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ORIGINAL ARTICLE

Active Brazilian crack cocaine users: nutritional, anthropometric, and drug use profiles

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Objective: To evaluate the nutritional status of crack users and to analyze its correlation with drug use profiles.
Methods: Cross-sectional study with 108 crack users. Anthropometric data were assessed through body mass index (BMI) and bioimpedance (BIA) measurements. A blood test to analyze hematocrit, hemoglobin, glucose, and lipid profiles was also performed. Crack use was determined through a standardized interview.
Results: Based on BMI and BIA, most individuals were eutrophic (about 70%). Regarding hematological parameters, we found that hemoglobin and hematocrit levels were below normal for 32.4 and 30.6% of patients, respectively. Considering normal parameters, a large part of the sample (60.2%) had low levels of HDL cholesterol and high levels of triglycerides (38%). There were no significant correlations between drug profile and nutritional variables.
Conclusion: This is a pioneering study that examines the nutritional status of crack users. Our results showed that most crack users present normal anthropometric findings and the prevalence of underweight is low. However, blood analysis showed changes and a specific type of malnutrition.

Keywords: Crack cocaine; nutritional assessment; nutritional status; biochemical parameters

Introduction

Crack cocaine (crack), a smoked form of cocaine, is a highly addictive and powerful stimulant that became popular in the mid-1980s and has been used worldwide ever since.[1,2] Brazil is considered the world's largest consumer of crack, with approximately 1.7 million regular users.[3,4] Recently, an epidemiological study estimated a prevalence of 0.81% of regular crack-cocaine users in Brazilian capitals, corresponding to 35% of all illicit drug users apart from marijuana.[4] Although rates of crack use are lower than those of other drugs, it has been observed that this illicit drug leads to more psychiatric hospitalizations and causes a greater demand for care.[5]

Crack use stimulates the central nervous system and activates the brain's reward systems by blocking the presynaptic reuptake of dopamine, norepinephrine, and serotonin. This increases the availability of these neurotransmitters in the synaptic cleft and causes intense feelings of pleasure.[6] Moreover, crack users often abuse other types of psychoactive substances, mostly alcohol and tobacco, which can aggravate their clinical and nutritional status.[7,8]

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The clinical consequences and comorbidities of crack use have been well described in a number of studies,[8-12] but there is a gap in literature regarding the nutritional profile of crack users. Malnutrition in this population may be multifactorial and could involve lower caloric intake, abnormal metabolic and gastrointestinal functions, and even deleterious drug effects.[13] Association between obesity and stimulant use (such as crack) is rare, since cocaine and amphetamines are appetite suppressants that tend to reduce body weight with their anorexic effects.[14,15]

Several studies have highlighted malnutrition and underweight in active multiple-drug users.[13,14,16-21] However, weight gain and binge eating have also been observed in people recovering from drug and alcohol dependence, which suggests the possibility that food and drugs may act on the same brain reward mechanisms.[14,22-24] Surprisingly, a pilot study conducted by our group found high rates of normal-weight and overweight crack users at hospital admission.[25] Considering that overweight is associated with multiple medical conditions, including diabetes, hypertension, and hyperlipidemia, the increased risk of morbidity and mortality could be particularly harmful when combined with the use of stimulants such as cocaine.[15,26]

This study focused on describing the previously-unknown nutritional profile of crack users, supporting early intervention based on scientific and personalized findings to aid clinical recovery. Our main hypothesis was that the severity of crack use may impair nutritional status. Due to the large number of crack users in Brazil and the increasing demand for health services, research is needed in this area.

This study aimed to evaluate the nutritional status of crack users and analyze its correlation with drug use profiles. In addition, we compared the performance of different nutritional assessment methods, such as anthropometric and biochemical parameters (blood tests).

Methods

Baseline subject characteristics and study design

A total of 108 individuals were consecutively recruited by convenience between April 2014 and April 2015 at the Serviço de Psiquiatria de Adição of the Hospital de Clínicas de Porto Alegre (HCPA), a large Brazilian teaching hospital affiliated with the Universidade Federal do Rio Grande do Sul (UFRGS) that provides free public services. All patients met the criteria for crack addiction as described by the DSM-5.[27] Crack addiction was diagnosed through a comprehensive clinical interview performed by a trained psychiatrist in charge of inpatient admissions. Inclusion criteria were: being a male crack cocaine user who screened positive for cocaine in a urine test at admission (Bioeasy® cocaine test, Alere™, Recife, Brazil); being at least 18 years old; and agreeing to provide blood samples and a signed informed consent form. Crack was required to be the drug of choice, but the use of other psychoactive substances was not an exclusion criterion. Subjects who presented symptoms compatible with dementia or psychosis or those who presented cognitive impairment that prevented comprehension of the study were excluded from the sample. These exclusion criteria were verified by a psychiatrist in a clinical interview, who used the standard recruitment center evaluation.

