

Relationship Between Serum Cortisol and Dietary Behavior in Non Diabetes Obese Cameroonian People

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Received: September 25,2019; Accepted: October 29,2019; Published: November 1,2019

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Abstract

Background: Dietary behavior refers to all the behaviors of an individual related to the consumption of food. In addition to environmental, socio-cultural and cognitive factors, the hormonal system plays an important role in the regulation of food intake. The aim of this study was to establish the relationship between the adrenocorticotrophic axis and dietary behavior in obese subjects in Cameroon.

Methods: This was a cross-sectional and analytical study over a period of 3 months at the National Obesity Center of Yaoundé. We included people who came to a dietetic consultation and had a BMI ≥ 30 kg / m². All patients with a known Cushing's syndrome, or who taking corticosteroids, as well as any other drug that has an influence on the bioavailability of dexamethasone were excluded. We evaluated the corticotrophic axis by quantitative determination of plasma cortisol before and after the overnight dexamethasone suppression test. After the clinical examination, the eating behavior was assessed using the Three-Factor Eating Questionnaire Test (TFEQ-R18).

Results: We included 25 patients (15 women and 10 men) aged 54 ± 10 years and with an average BMI of 37.8 kg / m². There was an association between BMI hyperphagia of disinhibition, cognitive restriction and hunger (P <0.0001). There was a significant association between disinhibition hyperphagia, susceptibility to hunger, cognitive restriction, and the level of cortisol after the overnight dexamethasone suppression test (P <0.0001).

Conclusion: The strong association between eating disorders, body mass index, and cortisol levels suggests that the adrenocorticotrophic axis would influence dietary behavior in obese subjects.

Keywords: Dietary disorders, Obesity, Serum cortisol.

Background

Dietary behaviour refers to all the behaviours of an individual vis-a vis the consumption of food. The physiological regulation of eating behavior is modulated by psychological, social and environmental factors that can disturb it, explaining the frequency of obesity [1]. The neuroendocrine events that preside in the short term to trigger and stop a food intake are modulated by endocrine factors that reflect the state of fat reserves of the body. In addition, there are other factors related to the environment, namely socio-cultural, psycho-affective, and cognitive factors that can also have a significant impact on eating behavior [2]. Food intake, which remains a voluntary behavior, is therefore subject to cognitive control by the individual [2,3]. This decision-making power can be overridden by external factors (view of the food ...) and can therefore settle eating disorders, responsible for significant weight abnormalities. During cognitive restriction, elevated secretion of cortisol was observed [4]. Some works incriminate the so-called "stress hormones", such as the

hormones of the corticotrophic axis, but also the growth hormone and insulin, both in eating disorders and in the development of the metabolic syndrome [5].

The practitioner, faced with obesity, always has the idea that there is an association between eating disorders and obesity [6]. The impact of eating behavior on hormonal changes or the metabolic syndrome is known [6,7]. The relationship between obesity and eating disorder is two-way: in fact, an eating disorder predisposes to obesity, and obesity predisposes to eating disorders including compulsion [8]. The aim of this study was to establish the relationship between the serum cortisol and the eating behavior in obese patients in Cameroon.

Methods

Data source and study subjects

It was a cross-sectional and descriptive study. The study took place over a period of 3 months at the National Obesity Center

(NOC) of Yaoundé. The study population consisted of obese subjects aged 18 to 60 years. We included all non-diabetic obese patients with BMI ≥ 30 kg / m² who give an informed consent. We excluded any patient with a known Cushing's syndrome, patients taking corticosteroids regardless of the route of administration, patients on estrogen / progestin and patients on drugs that can modify the bioavailability of dexamethasone. The study was proposed to the people coming in dietetic consultation.

Procedure

a. Information visit

We invited all people coming to routine dietetic consultation for obesity, to participate to the study. Capillary blood glucose was sampled in order to eliminate the presence of diabetes according diabetes diagnosis criteria.

b. Inclusion visit

Participants arrived at the NOC at 07A.M. The last meal must have been taken at 08P.M the day before the inclusion visit. In order to limit the stress induced by our interrogation which could have repercussions on the corticotropic axis, the patients were first sampled for biological work-up at laboratory. They observed a rest period of 30 minutes and after that, each participant was taken to the sampling room. All sampling started at 8:00 A.M.

