PAMJ One Health
Pan African Medical Journal

Research

# Macronutrient, physical activity, inflammatory biomarker, and lipid profile in obese adolescents

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Received: 10 Mar 2020 - Accepted: 23 Apr 2020 - Published: 06 May 2020

Domain: Child nutrition, Nutrition, Pediatrics (general)

Keywords: Macronutrient, TNF-Î±, hsCRP, lipid profile, adolescents, obesity

**Abstract**

**Introduction:** obesity causes oxidative stress, inflammation, and hypertrophy which cause various health complications. This study aims to analyze macronutrients, physical activity, inflammatory biomarkers, and lipid profiles in obese adolescents. **Methods:** a cross sectional study was conducted at the Pediatric Nutrition and Metabolic Diseases Outpatient Clinic, Dr Soetomo General Hospital, Surabaya from July to October 2018. Subjects were selected using total sampling technique. Weight and height measurement were performed on subjects to obtain a BMI. Macronutrient data was obtained through food recall. TNF-α and adiponectin examination were performed by the ELISA method. Correlation between variables was analyzed by correlation analysis with significant p <0.05. **Results:** this study found 59 central obesity adolescents, consisting of 32 (54.2%) adolescent boys and 27 (45.8%) adolescent girls. Adiponectin has a negative correlation with total calories and fat. There was no correlation between macronutrients with TNF-α and lipid profiles (p> 0.05). There was no correlation between sedentary physical activity and TNF-α, adiponectin, and lipid profile (p> 0.05). Sleep duration has a negative correlation with TNF-α (p = 0.017; r = -0.310). **Conclusion:** total calories and fat had a negative correlation with adiponectin. Sleep duration had a negative correlation with TNF-α. Macronutrient intake and lifestyle can be used as an early detection of inflammatory biomarker abnormalities in obese adolescents.

**Research | Volume 2, Article 3, 06 May 2020 | 10.11604/pamj-oh.2020.2.3.22283**

Available online at: https://www.one-health.panafrican-med-journal.com/content/article/2/3/full

## Introduction

The prevalence of obesity has increased in various countries and caused a health burden [1]. Obesity causes excess body metabolism. Excessive body metabolism causes an increase in stress response, inflammation and hypertrophy of cells. These conditions cause various health complications [2]. Metabolic syndrome, diabetes mellitus, cancer, cardiovascular disease and respiratory disorder are complications of obesity that can occur [3]. Cardiovascular disease causes more than 2/3 of deaths in obesity [1]. Excessive fat deposition causes accumulation of fat elsewhere, such as the liver which can trigger insulin resistance [2]. Genetic factors, lifestyle, diet, and physical activity influence inflammation in obesity [4]. Activation and infiltration of immune cells causes increased production of pro-inflammatory cytokines and causes inflammation [5]. Inflammatory conditions that occur in obesity are not accompanied by signs of infection or autoimmunity. Inflammation in obesity is called low grade inflammation or metainflammation [2]. The number of M1 macrophages and Th1 cells increases in obesity. Free fatty acids resulting from increased lipolysis stimulate TNF-α production [5]. In addition, the protective hormone produced by adipocytes is adiponectin which has a positive effect on inflammation in obesity [6]. Dietary patterns play a role in the occurrence of inflammation in obesity and complications due to obesity [2]. The content of inflammation in the diet associated with inflammation in obesity. Previous studies have suggested that decreased carbohydrate consumption and increased consumption of unsaturated fats and proteins improve inflammation [7]. Metabolic stress in obesity causes organelle dysfunction, especially the endoplasmic reticulum and mitochondria which regulate the metabolism of glucose, fat, protein, and cholesterol. In obesity can occur disorders of carbohydrate and fat metabolism [2]. The identification of risk factors for the emergence of complications from obesity and inflammatory biomarkers increases the chances of earlier therapy and better outcomes. This study aims to analyze macronutrients, physical activity, inflammatory biomarkers, and lipid profiles in obese adolescents.

## Methods

This research was a cross sectional study conducted at the Pediatric Nutrition and Metabolic Diseases Outpatient Clinic, Dr. Soetomo General Hospital, Surabaya from July to October 2018, Surabaya in obese adolescents. The study was conducted after obtaining ethical approval from the Ethics Commission. The study was conducted after parents sign the informed consent. A total of 59 obese adolescents aged 13-16 years were involved in this study. Exclusion criteria in this study included subjects taking dyslipidemia in the last 3 months, smoking, suffering from infections, endocrine and genetic disorders, consuming hormones, and suffering from secondary obesity due to other diseases. The amount of diet consumption, including total calories, carbohydrates, and fat are obtained through food recall. The duration of transient physical activity and sleep duration were obtained through interviews using a questionnaire. Physical examination carried out on subjects, including measurements of body weight and height to calculate BMI. Body weight was measured using a digital scale (Seca, Germany) with an accuracy of 0.1 kg. Body weight was measured in subjects who wore light, barefoot clothes and other accessories. Height was measured with a stadiometer (Seca, Germany) with an accuracy of 0.1 cm in subjects standing upright without bare feet and hats. Weight and height measurements were carried out by trained staff and calibrated equipment. BMI was calculated by the formula weight (kg) divided by height (meters) squared ($kg/m^2$). BMI

