THESIS

ASSOCIATION OF STUNTED IN OBESE ADOLESCENT AND THE RISK OF METABOLIC SYNDROME

BY

MUHAMMAD HARITS

NIM: 011911133088

MEDICAL STUDY PROGRAM

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To fulfill the requirements of Research Module lecture

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ASSOCIATION OF STUNTED IN OBESE ADOLESCENT AND THE RISK OF METABOLIC SYNDROME

ABSTRACT

Background : Indonesia has a high prevalence of stunting. It is even ranked fifth among countries with the highest burden of stunted children. The stunting rate for children in Indonesia based on Riskesdas is 30.8 percent in 2018. Stunting that is not treated at the age of 5 years will be at risk. In Indonesia from 1999 to 2004 the prevalence of overweight and obesity increased from 4.2 and 1.9% in childhood to 8.8 and 3.2% in adolescence. The increase in the incidence of obesity is in line with the increase in the incidence of metabolic diseases.

Purpose : To analyze the association of stunted in obese adolescents and the risk of metabolic syndrome.

Methodes : The type of the research is an analytic observational study. The design of the research is a cross sectional study using the medical records from the inpatient of the Child Health Department of Dr. Soetomo Hospital between October 2019 - December 2019. The sample is obese adolescents aged 13-18 years from Junior High School and Junior High School in Sidoarjo and Surabaya who meet the criteria for metabolic syndrome.

Result : Based on data taken randomly between male and female gender, it was found that there is no significant association in stunted obese adolescents. Central obesitas (92%) is the most common component of the metabolic syndrome experienced by the sample, followed by Low HDL (47%), Hypertension (33%), Hypertriglyceride (19%), Hyperglicemia (6%). Chi square analysis test was carried out to determine the association between obese adolescents with metabolic syndrome found p-value = 0.169 or had a non-significant association.

Conclusion : There are significant differences in BMI, FPG and Blood Pressure between obese adolescents who are stunted and the risk of metabolic syndrome.

Keyword : Stunted, Obese Adolescent, Metabolic Syndrome

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LIST OF ABBREVIATION

AT	: Adipose Tissue
BH	: Body Height
BL	: Body Length
CHD	: Coronary Heart Disease
Cm	: Centimeter
DBP	: Diastolic Blood Pressure
FPG	: Fasting Plasma Glucose
HDL	: High Density Lipoprotein
IFG	: Impaired Fasting Glucose
IGT	: Impaired Glucose Tolerance
LDL	: Low Density Lipoprotein
MetS	: Metabolic Syndrome
SBP	: Systolic Blood Pressure
SES	: Socioeconomic Status
T2D	: Type 2 Diabetes
WHO	: World Health Organization

LIST OF ADDENDUMS

No Addendums.

CHAPTER 1
PRELIMINARY

1.1 Background

Stunting has been identified as a major public health priority (Prendergast, 2014). According to the World Health Organization's (WHO) public health importance cut-off values for stunting, Indonesia has a high prevalence of stunting (Titaley et al., 2019) The country is even ranked fifth among countries with the highest burden of stunted children (Beal, T., et al, 2018) The stunting rate for children in Indonesia based on Riskesdas is 30.8 percent in 2018 (Riskesdas, 2018).

Stunting is caused by chronic malnutrition, and childhood malnutrition has been linked to an increased risk of adult obesity. Malnutrition has been linked to increased morbidity and mortality, impaired physical, neurodevelopmental, and economic ability, and an increased risk of metabolic disorders in adulthood, specifically increased metabolic fat, according to certain research (Grillo et al, 2016).

In Indonesia from 1999 to 2004 the prevalence of overweight and obesity increased from 4.2 and 1.9% in childhood to 8.8 and 3.2% in adolescence (Julia et al., 2008). A growing number of studies have found that nutritional stunting leads to a slew of long-term changes, including lower energy expenditure, increased susceptibility to the effects of high-fat diets, lower fat oxidation, and impaired food intake regulation, all of which increase the likelihood of becoming obese in the future (Sawaya, A.L., and Roberts, S., 2003).

