AUDITORY THRESHOLDS, OTOACOUSTIC EMISSIONS AND MEDIAL OLIVOCOCHLEAR SYSTEM OF EX-DRUG USERS

Limiares auditivos tonais, emissões otoacústicas e sistema olivococlear medial de ex-usuários de drogas

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ABSTRACT

Purpose: to analyze whether the use of illicit drugs may interfere with the peripheral and central auditory system. **Methods:** 17 subjects were divided according to the kind of consumed drug: 10 individuals in the cannabis group (G1) and seven in the group of crack/cocaine (G2). The groups were subdivided according to the time of drug use: five, six to 10 and more than 15 years. They were evaluated by anamneses, pure tone audiometry, tympanometry, transient evoked otoacoustic emissions (TEOAE) and TEOAE suppression effect. **Results:** comparing the pure tones of G1 and G2, the worst results were observed in the G2, with a statistically significant difference in the group of one to five years at 250, 500, 6000 and 8000 Hz in the right ear and six to 10 years of about 4000 and 8000 Hz in the left ear. For the users of for more than 15 years, there are pure tones above 25 dBHL from 3000 to 8000 Hz in the right ear. In TEOAE and TEOAE suppression effect, any statistically significant difference was found between G1 and G2 and between the time of drug use. The suppressive effect of TEOAE was present in 79% of the tested ears. **Conclusion:** the use of crack/cocaine has more deleterious effect in the auditory system if compared to marijuana. The time of use of the drug only influenced the results of the G1. The use of illicit drugs did not cause disorders in the medial olivocochlear system.

KEYWORDS: Hearing; Otoacoustic Emissions, Spontaneous; Street Drugs; Cannabis; Cocaine; Crack Cocaine

INTRODUCTION

Marijuana (*Cannabis sativa*) has as main component the hallucinogenic tetrahydrocannabinol (THC) and its effects on the body depend on the amount of THC present in the leaf[1]. The action of marijuana on the human body is mainly related to the central nervous system, causing, then, alterations in memory, learning, attention, processing speed and executive functions[2-4].

Crack and cocaine (*Erythroxylum coca*) are chemically identical, but they have different ways of preparing[5]. While cocaine is an alkaloid in the form of water-soluble salt, the crack is prepared by dissolving cocaine hydrochloride in water mixed with sodium bicarbonate. The hallucinogenic effects of crack are quick but they last less when compared to cocaine, which results in more frequent use and in drug dependence[6]. The clinical manifestations resulting from the use of these drugs include cardiac, pulmonary, psychiatric, gastrointestinal and endocrine alterations[7].

The effects of illicit drugs on hearing have been described in case reports of sudden hearing loss after overdose of cocaine and heroin. It is speculated that such hearing disorders have been caused by any of the following pathophysiologic mechanisms: cochlear hemorrhage, systemic toxemia, autoimmune reaction, cochlear hypoxia by vasoconstriction or temporary blockage of potassium

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channels in the outer hair cells (OHC)[8-10]. In studies with guinea pigs, repeated cocaine injections caused decreased blood flow to the cochlea and consequently this caused the injury in the referred structure[11].

Despite the existing theories on the drug pathophysiology in the peripheral auditory system, the effects regarding the use of marijuana, crack and cocaine at the hearing in a long term are not clear.

Pure tone audiometry (PTA) is still the standard method for monitoring the hearing. However, it does not consistently assess the place which is more vulnerable to injuries, the base of the cochlea. Transient otoacoustic emissions (TOAE) are more sensitive to early cochlear damage, because they detect alterations in auditory function occurs before significant alteration in auditory thresholds[12]. On hearing loss of a cochlear origin, the OHC are the first to suffer injuries. As a consequence, the absence of OAE in ears with normal hearing suggests alteration in cochlear amplifier[13].

The medial olivocochlear system (MOCS), responsible for the modulation of OHC, can be evaluated with the presentation of competitive sound stimulus in the contralateral ear during OAE acquisition. The integrity of the MOCS is related to the ability to detect signal in the noise, the thinning of the sequence selectivity, protection against acoustic overstimulation and focusing of attention to an acoustic phenomenon[14].

