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Association of Obesity with Depressive Symptomatology, Eating Habits, Interleukin-8 and Cortisol in a Young Population

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ABSTRACT

Obesity is the result of a complex combination of psychological, biological, and environmental factors. In this work, we evaluate whether obesity is related to eating habits, depressive symptomatology, as well as interleukin-8 and cortisol. A descriptive cross-sectional study was carried out in 232 university students. All youths were surveyed to determine their eating habits and depressive symptomatology. Anthropometric measures and a blood sample were taken to determine its biochemical profile and its concentration of interleukin-8 and cortisol. The results show that interleukin-8 increase in the overfat group. The altered eating behaviors were frequent in the studied group; they were associated with the presence of obesity and the variation of interleukin-8 and cortisol. Besides, we found correlations of interleukin-8 with age, glucose, and lipid profile in the overfat group. In conclusion, these results indicate that high adiposity is related to changes in the concentrations of interleukin-8 and eating habits, confirming that obesity is the consequence of a complex network of various factors.

KEYWORDS

Obesity; interleukin-8; cortisol; eating behaviors

Introduction

Obesity is a global epidemic and is associated with an increase in morbidity and mortality. It has been estimated that nearly 2 billion people worldwide were overweight and obese in 2015, which represents about 40% of the world population. If this trend continues, the percentage could reach 60% by the year 2030 (Chooi, Ding, and Magkos 2019; Smith and Smith 2016). In Mexico, this pandemic is even more severe since up to 70% of the population over 20 years old is overweight or obese (Barquera et al. 2013).

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Obesity is the result of a complex combination of environmental, biological, and psychological factors. Among them, one of the main elements involved in the development of obesity is diet. Different studies have shown that both what you eat and how you eat influence the loss or gain of weight and body fat (Hayes et al. 2018; Townshend and Lake 2017). In this way, it has been reported that in different populations, eating habits and behaviors influence the development of obesity. Especially in young people who are a highly vulnerable population group (da Silva Gasparotto et al. 2015; Gunes et al. 2012; Sogari et al. 2018).

On the other hand, it has also been recognized that obesity occurs with a chronic inflammatory process, in which cells and molecules of the immune system participate (Guzman-Flores and Lopez-Briones 2012; Stolarczyk 2017). It has been proposed that when there is an accumulation of adipose tissue, local inflammation is generated, which induces the activation of immune cells and the release of proinflammatory cytokines, such as interleukin-8 (IL-8). This cytokine is secreted mainly by macrophages, and its primary function is to induce neutrophil chemotaxis. The low-grade systemic inflammation can affect the central nervous system, causing alterations in its neurochemical tasks, including the metabolism of monoamines and the Hypothalamic-Pituitary-Adrenal Axis, in which cortisol participates (Delgado et al. 2018; Martinac et al. 2014). Cortisol is glucocorticoid involved in the metabolism of lipids and carbohydrates, as well as in the immune response (Delgado et al. 2018; Lee et al. 2014). Furthermore, these changes seem to be involved in the regulation of different aspects of behavior, such as the reduction in satiety, eating habits, inactivity, and depressive symptomatology, among others.

However, the relationship between these factors in obesity remains unclear. Therefore, this work aims to evaluate possible associations between eating habits, depressive symptomatology, cortisol, and IL-8 with obesity, in a young population from western Mexico.

Materials and methods

Study participants

A descriptive cross-sectional study was carried out in 232 students of the University of Guadalajara, in Jalisco, Mexico. Youths of both genders were included, with an age of 18 to 30 years. To know their lifestyle and socio-demographic information, a general survey was applied at the time of the recruitment. Those subjects who presented chronic or genetic diseases that could alter their nutritional status were excluded. In addition, study subjects with a low percentage of body fat were also excluded. The study was conducted following the ethical regulations and data protection. The provisions of the Declaration of Helsinki, as well as the national rules. The local ethics

committee reviewed and approved the present study (approval No: CUA/CEI/079/2016). All participants were informed about the purpose of the research and subsequently asked to sign an informed consent letter.