The increase in the incidence of obesity is in line with the increase in the incidence of metabolic diseases. Prevalence of metabolic syndrome in adolescents is increasing, in parallel with the increasing trends in obesity rates. As the proportion of the population with

obesity continues to rise, the prevalence of metabolic syndrome is increasing in adolescents. Children with metabolic syndrome have an increased risk of metabolic syndrome as adults, and possibly an increased risk of type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD) (Al Hamad, 2017).

According to the prevalence data listed above, it can be seen that the increasing prevalence of stunted adolescents who are obese is accompanied by an increase in the prevalence of adolescents with metabolic syndrome. This is caused by the nutrition transition, this transition can change in diet composition from conventional diets that are predominantly drawn from plant-based food sources that are low in fat and high in fibre. Obesity has raised the public health importance of noncommunicable diseases caused by nutrition, such as cardiovascular disease and diabetes (Kimani-Murage et al., 2010).

The definition of metabolic syndrome has been recommended by various organizations, including the World Health Organization (WHO), NCEP III, the International Diabetes Federation (IDF) and The National Heart, Lung and Blood Institute (NHLBI). However, for adolescents, the standard used is from the International Diabetes Federation, namely the IDF Criteria. With many consequences of nutritional deficiencies and complications of obesity, it can meet the criteria for the metabolic syndrome listed by the IDF criteria (IDF, 2006).

Similar study was conducted and included 59 obese adolescent between the ages of 13 and 16. Around 27 people (45.8%) had metabolic syndrome (Widjaja, et al., 2020) . From this research, it can be seen that the number of obese children with metabolic syndrome is quite high, so we would like to know more about the association between obese children who experience stunting with metabolic syndrome. Because to study the

relationship between adolescent obesity and metabolic syndrome is very useful to prevent the risk of similar diseases that will occur in the future. With the unprocessed data from previous studies, it will be easier to find out how likely it is that adolescents who are obese are stunted adolescents with the risk of metabolic syndrome. In addition, this research aims to provide opportunities for further research on stunted, obesity in adolescent, and metabolic syndrome.

1.2 Formulation of the Problem

Is there an association of stunted in obese adolescents and the risk of metabolic syndrome?

1.3 Research Purposes

Based on the formulation of the problem that has been described above, the objective of this research are:

1.3.1 General Purpose

To analyze the association of stunted in obese adolescents and the risk of metabolic syndrome.

1.3.2 Specific Purpose

1. To know the amount of stunted in obese adolescent
2. To know the amount of stunted in obese adolescent and metabolic syndrome
3. To analyze the association of obese adolescent in stunted and metabolic syndrome

4. To know the risk of hypertension in stunted and non stunted obese adolescent
5. To know the risk of triglycerides in stunted and non stunted obese adolescent
6. To know the risk of high density lipoprotein in stunted and non stunted obese adolescent
7. To know the risk of fasting plasma glucose in stunted and non stunted obese adolescent

1.4 Benefit of Research

1.4.1 Academic Benefits

This research is expected to increase understanding of the association between stunted and metabolic syndrome risk in adolescent obesity. This can also be useful in the application of the medical field to determine the possibility of children who can experience the risk of syndrome in obese adolescents who are stunted or not.

1.4.2 Practical Benefits

This research is expected to find out the prevalence of early detection of obese adolescents with short stature who experience stunted and metabolic syndrome by being able to provide knowledge to the public as parents to be careful and always take care of their children's health so that the risk of obesity and metabolic syndrome in stunted adolescents can be avoided. This research can also

be a reference to provide further research opportunities in stunting children, obesity in adolescents and metabolic syndrome.

CHAPTER 2
LITERATURE REVIEW

2.1 Stunting

Stunting condition is a condition of chronic malnutrition caused by inadequate nutritional intake for a long time due to feeding that is not in accordance with nutritional needs. Stunting status in school children is calculated using WHO anthropometric standards by calculating the Z-score TB/U respectively. -each child. The nutritional status assessment standards according to WHO are divided into two, namely the 2005 WHO growth standards for children under five and the 2007 WHO for school children up to the age of 18 years. Stunting conditions are identified by comparing the height/length of a child's body with the standard height of children in the normal population according to the same age and sex. Stunting conditions indicate a chronic growth disorder in which a child cannot reach his potential height due to nutritional and health problems.