Sociodemographic characteristics and drug use profile

The subjects' socioeconomic status was determined in accordance with the Brazilian Association of Research Companies (Associação Brasileira de Empresas e Pesquisa[28]) scale. This instrument includes questions about education, home appliances, housing characteristics, and access to public services to evaluate income level. Five classes are derived from these indicators: A (35-42 points); B (23-34 points); C (14-22 points); D (8-13 points); and E (0-7 points). Class A is the most advantaged, while class E is the poorest.

Crack use was determined through a standardized interview, using a questionnaire that included items related to the type, mode, and frequency of drug use. Severity of crack use was estimated by age of first use, years of use, and crack rocks used in the previous 30 days, as described in previous studies.[29-31] First crack use at the age of 11 was considered as 10 points, with a reduction of 1 point per year until age 20 (1 point) and 0 points for age 21 or older; 1 point was given for each year of per year crack use. The number of crack rocks used in the last 30 days was valued as follows: 1-5 = 1 point, 6-21 = 2 points, 22-40 = 3 points, 41-72 = 4 points, 73-103 = 5 points, 104-142 = 6 points, 142-200 = 7 points, 201-343 = 8 points, 344-515 = 9 points, and 516 or more = 10 points. The sum of these three variables was used to produce a crack use severity score, and the participants were categorized into a more severe or less severe group, which were divided by the median.

Daily alcohol consumption in the last 30 days was also assessed. Users mentioned the ingestion of only two types of beverage: *cachaça* (a distilled spirit made from fermented sugarcane juice, the most popular distilled alcoholic beverage in Brazil, with around 44% alcohol) and beer. For the statistical analyses, we used World Health Organization (WHO)[32] parameters, whereby 350 mL of beer or 30 mL of spirits correspond on average to 12 g of alcohol. We calculated the amount (in grams) of ethanol consumed daily. Data on the number of tobacco and marijuana cigarettes smoked in the last 30 days was also obtained in the clinical interview.

Anthropometry and body composition

The anthropometric evaluation was performed within 48 hours of admission. Weight and height were verified using an LD1050 scale (Líder®, São Paulo, Brazil) with 50 g precision and with a built-in stadiometer to determine body mass index (BMI) (weight in kg/height in m^2). BMI was classified according to WHO cutoff points.[33] Body composition (body fat percentage) was analyzed with tetrapolar bioimpedance (BIA) (Maltron BF 906, Maltron®, Rayleigh, UK). The test was administered according to manufacturer instructions, with patients having avoided exercise in the previous 12 hours, having fasted between 2 to 3 hours, and having their last urine elimination 30 minutes prior to the test. We used American Council on Exercise parameters to classify individuals based on body fat.[34]

Blood tests

Fasting blood samples were obtained for blood test analysis the morning after admission. Hematocrit and hemoglobin were analyzed with a Sysmex HST-402 (Sysmex, Hyōgo, Japan) platform that uses photometry and flow cytometry. For glucose and lipids (total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides), we used the enzyme assay method in a Cobas c702 analyzer (Roche, Basel, Switzerland). We measured the hematocrit and hemoglobin levels using the reference values proposed by Hoffbrand,[35] and the glucose and lipid profiles were based on references established by the Brazilian Society of Cardiology.[36] Human immunodeficiency virus (HIV) testing was performed on all patients using the immunoblot technique with HIV-1 and HIV-2 antigens. Student's *t*-test was used to verify the difference in variables between HIV-positive and HIV-negative individuals. Since there were no significant differences between groups, all individuals were recruited for the sample.

Ethics

This study was approved by the research ethics committee of the HCPA/UFRGS (project no. 140146). Data were collected after each individual was informed of the

procedures and objectives and gave permission to participate by signing an informed consent form.