Body composition

All measurements were made without footwear and with light clothing. The percentage of body fat was measured with a digital bioimpedance balance of the brand Tanita BC-545. The interpretation of body fat was made considering age and gender, the participants were classified as having low (BF% < 8), normal (BF% = 8–19.9), overfat (BF% = 20–24.9), or obese (BF% ≥ 25) body fat; for men. In women it was considered low (BF% < 21), normal (BF% = 21–32.9), overfat (BF% = 33–38.9), or obese (BF% ≥ 39) (Gallagher et al. 2000).

Biochemical analysis and measurement of cytokines

A venous blood sample was collected after 10 hours of fasting, at a time of 8 to 10 hours, in the morning. The serum was obtained by centrifugation and stored at −80 °C until analysis. The biochemical analysis included glucose, triglycerides, total cholesterol, and high-density lipoproteins. All biomolecules were determined by COBAS c111 (Roche Diagnostics; Mannheim; Germany). Low-density lipoprotein (LDL cholesterol) was calculated according to Friedewald et al (Friedewald, Levy, and Fredrickson 1972).

The ELISA technique using Cortisol Parameter Assay Kit (R&D Systems, USA) and ELISA MAX™ Deluxe Set Human IL-8 (Biolegend, USA) according to the manufacturer's instructions quantified the serum concentration of IL-8 and cortisol. Absorbance was measured using a microplate reader (Multiskan GO, Thermo Scientific, Finland).

Eating behavior and anxious and depressive symptomatology

The determination of the altered eating behavior was made through a test based on a previously published paper (Lazarevich, Irigoyen-Camacho, and Velazquez-Alva Mdel 2013). This questionnaire consisted of six questions: 1) Do you have difficulty in keeping a mealtime and to eat regularly? 2) Do you constantly feel hungry and overeat? 3) Is it difficult for you to stop eating once you have started even if you are satisfied? 4) Do you binge with the feeling that you cannot stop eating? 5) Do you prefer eating sweet things? 6) Do you have a tendency to snack frequently between meals? The options of the answers were according to the Likert scale, with options of never, sometimes, often, and always. The anxious and depressive symptomatology was identified using the questionnaire designed by Calderon. This instrument is validated in the Mexican population; in our study sample, it was estimated at >0.90 measured

by Cronbach's Alpha. Its interpretation allows classifying individuals into four groups: absence of anxious or depressive symptoms, depressive symptomatology and anxious symptomatology (Calderon-Narvaez 1997). The presence of other symptoms was not considered.

Statistical analysis

Descriptive analyses were performed for all variables. Data are presented as mean ± standard deviation and percentages. Significant differences in anthropometric data, biochemical, IL-8, cortisol, and dietary analysis among normal and obese individuals were determined by chi-squared, U from Mann-Whitney and Kruskal-Wallis ($p < .05$), besides the test of multiple comparisons of Dunn. After applying the corresponding normality tests. Statistical analysis was performed with SPSS 18.0 software (SPSS Science, Chicago, IL).

Results

A total of 232 youths were included in the study, of which 172 (74.1%) were women and 60 (25.9%) men. The study subjects were classified according to the percentage of body fat: normal, overfat, and obese. Table 1 shows the general characteristics concerning obesity. In any case, none of the factors studied were associated with obesity.

As can be seen in Table 2, the altered behaviors that were most frequently presented were: have difficulty in keeping mealtime and to eat regularly; and have a tendency to snack commonly between meals (87.5% and 89.7%, respectively). On the contrary, the ones that were least reported were: "difficult for you to stop eating once you have started even if you are satisfied"; and "binge with the feeling that you cannot stop eating" (28.9% and 35.8%, respectively).

Subsequently, we carry out the analysis of altered eating behaviors concerning the different groups, according to their body fat percentage (Table 2). Our results show that the rate of youths with binge eating is higher in the overfat and obese group. Also, the obese group presents more frequently difficulty

Table 1. General characteristics of the study population, according to fat percentage.

