

Insulin Resistance, Visceral Fat, and Vitamin D in Overweight and Obesity Adolescents

Rahma Labatjo^{1,*}, Imran Tumenggung¹, Agus Hendra Al Rahmad²

¹Department of Nutrition, Gorontalo Polytechnic of Health, Indonesia

²Department of Nutrition, Aceh Polytechnic of Health, Indonesia

Received December 6, 2022; Revised April 27, 2023; Accepted June 9, 2023

Cite This Paper in the Following Citation Styles

(a): [1] Rahma Labatjo, Imran Tumenggung, Agus Hendra Al Rahmad, "Insulin Resistance, Visceral Fat, and Vitamin D in Overweight and Obesity Adolescents," *Universal Journal of Public Health*, Vol. 11, No. 4, pp. 463 - 471, 2023. DOI: 10.13189/ujph.2023.110411.

(b): Rahma Labatjo, Imran Tumenggung, Agus Hendra Al Rahmad (2023). *Insulin Resistance, Visceral Fat, and Vitamin D in Overweight and Obesity Adolescents*. *Universal Journal of Public Health*, 11(4), 463 - 471. DOI: 10.13189/ujph.2023.110411.

Copyright©2023 by authors, all rights reserved. Authors agree that this article remains permanently open access under the terms of the Creative Commons Attribution License 4.0 International License

Abstract Overweight and obesity are the causes of health problems. This condition is detrimental to growth in adolescents. Furthermore, being overweight and obese can affect insulin performance, visceral fat levels, and vitamin D status in the body. This study aimed to investigate the relationship between overweight in adolescents and insulin resistance, visceral fat, and vitamin D. This study was cross-sectional. All samples were purposively selected. Types of data in the form of anthropometric data (age, weight, and height) include fasting glucose levels, TyG index value, visceral fat composition, and vitamin D. The study was conducted in Gorontalo city, with the research sample being teenagers with overweight problems. Data were analyzed using univariate analysis in the form of the mean value. Meanwhile, the multivariate analysis employed the Spearman Rank correlation. The results showed that body weight was significantly correlated with visceral fat content (p -value = 0.000), bone density (p -value = 0.007) and vitamin D (p -value = 0.000). Visceral fat content was correlated with vitamin D (p -value = 0.000) and insulin resistance (p -value = 0.013).

Keywords Overweight, Obesity, Insulin Resistance, Visceral Fat, Adolescent

accumulation that can harm health. Such a concerning health problem is related to non-communicable diseases (NCDs), e.g., cardiovascular diseases (CVDs) including heart disease and stroke, type 2 diabetes, certain types of cancers of the digestive tract and reproductive system, and disorders of the bones and joints such as osteoarthritis [1, 2]. Obesity in adolescence is detrimental to growth. Youth growth is crucial in preparing for healthier growth later in adulthood [3]. In other words, weight problems during the growth period will be fatal to an adult's health.

The prevalence of overweight and obesity problems continues to increase globally. The World Health Organization (WHO) notes that more than 340 million children aged 5-19 years are overweight and obese [4]. Meanwhile, in Indonesia, based on the 2018 Basic Health Research (Riskesdas), the prevalence of overweight and obesity in the 13-15 year age group was 11.2% and 4.8%, respectively. In the 16-18 year age group, 9.5% were overweight, and 4.8% were obese [5]. Meanwhile, in Gorontalo, the prevalence of overweight and obesity in the 16-18 year age group was 8.6% and 3.8%, respectively. Riskesdas 2018 also noted that adolescents mostly experienced obesity in urban areas, accounting for 10.7% [5].

Insulin is a hormone that plays a role in glucose metabolism. It is produced by the beta cells of the pancreas [6]. Since the circulatory system of glucose in the blood requires insulin, it is released in response to rising levels of glucose in the bloodstream after a meal. In addition, insulin plays a role in fat metabolism [7]. When insulin is released, it signals adipose tissue (fat cells) to take up glucose and

1. Introduction

Being overweight or obese is a state of excess fat

fatty acids from the bloodstream [8, 9]. The glucose and fatty acids are then converted into triglycerides (a type of fat) and stored in the adipose tissue for later use as energy [10]. Insulin also inhibits the breakdown of stored fat (lipolysis), thereby promoting fat storage [11].

Furthermore, overweight and obesity can affect insulin performance. This is due to a significant relationship between the occurrence of inflammation and obesity. Insulin signalling in adipose and hepatocytes can be inhibited by inflammation by several mechanisms, resulting in insulin resistance [9]. Insulin resistance is a condition in which cells throughout the body do not respond to insulin as effectively as they should [12]. Insulin resistance occurs when the normal amount of insulin secreted by the pancreas is not enough to overcome the resistance of the cells to insulin's actions. This leads to an impaired ability of cells to take up glucose from the bloodstream, which can cause high blood sugar levels. Insulin resistance is negatively impactful on health. These disorders include impaired blood glucose circulation, CVDs, kidney disease, cancer, Alzheimer's disease, and visual impairment [9].