The process of stunting begins with weight loss, especially at the age of 2 years. Continued weight loss will result in suboptimal body length. Based on the height velocity graph, the body length is divided into 3 phases (Sterling R et al, 2012) , namely:

a. Infant phase (infant) aged 0-2 years

Age 0-12 months (1 year) increase in body length reaches 24-27 centimeters (cm) every year, while at the age of 1-2 years the increase in body length can reach 12 cm in a year.

b. childhood phase (child)

- At the age of 2-3 years, the body length increases 8 cm every year
- At the age of 3-5 years, the body length increases 7 cm every year

- At the age of 5-puberty body length increase of 8 cm every year

c. Puberty phase

- Women aged 10-12 years increase in height 8-12 cm every year

- Boys aged 12-14 years increase in height 12-14 cm every year.

Sterling's research shows that every decrease of 1 SD PB/U at birth correlates with a decrease of 0.7 SD TB according to age during adolescent, both boys and girls, so that there is a 9.7 times risk of becoming stunted (95% CI 3.3-28, 6). Each decrease in 1 SD PB/U in the first 30 months of life correlated with a decrease in TB/U in adolescents 0.4 SD in male adolescents and 0.6 SD in female adolescents, with a 5.8 times greater risk of stunting (95% CI 2.6-13.5) (Soetjiningsih G et Ranuh, 2013).

2.1.1 Risk Factors of Stunting

Several risk factors that cause stunting are poor maternal health, a lack of antenatal care facilities, insufficient feeding and care, and insufficient infrastructure and healthcare services are all key risk factors for stunting. Other risk factors for stunting, including socioeconomic, demographic, and environmental factors, are important in predicting stunting and obesity, as well as other aspects like nutrition and lifestyle (Keino S, 2014).

Growth is a quantitative change, namely an increase in the number, size, dimensions at the level of cells, organs, and individuals. Children not only grow physically, but also the size and structure of organs and brain. For example, the result of brain growth is that children have a greater capacity to learn, remember, and use their minds. So the child grows both physically and mentally. Physical

growth can be assessed by measuring weight (grams, pounds, kilograms), length (cm, meters), bone age, and secondary sex characteristics (WHO, 2014).

Normal growth is the result of the right interaction between internal and external factors. Genetic factors (nature) as internal factors can play a role in determining the basic growth potential, but external or environmental factors (nurture) have an important influence on the profile achieved. External factors include prenatal, natal and post-natal factors. Nutritional factors are external factors that cause stunting.

In general, the causes of stunting are classified into direct and indirect causes. Several epidemiological studies have shown that the cause is inadequate nutritional intake in terms of quantity, quality and variety of food. In addition to this, repeated infections and adequate health care are the causes of stunting. As indirect causes are poverty, inequality, social norms, maternal education and the social status of women. Other theories regarding the determinants of stunting according to WHO include family and household factors, nutritional factors (inadequate intake), factors for consuming breast milk (ASI), infections and social and community factors (Beal T et al, 2018).

Based on the WHO conceptual framework, there are eight elements of household and family factors that influence the incidence of stunting. The main factor in this element is nutritional deficiency in the first 1,000 days of life (HPK) of toddlers. Nutrition plays a role in children's linear growth. Golden et al., conducted a study on the effect of various nutrients on linear growth and divided them into type 1 and type 2 nutrients. Deficiency of type 1 nutrients did not directly

affect linear growth, while type 2 nutrient deficiency directly caused linear growth retardation (WHO, 2018).