Biological work-up include the lipid profile; basal cortisol levels and the overnight dexamethasone suppression test (ODST).

Next step consisted in filling the TFEQ-R18 by the participant himself in the consultation box. Sometimes, investigator could help if there were any concerns in the questionnaire. After all, we completed the visit by a physical exam.

Exploration of the corticotropic axis: Overnight Dexamethasone Suppression Test (ODST):

We gave 2 tablets of dexamethasone dosed at 0.5 mg tablet. The participant had to take 1mg of dexamethasone at home between 11pm and midnight. He returned the next day from 7:30 for a 2nd blood sample at 8 am.

c. Dietary assessment

The Three-Factor Eating Questionnaire Test (TFEQ)

The TFEQ is a standardized questionnaire with 51 questions. It assesses the eating habits of an individual in three main modes namely:

- The cognitive restriction capacity of food intake
- The susceptibility to hunger
- Disinhibition of hyperphagia.

In our study we used the revised 18-item test: TFEQ R- 18. A 10-15 minutes period was required to complete the questionnaire.

Each answer of the 18 items of the questionnaire is scored from 1 to 4; a score of 4 indicating a strong addiction to the behavior studied. Cognitive restriction, Disinhibition and Hunger were considered as low if the values were 0–10, 0–8 and 0–7, respectively. The values above 10, 8 and 7 of cognitive restriction,

disinhibition and Hunger respectively, were high. The results are presented as mean scores. Each question in the test is related to a type of eating behavior; and each factor measured by the test has an alpha Cronbach coefficient of 0.83-restrictive cognition, 0.77-dishinhibition and 0.80-feeling hunger [9].

The table I below represents the questions related to each eating behavior.

Eating behavior	Number of the question of the TFEQ related to a eating behaviour	Total of points
Cognitive Restriction	4, 6, 10, 14, 15, 18	24
Susceptibility to Hunger	3, 5, 8, 12, 17	20
Disinhibition of hyperphagia	1, 2, 7, 9, 11, 13, 15, 16	32

Statistical analyzes

The clinical and biological data obtained were stored in an Epi Data database and analyzed by SPSS software version 18.0. The results are expressed in terms of frequency and mean \pm standard deviation. The comparison between different groups was done by the Student's test. The Fisher test was used to study correlations between quantitative variables. We performed ANOVA test for multivariate analyzes. The threshold of significance is set at a value $p < 0.05$.

Results

Characteristics of the population studied

1. Population distribution by gender and clinical characteristics of the population:

We recruited 25 people who agreed to participate in our study. The study population consisted of 40% (n=10) men and 60% (n=15) women. The mean age of our subjects was 54 ± 10 years.

The average body mass index was of 37.8 kg / m². However, there was no elevation of blood pressure in these subjects (Table II)

Clinical Characteristics	Median [II Q]
Age (years)	54[40.5-60.5]
BMI(Kg/m ²)	37.8[32.8-44.9]
Fat Mass(Kg)	160[93-117.5]
Systolic Blood pressure(mmHg)	131[117.5-140.5]
Diastolic Blood pressure(mmHg)	81[73-89]
Height(cm)	164[160-172]
Waist circumference(cm)	114[107-121]
Hip circumference(cm)	120[111-134]

Biological data

No patient had a fasting glucose disorder. Regarding the lipid profile, our subjects had a high LDL-cholesterol level > 1g/l. The average basal 8AM cortisol was strictly normal at 162.7nmol/l. The determination of the basic 8 AM cortisol showed a maximum at 391nmol/l and the minimum at 9.7nmol/l. After the ODST, there was a suppression of cortisol with a maximum at 163nmol/l and a minimum of 9.18nmol/l (Figure 1). In addition, the ODST resulted in a suppression of > 50% of basal cortisol in 76% (n=19) participants and a significant absence of suppression in 25%(n= 6) participants.