Visceral fat is a type of body fat that is stored deep within the abdominal cavity, surrounding and protecting vital organs such as the liver, pancreas, and intestines [13, 14]. Visceral fat is different from subcutaneous fat, which is the fat located directly beneath the skin [15]. Increased visceral fat levels correlate with the incidence of CVDs, increased levels of glucose and triglycerides, and decreased levels of High-Density Lipoprotein (HDL) [16]. Visceral fat can be measured using imaging techniques such as CT scans or MRI, or by measuring waist circumference. A waist circumference of 35 inches or more for women, or 40 inches or more for men, is considered a risk factor for the development of health problems associated with excess visceral fat [17]. Overweight and obesity affect visceral fat levels. Visceral fat levels in overweight and obese patients tend to be high [18]. Therefore, overweight and obese people are more likely to suffer from dangerous comorbidities like CVDs and NCDs.

Vitamin D is a critical nutrient that plays a significant role in several essential bodily functions. One of its primary functions is to help the body absorb calcium, which is necessary for healthy bones and teeth [19]. When vitamin D is consumed or produced in the skin through exposure to sunlight, it travels through the bloodstream to the intestines, where it helps the body absorb calcium from food [20]. This calcium is then used to build and maintain bones, regulate heart rhythm, and facilitate nerve function. Vitamin D deficiency in adolescents can result in osteomalacia, a condition where bones weaken [21]. In addition, inadequate levels of vitamin D can result in muscle weakness and muscle pain [22]. Overweight and obesity conditions can affect vitamin D levels. Overweight and obese patients tend to have vitamin D deficiency [23]. Thus, adolescents who are overweight and obese are at risk

for vitamin D deficiency and are at risk of suffering from comorbidities caused by these conditions.

It is necessary to conduct research that focuses on overweight and obesity in adolescents, given the increasing trend of overweight and obesity problems in adolescents and their negative consequences. An element of novelty in this study is a more specific investigation into the relationship between insulin resistance, vitamin D status, and visceral fat levels in adolescents with weight problems.

This research will be carried out in collaboration with partner institutions, which in this case are Poltekkes, and the Ministry of Health Aceh. Based on literature searches and the focus of lecturer research carried out by the Health Poltekkes of the Aceh Ministry of Health, research on obesity and overweight has been widely carried out. However, the variables studied did not include insulin resistance, visceral fat, and vitamin D in overweight and obese adolescents. Thus, the present research is expected to contribute positively to formulating obesity prevention programs in adolescents. This study examines the relationship between insulin resistance, visceral fat levels, and vitamin D status with overweight and obesity in adolescents.

2. Research Methods

This research used a cross-sectional study design. This research was conducted in Gorontalo City in August-November 2021. A blood sample examination was conducted at the Prodia Laboratory of Gorontalo City. The research sample was obtained using the minimum sample size formula for a cross-sectional study [24]. The total sample for this study is 22 samples. The sample is teenagers in Gorontalo City with the following inclusion and exclusion criteria.

2.1. Inclusion Criteria

- a. Physically and mentally healthy.
- b. Aged 16-18 years.
- c. Can communicate actively.
- d. Domiciled in Gorontalo City.
- e. Willing to be a research sample.
- f. Not suffering from NCDs and CVDs and their complications.

2.2. Exclusion Criteria

- a. Not in place at the time of data collection.
- b. Decided to withdraw at the time of data collection.

2.3. Data Collection

2.3.1. Anthropometric Data

Data on body weight (BB) were obtained using a digital weight scale with an accuracy of 0.1 kg. The height (TB) of

the sample was measured using a stadiometer with an accuracy of 0.1 cm. Data regarding the age of the sample was obtained from the records on the sample's Family Card (KK). Body Mass Index data is obtained using the following formula.

$$\text{Body Mass Index} = \frac{BB \text{ (kg)}}{TB^2 \text{ (m)}}$$

2.3.2. Weight Classification

Classification of sample weight is grouped into three categories based on the BMI calculation value. The nutritional status categories of the sample are as follows [25].

- Not overweight and obese, if the BMI value = 18.5 – 23 kg/m²
- Overweight, if the BMI value = >23 – 27.5 kg/m²
- Obesity, if the BMI value = >27.5 kg/m²

2.3.3. Insulin Resistance Data

Insulin resistance was measured using the TyG index method. TyG index is an early indicator of insulin resistance in groups at risk for type 2 diabetes [26]. The formulation of the TyG index formula is as follows.

$$= \frac{\ln(\text{fasting Triglyceride} \times \text{fasting glucose})}{2}$$

Description:

Fasting Triglyceride = Fasting Triglyceride value (mg/dl)

Fasting glucose = fasting blood sugar (mg/dl)

ln = logarithm

Insulin resistance based on the value of the TyG index was at the *cut-off point* of 4.49. The TyG index value of 4.49 and above may indicate that insulin resistance occurs [27]. Thus, insulin resistance data can be categorized into the following.

- Resistant if TyG index value ≥ 4.49
- Not resistant if the value of TyG index < 4.49

2.3.4. Data on Visceral Fat Content

A popular technique for assessing Body Composition (BC) is bioimpedance analysis (BIA), which examines the electrical characteristics of body tissue and calculates BC parameters including total body water (TBW) and FFM BC parameters. In both clinical and nonclinical contexts, BIA is a reliable, inexpensive, and non-invasive approach for assessing BC. The fundamental tenet of the BIA approach is that the BC characteristics determine how quickly a low-voltage electric current travels through the body. The chemical makeup of Fat-Free Mass, which consists of water, proteins, glycogen, and minerals, as well as the significant inter- and intraindividual variability brought on by changes in FFM with development, maturation, aging, and disease states, pose limits to this research [28]. Visceral fat content in this research was measured by the *Bioelectric Impedance Analysis* (BIA) method.