Characteristic	Total n = 232; (%)	Normal n = 131; (%)	Overfat n = 42; (%)	Obese n = 59; (%)
Gender				
Women	172 (74.1)	101 (77.1)	31 (70.8)	40 (67.8)
Men	60 (25.9)	30 (22.9)	11 (26.2)	19 (32.2)
Psychology				
Absence of anxious or depressive symptoms	152 (70.7)	83 (66.9)	25 (69.4)	40 (80.0)
Depressive symptomatology	23 (10.7)	16 (12.9)	3 (8.3)	4 (7.3)
Anxious symptomatology	40 (18.6)	25 (20.2)	8 (16.7)	7 (25.6)

Table 2. Altered eating behaviors concerning fat percentage.

Eating behavior	Total n = 232 (%)	Normal n = 131 (%)	Overfat n = 42 (%)	Obese n = 59 (%)
Keeping mealtime				
Never	29 (12.5)	17 (13.0)	6 (14.3)	6 (10.2)
Sometimes to always	203 (87.5)	114 (87.0)	36 (85.7)	53 (89.8)
Constantly feel hungry				
Never	49 (21.1)	35 (26.7)	6 (14.3)	7 (13.6)
Sometimes to always	183 (78.9)	96 (73.3)	36 (85.7)	52 (86.4)[a]
Binge eating				
Never	149 (64.29	97 (74.0)	23 (54.8)	29 (49.2)
Sometime to always	83 (35.8)	34 (26.0)	19 (45.2)[a]	30 (50.8)[a]
Difficult stop eating				
Never	165 (71.1)	101 (77.1)	31 (73.8)	33 (55.9)
Sometimes to always	67 (28.9)	30 (22.9)	11 (26.2)	26 (44.1)[a]
Sweet foods				
Never	51 (22.0)	30 (22.9)	9 (21.4)	12 (20.3)
Sometimes to always	181 (78.0)	101 (77.1)	33 (78.6)	47 (79.7)
Frequent snacking				
Never	24 (10.3)	15 (11.5)	5 (11.9)	4 (6.8)
Sometimes to always	208 (89.7)	116 (88.5)	37 (88.1)	55 (93.2)

[a] $p < 0.05$ for the normal group.

stopping eating and always felt hungry, compared to the normal group. The other altered eating behaviors were not associated with overfat or obesity.

We also measure different biochemical parameters, in addition to the cortisol and IL-8 serum concentration (Table 3). As expected, obese subjects had higher glucose and lipids, except HDL-C, compared to the normal group. Interestingly, IL-8 was found elevated in the overfat group compared to the fit and obese group. Regarding cortisol, no changes were observed among the groups studied.

On the other hand, we investigated if cortisol and IL-8 serum concentration were modified according to eating behaviors (Table 4). Those subjects who never had difficulty maintaining a stable mealtime had lower cortisol concentration, compared to youths who did have trouble in their meal schedules. Besides, youths who responded never to eat sweets or snacks had higher IL-8, compared to youths with the tendency to eat these foods.

Finally, correlations between biochemical parameters, IL-8, and cortisol were analyzed (Table 5). IL-8 correlated negatively with glucose, age, total

Table 3. Biochemical parameters in the different studied groups.

Biochemical parameters	Total n = 232	Normal n = 131	Overfat n = 42	Obese n = 59
Age (years)	20.3 ± 2.2	19.9 ± 1.7	20.6 ± 2.5	20.9 ± 2.9
Glucose (mg/dL)	86.5 ± 13.1	84.6 ± 12.6	83.8 ± 13.3	92.3 ± 12.6[a,b]
CHOL (mg/dL)	159.3 ± 28.3	157.4 ± 27.8	161.7 ± 28.8	161.7 ± 28.8
TGs (mg/dL)	93.2 ± 44.3	84.9 ± 34.3	96.6 ± 39.6	108.8 ± 60.2[a]
HDL-C (mg/dL)	49.3 ± 12.71	52.1 ± 11.7	46.7 ± 12.3	45.1 ± 11.3[a]
LDL-C (mg/dL)	91.3 ± 25.8	88.3 ± 25.7	95.0 ± 21.9	95.3 ± 28.1
VLDL-C (mg/dL)	18.6 ± 8.8	16.9 ± 6.8	19.3 ± 7.9	21.7 ± 12.0[a]
Cortisol (pg/mL)	8.7 ± 3.9	8.6 ± 2.8	8.5 ± 4.1	9.3 ± 5.6
IL-8 (pg/mL)	42.2 ± 31.2	37.9 ± 25.1[b]	59.6 ± 45.9	36.0 ± 20.8[b]