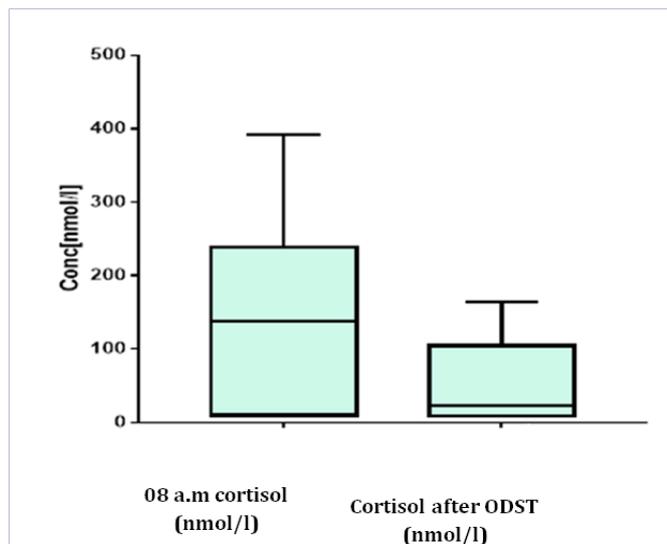


Figure 1: Variation of cortisol in the population before and after ODST

Study of dietary behavior

Regarding disinhibition of hyperphagia the highest score was 25 and the lowest score was 10/32. Regarding the feeling of hunger, the maximum score was 17 with a minimum of 5/20. For cognitive restriction we had a maximum score at 18 and a minimum of 8/24. (Figure 2)

Measures of association

Tables III, IV and V showed a high association between disinhibition of hyperphagia and suppression of cortisol after ODST by more than 50% (Eta = 0.737), between feeling hungry and a suppression > 50% of cortisol post ODST (Eta = 0.591) and also between cognitive restriction and post ODST suppression of cortisol > 50% (Eta = 0.824). After calculating the percentage of cortisol suppressed after ODST, we performed multivariate analyzes between different eating behaviours and the difference in cortisol concentration. Our results show that there is a strong association between the difference in cortisol suppressed and each eating behaviour P <0.001. There was also a very strong association (p <0.0001) between body mass index, eating behaviour, and percentage of suppression of cortisol (table VI). The multivariate analysis of each parameter of the lipid profile has shown that there is a strong association between these different parameters and the disorders of the studied eating behaviour. P <0.0001.

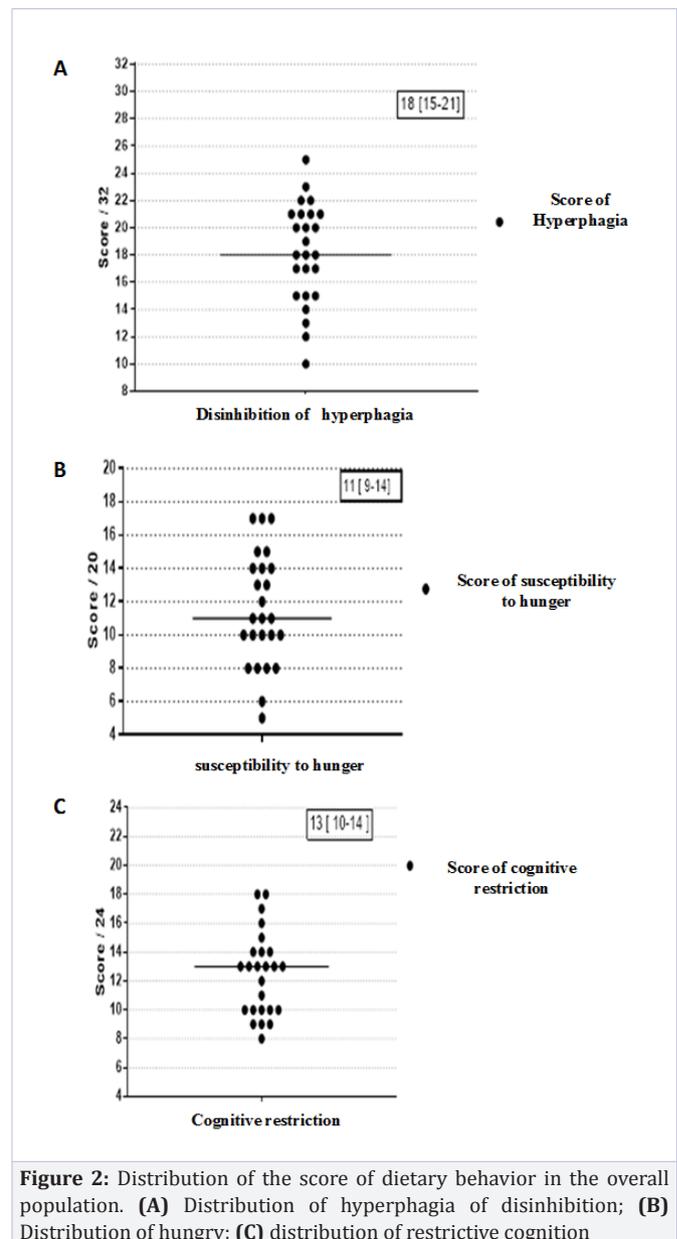


Figure 2: Distribution of the score of dietary behavior in the overall population. (A) Distribution of hyperphagia of disinhibition; (B) Distribution of hungry; (C) distribution of restrictive cognition