2.3.5. Vitamin D Status Data

Vitamin D levels in blood plasma were examined using the *Chemiluminescence Immunoassays* (CLIA) method. Vitamin D is connected to an isoluminol derivative, and it is specifically an antibody to vitamin D that is utilized to coat magnetic particles (solid phase). 25-hydroxyvitamin D separates from its binding protein during incubation and competes with labelled vitamin D for antibody binding sites. Following incubation, a wash cycle is used to remove the unbound material. The starter reagents are then introduced, and a flash chemiluminescent reaction is subsequently started. The amount of 25-hydroxyvitamin D contained in samples is inversely proportional to the light signal, which is quantified by a photomultiplier as relative light units [29]

The category of vitamin D status is classified according to the following categories: (1) Vitamin D status is normal if the serum 25-hydroxy vitamin D (25(OH)D) is ≥ 30 – 100 ng/ml; (2) Vitamin D deficiency status if serum 25-hydroxy vitamin D (25(OH)D) < 30 ng/ml [30].

2.3.6. Data Analysis

Data analysis was performed employing univariate analysis using the mean, minimum and maximum values for the variables of insulin levels, visceral fat, and vitamin D status in each sample group. The results are presented in the form of a frequency distribution table. Correlation between insulin resistance, visceral fat levels, vitamin D status, and body weight using the Spearman Rank Correlation test.

3. Results and Discussion

3.1. Research Result

Sample Characteristics: The information presented in Table 1 provides details about the characteristics of the research sample, which are important to understand the demographic and physical characteristics of the participants in the study. The table displays the mean (average) values for each of the four characteristics measured (age, weight, height, and BMI) as well as the minimum and maximum values for each of these characteristics, indicating the range of values among the participants.

Table 1. Sample Characteristics

Sample Characteristics	Minimum Value	Average value	Maximum Value
Age (years)	16	17.59	18
Body weight (kg)	38,90	55.38	101.40
Height (cm)	138	151.73	168
Body Mass Index (kg/m ²)	15.01	23.89	35.93
Bone density (kg)	1.50	2.08	3.50
Visceral fat (%)	9.10	31.78	48.90

Source: primary data

Based on Table 1, the average age of the sample was 17.59 years, indicating that the majority of participants were teenagers. The minimum age was 16 years, and the maximum age was 18 years, indicating that the study participants were limited to a specific age range. The average body weight was 55.38 kg, with a minimum weight of 38.90 kg and a maximum weight of 101.40 kg, indicating the range of body weights among the participants.

The average height of the participants was 151.73 cm, with a range of 138 cm to 168 cm, which indicates the variability in height among the participants. Finally, the average BMI was 23.89 kg/m², which falls within the healthy weight range, with a range of 15.01 kg/m² to 35.93 kg/m², indicating that some participants were underweight or overweight/obese. Furthermore, laboratory examination data are presented in the following Table 2.

Table 2. Laboratory Examination

Check Variable	Minimum Value	Average value	Maximum Value
Fasting glucose (mg/dl)	64	80.14	106
Triglycerides (mg/dl)	38	102.55	204
Vitamin D (ng/ml)	8.70	18.47	31.30
TyG index	3.96	4.46	4.89

Source: primary data

Table 2 presents the findings of the study regarding the various measurements taken. The average value of fasting glucose levels was 80.14 mg/dl, with a minimum value of 64 mg/dl and a maximum value of 106 mg/dl. Triglyceride levels had an average value of 102.55 mg/dl, with a minimum value of 38 mg/dl and a maximum value of 204 mg/dl. The average blood level of vitamin D was 18.47 ng/ml, with a range of 8.70 ng/ml to 31.30 ng/ml. The TyG index, which is a measure of insulin resistance, had an average value of 4.46, with a range of 3.96 to 4.89.

To further explain, fasting glucose levels are used to measure the amount of glucose in the blood after a period of fasting. Triglycerides are a type of fat found in the blood that can increase the risk of heart disease when elevated. Vitamin D is an essential vitamin that helps the body absorb calcium and maintain strong bones. The TyG index is a relatively new measure that combines fasting glucose and triglyceride levels to estimate insulin resistance, which is a precursor to type 2 diabetes.

3.2. Correlation between Insulin Resistance, Visceral Fat Level, Vitamin D Status, and Body Weight

The relationship between the variables of insulin resistance, visceral fat content, vitamin D status, and body weight was identified using the Spearman Rank correlation test. The test results can be seen in the following Table 3.