was plotted into the 2000 CDC IMT curve according to age and sex. The diagnosis of obesity was obtained if BMI> P95 according to age and sex based on the Centers for Disease Control and Prevention (CDC) 2000 curve. Blood examinations were measured by taking blood from a vein and centrifugation was done to get serum. Lipid profile examination was carried out included total cholesterol, HDL, and LDL. LDL was measured using Cholestest®LDL. The HDL cholesterol examination was carried out using Cholestest®N HDL. Total cholesterol checks were performed using Pureauto® S CHO-N (Sekisui Medical Co., Ltd., Japan). HDL, LDL, and total cholesterol levels were measured in mg/dL units. TNF-α examination was performed using the ELISA method. Macronutrient levels, lipid profiles and inflammatory biomarkers were described with mean values and standard deviations. Correlations between variables were analyzed using Pearson correlation if the data were normally distributed and Spearman's rho if the data were not normally distributed. Statistical analysis was performed using SPSS with a p value less than 0.05 considered significant.

## Results

This study found 59 obese adolescents, consisting of 32 (54.2%) adolescent boys and 27 (45.8%) adolescent girls as in Table 1. Macronutrient had a negative correlation with adiponectin (Table 2.). There was no relationship between macronutrients, TNF-α, and lipid profile. Table 3 showed that there was no correlation between sedentary physical activity and TNF-α, adiponectin, and lipid profile. There was no relationship between sleep duration with adiponectin and lipid profile. Sleep duration had a negative correlation with TNF-α (p=0,017; r=-0,310).

## Discussion

Obesity is a chronic condition and is associated with inflammation [8]. Inflammation in obesity causes various health complications, such as diabetes mellitus, metabolic syndrome, cancer, and cardiovascular disease [3]. Control of inflammatory mediators through diet, lifestyle, and physical examination is one of the prevention of cardiovascular disease [9]. Adipose tissue produces adipocytokines which regulate energy metabolism, insulin sensitivity, cell proliferation, and inflammation [3]. Adiponectin is a part of adipokin that is produced by fat cells and has a protective effect on inflammation [10]. Adiponectin plays a role in maintaining metabolic hemostasis [11]. Adiponectin levels decrease in obesity [6]. An increase in visceral fat in obesity is associated with an increase in inflammatory biomarkers and a decrease in adiponectin [12]. A pro-inflammatory-rich diet is associated with an increased incidence of obesity [13], regain weight after weight loss in obesity [14], HDL cholesterole level, trygliseride, and blood pressure [15]. The Western diet is positively correlated with inflammatory biomarkers, while the vegetable and fruit diet is negatively correlated with inflammatory biomarkers [16]. The relationship of the inflammatory index on the diet with obesity in adolescents is still being studied because other studies provide meaningless results [17]. This study showed that adiponectin had a negative correlation with total calories and fat. Previous studies suggested that a low-calorie diet can increase adiponectin levels [18]. Calorie restriction improves metabolism due to upregulation of adiponectin levels [19]. Adiponectin has a role in fat metabolism. Increased adiponectin levels improve blood lipid levels [10]. Increased free fatty acids in the blood increase lipid profiles, such as total cholesterol and triglycerides [20].

