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Proinflammatory, anti-inflammatory cytokines and adiponkines in students with central obesity

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ABSTRACT

Several studies have investigated the correlation between central obesity and inflammatory cytokines and the anti-inflammatory cytokine adiponectin. But, the correlation between central obesity and the anti-inflammatory cytokines IL-4, IL-5 has not been studied yet. Thus, we aimed to study the IL-4 and IL-5 correlation to central obesity in adolescent Egyptian girls among proinflammatory and anti-inflammatory cytokines. The study was carried out on 86 obese adolescent girls (BMI > 95 percentile) divided into two groups according to central obesity. The group I with waist to hip ratio <0.8 as a control and group II with waist to hip ratio >0.8 (central obesity). There was a significant increase in TNF-alpha ($p < 0.0001$), and IL-1β ($p < 0.0001$), as proinflammatory cytokines in group II, as compared to their corresponding group I. Group II showed a significant increase in the anti-inflammatory cytokines IL-4 and IL-5 than group I at ($p < 0.0001$) and ($p < 0.0005$) respectively. In addition there was a significant decrease in the anti-inflammatory adiponectin and an increase in the inflammatory leptin levels in group II at ($p < 0.0001$) and ($p < 0.0001$) respectively in comparison to group I. A high positive correlation has been observed between waist to hip ratio, leptin, TNF-α, IL-1-β, IL-4 and IL-5 at ($r = 0.331$, $p < 0.03$), ($r = 0.559$, $p < 0.001$), ($r = 0.435$, $p < 0.004$), ($r = 0.509$, $p < 0.001$), ($r = 0.550$, $p < 0.0015$), in group II respectively and a high negative one with adiponectin at ($r = -0.410$, $p < 0.0001$). We concluded that central obesity lowers adiponectin plasma level through increasing proinflammatory adipokines such as TNF-α, IL-1β, leptin. Further studies are needed to explore the positive correlation we found between central obesity and the anti-inflammatory cytokines IL-4 and IL-5 known to be associated with bronchial asthma.

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1. Introduction

Abdominal obesity known as central obesity is the accumulation of abdominal fat resulting in an increase in waist size [1]. Recently a study released by Jay and colleagues [2], revealed that weight gain results in lipid accumulation and adipocyte stress-factors known to disrupt the balance of systemic cell signaling (adipokines and cytokines) [3]. This bioactive substance my directly contributes to the pathogenesis of conditions associated with obesity [4], and favors inflammation [3]. A study done by Matsuzawa revealed that a substantial proportion of adipocytokines are involved in the inflammatory stimulation and response, as either proinflammatory or anti-inflammatory adipocytokines [5]. Initial studies have focused mainly on the association of obesity with TNF-α, IL-1β [6], leptin [7], adiponectin [8], IL-4 [9]. In addition Lamkhioued et al. [10], observed the presence of IL-5 in asthmatic obese individuals. TNF-α has come to be recognized as an important adipocytokines, and that adipose TNF-α mRNA and plasma TNF-α protein are increased in most animal models and humans with obesity [11]. In addition to TNF-α, IL-1β has been shown to be secreted from adipose tissue and linked to inflammation and inflammatory response [12]. Leptin the inflammatory adipokine [13], is secreted primarily by fat cells and acts centrally particularly in the hypothalamus to reduce food intake and body weight [14]. An excess of leptin in the circulation in obesity and overweight [15], acts as a nonmetabolic switch, connecting the body's metabolism to high energy consuming process such as immune response [16], reflects the amount of energy stored in adipose tissue and remains proportional to overall adipose mass [17]. Adiponectin is one of the most abundant adipokine produced by adipocytes [8], this strong anti-inflammatory adipokine was found to be inversely related to adiposity [18]. Furthermore, it has recently been suggested that adiponectin also acts in the brain to increase energy expenditure and cause weight loss [19]. These observations suggest that adiponectin is an important negative regulatory adipokine in immune and inflammatory systems indicating that it may be involved in terminating inflammatory responses through its inhibitory functions [20]. IL-4 is involved in lipid metabolism by

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inhibiting lipid accumulation in fat tissues, which leads to decreased weight gain and fat mass [9]. Spergel et al. [21], revealed that IL-4 regulates some proinflammatory chemokines, glucose and lipid metabolism by promoting insulin sensitivity, glucose tolerance and inhibiting lipid deposits [9]. Interleukin 5 (IL-5) is the main factor that promotes the differentiation of eosinophils progenitors and enhances the effector capacity of mature eosinophils [22], mediating its expansion priming, recruitment and prolonged tissue survival in response to allergic stimuli at the level of bone marrow, blood and tissue [23], in addition it plays an important role in enhancing the attraction of eosinophils to the airways and in prolonging their survival into the lung parenchyma of obese individuals [10]. So, the proposal of this research is to investigate the effect of central obesity in obese adolescent girls on the promotion of the secretion of pro- inflammatory (TNF-α, IL-1β, leptin) and anti-inflammatory adiponectin, (IL-4, IL-5) adiponkines. A further study is needed to clarify the role of IL-5 if it can be considered as a predictor of asthma in children with central obesity.