Table 3. Spearman's Rank Correlation Test Results

		Weight	Bone Density	Visceral Fat Content	Vitamin D	Insulin Resistance	
<i>Spearman's rho</i>	Weight	<i>Correlation Coefficient</i>	1,000	.411 *	.871 **	.797 **	-.357
		<i>Sig. (2-tailed)</i>	.	.007	.000	.000	.103
		N	22	22	22	22	22
	Bone Density	<i>Correlation Coefficient</i>	.411 *	1,000	-.335	.182	-.262
		<i>Sig. (2-tailed)</i>	.007	.	.127	.419	.238
		N	22	22	22	22	22
	Visceral Fat Content	<i>Correlation Coefficient</i>	.871 **	-.335	1,000	.710 **	-.520 *
		<i>Sig. (2-tailed)</i>	.000	.127	.	.000	.013
		N	22	22	22	22	22
	Vitamin D	<i>Correlation Coefficient</i>	.797 **	.182	.710 **	1,000	-.128
		<i>Sig. (2-tailed)</i>	.000	.419	.000	.	.570
		N	22	22	22	22	22
	Insulin Resistance	<i>Correlation Coefficient</i>	-.357	-.262	-.520 *	-.128	1,000
		<i>Sig. (2-tailed)</i>	.103	.238	.013	.570	.
		N	22	22	22	22	22
** . Correlation is significant at the 0.01 level (2-tailed).							
* . Correlation is significant at the 0.05 level (2-tailed).							

Source: primary data

Based on the results of the *Spearman Rank correlation* test, body weight was significantly correlated with levels of visceral fat (p -value = 0.000), bone density (p -value = 0.007), and vitamin D (p -value = 0.000). Visceral fat content was correlated with vitamin D (p -value = 0.000) and insulin resistance (p -value = 0.013). The results of the Spearman Rank correlation test showed that there were significant correlations between various variables in the study. Body weight was found to be significantly correlated with levels of visceral fat, bone density, and vitamin D. This suggests that as body weight increases, so do the levels of visceral fat, bone density, and vitamin D. Meanwhile, visceral fat content was found to be correlated with vitamin D and insulin resistance, indicating that as visceral fat levels increase, so does the risk of vitamin D deficiency and insulin resistance. The p -values for these correlations were all less than 0.05, indicating that the results were statistically significant. Overall, these findings provide important insights into the relationships between various factors that can impact health outcomes in the population studied.

4. Discussion

The age range of the research sample ranged from 16-18 years. This age group is included in the puberty period, which is a period when the growth of long bones (growth spurt) reaches its peak [31]. From childhood to adolescence, about 80% of bone growth occurs [31, 32]. Maximum bone mass growth takes place during adolescence. Changes in bone structure mostly occur at this phase in the form of physical growth, resulting in rapid osteogenesis and bone remodelling [33]. Because the process of bone mass formation decreases with age, if the maximum bone mass is low in adolescence, the risk of fracture or osteoporosis at a young age and in later years may increase.

Body weight in adolescence is associated with nutritional status based on BMI values. In this study, the average BMI of the sample was 23.89 kg/mg². In other words, the average teenager in the study sample is in the overweight category. Body weight can have a positive effect on bone density where every weight gain will trigger the process of bone formation, increase the secretion of the hormone estrogen by fat tissue, and prevent osteoclast osteolysis [34]. However, this is not the case with fat content. Increased fat mass is negatively correlated with bone density. The increase in fat mass will cause a decrease in bone mass [35]. On that ground, fat mass is a predictor of inhibiting bone mass growth.

The fasting glucose variable in this study sample averaged 80.14 mg/dl. A normal fasting glucose level in the > 12-year age group is <100 mg/dl [36]. Thus, the average fasting glucose of the study sample was categorized within normal limits. It is important to keep glucose levels within normal levels. In normal people or those without impaired glucose metabolism, normal

glucose levels can provide benefits, such as preventing weight gain, helping to achieve ideal weight, reducing the risk of insulin resistance and type 2 diabetes, and reducing stress and inflammatory hormone [37].

The triglyceride levels of the research sample had an average value of 102.55 mg/dl. A normal triglyceride level is <150 mg/dl [38]. Thus, the triglyceride levels of the research sample were included in the normal category. Triglycerides are fat found in the blood and stored in fat cells. It will be broken down by hormones and used as energy [38]. High triglycerides can cause hardening of the arteries or thickening of the walls of the arteries (arteriosclerosis), which increases the risk of stroke, heart attack, and heart disease. High triglycerides can also cause acute inflammation of the pancreas (pancreatitis) [38]. High triglycerides are often signs of other conditions that increase the likelihood of heart disease, stroke, obesity, and metabolic syndrome (including excessive fat around the waist, high blood pressure, high triglycerides, high blood sugar, and abnormal cholesterol levels). High triglycerides can also be a symptom of: type 2 diabetes or prediabetes; metabolic syndrome, which is a condition when high blood pressure, obesity and high blood sugar coexist, which in turn increases the risk of heart disease; low levels of thyroid hormones (hypothyroidism); and certain rare genetic conditions impacting the way the human body converts fat into energy [38].

The average vitamin D level of the study sample was 18.47 ng/ml. Normal serum vitamin D levels are \geq 30-100 ng/ml [30]. For this reason, the vitamin D serum of the study sample was categorized as low. Vitamin D is a nutrient we eat and a hormone made by our bodies. Vitamin D is a fat-soluble vitamin that has long been recognized for its function in helping the body absorb and retain calcium and phosphorus. Calcium and phosphorus are essential for building bones. Laboratory studies show that vitamin D can reduce the growth of cancer cells, help control infection and reduce inflammation. Many body organs and tissues have receptors for vitamin D, which suggest their paramount roles in addition to bone health, and scientists are actively investigating its possible functions [39, 40]. Vitamin D deficiency can occur from inadequate intake of food, poor absorption, or a metabolic need for higher amounts. Such a deficiency may develop if a person does not consume enough vitamin D and does not receive sufficient exposure to ultraviolet light for a long time. Similarly, people who cannot tolerate or do not eat dairy, eggs, and fish, such as those who are lactose intolerant or who follow a vegan diet, are at higher risk of deficiency [40].