The aim of this study was to analyze if the use of marijuana, cocaine and crack can have effects on the peripheral and central auditory systems.

METHODS

This study was reviewed and approved at *Universidade Federal de Santa Maria* Research Ethics Committee under the number 23081.019003/2010-40.

This is a cross-sectional, a descriptive, and a non-experimental quantitative study. This work was carried out in the audiology clinic of *Hospital Universitario de Santa Maria* during the period from April to July 2011.

The sample was composed of subjects who attend the Centers for Psychosocial Care (CAPS) *"Caminhos do Sol"* and *"Cia do Recomeço"* and support groups for ex-users of alcohol and/or other drugs such as *"Amor Exigente"* from Santa Maria/RS.

32 subjects of both genders agreed to participate of the research. However, 18 males, aged between 15 and 35 years attended to the evaluation. Involved subjects, parents and/or guardians read and signed the informed consent.

Only subjects who did not present any causal factor of hearing loss, such as an occupational noise exposure, family history of hearing loss and the use of ototoxic medication were included in the sample. Besides, subjects aged over 35 years, with the presence of air-bone gap in PTA and who did not present tympanometric curve type A were also excluded from the sample. This way, a subject was excluded from the sample once he presented tympanogram B-type in both ears.

So, the sample consisted of 17 subjects, divided into two groups, according to the most consumed type of drug: 10 subjects in the marijuana group (G1) and seven subjects in the crack/cocaine group (G2).

G1 and G2 groups were divided according to the time of drug use: one to five years, six to 10 years, 11 to 15 years and greater than 15 years.

It is important to mention that four members of the group eventually consumed marijuana and crack and cocaine, but in less quantity and in less time. Users of crack and cocaine were grouped due to the chemical similarity of the substances.

Anamnesis, meatoscopy, pure tone audiometry (PTA), acoustic impedance measurements (AIM) and transient otoacoustic emissions (TOAE) with and without competing noise were performed.

The anamnesis searched for information regarding hearing complaints, otologic history and chemical dependency (Figure 1). Meatoscopy tried to discard alterations in external and middle ear.

The PTA was carried out with the audiometer *Sibelmed* brand, model AC50 - D. Hearing thresholds of air at frequencies of 250, 500, 1000, 2000, 3000, 4000, 6000 and 8000 Hz and bone conduction at frequencies of 500, 1000, 2000, 3000 and 4000 Hz, through the descending–ascending method were researched. The level of hearing loss was classified according to Lloyd and Kaplan (1978)[15].

The AIM were researched due to the criterion that excludes from the sample subjects with alterations of medium and/or external ear, but they were not analyzed during this work. The AIM were carried out with middle ear analyzer brand *Interacoustics* model AZ6. Tympanometry curves were classified as type A, B, C, As and Ad[16].

TOAE were surveyed through the cochlear analyzer from *Intelligent Hearing Systems* (IHS). TOAE were considered present when signal/noise relation (S/N) is equal to or greater than 6 dB, the overall reliability was less than 50% and the stability was less than 70%. The stimulus was not of linear type, at the intensity of 80 dB SPL (decibel sound pressure level).

Name_____
Age_____ B.D.___/____/_____ Gender ( )F ( ) M
Date___/____/____

1. AUDIOLOGICAL ANAMNESIS

1.0 Do you have difficulty in hearing? ( ) No ( ) Yes ( ) RE ( ) LE ( ) BE
1.1 Do you present tinnitus? ( ) No ( ) Yes ( ) RE ( ) LE ( ) BE
1.2 Do you present dizziness? ( ) No ( ) Yes
1.3 Do you have difficulty in understanding speech in noisy environments? ( ) No ( ) Yes
1.4 Have you ever presented episodes of otitis? ( ) No ( ) Yes
1.5 Have you ever made use of ototoxic medication? ( ) ( ) No ( ) Yes
1.6 Have you worked or still work with activities in environments with loud noise? ( ) No ( ) Yes
1.7 Is there a family history of hearing loss? ( ) No ( ) Yes
1.8 Do you have a health problem( ) No ( ) Yes Which one? _____
1.9 Do you take a regular medication? ( ) No ( ) Yes Which one? _____