2. Materials and subjects

2.1. Subjects

The current study was carried out on obese adolescent girls with age range from 13 to 18 years old, through a project conducted in the National Research Centre, Egypt, to estimate the prevalence of obesity among school adolescent girls. It was a cross-sectional survey. Four local public schools Cairo were enrolled in this study regarding adolescents (two preparatory schools and two secondary schools). Permission to perform the study was granted by the Ministry of Education, and the directors of the school included in the research.

The protocol was approved by the ''Ethical Committee'' of the ''National Research Centre''. In accordance with the code of ethics of the world medical association (Declaration of Helsinki) of the total sample, 86 adolescent girls with the complaint of obesity were included in the current research after obtaining written informed consent from their parents. Student assent was also obtained. The adolescents were required to meet the following inclusion criteria: age, 13–18 years and BMI > 95 percentile for age and gender, students were excluded if they had a prior major illness, including type I or II diabetes, took medications or had a condition known to influence body composition, and insulin secretion (e.g. Glucocorticoids therapy, hypothyroidism, Cushing's disease). Each adolescent underwent a complete physical examination, including anthropometric measures. The height and the weight were recorded. The height was measured to the nearest 0.5 cm on a Holtain portable anthropometer, and the weight was determined to the nearest 0.1 kg on a Seca scale Balance with the subject dressed minimum clothes and no shoes. Body mass index (BMI) was calculated as weight (kg)/height (m^2). Waist circumference was measured to the nearest 0.1 cm by using nonstretchable stain steel tape, considering the smaller circumference between the iliac crest and first rib as the anatomical point to perform the measurement [24]. Hip circumference was measured to the nearest 0.1 cm at the maximal gluteal protrusion or at the most prominent area of the buttocks at the level of the symphysis pubis in a horizontal plane the tape measure was held snugly against the body but without compression [25]. The 90th percentile values for waist circumference for gender and age generated in the national health and nutrition examination survey (NHANES III) were used as cut off values to identify children with abdominal obesity [26]. Then, WHR (waist to hip ratio) was calculated by dividing waist (cm) by hip circumference (cm), and abdominal obesity was diagnosed when the WHR was 0.80 in girls [27].

The participating adolescent girls were divided into two groups according to central obesity Group I (as controls): with WHR < 0.8 and Group II with WHR > 0.8 (with central obesity) .Blood samples were drawn from the students and the serum was separated and kept frozen at −70 °C until analysis.

2.2. Biochemical assays

(1) Serum IL-4 levels were determined with an enzyme- linked immunosorbant assay (ELIZA) technique using commercial kits (Orgenium laboratories, FIN-00790, Helsinki, Finland) and the sensitivity of detection level was <7 pg/ml.
(2) IL-5 was measured using ELIZA Kit Bender MedSystems GmbH Campus Vienna Biocenter A-1030 Vienna, Austria, Europe and the sensitivity were 1.45 pg/ml.
(3) Serum levels of cytokines TNF-alpha were measured using commercially available ELIZA kits (Ani Biotech Orgenium Laboratories Business Unit, Finland) Bender MedSystems GmbH Campus Vienna Biocenter and the sensitivity of detection for TNF-was 2.3 pg/ml.
(4) Adiponectin (Acrp 30) levels were determined with an (ELIZA) procedure using commercial kits (Orgenium laboratories Viikinkaari 6, FIN-00790, Helsinki, Finland) and the sensitivity of detection level was <3 ng/ml.
(5) Serum levels of IL-1β were measured using ELISA kit purchased from IMMUNOTECH SAS-130 AV. De latter de Tassigny- B.P. 177-13276 Marseille Cedex 9 France. And the sensitivity of detection was 1.5 pg/ml in the protocol 0–250 pg/ml.
(6) .Leptin levels were determined with an (ELIZA) procedure using commercial kits (Diagnostics Biochem Canada Inc., 1020 Hargrieve Road) and the sensitivity of detection level was 0.5 ng/ml.

2.3. Statistical analysis

All values are expressed as mean ± SE and the differences between the two groups were calculated by Student's *t* test. The correlation was done between different parameters using Pearson correlation. All analyses were carried out using SPSS version 14 (IBM, Chicago IL, USA. Statistical software).

3. Results

Data and the difference between group I and group II regarding waist circumference and WHR showed a highly significant difference ($p < 0.001$) between the two studied groups noting that group II recorded the higher values (Table 1).

Table 1
Comparison between group I and II regarding studied parameters.

Group	Total number	Age	Number	Waist circumference (cm) mean ± SE	WHR mean ± SE
Group I	43	13–18	43	79.11 ± 0.75	0.74 ± 0.0045
Group II	43	13–18	43	102 39 ± 2.72	0.89 ± 0.017
Total	86	13–18	86	$p < 0.001$	$p < 0.001$

$p < 0.0001$ = very highly significant difference.

Table 2
Comparison between proinflammatory cytokines and adipokines in group I and II.

Proinflammatory cytokines and adipokine	Group I mean ± SE	Group II mean ± SE	Significance
TNF-α pg/ml	2.24 ± 0.59	30.4 ± 1.735	$p < 0.0001$
IL-1β pg/ml	4.21 ± 0.436	60.83 ± 4.43	$p < 0.0001$
Leptin ng/ml	7.24 ± 0.314	16.25 ± 0.41	$p < 0.0001$

$p < 0.0001$ = very highly significant difference.

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