Other groups at high risk of vitamin D deficiency include the following: (1) People with inflammatory bowel disease (ulcerative colitis, Crohn's disease) or other conditions that interfere with the normal digestion of fat. Vitamin D is a fat-soluble vitamin that depends on the ability of the intestines to absorb dietary fat; (2) people who are obese tend to have lower blood levels of vitamin D.

Vitamin D accumulates in excess fatty tissue but is not readily available for use by the body when needed. In contrast, blood levels of vitamin D increase when obese people lose weight; (3) people who have had gastric *bypass* surgery usually remove the small intestine's upper part where vitamin D is absorbed [39-42].

Meanwhile, prolonged vitamin D deficiency conditions include the following conditions: (1) Rickets: a condition in infants and children of soft bones and bone deformities caused by the failure of bone tissue to harden; (2) Osteomalacia: a condition in adults with weak, soft bones that can be treated with supplementation. This condition contrasts with osteoporosis, where the bones are porous and brittle, and the condition is irreversible [39].

In this study, the average adolescent sample was in the overweight or overweight category. This category is based on BMI scores. BMI is a reasonably strong predictor in terms of determining the status of overweight and obesity [43]. Although the correlation between BMI and visceral fat is not as strong as that between fat mass and visceral fat [44], BMI can be used as a quick and safe indicator to determine obesity status in adolescents. Such is because this method only uses weight and height measurements. Based on the correlation test, the body weight of the study sample was positively correlated with bone density. The average bone density of the sample is categorized as normal, and body weight is in the overweight category. It is concluded that weight gain can increase bone density or bone mass in adolescents. Higher bone density and bone minerals in obese people are associated with many factors [45]. Such a condition is due to significantly increased mechanical load on bone and an altered hormonal environment; while higher serum adipokine levels are associated with obesity [45]. Other genetic and environmental factors such as smoking, dietary intake, and lifestyle also play independent roles in influencing bone mass in obesity [45].

It is different from vitamin D. Compared with leaner individuals, in obese people, serum levels of 25-hydroxy vitamin D (25 (OH) D) are lower [45]. Serum 25-hydroxy vitamin D (25(OH)D) has a specific action on bone. Lower serum 25-hydroxy vitamin D (25(OH)D) levels in obese individuals may be due to storage in adipose tissue. Low serum 25-hydroxy vitamin D (25 (OH) D) and excess fat accumulation harm each other, which is caused by excessive metabolic processes and enzymatic disorders. This condition causes a decline in the activity of the alpha-hydroxylase enzyme, i.e., the main enzyme in the process of calciferol biotransformation in the fatty liver [46]. Vitamin D deficiency is also associated with insulin resistance. The correlation between these two variables is negative. Vitamin D deficiency will increase the risk of insulin resistance. This will be more influential on people with obesity. In obesity, vitamin D affects insulin secretion, tissue sensitivity to insulin, and systemic inflammation. The direct and paracrine effects of vitamin D trigger vitamin D receptor (VDR) activation in pancreatic beta

cells, CYP27B1 expression and local synthesis of 1,25(OH)₂D [46].

Visceral fat is associated with insulin resistance. In obese patients with high visceral fat accumulation, insulin performance tends to experience resistance [47]. Visceral fat secretes a *retinol-binding protein 4* (RBP4), which has been shown to increase insulin resistance [47]. Adipose cells and adipocytes are the determining factors in the pathogenesis of insulin resistance which is correlated with obesity. Adipocyte hypertrophic dysfunction, often found in visceral and subcutaneous fat, is highly lipolytic in nature, thereby increasing the release of free fatty acids (FFA) [48]. Two main hypotheses explaining how increased FFA may be associated with central obesity or visceral fat and insulin resistance are the portal hypothesis and the *spillover* hypothesis (or ectopic fat). In accordance with the portal theory, increased visceral fat leads to increased delivery of FFA to the liver via its portal vein drainage. As a result, it increases the problem of insulin resistance in the liver, thereby promoting glucose production. Increased portal FFA, an inflammatory cytokine released by visceral fat into the portal vein, also causes insulin resistance [48].

Following the spillover hypothesis or the 'overflow' hypothesis, in the face of a positive energy balance, a reduced or limited capacity of adipose tissue (especially the peripheral subcutaneous compartment) will lead to an overflow of FFA to the visceral fat compartment and non-adipose tissue (e.g., liver, muscle, pancreas, kidney, bone). Due to the limited ability of non-adipose tissue to oxidize and/or store FFA, ectopic accumulation of FFA and/or its metabolically active derivatives will cause insulin resistance and cell lipotoxicity apoptosis (cell death process) that disrupt organ function [48]. Hypertrophied adipocytes can cause local hypoxia, which promotes endoplasmic reticulum (ER) stress, adipocyte death, and macrophage infiltration. The latter event increases the secretion of inflammatory cytokines, such as TNF- and interleukin (IL)-6 and monocyte *chemoattractant* protein (MCP)-1, leading to low-grade local and systemic inflammation and consequently impaired insulin signaling [48].