Statistical analysis

The Kolmogorov-Smirnov method (with Lilliefors significance correction) was used to verify the normality of the variables. For the descriptive analysis, we used mean and standard deviation or median and interquartile range. The Pearson and Spearman tests were used for correlations and the Mann-Whitney *U* test and Student's *t*-test were used to compare groups. Statistical analysis was processed in SPSS version 18.0.

Results

As shown in Table 1, we assessed the subjects' baseline characteristics. Users were mostly Caucasian and came from poor socioeconomic backgrounds. Considering their BMI and BIA, few individuals were underweight or had low body fat; most were eutrophic, overweight, or obese.

The blood tests presented in Table 2 show that the patients' hemoglobin and hematocrit levels were below normal in approximately 30% of the sample, and a large part of the sample had low levels of HDL cholesterol and high levels of triglycerides. LDL cholesterol, total cholesterol, and glucose also presented alteration, but at lower percentages. HIV prevalence was 11.1%.

Table 3 shows the participants' drug use profiles (crack, alcohol, marijuana, and/or tobacco). Crack rocks consumed per day ranged from one to 150 and the consumption period ranged from four months to 32 years. Sixty-six (61.1%) patients reported daily consumption of alcoholic beverages (*cachaça* or beer). Ethanol consumption ranged from 34.28 g to 2,000 g per day. A total of 75.9% of the subjects reported daily use of tobacco cigarettes. A total of 50 individuals (46.29% of the sample) reported marijuana consumption. Among marijuana users, consumption ranged from one to 15 cigarettes per day. As a means of evaluating the influence of ethanol on crack consumption, a Mann-Whitney *U* test was used to assess the difference between drinkers (n=66) and non-drinkers (n=42). The difference between groups was significant ($p = 0.040$), demonstrating that individuals who consumed alcohol used crack less.

There were no significant correlations between crack consumption and anthropometric measures/blood tests. The main correlations are presented in Table 4. We found positive correlations between BMI, BIA, triglycerides, cholesterol, and glucose.

Using the median crack use severity, we divided the sample of users into two groups ($\leqslant 19$ and > 19). There was no significant difference in the nutritional, biochemical, or drug use variables analyzed between the groups (data not shown).

Table 1 Sociodemographic and nutritional baseline characteristics of crack users (n=108)

Variables	Reference value	n (%)
Age, mean (SD)	-	34.51 (9.22)
Race/ethnicity		
Caucasian	-	80 (74.1)
Black	-	28 (25.9)
Socioeconomic class		
A/B	-	0 (0.0)
C	-	5 (4.6)
D/E	-	103 (95.4)
BMI (kg/m^2)		
Underweight	< 18.5	6 (5.6)
Normal	18.5-24.9	69 (63.9)
Overweight + obese	> 25.0	33 (30.6)
BIA (body fat %)		
Essential fat	$\leqslant 5.0$	4 (3.7)
Average/normal	6.0-24.0	84 (77.8)
Obese	$\geqslant 25.0$	20 (18.5)

Data presented as n (%), unless otherwise specified.
BIA = tetrapolar bioimpedance; BMI = body mass index; SD = standard deviation.
BMI was classified according to World Health Organization cutoff points.[33] The subjects' body fat was classified according to American Council on Exercise parameters.[34]

Table 2 Assessment of nutritional parameters using blood tests (n=108)

Variables/reference value	n (%)	Mean (SD)
Hematocrit (%)		41.4 (3.6)
40.0-52.0	75 (69.4)	
< 40.0	33 (30.6)	
Hemoglobin (mg/dL)		14.0 (1.2)
13.5-17.5	73 (67.6)	
< 13.5	35 (32.4)	
Total cholesterol (mg/dL)		151.2 (36.5)
$\leqslant 200.0$	98 (91.6)	
> 200.0	9 (8.4)	
HDL cholesterol (mg/dL)		42.2 (14.1)
> 40.0	43 (39.8)	
$\leqslant 40.0$	65 (60.2)	
LDL cholesterol (mg/dL)		85.9 (29.5)
< 129.0	101 (93.5)	
$\geqslant 129.0$	7 (6.5)	
Triglycerides (mg/dL)		120.1 (63.1)
< 150.0	67 (62.0)	
$\geqslant 150.0$	41 (38.0)	
Glucose (mg/dL)		94.4 (34.6)
< 100.0	95 (88.0)	
$\geqslant 100.0$	13 (12.0)	
HIV		
Negative	96 (88.9)	
Positive	12 (11.1)	