2. HISTORY OF DRUG USE:

2.0 Which illicit drugs have you ever used? _____
2.1 How long have you used? _____
2.2 How long are you without using drugs?_____
2.3 Have you used more than one drug at the same time? ( ) Yes ( ) No
Which ones?_____
2.4 Have you had an episode of sudden hearing loss? ( ) Yes ( )N o
2.4.1 How long did you stay without hearing?_____
2.4.2 The hearing loss affected... ( ) RE ( ) LE ( ) BE
2.4.3 What kind of drug were you using when that happened?_____

Figure 1 - Anamnesis

To verify the presence of the suppressive effect of the OAE, the TOAE was carried out in the absence and then in the presence of noise in the contralateral ear. The stimulus used for this verification was the *click* of the nonlinear type in 80dBSPL. The contralateral white noise generated by the cochlear analyzer through the earphone TDH-39, at the intensity of 60 dB was used as a suppressor acoustic stimulus. The calculation of TOAE suppression was calculated by subtracting the S/N relation of the TOAE with and without contralateral acoustic stimulation. Positive values indicate the presence of TOAE suppression and negative values or zero indicated the absence of the phenomenon. The effect of TOAE suppression was analyzed by ear.

Subjects who presented alterations in any of the applied evaluations were referred for evaluation and otolaryngologic conduit.

The collected data were tabulated and analyzed using descriptive statistics and the nonparametric tests of Chi-square and Mann-Whitney, being chosen the statistical significance level of 5 % ($p \leq 0.05$).

RESULTS

Table 1 presents the distribution of G1 and G2 according to the time of drug use, showing that there were no subjects who fit the period between 11 and 15 years.

Table 1 - Distribution of the sample according to time and type of consumed drug

	Marijuana (N)	*Crack*/cocaine (N)	Total (N)
1 – 5 years	4	3	**7**
6 – 10 years	4	4	**8**
>15 years	2	0	**2**
Total	**10**	**7**	**17**

In the anamnesis, the subjects reported one month to eight years of abstinence, but the use of alcohol and cigarettes before the evaluations were observed.

The data obtained in the anamnesis showed that only one subject expressed no hearing complaints.

Table 2 presents the number of subjects in groups G1 and G2 who showed that complaints of hearing loss, tinnitus and difficulties regarding understanding of speech in noise. There was no statistically significant difference in the occurrence of complaints between groups (p = 0.985) .

Table 2 - Presence of hearing complaints in G1 and G2

	Hearing loss	Tinnitus	Difficulty in understanding speech in noise
G1 (N)	3	3	8
G2 (N)	4	3	5

Mann-Whitney Test (p=0,985)

The difficulty in understanding speech in noisy environments was the most frequent symptom in both groups. Eight of 10 subjects in G1 and five of the six subjects of G2 presented that complaint (Table 2). Considering that the same subject could have more than one complaint, this table shows an N greater than 17.

The majority (70.6 %) of the subjects had thresholds lower than 25 dB HL in the PTA. However, two subjects presented sensorineural hearing loss with a mild level in the right ear, other one presented neurossensorial hearing loss moderately severe in the right ear and moderate in the left ear; and two others presented normal tone average, but with hearing loss from 2000 Hz, a subject presented unilateral impairment and other bilateral one.

In relation to the subjects from both groups who used drugs within one to five years there was a statistically significant difference in the frequencies of 250, 500, 6000 and 8000 Hz in the right ear (Table 3).

On the other hand, in relation to the subjects who used drugs for six to 10 years, it was verified a statistically significant difference between G1 and G2 for the frequencies 4000 and 8000 Hz in the left ear (Table 4).

The group of subjects who used drugs in a greater period than 15 years was constituted only by marijuana users, so it was not possible to make a comparison between G1 and G2. The mean of pure tone thresholds for this group is in Table 5.

About half of the subjects had TOAE: 59 % in the right ear and 53 % in the left ear.