5. Conclusions

Based on the results of the *Spearman Rank* correlation test, body weight was significantly correlated with levels of visceral fat, bone density, and vitamin D. The average nutritional status of adolescents based on BMI values is included in the overweight category and did not experience insulin resistance. The study is significant as it sheds light on the relationship between overweight in adolescents and various health-related parameters such as insulin resistance, visceral fat, and vitamin D status. The findings of this research are relevant to the field of public health, particularly in addressing the health problems associated

with overweight and obesity in young adults. The study highlights the need for interventions aimed at improving the nutritional status of adolescents and addressing issues such as vitamin D deficiency, insulin resistance, and visceral fat accumulation.

The use of a cross-sectional design and purposive sampling method in this study may limit the generalizability of the findings. Thus, further research using longitudinal designs is needed to establish causal relationships and determine the impact of interventions over time. Nonetheless, this research serves as an important contribution to the existing body of knowledge on the health implications of being overweight or obese in adolescence. It provides useful information that can help health professionals develop effective strategies for managing and preventing obesity-related health problems in this population. Due to the complexity of the factors that influence the incidence of overweight and obesity in adolescents, it is also necessary to design a prevention and treatment program for these problems that does not only focus on total calorie intake and physical activity. Micronutrient variables such as vitamin D also need to focus on nutritional management in adolescents.

REFERENCES

- [1] Darweish S, "Obesity in children and teenagers," *Primary Health Care*, 22(7), 28-31, 2012. doi: 10.7748/phc2012.09.22.7.28.c9267
- [2] Swinburn BA, Kraak VI, Allender S, Atkins VJ, Baker PI, Bogard JR, Brinsden H, Calvillo A, De Schutter O, Devarajan R, Ezzati M, Friel S, Goenka S, Hammond RA, Hastings G, Hawkes C, Herrero M, Hovmand PS, Howden M, Jaacks LM, Kapetanaki AB, Kasman M, Kuhnlein HV, Kumanyika SK, Larijani B, Lobstein T, Long MW, Matsudo VKR, Mills SDH, Morgan G, Morshed A, Nece PM, Pan A, Patterson DW, Sacks G, Shekar M, Simmons GL, Smit W, Tootee A, Vandevijvere S, Waterlander WE, Wolfenden L, Dietz WH, "The global syndemic of obesity, undernutrition, and climate change: The Lancet Commission report," *Lancet*, 393(10173), 791-846, 2019. doi: 10.1016/S0140-6736(18)32822-8.
- [3] Zalewska M, Maciorkowska E. Selected nutritional habits of teenagers associated with overweight and obesity. *PeerJ*, 5(e3681), 2017. doi: 10.7717/peerj.3681.
- [4] World Health Organization, "Obesity and overweight", WHO, <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight> (accessed Jan. 2, 2020).
- [5] Agency for Health Research and Development, "National Report RISKESDAS 2018", Riskesdas, http://labmandat.litbang.depkes.go.id/images/download/laporan/RKD/2018/Laporan_Nasional_RKD2018_FINAL.pdf (accessed: Oct. 28, 2019).
- [6] Kumar V, Abbas AK, "Robbins basic pathology," Elsevier Saunders, 2014.
- [7] Petersen MC, Shulman GI, "Mechanisms of insulin action and insulin resistance," *Physiol Rev.*, 98(4), 2133-2223, 2018. doi: 10.1152/physrev.00063.2017. PMID: 30067154.
- [8] Kadowaki T, Kadowaki H, "TCF7L2 and glucose metabolism: time to look beyond the pancreas," *Diabetes, Obesity and Metabolism*, 17(3), 221-228, 2015. doi: 10.1111/dom.12398
- [9] Ye J, "Mechanisms of insulin resistance in obesity," *Front Med*, 7(1), 2013. doi: 10.1007/s11684-013-0262-6.
- [10] Perry RJ, Shulman GI, "Mechanisms of insulin resistance in obesity", In *Handbook of obesity: epidemiology, etiology, and physiopathology*, CRC Press, 2020, pp. 199-215. doi: 10.1201/9780429298842-11
- [11] Boucher J, Kleinridders A, Kahn CR, "Insulin receptor signaling in normal and insulin-resistant states," *Cold Spring Harbor Perspectives in Biology*, 14(1), a040766, 2022. doi: 10.1101/cshperspect.a040766
- [12] Li L, Feng Q, Ye J, Li Y, "Insulin resistance in skeletal muscle: An updated review," *Journal of Cellular Physiology*, 236(2), 799-814, 2021. doi: 10.1002/jcp.29860.
- [13] Dimitriadis G, Mitrou P, Lambadiari V, Maratou E, Raptis SA, "Insulin effects in muscle and adipose tissue," *Diabetes Res Clin Pract*, 93(Suppl 1S5), 2-9, 2011. doi: 10.1016/S0168-8227(11)70014-6. PMID: 21864752.
- [14] Després JP, "Body fat distribution and risk of cardiovascular disease: An update," *Circulation*, 126(10), 1301-1313, 2013. doi: 10.1161/CIRCULATIONAHA.111.067264
- [15] Harvey I, Boudreau A, Stephens JM, "Adipose tissue in health and disease," *Open Biol*, 10(12), 200291, 2020. doi: 10.1098/rsob.200291. PMCID: PMC7776562.
- [16] Ozato N, Saito T, Yamamoto H, Ando K, Miyamoto Y, Yoshimura E, Tsujimoto T, "Association between nutrients and visceral fat in healthy Japanese adults: A 2-year longitudinal study," *Nutrients*, 11(11), 2698, 2019. doi: 10.3390/nu11112698.
- [17] Gómez-Ambrosi J, Silva C, Galofré JC, Escalada J, Santos S, Millán D, Vila N, Ibañez P, Gil MJ, Valentí V, Rotellar F, Ramírez B, Salvador J, Frühbeck G, "Body mass index classification misses subjects with increased cardiometabolic risk factors related to elevated adiposity," *Int J Obes (Lond)*, 36(2), 286-94, 2012. doi: 10.1038/ijo.2011.100. PMID: 21587201.
- [18] Elffers TW, de Mutsert R, Lamb HJ, de Roos A, "Body fat distribution, in particular visceral fat, is associated with cardiometabolic risk factors in obese women," *The American Journal of Clinical Nutrition*, 105(1), 44-51, 2017. doi: 10.3945/ajcn.116.140558.
- [19] Migliaccio S, Greco EA, Aversa A, Lenzi A, "Obesity and hypovitaminosis D: causality or casualty?" *International journal of obesity supplements*, 9(1), 1-17, 2019. doi: 10.1038/s41367-019-0005-2.
- [20] Nair R, Maseeh A, "Vitamin D: The 'sunshine' vitamin," *J Pharmacol Pharmacother*, 3(2), 118-26, 2012. doi: 10.4103/0976-500X.95506. PMCID: PMC3356951.
- [21] Winzenberg T, Jones G, "Vitamin D and bone health in childhood and adolescence," *Calcif Tissue Int.*, 92(2), 140-50, 2013. doi: 10.1007/s00223-012-9615-4. PMID:

- 22710658.
- [22] Mansournia MA, Ostadmohammadi V, Doosti-Irani A, Ghayour-Mobarhan M, Ferns G, Akbari H, Ghaderi A, Talari HR, Asemi Z, "The effects of vitamin d supplementation on biomarkers of inflammation and oxidative stress in diabetic patients: A systematic review and meta-analysis of randomized controlled trials, *Horm Metab Res.*, 50(6), 429-440, 2018. doi: 10.1055/a-0630-1303. PMID: 29883970.
- [23] Pereira-Santos M, Costa PRF, Assis AMO, Santos CAS, Santos DB, "Obesity and vitamin D deficiency: a systematic review and meta-analysis," *Obesity Reviews: An Official Journal of the International Association for the Study of Obesity*, 16(4), 341-349, 2015. doi: 10.1111/obr.12239
- [24] Lemeshow S, David W. "Sampling of populations: methods and applications." Wiley, 1997.
- [25] Nishida, C, "Appropriate body mass index for asians populations and its implications for policy and intervention strategies," *Lancet*, 363, 157-63, 2004.
- [26] Navarro-González D, Sánchez-Íñigo L, Pastrana-Delgado J, Fernández-Montero A, Martínez JA, "Triglyceride-glucose index (TyG index) in comparison with fasting plasma glucose improved diabetes prediction in patients with normal fasting glucose: The Mollerussa cohort study," *The Journal of Clinical Endocrinology & Metabolism*, 101(3), 1343-1350, 2016. doi: 10.1210/jc.2016-3385.
- [27] Salazar J, Bermúdez V, Ramos E, Silva E, Carvajal A, Mendoza V, Córdova M, Maiz E, Rojas J, Chávez-Castillo M, Arraiz N, Camacho N, Molina-Guarneros J, Fernández M, Olivar LC, Acosta H, Pineda J, Añez R, Mosquera J, "Optimal cutoff for the evaluation of insulin resistance through triglyceride-glucose index: A cross-sectional study in a Venezuelan population," *Annals of Medicine and Surgery*, 20, 15-20, 2017. doi: 10.1016/j.amsu.2017.05.006
- [28] Marra M, Sammarco R, De Lorenzo A, Iellamo F, Siervo M, Pietrobelli A, "Assessment of body composition in health and disease using bioelectrical impedance analysis (BIA) and dual energy X-ray absorptiometry (DXA): A critical overview, *Contrast Media & Molecular Imaging*, 3548284, 2019. doi: 10.1155/2019/3548284.
- [29] Snellman G, Melhus H, Gedeberg R, Olofsson S, Wernroth L, Sundström J. Determining vitamin D status: A comparison between commercially available assays. *PLoS one*, 5(7), e11555, 2010. <https://doi.org/10.1371/journal.pone.0011555>
- [30] Hartant YB, Setiati S, Sutrisna B, Nafrialdi N, Ismail THT, "Vitamin D deficiency in hospitalized chronic heart failure patients, *Indonesian Journal of Cardiology*, 39(4), 263-267, 2018.
- [31] Chang CY, Arasu K, Wong SY, et al. Factors associated with bone health status of Malaysian pre-adolescent children in the PREBONE-Kids Study. *BMC Pediatr* 21, 382, 2021. <https://doi.org/10.1186/s12887-021-02842-6>
- [32] Kelley JC, Crabtree N, Zemel BS, "Bone Density in the Obese Child: Clinical Considerations and Diagnostic Challenges," *Calcified Tissue International*, 100(5), 514-527, 2017. doi: 10.1007/S00223-016-0233-4.
- [33] Kim A, Lee JE, Yoon H, Kim JK, Cho S, "Bone Mineral Density of Femur and Lumbar and the Relation between Fat Mass and Lean Mass of Adolescents: Based on Korea National Health and Nutrition Examination Survey (KNHNES) from 2008 to 2011," *International journal of environmental research and public health*, 17(12), 4471, 2020. doi:10.3390/ijerph17124471