Discussion

We conducted a study that evaluated the implication of cortisol in the eating behavior of obese subjects. The burden of obesity is growing in developing countries especially in Cameroon, but data are lacking. Knowing the pathophysiological mechanisms involved in eating disorders could improve nutritional management of our obese patients. Our results show that more women than men are obese in 60% of cases. Mbanya in 2006 [10] also found that the prevalence of obesity was higher among women (17.1%) than men, especially since one is in urban areas. These same findings were also made by Sobngwi in 2002 [8]. This is explained by the fact that over the last 10 years in Cameroon, the lifestyle of the population has changed; physical activity is reduced, and moreover, women are active in the workplace. For this purpose, they practice less field activities

Table III: Association measure between the hyperphagia score and the difference in cortisol

		Cortisol decreased more than 50% of		Total	Eta
		baseline			
		NO	YES		
Disinhibition of hyperphagia score /32	10	1	0	1	0.737**
	12	1	0	1	
	13	0	1	1	
	14	0	1	1	
	15	0	3	3	
	17	1	2	3	
	18	1	2	3	
	19	0	1	1	
	20	0	3	3	
	21	1	3	4	
	22	0	2	2	
23	0	1	1		
Total	25.00	1	0	1	
		6	19	25	

*Eta tends to 0 = low degree of association,
** Eta tends to 1= high degree of association

Table IV: Association measurement between the feeling of hunger score and the difference in cortisol

		Cortisol decreased more than 50%		Total	Eta
		baseline			
		NO	YES		
Hungry sensation score/20	5	0	1	1	0.591**
	6	0	1	1	
	8	2	2	4	
	10	1	4	5	
	11	0	3	3	
	12	0	1	1	
	13	0	2	2	
	14	2	1	3	
	15	1	1	2	
	17	0	3	3	
Total		6	19	25	

*Eta tends to 0 = low degree of association
** Eta tends to 1= High degree of association

Table V: Association Measure Between the Cognitive Restriction Score and the Cortisol Difference

		Cortisol decreased more than 50%		Total	Eta
		NO	YES		
Cognitive restriction score/24	8	0	1	1	0.824**
	9	2	1	3	
	10	1	4	5	
	11	0	1	1	
	12	0	1	1	
	13	0	6	6	
	14	0	3	3	
	15	1	0	1	
	16	1	0	1	
	17	1	0	1	
18	0	2	2		
Total		6	19	25	

*Eta tends to 0 = Low degree of association
** Eta tends to 1= High degree of association

Table VI: Multivariate analyzes of dietary behavior disorders versus the difference in cortisol concentration and BMI

Compared variables	P value
Cortisol difference vs. Disinhibition of hyperphagia	<0.0001**
Cortisol difference vs. Hungry sensation	<0.0001**
Cortisol difference vs. Cognitive restriction	<0.0001**
One- way ANOVA: *p > 0.05 = No association **p < 0.05 = Association	

and other household tasks involving physical effort. In addition, physiologically, women, because of their higher estrogen levels and maternity, tend to gain more weight than men especially if it is in an obesogenic environment.

The 8 A.M cortisol of the participants was 167nmol / l. Indeed, there is a circadian rhythm secretion of cortisol with a peak of secretion early in the morning and which will gradually decline throughout the day with the lowest threshold between 11 P.M and midnight [11]. Thus, the diagnosis of adrenal insufficiency will be made on a morning cortisolemia at 8 o'clock when it is physiologically supposed to be high and the diagnosis of hypercortisolisms is made with regard to the midnight cortisol level [12]. The morning cortisol at 167nmol / l found in our subjects, allows us to affirm that none of our participants had a corticotropic insufficiency, confirming that there was no hidden uptake of corticosteroids which could explain the obesity in some participants.