In the WHO conceptual framework, maternal height is also a factor that causes stunting. Several studies in Indonesia show that there is a strong strong association between short mothers and stunted children. A cross-sectional study of the Indonesia Nutrition Surveillance System (NSS) in nine provinces, found that mothers with height <145 cm were associated with stunting children with an AOR value of 2.32 (95% CI [2.25-2.40]) (Golden M. H, 1995). In contrast to mothers who have a height > 150 cm, the results of the study found that mothers who were taller, reduced the incidence of stunting in children aged 6-59 months (Bardosono et al, 2007).

Other household factors that can cause stunting are inadequate stimulation and activity of children, availability of water, poor sanitation, poor parenting practices, including low levels of maternal knowledge about health and nutrition. In a study conducted in Indonesia, it was found that stunting children were associated with poor parenting practices, poor sanitation and water availability, food insecurity and low levels of caregiver education (Dewey K et Brown K, 2003). A cross-sectional study in Indonesia that shows the relationship between stunting children and parenting practices is bad, the results of the study found that there was no strong association (WHO, 2003).

Other factors that play a role as a cause of stunting are inadequate complementary feeding (MPASI), in this case including poor food quality, wrong feeding practices, and food safety. A study conducted by Dewey and Brown

showed that at the age of 6 months, breast milk contains only 80% protein and 21% zinc of the daily requirement (Semba R et al, 2011). Therefore, at the age of 6 months WHO recommends complementary feeding to complement nutritional deficiencies. The WHO Global Strategy for Feeding Infant and Young Children in 2003 recommended that the provision of complementary foods meet 4 requirements (Koletzko B et al, 2009), namely:

1. Timely, meaning that complementary foods must be given when exclusive breastfeeding is no longer able to meet the baby's nutritional needs
2. Adequate, meaning that MPASI contains energy, protein, and micronutrients that can meet the macronutrient and micronutrient needs of infants according to their age.
3. Safe, meaning that solid food is prepared and stored in a hygienic manner, given using clean hands and eating utensils.
4. Given properly (properly fed), meaning that complementary foods are given by paying attention to the signal of a child's hunger and satiety. The frequency of feeding and the method of feeding should be able to encourage the child to actively consume food in sufficient quantities using hands, spoons, or self-feeding (adjusted for the age and stage of development of a child).

Another study stated that intake of multiple micronutrient (MMN)-fortified milk was associated with a reduction in the incidence of stunting at the age of 6-59 months in rural areas (UOR 0.87, 95% CI [0.85, 0.90] and urban areas (UOR 0.87, 95% CI [0.85, 0.90]) and urban areas (UOR 0.80, 95% CI [0.76, 0.85]) (Herrador Z et al, 2014). Research on the effect of zinc supplementation in preventing stunting

has yielded varying results, while protein is consistently able to prevent stunting (Ahmad A et Miko A, 2016). Essential amino acids are the most important types of protein in preventing stunting. stunting. A study showed that the height of children who received 15% protein from the total calorie intake was higher than those who received 7.5% of the total calorie intake. The first source of protein in the 15% protein diet was animal protein (milk, eggs, chicken) which contains essential amino acids.This shows that apart from quantity, protein quality also affects linear growth (Arifin D et al, 2012).

One of the important nutritional intakes in supporting the growth and development of toddlers is exclusive breastfeeding. Exclusive breastfeeding is breastfeeding without adding and or replacing with other foods or drinks to babies from birth to six months of age. Breast milk (ASI) is a single food for infants at the beginning of life that contains all the nutrients needed by infants in sufficient and balanced quantities (Hasanah Z et al, 2018).

Toddlers who experience nutritional deficiencies before the sensitive period can still be improved with good and adequate intake so that they can undergo growth and development according to their age of growth and development. However, if the intervention is late, the toddler will not be able to catch up with the growth delay which is known as failure to thrive, which leads to stunting (Prendergast A et al 2014).

Research conducted in Banda Aceh City stated that the incidence of stunting was caused by low family income levels, not being given exclusive breastfeeding, poor complementary feeding, and incomplete immunization. Of the four factors,

the problem of breastfeeding is the most influential on the incidence of stunting (Mendez M et Adair L, 1999). The same results were carried out by Arifin in 2012 with a study which stated that stunting was influenced by birth weight, nutritional intake of children under five, breastfeeding, history of infectious diseases, knowledge of maternal nutrition under five, family income, and distance between births. However, the most dominant factor is breastfeeding (Martorell R et al, 2010).

