlying environmental risk factors". *Psychiatry Research* 227.2-3 (2015): 144-151.
- 13. Smith MJ., *et al.* "Cannabis-Related Episodic Memory Deficits and Hippocampal Morphological Differences in Healthy Individuals and Schizophrenia". *Hippocampus* 25.9 (2015): 1042-1051.
- 14. Howell LL and Cunningham KA. "Serotonin 5-HT2 Receptor Interactions with Dopamine Function: Implications for Therapeutics in Cocaine Use Disorder". *Pharmacological Reviews* 67.1 (2015): 176-197.
- 15. Calatyud J and González A. "História do desenvolvimento e evolução da anestesia local desde a folha de coca". *Anestesiologia* 98.6 (2003): 1503-1508.
- 16. Goldstein RA., et al. "Cocaína: história, implicações sociais e toxicidade uma revisão". Disease-a-Month 55.1 (2009): 6-38.
- 17. Baik JH. "Dopamine signaling in reward-related behaviors". Frontiers in Neural Circuits 7 (2013): 152.
- 18. Volpe FM., et al. "Vasculite cerebral e uso de cocaína e crack". Revista Brasileira de Psiquiatria 21.3 (1999): 174-176.
- 19. Levine SR., *et al.* "Cerebrovascular Complications of the Use of the Crack Form of Alkaloidal Cocaine". New England Journal of Medicine 323.11 (1990): 699-704.
- 20. Neiman J., *et al.* "Neurological complications of drug abuse: pathophysiological mechanisms". *European Journal of Neurology* 7.6 (2000): 595-606.
- 21. Merigian KS., *et al.* "Adrenergic crisis from crack cocaine ingestion: report of five cases". *Journal of Emergency Medicine* 12.4 (1999): 485-940.
- 22. Levine SR., *et al.* "A comparative study of the cerebrovascular complications of cocaine: alkaloidal versus hydrochloride--a review". *Neurology* 41.8 (1991): 1173-1177.

*Citation:* Marco Orsini., *et al.* "Neurological Complications Due to the Use of Illicit Drugs: A Mini-Review". *EC Neurology* 8.6 (2017): 190-194.

- 23. Cunha PJ., *et al.* "Alterações neuropsicológicas em dependentes de cocaína/crack internados: dados preliminaries". *Revista Brasileira de Psiquiatria* 26.2 (2004): 103-106.
- 24. Halberstadt AL. "Recent advances in the neuropsychopharmacology of serotonergic hallucinogens". *Behavioural Brain Research* 277 (2015): 99-120.
- 25. Escobar JAC and Roazzi A. "Panorama Contemporâneo do Uso Terapêutico de Substâncias Psicodélicas: Ayahuasca e Psilocibina". *Neurobiologia* 73.3 (2010): 159-172.
- 26. Ede Frecska. "Therapeutic guidelines: dangers and contraindications in therapeutic applications of halluciongens". *Psychedelic Medicine: Social, Clinical, and Legal Perspectives* 4 (2006): 69-95.
- 27. Lieberman AN., et al. "Carotid artery occlusion following ingestion of LSD". Stroke 5.2 (1974): 213-215.
- 28. Aki Kawasaki MD and Valerie Purvin M. "Persistent Palinopsia Following Ingestion of Lysergic Acid Diethylamide (LSD)". Archives of Ophthalmology 114.1 (1996): 47-50.

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