There were no statistically significant differences in S/N ratio of TOAE between G1 and G2, both for users of one to five years and for users from six to 10 years (Table 6).

Table 3 – Mean of the pure tone thresholds for G1 and G2 with one to five years of drug use

Frequency (Hz)	Right Ear			Left Ear		
	G1 (dBNA)	G2 (dBNA)	p value	G1 (dBNA)	G2 (dBNA)	p value
250	15,00	30,00	0,042*	17,00	15,00	1,000
500	17,00	42,50	0,032*	15,00	17,5	0,843
1000	17,00	40,00	0,121	12,00	22,50	1,000
2000	15,00	55,00	0,121	7,00	22,50	1,000
3000	12,00	50,00	0,051	10,00	20,00	1,000
4000	7,00	47,50	0,051	8,00	17,50	0,687
6000	15,00	50,00	0,044*	14,00	22,50	0,554
8000	11,00	47,50	0,044*	6,00	15,00	0,839

*value of p < 0,05 (Mann-Whitney Test)

Table 4 – Mean of pure tone thresholds for G1 and G2 with six to 10 years of drug use

Frequency (Hz)	Right Ear			Left Ear		
	G1 (dBNA)	G2 (dBNA)	(Hz)	G1 (dBNA)	G2 (dBNA)	(Hz)
250	12,50	20,00	0,306	15,00	15,00	1,000
500	10,00	16,25	0,383	11,25	13,75	0,661
1000	10,00	15,00	0,549	6,25	17,50	0,077
2000	6,25	17,50	0,105	3,75	17,50	0,072
3000	6,25	20,00	0,374	3,75	23,75	0,057
4000	5,00	18,75	0,538	2,50	27,50	0,025*
6000	7,50	13,75	0,556	10,00	20,00	0,101
8000	7,50	26,25	0,380	6,25	28,75	0,020*

*value of p < 0,05 (Mann-Whitney Test)

Table 5 – Mean of pure tone thresholds for 2 marijuana users (G1) for over 15 years

Frequencies (Hz)	250	500	1000	2000	3000	4000	6000	8000
Right Ear (dBNA)	10,00	15,00	20,00	17,50	27,50	32,50	35,00	30,00
Left Ear (dBNA)	10,00	10,00	5,00	7,50	15,00	22,50	20,00	7,50

Table 6 - Comparison of the means of the signal/noise ratio of transient otoacoustic emissions for G1 and G2 with one to five years and six to 10 years of drug use

	1 – 5 years			6 – 10 years		
	G1 (dB)	G2 (dB)	p value	G1 (dB)	G2 (dB)	p value
Right Ear	7,16	5,32	1,000	7,94	5,04	0,386
Left Ear	4,57	4,81	1,000	9,72	4,16	0,148

(Chi-square test)

Although it is not statistically significant, the G1 had the mean for the S/N relation of TOAE higher than the mean found for the G2. Considering that the OAE are present when the S/N ratio is greater than 6 dB, TOAE were present in the right ear for subjects from G1 with one to five years of drug use and in both ears for the G1 with six to 10 (table 6). And subjects from G1 with over 15 years of drug use showed no TOAE in both ears.

It was observed that about half of subjects with tinnitus (58 %) and with difficulty to understand speech in noisy environments (53 %) showed absence of otoacoustic emissions (Table 7).

Table 7 - Relationship between hearing complaints and the occurrence of transient otoacoustic emissions

Complaint	TOAE present	TOAE absent
Hearing loss	7	7
Tinnitus	5	7
Difficulty in understanding speech in noise	12	14
No complaints	2	0

Among the 19 ears that presented TOAE, 79 % of them also had this suppressive effect present.

The difference of the S/N ration of TOAE with and without noise suppressor is in Table 8. There was no statistically significant difference in the level of OAE suppression between G1 and G2 (p = 1.000) groups.

The absence of TOAE suppression effect was more frequent (75 %) in subjects complaining of difficulty to understand speech in noisy environments than in subjects who did not show complaints (table 9), although this relation was not statistically significant (p = 0.834).