2.1.2 The Effect of Stunting

Growth retardation caused by nutritional deficiencies and repeated infections tend to be more at risk of causing morbidity and mortality. In general, the impact of stunting can be divided into short-term and long-term impacts. In the short term, stunting can cause an increase in morbidity and mortality, cognitive and motor development, not optimal, and increase health costs. The long-term impact of stunting is that the body posture is not optimal as an adult so that children are shorter than normal children, and the risk of obesity and other diseases increases, reproductive health decreases, learning capacity and performance during school time become less than optimal, and can lead to productivity and capacity not optimal work (Prendergast A et Humphrey J, 2014).

Malnutrition that occurs in stunted children is the cause of 45% of deaths of children under 5 years old in developing countries. The results of a review of several studies conducted by Prendergast and Humpey found that children with poor linear growth were 1.5 times more likely to experience respiratory infections and diarrhea than normal children, while stunted children were six times more

likely to have these disorders (De Onis M et Branca F, 2016). Children with very short conditions also have three times more risk of dying from infections such as sepsis, meningitis, tuberculosis, hepatitis and cellulitis.

Stunting conditions when they were young caused their adult height to be 1% lower than normal height and decreased productivity by 1.4%. In addition, stunting reduces a person's level of intelligence (IQ) by 5 to 11 points. According to research conducted by Mendez et al in the Philippines, children who are short in the first 2 years have lower school grades than normal children, especially children with severe shortness (severe) (Adair L et al, 2013). Stunting children also tend to be slower to start school and advance to grades. and tend not to attend school (Shekar M, 2017). Martorel et al stated that adults with a history of stunting at the age of 2 years experienced a one-year delay in completing school (Kepmenkes R.I., 2016) Adair et al stated that improving linear growth of one standard deviation in children under two years of age could improve half times better at school achievement.

Stunting is also an indirect factor in increasing the risk of degenerative diseases or diseases that arise with age. Children with stunting have a high risk for obesity, diabetes, heart and blood vessel disease, stroke, cancer, and disability in old age. The Indonesian government suffered heavy losses due to the high incidence of stunting due to increased government spending, especially national health insurance related to non-communicable diseases such as heart disease, stroke, diabetes or kidney failure. The results of World Bank research illustrate that the

losses caused by stunting reach 4-11% of Gross Domestic Product (GDP) in Asia and Africa (Black M.M., 2018).

2.1.3 How to Measure Body Length or Height

Accurate measurement of the length or height of children under 2 years is done in a supine position, while those over 2 years old or above 85 cm are performed in a standing position. The way to measure in a lying position is by using an infantometer. Measurements should be carried out by two people, namely by laying the baby on his back on a flat base, then the baby's head is positioned against the number barrier. The first person is in charge of holding the baby's head so that it remains attached to the number 0 barrier (head barrier), while the second person is in charge of pressing the baby's knees so that they are straight using their left hand, then the right hand presses the foot limit to the sole of the foot The measurement results are determined by looking at the numbers on the outside of the gauge by a second person. If children aged 0 - 24 months are measured standing up, the measurement results are corrected by adding 0.7 cm (Hadders-Algra, M., 2010).

The second measurement method is in a standing position using a stadiometer/microtoise. In the measurement with a standing position, the child was asked to stand without wearing footwear with an upright position facing forward. The position of the back, buttocks and heels against the measuring pole. Once the position is correct, the upper limit of the gauge is lowered until it sticks to the crown. The measurement results are read at that limit. If measurements in children

aged over 24 months are carried out in a supine position, then the measurement results are corrected by subtracting 0.7 cm (Hadders-Algra, M., 2010).