[a] $p < 0.05$ for the normal group. [b] $p < 0.05$ for the overfat group. The data are shown as mean ± standard deviation. CHOL: total cholesterol. TGs: triglycerides. HDL-C: high-density lipoprotein cholesterol. LDL-C: low-density lipoprotein cholesterol. VLDL-C: very-low-density lipoprotein. IL-8: interleukin-8.

Table 4. Serum cortisol and IL-8 according to altered eating behaviors.

Eating behavior	Cortisol		IL-8	
	Never	Sometimes to always	Never	Sometimes to always
Keeping mealtime	7.5 ± 2.6	8.9 ± 4.1*	52.0 ± 46.6	40.5 ± 27.7
Constantly feel hungry	8.0 ± 2.2	8.9 ± 4.3	38.5 ± 29.3	43.0 ± 31.7
Binge eating	8.9 ± 4.1	8.5 ± 8.7	44.1 ± 33.3	39.5 ± 27.9
Difficult stop eating	8.9 ± 4.3	8.2 ± 3.0	42.3 ± 32.7	42.0 ± 28.3
Sweet foods	8.3 ± 3.6	8.8 ± 4.0	51.0 ± 34.7	40.2 ± 30.2*
Frequent snacking	8.0 ± 2.8	8.8 ± 4.0	59.5 ± 39.0	40.6 ± 30.1*

*P < 0.05 for the never group.

Table 5. Correlation analysis between cortisol and IL-8 with biochemical parameters in the different study groups.

	Total n = 232		Normal n = 131		Overfat n = 42		Obese n = 59	
	Cortisol	IL-8	Cortisol	IL-8	Cortisol	IL-8	Cortisol	IL-8
Age (years)	0.147*	0.003	0.177*	0.107*	−0.033	−0.411*	0.256	0.145
Glucose	0.027	−0.559*	−0.036	−0.493*	0.210	−0.627*	−0.059	−.0611*
Cholesterol	0.106	−0.115	0.096	−0.159	0.266	−0.409*	0.047	−0.017
Triglycerides	0.167*	0.011	0.228*	0.069	0.014	−0.123	0.166	−0.003
HDL-C	0.105	−0.133	0.048	−0.161	0.060	0.021	0.220	−0.168
LDL-C	0.028	−0.078	0.046	−0.125	0.181	−0.407*	−0.082	0.101
VLDL-C	0.167*	0.011	0.228*	0.069	0.014	−0.123	0.166	−0.003

*P < 0.05, the number shown corresponds to the correlation coefficient (*r*).

cholesterol, and LDL-C in the overfat group, the same group where an increase in this cytokine was observed. We also found some correlations with cortisol, but these were weak.

Discussion

This research work addresses the relationship of obesity with eating habits, depressive symptomatology, cortisol, and IL-8. Our results show that young people from Jalisco (Mexico) showed a higher percentage of altered eating behavior than those reported in a previous study conducted in the city of Mexico (Lazarevich, Irigoyen-Camacho, and Velazquez-Alva Mdel 2013). Some of these behaviors were also associated with obesity. Our results are consistent with previous studies carried out in other parts of the world in the university population (da Silva Gasparotto et al. 2015; Gunes et al. 2012; Lazarevich, Irigoyen-Camacho, and Velazquez-Alva Mdel 2013). Therefore, these results show that personal habit is associated with obesity.

Moreover, in the present study, we found no evidence of an association between anxious or depressive symptoms and obesity. These results differ with others previously reported (Lazarevich et al. 2016; Lazarevich, Irigoyen-Camacho, and Velazquez-Alva Mdel 2013; Zavala et al. 2018). However, it is important to consider that the depressive symptomatology was made through a questionnaire designed by Calderon (Calderon-Narvaez 1997).