Table 8 - Suppression effect of otoacoustic emissions for G1 and G2

	1 – 5 years		6 – 10 years	
	G1 (dB)	G2 (dB)	G1 (dB)	G2 (dB)
Right Ear	0,83	1,25	0,57	0,88
Left Ear	2,13	0,43	1,15	1,48

(Chi-square test; p = 1,000)

Table 9 - Relationship between difficulty in understanding speech in noisy environments and the suppression effects of otoacoustic emissions

Difficulty in understanding	Suppression effect present	Suppression effect absent
Yes	8	3
No	7	1

Chi-square test (p=0,834)

Table 10 - Relationship between the results of pure tone audiometry and transient otoacoustic emissions

	TOAE present n = ears	TOAE absent n = ears
PTA normal	16	11
PTA altered	3	4
p = 0,615		

(Chi-square test)

In Table 10, we observed the relation between the results and the occurrence of PTA and the occurrence of TOAE, however this relation was not statistically significant.

DISCUSSION

The effects of drug use on the hearing, more specifically on the cochlea, have been described in isolated case reports and also in studies with guinea pigs[8-11,17,18].

The most frequent complaint hearing for both groups was the difficulty in understanding speech in noise, followed by hearing loss and tinnitus (Table 2). Nigri et al. (2009)[19] in a research with 40 crack users and multiple drugs, the most common complaints that users presented were tinnitus, hyperacusis, auditory hallucination and balance alterations. Thus, the only symptom in common in both studies was the tinnitus.

In this case basis, 30 % of subjects presented some level of hearing loss. Indeed, the literature on case reports of sudden hearing loss after the use of cocaine and multiple drugs, the subjects showed labyrinthine hemorrhage[8], sensorineural hearing loss of moderate level in both ears[9], sensorineural hearing loss of severe level in both ears[10] and normal average tritone, but with hearing loss at frequencies from 2000 Hz[18].

Regardless of the time of drug use, the G2 group showed pure tone thresholds superior to G1 in both ears (Tables 3 and 4). These results might be related to the kind of drug that was used, once studies on rats show that cocaine causes decreased blood flow in the cochlea and, with this sensitive structure to hypoxia, the organ of Corti and spiral ganglion were susceptible to degenerative damages[11,17,20].

Considering that the subjects reported abstinence for more than a month, the possible effects of marijuana on the hearing could have already been recovered in this period and therefore they showed normal hearing thresholds and OAE present. However, those who used the drugs for more than 15 years had hearing loss in PTA and/or TOAE absent.

In relation to the crack/cocaine, Ciorba et al. (2009)[10] in report cases of sudden deafness after overdose showed that the first evaluation, OAE were absent and 30 days later were present when the outer hair cells recovered their function. In this case, it is supposed that cocaine only causes a disturbance of cochlear homeostasis, blocking potassium channels in the outer hair cells.

However, in cases of permanent hearing loss, as observed in this study, the cochlear damage possibly occurs by the reduction of oxygen in the cochlea, due to the vasoconstriction caused by the drug[10]. Thus, TOAE in users of crack/cocaine, could be explained as a result of deterioration of hair cells, resulting from prolonged hypoxia (use superior to 10 years) caused by drug use[11, 17].

Comparing the results found in users of illicit and licit drugs, it is observed that, in this study, even subjects with normal hearing in PTA showed TOAE (table 10), opposing to what was observed by Vinay (2010) [21] who investigated the ototoxicity of the cigarette in 50 normal-hearing, in which all subjects had TOAE, but with lower TOAE amplitude when compared to the control group. The same result was observed when they were evaluated with the OAE by product of distortion[22].

About half of the subjects who complained of difficulty in understanding speech in noise and tinnitus had TOAE absent. These results corroborate with researches carried out with subjects with normal hearing and tinnitus complaints, who perceived the relationship between the presence of the complaint and decreased amplitude and occurrence OAE[23,24]. Similar findings between these studies show that, no matter the sample, the presence of this symptom is generally associated with the absence of OAE.