2.1.4 The Difference Between Stunting and Stunted

Stunted is a cyclical process because adolescents who were stunting as children have stunting offspring, producing a difficult-to-break intergenerational cycle of poverty and diminished human capital. The mechanisms driving linear growth failure at various ages are detailed, as well as the short-, medium-, and long-term effects of stunting. The evidence supports windows of opportunity over the life cycle to focus interventions at the stunting syndrome is assessed (Prendergast A et Humphrey J, 2014).

2.2 Obesity

2.2.1 Definition

The World Health Organization (WHO) defines obesity as an abnormal accumulation of fat in the body that can affect health. Obesity occurs due to an imbalance between energy intake and energy output, resulting in excess energy being stored in the form of fat tissue (WHO, 2018). Based on an anthropometric assessment using the CDC (Centers for Disease Control) curve from 2000, BMI is above the 95th percentile (P>95). Waist circumference does not need to be measured if BMI is greater than 30 kg/m2, as central obesity can be inferred (IDF, 2006).

2.2.2 Diagnosis

According to the Indonesian Pediatrician Association (2014), overweight or obesity in adolescents is established based on interviews (anamnesis), physical examination, anthropometric evaluation, and early detection of comorbidities through supporting examinations (Sjarif et al., 2014):

A. Interview.

The interview included an evaluation of diet, physical activity patterns, as well as a family history of obesity, a family history of health risks associated with obesity such as early cardiovascular disease, hypertension, type 2 diabetes mellitus. Children with obesity may complain of difficulty breathing at night, resulting in sleepiness. at noon.

B. Physical examination

General physical examination in obese children usually found a rounded face, chubby cheeks and double chin. The neck formation is relatively short, with a puffy chest and enlarged breasts. Stomach looks bulged with abdominal wall folds. The limbs are generally X-shaped, and the genitalia appear small.

C. Anthropometric evaluation

Anthropometric evaluation carried out included measurements of height, weight, body mass index (BMI). If the measurement results show overnutrition potential (Z score > +1SD) or weight by height > 110%, then the BMI chart according to age and gender is used to determine the presence of obesity. Obesity in children aged < 2 years is confirmed if Z score > +3

SD using the 2006 WHO BMI chart, while in children aged 2-18 years using the 2000 CDC (Center for Diseases Control) BMI chart by plotting the BMI calculation results on the CDC BMI curve (BMI by age) with values According to the 2000 CDC BMI chart, overweight is if it is above P85–P95, while obesity is more than P95 on the 2000 CDC BMI chart (IDAI, 2014).

2.2.3 Epidemiology

In 2016 WHO stated that more than 1.9 billion adults aged 18 years and over were overweight, with 650 million of them suffering from obesity. In 2016 it was estimated that 41 million children aged less than 5 years were overweight and obese, almost half of children aged less than 5 years who were overweight and obese were from Asia (WHO, 2018).