In this study there was no relationship between adiponectin and carbohydrates. Previous studies suggested that adiponectemia can decrease due to increased carbohydrate intake. Adiponectemia can be influenced by anthropometric size and TNF-α [21]. Genetic variations can affect adiponectin levels [22]. Adiponectin gene single-nucleotide polymorphism affects serum adiponectin levels [23]. In this study, there was no correlation between macronutrients with TNF-α and lipid profiles (p> 0.05). Previous studies suggested that diet affects TNF-α levels. A high-carbohydrate diet increases TNF-α levels in the liver mice, but not with a high-fat diet [24]. Studies in obese adolescents suggest that TNF-α levels, and lipid profiles are influenced by single-nucleotide polymorphism (SNP) [23]. Physical exercise is one of the strategies to reduce obesity and improve body weight [25]. In this study no correlation was found between sedentary physical activity and TNF-α, adiponectin, and lipid profile (p> 0.05). Physical exercise can improve metabolism due to upregulation of adiponectin levels or activation of adiponectin receptors [26]. Previous studies suggested that physical exercise can increase adiponectin levels [18]. Physical exercise can affect adiponectin levels, but depending on study population, training intensity, and exercise type [27]. In obesity can occur sleep disorder. Sleep has a role in neuroendocrine function and plays a role in glucose metabolism, increasing glucose tolerance, cortisol and ghrelin levels, and decreasing leptin levels [28]. Sleep duration had a negative correlation with TNF-α (p = 0.017; r = -0.310). Previous studies have suggested that lack of sleep increases inflammatory biomarkers, such as IL-6 [29], TNF-α, and CRP [30]. However, another study stated that sleep duration> 8 hours increases marker inflammation and is not related to quality of sleep [31]. This research has several weaknesses. First, the number of subjects was limited. Second, measurements of inflammatory biomarkers and lipid profiles were not carried out serially. Further studies with a greater number of subjects and serial inflammatory biomarker measurements are needed to analyze the relationship of macronutrient, inflammatory biomarkers, and lipid profiles in obese adolescents in developing countries.

## Conclusion

Macronutrient intake and lifestyle can be used as an early detection of inflammatory biomarker abnormalities in obese adolescents.

**What is known about this topic**

- Adiponectin has a negative correlation with total calories and fat;
- Sleep duration has a negative correlation with TNF-α.

**What this study adds**

- Macronutrient intake can be used as an early detection of inflammatory biomarker abnormalities in obese adolescents;
- Lifestyle parameter can be used as an early detection of inflammatory biomarker abnormalities in obese adolescents.

## Competing interests

The authors declare no competing interests.

## Authors' contributions

Nur Aisiyah Widjaja, Rendi Aji Prihaningtyas, Roedi Irawan, Meta Herdiana Hanindita: conceived and design analysis, collect the data, contributed data and analysis tool, performed the analysis, drafting and writing the article. Retno Handajani, IDG Ugrasena: contributed data analysis. All the authors have read and agreed to the final manuscript.

## Acknowledgments

The authors thank the Director and staff of Dr Soetomo General Hospital, Surabaya for supporting this study.

## Tables

**Table 1**: subject characteristics

**Table 2**: correlation between diet and TNF-α, adiponectine, and lipid profile

**Table 3**: correlation between physical activity and TNF-α, adiponectine, and lipid profile

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| **Table 1**: subject characteristics | |
|---|---|
| **Variable** | **n (%)** |
| Sex | |
| Male | 32 (54.2) |
| Female | 27 (45.8) |
| | Mean ± SD |
| Weight (kg) | 80.77 ± 13.35 |
| Height (cm) | 158.76 ± 7.13 |
| Body Mass Index (kg/m2) | 31.99 ± 3.67 |
| TNF-α (ng/l) | 147.17 ± 46.16 |
| Adiponectine (ng/ml) | 7838.80 ± 3808.19 |
| Total Cholesterole (mg/dl) | 176.13 ± 32.87 |
| LDL (mg/dl) | 117.81 ± 27.70 |
| HDL (mg/dl) | 44.42 ± 7.73 |
| Triglyseride (mg/dl) | 118.12 ± 63.74 |

| **Table 2:** correlation between diet and TNF-α, adiponectine, and lipid profile | | | | | | |
|---|---|---|---|---|---|---|
| | **Adiponectine** | **TNF-α** | **LDL** | **HDL** | **Total Cholesterole** | **Triglyseride** |
| Calory | | | | | | |
| r | -0.287 | -0.090 | -0.029 | -0.083 | 0.007 | 0.158 |
| p | 0.028* | 0.496 | 0.828 | 0.531 | 0.957 | 0.231 |
| Fat | | | | | | |
| r | -0.270 | -0.033 | -0.042 | -0.201 | -0.015 | 0.202 |
| p | 0.039* | 0.807 | 0.750 | 0.126 | 0.911 | 0.124 |

| **Table 3**: correlation between physical activity and TNF-α, adiponectine, and lipid profile | | | | | | |
|---|---|---|---|---|---|---|
| | **Adiponectine** | **TNF-α** | **LDL** | **HDL** | **Total Cholesterole** | **Trigliseride** |
| Sleep duration | | | | | | |
| r | 0.059 | -0.310 | -0.019 | -0.145 | -0.058 | -0.015 |
| p | 0.657 | 0.017* | 0.889 | 0.274 | 0.662 | 0.912 |
| Sedentary Activity Duration | | | | | | |
| r | -0.087 | 0.041 | 0.091 | -0.043 | 0.077 | 0.213 |
| p | 0.510 | 0.758 | 0.493 | 0.746 | 0.560 | 0.106 |