HIV = human immunodeficiency virus; SD = standard deviation.
Hematocrit and hemoglobin levels were measured using the reference values proposed by Hoffbrand[35]; glucose and lipid profiles were based on references established by the Brazilian Society of Cardiology.[36]

Discussion

This study provides new insights into the nutritional assessment of drug addicts. Regarding anthropometric parameters (BMI and BIA), few individuals presented low weight

Table 3 Distribution of variables associated with the sample's drug use profile (n=108)

Variables	Mean (SD)	Median (IR)
Years of crack use	10.40 (7.01)	9.0 (5.0-14.0)
Crack rocks used (per day)	16.27 (23.90)	8.0 (4.0-15.5)
Age of first crack use	24.10 (8.70)	22.0 (17.0 30.8)
Severity of crack use	19.90 (8.20)	18.5 (14.0-25.0)
Associated drugs (alcohol and tobacco), n (%)		
Only crack	5 (4.63)	-
Crack + tobacco	17 (15.74)	-
Crack + tobacco + alcohol	26 (24.07)	-
Crack + tobacco + marijuana	16 (14.81)	-
Crack + tobacco + alcohol + marijuana	23 (21.30)	-
Crack + alcohol	10 (9.26)	-
Crack + alcohol + marijuana	7 (6.48)	-
Crack + marijuana	4 (3.70)	-
Ethanol (g/day) (n=66)	-	400.0 (154.3-800.0)
Comparison of crack use between groups (rocks/day)*		
With ethanol (n=66)	-	7.0 (3.0-10.0)
Without ethanol (n=42)	-	10.0 (6.0-20.0)

IR = interquartile range; SD = standard deviation.
* Mann-Whitney *U* test, p = 0.040.

Table 4 Correlations between body mass index, bioimpedance, and blood nutritional parameters (n=108)

	BIA	Hematocrit	Hemoglobin	TC	HDL	LDL	Glucose	Triglycerides	Ethanol (g/day)	Tobacco cigarettes/ day	Severity crack use
BMI*	**0.879**	-0.019	0.043	**0.278**	-0.058	**0.194**	0.171	**0.414**	0.094	-0.054	-0.023
	(> 0.001)	(0.844)	(0.659)	**(0.004)**	(0.548)	**(0.044)**	(0.077)	**(> 0.001)**	(0.336)	(0.576)	(0.814)
BIA*	1	-0.053	0.020	**0.302**	-0.035	**0.220**	**0.224**	**0.380**	0.086	-0.052	0.058
		(0.587)	(0.834)	**(0.001)**	(0.719)	**(0.022)**	**(0.020)**	**(> 0.001)**	(0.373)	(0.596)	(0.549)
Hematocrit*		1	**0.932**	0.007	-0.186	0.025	-0.144	0.181	-0.102	0.167	-0.176
			(> 0.001)	(0.942)	(0.055)	(0.798)	(0.137)	(0.061)	(0.293)	(0.084)	(0.068)
Hemoglobin*			1	0.052	-0.184	0.021	-0.095	**0.263**	-0.071	0.155	-0.152
				(0.596)	(0.056)	(0.830)	(0.330)	**(0.006)**	(0.465)	(0.108)	(0.116)
TC†				1	**0.403**	**0.832**	0.163	**0.399**	0.142	0.031	0.001
					(> 0.001)	**(> 0.001)**	(0.091)	**(< 0.001)**	(0.142)	(0.753)	(0.991)
HDL†					1	0.146	0.094	**-0.341**	**0.326**	-0.120	-0.089
						(0.131)	(0.334)	**(< 0.001)**	**(0.001)**	(0.217)	(0.362)
LDL†						1	0.080	**0.213**	-0.078	0.116	-0.004
							(0.411)	**(0.027)**	(0.422)	(0.230)	(0.964)
Glucose†							1	0.183	0.064	-0.052	0.047
								(0.059)	(0.510)	(0.590)	(0.627)
Triglycerides†								1	0.044	0.043	0.023
									(0.649)	(0.657)	(0.813)
Ethanol (g/day)†									1	-0.064	0.016
										(0.512)	(0.871)
Tobacco cigarettes/day†										1	-0.026
											(0.786)
Severity crack use†											1

TC = total cholesterol.
Values in bold represent correlations coefficients (r) with $p < 0.05$.
* Variable with normal distribution.
† Variable with asymmetric distribution.

and body fat, with most being normal weight, overweight, or obese. Although a number of studies have reported malnutrition and underweight in drug users,[13,14,16-21] our study found a low prevalence. Nevertheless, this does not exclude other specific deficiencies. The blood work revealed important alterations in a significant proportion of our sample, such as low levels of hemoglobin and hematocrit, which can be associated with protein-energy malnutrition and anemia. However, we found no significant correlations between crack use variables and any of the nutritional parameters we evaluated.