The proper functioning of MOCS was evidenced by the presence of the suppressive effect of TOAE in 79% of the evaluated ears infers, in the subjects who were studied, the integrity of the auditory efferent pathway in ex-users of illicit drugs. In the study carried out by Perez, Kós e Frota (2006)[25] 29 women

with normal hearing and no history of drug use the TOAE suppression effect was present in 89.7% of right ears and 79.3% of left ears, which agrees with the results of our research. It is noteworthy that the suppressive effect of TOAE could only be investigated in 19 ears with TOAE.

In this study, 75 % of subjects who presented no suppressive effect of TOAE complained of difficulty in understanding speech in noisy environments (Table 9). The relationship between such complaint and abnormal auditory efferent pathway was demonstrated by Lautenschlager et al. (2010)[26] in a study with 24 normal hearing subjects and who showed difficulty of speech recognition in noise.

Although not statistically significant, when comparing the results of PTA with the occurrence of TOAE (Table 10), it was revealed that four ears with abnormal hearing thresholds showed TOAE absent, suggesting that hearing loss is related to alterations of the cochlea[13,27,28].

The 11 ears (32%) with normal PTA and TOAE absent (Table 10) suggest that the use of illicit drugs causes alterations in cochlear function even before modifying the tone thresholds. In a study of smokers, the authors found that 13.9% of subjects with normal hearing thresholds had EOA absent[29], lower rate if we consider the results of the present study.

In this study it was observed significant alterations in the ratings of ex-users of crack/cocaine (G2): higher auditory thresholds for frequencies of 250, 500, 4000, 6000 and 8000 Hz and the absence of TOAEs. The agreement of these results with the studies with guinea pigs exposed to cocaine[11,17] infers that crack and cocaine may be potentially ototoxic to the cochlea.

In general, the group of ex-marijuana users (G1) showed responses within normal limits for both PTA and for TOAE, except for those ones who had a period of use of drugs greater than 15 years. These results lead us to studies showing that the effect of marijuana on the body is transient[3] and therefore any alteration in the auditory system may have been recovered after the abstinence.

The results support the need to study the auditory system of illicit drug users in a long turn with a greater case basis.

CONCLUSION

Ex-users of crack/cocaine showed higher hearing thresholds and TOAE absent, no matter the time of drug use. However, for ex-marijuana users such alterations were only observed with more than 15 years of drug use.

In this sample, the use of marijuana and crack/cocaine had no deleterious effect on the olivocochlear system, when the two investigated groups were compared.

RESUMO

Objetivos: analisar se o uso de drogas ilícitas pode interferir nos sistemas auditivos periférico e central. **Métodos:** a amostra foi composta por 17 indivíduos distribuídos conforme o tipo de droga mais consumida: 10 indivíduos no grupo maconha (G1) e sete no grupo *crack*/cocaína (G2). Os grupos foram subdivididos segundo o tempo de uso de drogas: um a cinco, seis a 10 e mais que 15 anos. Foram avaliados por meio de anamnese, audiometria tonal liminar, imitânciometria, emissões otoacústicas transientes (EOAT) e efeito supressor das EOAT. **Resultados:** comparando os limiares tonais do G1 e G2, observaram-se limiares elevados para o G2, com diferença estatisticamente significante no grupo de um a cinco anos para 250, 500, 6000 e 8000Hz na orelha direita e de seis a 10 anos para 4000 e 8000Hz na orelha esquerda. Para usuários por mais que 15 anos, observou-se limiares superiores a 25dBNA de 3000 a 8000Hz na orelha direita. Nas EOAT e efeito supressor das EOAT não houve diferença estatisticamente significante entre G1 e G2 e entre os tempos de uso das drogas. O G1 apresentou relação sinal/ruído das EOAT superior ao G2. O efeito supressor das EOAT esteve presente em 79% das orelhas avaliadas. **Conclusão:** na amostra estudada, o *crack*/cocaína apresentou efeito mais deletério sobre o sistema auditivo do que a maconha. O maior tempo de uso de drogas influenciou nos resultados do G1. O uso de drogas ilícitas não provocou alterações no SOCM.

DESCRITORES: Audição; Emissões Otoacústicas Espontâneas; Drogas Ilícitas; Cannabis; Cocaína; Cocaína Crack

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