- [34] Kim A, Choi SW, "Comparative analysis on change of body composition of middle school girls according to bone mineral density classification," *Korea Journal of Sport Science*, 25, 137-147, 2016.
- [35] Park JH, Hong JW, Kim KH, Lee HJ, Shin DH, Cho NH, "The association between fat and lean mass and bone mineral density: the Healthy Twin Study," *Bone*, 50(4), 1006-1011, 2012. doi:10.1016/J.BONE.2012.01.015.
- [36] American Diabetes Association. (2021). Standards of medical care in diabetes--2021. *Diabetes Care*, 44(Suppl. 1), S1-S232. doi: 10.2337/dc21-S001.
- [37] Röder PV, Wu B, Liu Y, Han W, "Pancreatic regulation of glucose homeostasis," *Exp Mol Med*, 48(3), e219, 2016. doi: 10.1038/emm.2016.6. PMID: PMC4892884.
- [38] Mayo Clinic, "Triglycerides: Why do they matter? - Mayo Clinic," <https://www.mayoclinic.org/diseases-conditions/high-blood-cholesterol/in-depth/triglycerides/art-20048186> (Accessed: 28 November 2021).
- [39] Ross AC, Manson JE, Abrams SA, Aloia JF, Brannon PM, Clinton SK, ... Shapses SA, "Dietary Reference Intakes for Calcium and Vitamin D. Washington," National Academies Press (US), 2011. doi: 10.17226/13050.
- [40] Avenell A, Mak JCS, O'Connell D, "Vitamin D and vitamin D analogues for preventing fractures in post-menopausal women and older men," *Cochrane Database of Systematic Reviews*. John Wiley and Sons Ltd, 2014.
- [41] Cauley JA, LaCroix AZ, Wu L, Horwitz M, Danielson ME, Bauer DC, Lee JS, Jackson RD, Robbins JA, Wu C, Stamm E, Wactawski-Wende J, Robbins J, Heyse S, Cummings SR, "Serum 25-hydroxyvitamin D and the risk of hip and nonspine fractures in older men," *Journal of Bone and Mineral Research*, 25(3), 545-53, 2010. doi: 10.1359/JBMR.090826.
- [42] Pittas AG, Dawson-Hughes B, Sheehan PR, Rosen CJ, Ware JH, Knowler WC, ... Hu FB, "Vitamin D supplementation and prevention of type 2 diabetes,," *The New England Journal of Medicine*, 381(6), 520-530, 2019. doi: 10.1056/NEJMOA1900906.
- [43] Brambilla P, Bedogni G, Moreno LA, Goran MI, Gutin B, Fox KR, Peters DM, Barbeau P, De Simone M, Pietrobelli A, "Waist circumference-to-height ratio predicts adiposity better than body mass index in children and adolescents," *International Journal of Obesity*, 37(7), 943-946, 2013. doi: 10.1038/ijo.2013.32.
- [44] Wang J-J, Wang Z-Z, Shi L-P, Luo Y, "The association between body mass index, waist circumference with body fat percent, and abdominal fat rate in overweight and obese pupils," *Chinese Journal of Preventive Medicine*, 47(7), 603-609, 2013.
- [45] Shapses, SA, Sukumar D, "Bone Metabolism in Obesity and Weight Loss" *Annual review of nutrition*, 32, 287, 2012. doi: 10.1146/ANNUREV.NUTR.012809.104655.

- [46] Zakharova I, Klimov L, Kuryaninova V, Nikitina I, Malyavskaya S, Dolbnya S, "Vitamin D insufficiency in overweight and obese children and adolescents. *Frontiers in Endocrinology*, 10(MAR), 103, 2019. doi: 10.3389/fendo.2019.00103
- [47] de Mutsert R, Sun Q, Willett WC, Hu FB, van Dam RM, "Overweight in early adulthood, adult weight change, and risk of type 2 diabetes, cardiovascular diseases, and certain cancers in men: a cohort study," *Am J Epidemiol*. 179(11), 53-65, 2014. doi: 10.1093/aje/kwu052. PMID: PMC4036209.
- [48] Castro AVB, Kolka CM, Kim SP, Bergman RN, "Obesity, insulin resistance and comorbidities – Mechanisms of association," *Arquivos Brasileiros de Endocrinologia & Metabologia*, 58(6), 600–609, 2014. doi: 10.1590/0004-273000000322