In one of the first studies on nutrition in drug addicts, Santolaria-Fernández et al.[16] demonstrated that 90% of drug users suffer from protein-energy malnutrition. Underweight (BMI < 18.5 kg/m^2) has already been demonstrated in multidrug users (60% prevalence)[18] and injection drug users (50% prevalence).[20] Despite this, we found no prevalence of low weight. One important bias is that the other studies involved multiple-drug users

from other locations with different drug profiles, usage types and frequency, while ours included only crack cocaine users with associated alcohol and tobacco consumption. Since this is the first study to ever sample such a profile, we cannot compare it confidently with studies covering different types of drug users.

There are data on overweight and obesity in recovering drug users, but not in current drug users.[14,22-24] For example, Cowan & Devine[14] studied drug addicts at different stages of recovery and reported that most have poor diets during active addiction, are generally undernourished at the beginning of treatment, and become overweight and obese during recovery. During abstinence, they may seek alternative ways of activating the brain reward system and the inhibition of dopamine reuptake, with one common outlet being overeating.[37] To reinforce this hypothesis, Ersche et al.[38] reported that cocaine-dependent men had a higher food intake than non-users, specifically foods high in fat and carbohydrates, but had no concomitant increase in body weight. The authors suggest that this is due to cocaine's interference with normal metabolic processes, resulting in an imbalance between fat intake and storage.

Regarding hematological parameters, we found that hemoglobin and hematocrit levels were below normal in 32.4% and 30.6% of patients, respectively. These decreased levels may indicate protein-energy malnutrition and anemia. In these cases, anemia may be associated with a diet poor in micronutrients, especially iron, as well as insufficient protein intake and clinical problems (decreased hydrochloric acidproduction, decreased intrinsic factor secretion, intestinal perforations, bacterial or infectious diseases).[39] Supporting our findings that BMI alone is a poor indicator of nutritional status, Nazrul et al.[18] demonstrated that 74% of drug addicts showed clinical signs of nutrient deficiency, with significantly lower hemoglobin and total serum protein levels. Meanwhile, Ross et al.[19] found blood markers indicating that 50% of all subjects had iron or vitamin deficiencies.

Compared to normal parameters, a large proportion of our sample (60.2%) had low levels of HDL cholesterol and high levels of triglycerides (38%). LDL cholesterol (6.5%), total cholesterol (8.4%), and glucose (12%) also presented alteration, but at lower percentages. This can probably be ascribed to the subject's low quality of life, lack of access to healthy food and physical activity, and high alcohol consumption. Tang et al.[20] studied HIV-positive and HIV-negative drug addicts and identified other aspects of nutritional deficiencies, such as food insecurity and low levels of caloric and protein intake. Our study also found low levels of HDL, which may be associated with a lack of social, economic, and physical activities. In a study on specific deficiencies in multiple-drug users, Nazrul Islam et al.[17] found lower concentrations of antioxidant vitamins E, C, and A in this population, suggesting a lack of access to certain foods. The damage and consequences of crack use can lead to numerous specific nutritional deficiencies and may require further investigation.

We found positive correlations between BMI, BIA, triglycerides, cholesterol, and glucose, which reinforces that body fat is associated with high serum lipid levels and glucose alterations. Changes in lipid and glucose profiles, alcohol consumption, and smoking are risk factors for the development of chronic diseases such as diabetes, dyslipidemias, hypertension, and metabolic syndrome. Considering that these individuals are at risk for cardiovascular problems due to drug use, the sum of these factors may increase the probability of disease and malnutrition.