The serum concentration of IL-8 increased in the overfat group, as reported in other studies. Although, in these works, the subjects were diagnosed as obese according to their BMI; while we did it through the percentage of fat, a method that considers the age, gender, and the presence of muscle mass. (Dorneles et al. 2016; Kim et al. 2006; Straczkowski et al. 2002; Tam et al. 2010). The increase of IL-8 may be explained because this cytokine is secreted mostly by visceral adipose tissue (Bruun et al. 2004). This variable unfortunately, we do not measure in specific. Another possible explanation for the increase in IL-8 may be that this cytokine is a potent chemotactic factor of neutrophils, and these cells participate mainly in the initial stages of inflammation. Therefore, it may decrease when the inflammation has become chronic, being the case, the obesity group. Also, the measurement of other parameters used as inflammation markers would be useful to understand the role of IL-8 in the inflammatory circuit present in obesity.

We find an association in the subjects who tend to consume more sweet foods and snacks with lower levels of IL-8. A study conducted in subjects with anorexia nervosa analyzing IL-8, show controversial results. Some molecules involved in inflammation decreased, such as TNF-β, while they increased, such as IL-6 and IL-15. It is interesting to note that IL-8 did not have a variation between the two groups studied (Dalton et al. 2018). However, it should be noted that our study subjects did not present a condition as dangerous as anorexia nervosa, and our study includes a larger sample. Therefore, eating habits can induce an inflammatory state, and possibly IL-8 has some participation in this process.

Interestingly, our results show negative correlations of IL-8 with biochemical parameters, especially in the overfat group, although this data should be taken with caution. The negative correlation between IL-8 and glucose was an unexpected result due to the pro-inflammatory nature of this cytokine. Previous studies have shown that pro-inflammatory molecules, such as TNF-alpha, IL-6, as well as IL-8, are associated with insulin resistance (Guzman-Flores and Lopez-Briones 2012; Kim et al. 2006; Stolarczyk 2017). However, research conducted in smooth muscle cells reported that mRNA expression was diminished by high glucose (Temaru et al. 1997). Besides, another research group reported that IL-8 upregulates the expression of GLUT3, thereby increasing glycolysis, so a high concentration of IL-8 would induce a decrease in serum glucose (Shimizu and Tanaka 2019).

We did not find a statistical difference in the concentration of cortisol in the study groups; this may be due to the sample because other studies have preferred to measure this molecule in saliva and even in the hair (Lee et al. 2014). Concerning eating behaviors, we found that cortisol increased in those people who did not keep mealtime. A previous investigation carried out in adults showed that those people with eating disorders had high cortisol at different times of the day, but mainly in the morning (Filaire et al. 2015).

Another study conducted in children correlated cortisol with the consumption of sweet foods (Michels et al. 2013). In any case, one of the limitations of the present study was the measurement of serum cortisol and not saliva, and only one sample was taken in the morning.

Finally, our results could be integrated as follows. Eating behaviors, predispose to the gain of body fat. Excess fat increases IL-8 concentration, but not cortisol. However, this molecule is unable to activate the Hypothalamo-Pituitary-Adrenal axis, so the behavior is not altered, and depressive symptomatology is not observed. On the other hand, alterations in eating behaviors change IL-8, which is consistent with the negative correlation of IL-8 with glucose and hyperglycemia found in the overfat group.

One of the limitations of the present study is the type of methodological design; since it is a cross-sectional study, it is not possible to determine a cause-effect, we are only able to decide on associations. Although we try to make a comprehensive study of obesity, covering different factors, it is advisable to carry out more studies to clarify the multifactorial origin of obesity.

In general, our results show that IL-8 is possibly associated with overfat, age, and glucose. This cytokine and cortisol are also associated with eating behaviors, which have a high prevalence in young people from Mexico. On the other hand, we did not find depressive symptoms associated with obesity. Therefore, these results show that obesity is a complex network of several factors.

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Disclosure statement

The authors declare no conflict of interest.

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