The diagnosis of a hypercortisolism involves several tests including either the ODST, or the increase of the urinary free

cortisol (UFC) on the 24-hour urine . No test has a superiority over the other. ODST was suitable in this study because it is easily achievable and reproducible, therefore it represents the ideal test for outpatient screening of hypercortisolism [12].

After ODST, 76% patients suppressed cortisol by at least 50% baseline, while 24% had an insufficient suppression of cortisol. This might suggest, that these subjects would have hyperactivation of their corticotropic axis and thus subclinical Cushing's syndrome as described in the literature [13-14]. Indeed, subclinical Cushing's syndrome predisposes to a higher prevalence of obesity and other elements of the metabolic syndrome. This situation needs to be confirmed by others tests as late midnight salivary or serum cortisol or UFC; because effect exists in individuals a threshold of sensitivity to DXM [14]. The analyzes showed that there is a strong association between disinhibition of hyperphagia, cognitive restriction, feeling hungry and cortisol suppression by > 50% post ODST (eta = 0.7; 0.5 and 0.8 respectively). Studies have shown that disinhibition of hyperphagia and feeling of hunger influence weight gain. In Algeria, Koceir et al found that disinhibition of hyperphagia and hunger positively correlated with BMI [15]. In our study, BMI is strongly associated with disinhibition of hyperphagia (P <0, 0001). These results are similar to those of Anglé in 2009 in Finland [16]. In fact, disinhibition of hyperphagia is a frequent eating disorder in obese subjects, the weight gain occurs because of the imbalance of the energy balance with a massive food intake. All this is responsible for the onset of the metabolic syndrome and hormonal dysfunction including hormones involved in energy metabolism (cortisol, insulin resistance). Severe obesity is recognized as a cause of pseudo-Cushing, which could explain absence significant suppression of cortisol observed in some of our obese participants.

Regarding the different eating disorders we found that our participants had high scores, with a maximum of disinhibition of hyperphagia of 25/32, 17/20 and 18/24 respectively for the feeling of hunger and cognitive restriction. Several data in the literature have focused on the relationship between disinhibition hyperphagia and BMI in obese subjects [17]. Other studies have shown that disinhibition of hyperphagia is the major determinant of weight gain. Strong disinhibition is associated with the degree of adiposity [18]. Indeed, despite a high leptinemia, in obese patients, the sensation of hunger and the decrease in energy expenditure were strongly correlated with eating disorders [3]. Thus, a strong leptinemia does not decrease the appetite of the obese but rather continues to be accompanied by hyperphagia and weight gain. This is explained by a leptin resistance at both the peripheral and central level, as it has been proposed for insulin [19].

Conclusion

Dietary disorders had been identified in our population, and disinhibition of hyperphagia seems to be the major form eating disorders in our participants. There is a strong association between the 3 factors of eating behaviors and the suppression cortisol > 50% after ODST. There is also a strong association between dietary behavior scores, body mass index and lipid

profile. This might suggest that the adrenocorticotrophic axis would influence the dietary behavior of obese people in our population.

Abbreviations

BMI:	Body Mass Index
DXM:	Dexamethasone
HDL-C:	High density lipoprotein-cholesterol
LDL-C:	Low density lipoprotein-cholesterol
NOC:	National Obesity Center
ODST:	Overnight Dexamethasone Suppression Test
TC:	Total cholesterol
TFEQ:	Three Factor Eating Questionnaire
TG:	Triglycerides
UFC:	Urinary Free Cortisol

Acknowledgements

We gratefully acknowledge all the participants who have accepted to take part in this study.

Authors' contributions

JCM, ES conceived and designed the study; MCEE, WTBN, AOB, collected the data and retrieved the data; MCEE wrote the manuscript; AOB revised the language of the entire manuscript; GM and ENN analyzed and interpreted the data; All authors have read and approved the final manuscript.

Funding

The research received no funding

Availability of data and materials

Data will be made available by the corresponding author upon reasonable request

Ethics approval and consent to participate

The study was approved by the Regional Ethical Research Committee N° 0748/CRERHC2019. The was conducted in accordance with the guidelines of the Helsinki Declaration. All participants provided written informed concern prior to enrolment.

Competing interests

The authors declare that they have no competing interests.

Disclosures

No funding was received in the writing and publication of this article.

Competing interests

All authors declare no conflict of interest for tis article.

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