There was no significant correlation between severity of crack use and anthropometric or biochemical variables. However, individuals who consumed alcohol used crack less ($p < 0.05$), suggesting a compensatory behavior involving ethanol. Crack use has been reported to induce intestinal perforations, gastric ulcerations, retroperitoneal fibrosis, abdominal pain, nausea, mesenteric ischemia, and esophagitis, thus impairing absorption, digestion, and metabolism[9] and leading to malnourishment. As for cardiovascular consequences, cocaine can cause increased heart rate and blood pressure, endothelial dysfunction, arrhythmia, and atherosclerosis. Combining cocaine with other substances (such as alcohol, marijuana, and/or tobacco) and other risk factors, such as overweight and obesity, can cause cumulative health damage.[26,40]

The high level of alcohol consumption (61%) among crack users was an unexpected finding. They reported consuming it in large quantities, mainly in the form of *cachaça*, exceeding the maximum daily amount of ethanol (30 g) recommended by the WHO.[32] Our results show high alcohol consumption, indicating that this factor should also be a concern in the treatment of crack addiction. The combined use of cocaine/crack and alcohol induces the biotransformation of cocaine, leading to the transesterification of a metabolite, which results in a substance known as cocaethylene. This increases the duration of euphoric effects and is more cardiotoxic than consuming each drug separately.[7] We also observed a high prevalence of tobacco and marijuana use, with 75.9% and 46.29% of the individuals involved in this behavior, respectively. It has been well documented that smoking can cause numerous diseases, such as cancer, emphysema, and cardiovascular diseases. The nutritional effects of smoking include an increase in free radicals and a decrease in antioxidants.[41] Although crack is much more deleterious than tobacco or marijuana, the effects of chronic associated use can be compounded.

We found that users from classes D and E were mostly Caucasian, characterizing a low-income population with little access to education. HIV prevalence in this group is high (11.1%) compared to the general population, confirming that crack users tend to engage in high-risk behaviors.[8,42] These social and clinical factors, when associated with crack use, can cause even further damage to their nutritional status. Since crack use is associated with complex social issues,[43] these individuals could benefit from lifestyle intervention programs, which have had positive results in the obese, diabetics, and binge eaters.[44-47]

Among the present study's limitations is the fact that we did not evaluate the impact of alcohol and marijuana consumption on nutritional status, and our sample consisted exclusively of male subjects. Moreover, we did not evaluate control subjects, which would have been important

for comparison with the crack group. We also evaluated subjects seeking inpatient treatment who, therefore, do not represent the general population of crack-cocaine users. However, the present study's pioneering results could serve to guide further research on the matter. In fact, we are already developing a case-control trial protocol to better elucidate the nutritional profile of crack users and evaluate the influence of nutritional aspects on treatment prognosis.

In summary, this was a pioneering study that examined the nutritional status of crack users. The BMI of most of them was within the normal, overweight, or obese range. However, their blood tests indicated alterations in hematocrit and hemoglobin levels and in glucose and lipid profiles. This shows that, although crack users are not necessarily underweight, they present other specific nutritional deficiencies that qualify them for a diagnosis of malnutrition. Furthermore, their condition may be associated with other issues that deserve attention, such as alcohol consumption and social problems. This study is the first step towards highlighting the importance of nutrition in the treatment of drug addiction for this marginalized population. We intend to follow these users and evaluate their changes in nutritional status during recovery, as well as food intake and food-related preferences during this period.

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Disclosure

The authors report no conflicts of interest.

References

1 Berridge V, Mars S. History of addictions. J Epidemiol Community Health. 2004;58:747-50.
2 Dias AC, Araújo MR, Dunn J, Sesso RC, de Castro V, Laranjeira R. Mortality rate among crack/cocaine-dependent patients: a 12-year prospective cohort study conducted in Brazil. J Subst Abuse Treat. 2011;41:273-8.
3 Alves HN, Ribeiro M, Castro DS. Cocaína e crack. In: Diehl A, Cordeiro DC, Laranjeira R, organizadores. Dependência química: prevenção, tratamento e políticas públicas. Porto Alegre: Artmed; 2011. p. 170-9.
4 Bastos FIPM, Bertoni N. Pesquisa Nacional sobre o uso de crack: quem são os usuários de crack e/ou similares do Brasil? Quantos são nas capitais brasileiras? Rio de Janeiro: ICICT; 2014.
5 Halpern SC, Scherer JN, Roglio V, Faller S, Sordi A, Ornell F, et al. [Clinical and social vulnerabilities in crack users according to housing status: a multicenter study in six Brazilian state capitals]. Cad Saude Publica. 2017;33:e00037517.
6 Carrera MR, Meijler MM, Janda KD. Cocaine pharmacology and current pharmacotherapies for its abuse. Bioorg Med Chem. 2004; 12:5019-30.
7 Harris DS, Everhart ET, Mendelson J, Jones RT. The pharmacology of cocaethylene in humans following cocaine and ethanol administration. Drug Alcohol Depend. 2003;72:169-82.
8 Duailibi LB, Ribeiro M, Laranjeira R. Profile of cocaine and crack users in Brazil. Cad Saude Publica. 2008;24:s545-57.
9 Muñiz AE, Evans T. Acute gastrointestinal manifestations associated with use of crack. Am J Emerg Med. 2001;19:61-3.
10 Pianca TG, Rohde LA, Rosa RL, Begnis AP, Ferronatto PB, Jensen MC, et al. Crack cocaine use in adolescents: clinical characteristics and predictors of early initiation. J Clin Psychiatry. 2016;77:e1205-10.
11 Stewart MJ, Fulton HG, Barrett SP. Powder and crack cocaine use among opioid users: is all cocaine the same? J Addict Med. 2014; 8:264-70.
12 Devlin RJ, Henry JA. Clinical review: major consequences of illicit drug consumption. Crit Care. 2008;12:202.
13 Quach LA, Wanke CA, Schmid CH, Gorbach SL, Mwamburi DM, Mayer KH, et al. Drug use and other risk factors related to lower body mass index among HIV-infected individuals. Drug Alcohol Depend. 2008;95:30-6.
14 Cowan J, Devine C. Food, eating, and weight concerns of men in recovery from substance addiction. Appetite. 2008;50:33-42.
15 Vanbuskirk KA, Potenza MN. The treatment of obesity and its co-occurrence with substance use disorders. J Addict Med. 2010;4:1-10.
16 Santolaria-Fernández FJ, Gómez-Sirvent JL, González-Reimers CE, Batista-López JN, Jorge-Hernández JA, Rodríguez-Moreno F, et al. Nutritional assessment of drug addicts. Drug Alcohol Depend. 1995;38:11-8.
17 Nazrul Islam SK, Jahangir Hossain K, Ahsan M. Serum vitamin E, C and A status of the drug addicts undergoing detoxification: influence of drug habit, sexual practice and lifestyle factors. Eur J Clin Nutr. 2001;55:1022-7.
18 Nazrul Islam SK, Jahangir Hossain K, Ahmed A, Ahsan M. Nutritional status of drug addicts undergoing detoxification: prevalence of malnutrition and influence of illicit drugs and lifestyle. Br J Nutr. 2002;88:507-13.
19 Ross LJ, Wilson M, Banks M, Rezannah F, Daglish M. Prevalence of malnutrition and nutritional risk factors in patients undergoing alcohol and drug treatment. Nutrition. 2012;28:738-43.
20 Tang AM, Bhatnagar T, Ramachandran R, Dong K, Skinner S, Kumar MS, et al. Malnutrition in a population of HIV-positive and HIV-negative drug users living in Chennai, South India. Drug Alcohol Depend. 2011;118:73-7.
21 Saeland M, Haugen M, Eriksen FL, Wandel M, Smehaugen A, Böhmer T, et al. High sugar consumption and poor nutrient intake among drug addicts in Oslo, Norway. Br J Nutr. 2011;105:618-24.
22 Hodgkins CC, Jacobs WS, Gold MS. Weight gain after adolescent drug addiction treatment and supervised abstinence. Psychiatr Ann. 2003;33:112-6.
23 Jackson TD, Grilo CM. Weight and eating concerns in outpatient men and women being treated for substance abuse. Eat Weight Disord. 2002;7:276-83.
24 Fenn JM, Laurent JS, Sigmon SC. Increases in body mass index following initiation of methadone treatment. J Subst Abuse Treat. 2015;51:59-63.
25 Willhelm FF, Escobar M, Perry IDS. Alterações na composição corporal e em parâmetros antropométricos de dependentes de crack internados em unidade de adição. J Bras Psiquiatr. 2013;62:183-90.
26 Stoutenberg M, Rethorst CD, Vidot DC, Greer TL, Trivedi MH. Cardiorespiratory fitness and body composition of stimulant users: a baseline analysis of the STRIDE cohort. J Subst Abuse Treat. 2017;78:74-9.
27 American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5). Arlington: American Psychiatric Publishing; 2013.
28 Associação Brasileira de Empresas e Pesquisa (ABEP). Critério de classificação econômica do Brasil 2014 [Internet]. [cited 2017 Oct 24]. http://www.abep.org/Servicos/DownloadCodigoConduta.aspx?id=02.
29 Scherer JN, Schuch S, Ornell F, Sordi AO, Bristot G, Pfaffenseller B, et al. High levels of brain-derived neurotrophic factor are associated with treatment adherence among crack-cocaine users. Neurosci Lett. 2016;630:169-75.
30 von Diemen L, Kapczinski F, Sordi AO, de Magalhães Narvaez JC, Guimarães LS, Kessler FH, et al. Increase in brain-derived neurotrophic factor expression in early crack cocaine withdrawal. Int J Neuropsychopharmacol. 2014;17:33-40.
31 Sordi AO, Pechansky F, Kessler FH, Kapczinski F, Pfaffenseller B, Gubert C, et al. Oxidative stress and BDNF as possible markers for the severity of crack cocaine use in early withdrawal. Psychopharmacology (Berl). 2014;231:4031-9.
32 World Health Organization (WHO). International guide for monitoring alcohol consumption and related harm. Geneva: WHO; 2000.

33 World Health Organization (WHO). Physical status: the use of and interpretation of anthropometry. Technical Report Series No. 854. Geneva: WHO; 1995.
34 Bryant CX, Green DJ. ACE lifestyle & weight management consultant manual: the ultimate resource for fitness professionals. San Diego: American Council on Exercise; 2007.
35 Hoffbrand AV, Moss PA. Hoffbrand's essential haematology. New York: John Wiley & Sons; 2015.
36 Xavier H, Izar M, Faria Neto J, Assad M, Rocha V, Sposito A, et al. V Diretriz brasileira de dislipidemias e prevenção da aterosclerose. Arq Bras Cardiol. 2013;101:1-20.
37 Volkow ND, Wang GJ, Tomasi D, Baler RD. Obesity and addiction: neurobiological overlaps. Obes Rev. 2013;14:2-18.
38 Ersche KD, Stochl J, Woodward JM, Fletcher PC. The skinny on cocaine: insights into eating behavior and body weight in cocaine-dependent men. Appetite. 2013;71:75-80.
39 Batista Filho M, de Souza A, I Bresani CC. [Anemia as a public health problem: the current situation]. Cien Saude Colet. 2008;13:1917-22.
40 Schwartz BG, Rezkalla S, Kloner RA. Cardiovascular effects of cocaine. Circulation. 2010;122:2558-69.
41 Preston AM. Cigarette smoking-nutritional implications. Prog Food Nutr Sci. 1991;15:183-217.
42 de Azevedo RC, Botega NJ, Guimarães LA. Crack users, sexual behavior and risk of HIV infection. Rev Bras Psiquiatr. 2007;29:26-30.
43 Narvaez JC, Pechansky F, Jansen K, Pinheiro RT, Silva RA, Kapczinski F, et al. Quality of life, social functioning, family structure, and treatment history associated with crack cocaine use in youth from the general population. Rev Bras Psiquiatr. 2015;37:211-8.
44 Verstuyf J, Vansteenkiste M, Soetens B, Soenens B. Motivational dynamics underlying eating regulation in young and adult female dieters: relationships with healthy eating behaviours and disordered eating symptoms. Psychol Health. 2016;31:711-29.
45 Mama SK, Schembre SM, O'Connor DP, Kaplan CD, Bode S, Lee RE. Effectiveness of lifestyle interventions to reduce binge eating symptoms in African American and Hispanic women. Appetite. 2015; 95:269-74.
46 Lofrano-Prado MC, Antunes HK, do Prado WL, de Piano A, Caranti DA, Tock L, et al. Quality of life in Brazilian obese adolescents: effects of a long-term multidisciplinary lifestyle therapy. Health Qual Life Outcomes. 2009;7:61.
47 Gorin AA, Niemeier HM, Hogan P, Coday M, Davis C, DiLillo VG, et al. Binge eating and weight loss outcomes in overweight and obese individuals with type 2 diabetes: results from the Look AHEAD trial. Arch Gen Psychiatry. 2008;65:1447-55.