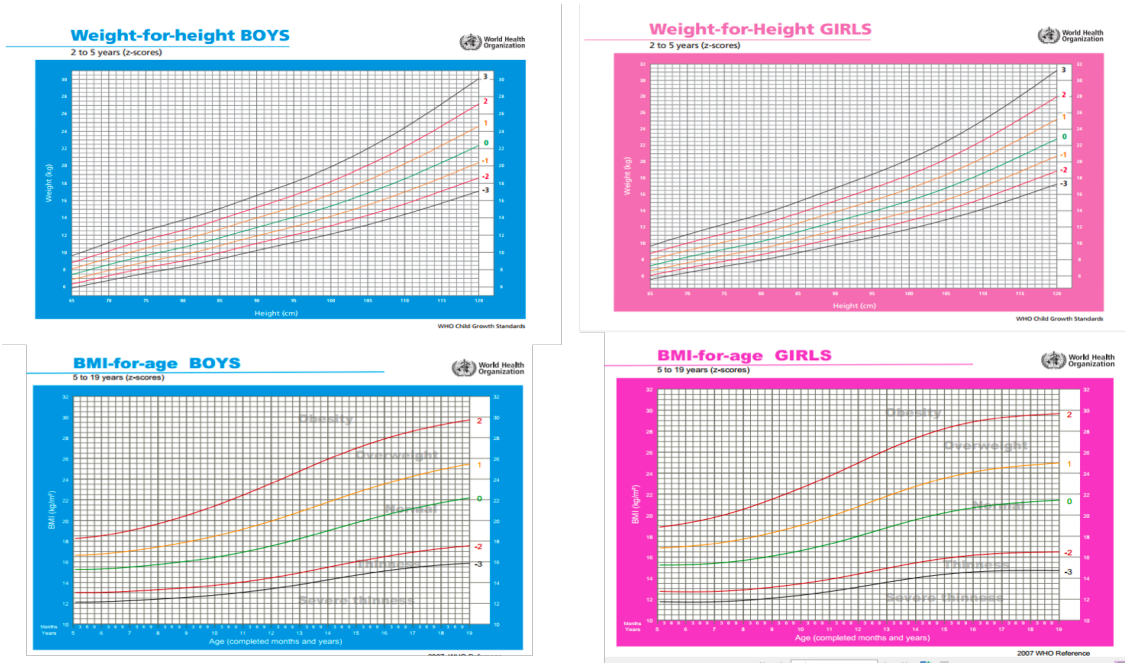


Figure 2.1 WHO growth curve used in establishing obesity according to age and sex (WHO, 2018).

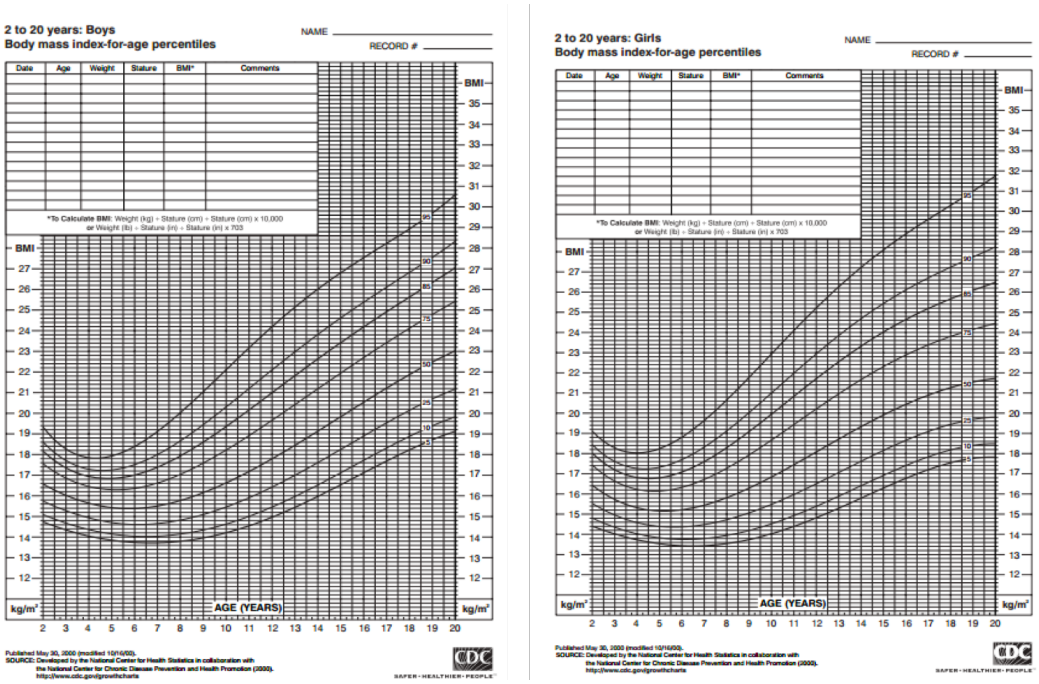


Figure 2.2 The CDC's BMI growth curve used in establishing obesity in adolescents according to age and sex (Sondik et al., 2002).

The prevalence of overweight and obesity increased sharply from 4% in 1975 to more than 18% in 2016. This increase occurred in both boys and girls. Only less than 1% of children and adolescents aged 5-19 years were obese in 1975, but in 2016 more than 124 million children and adolescents were obese (6% of girls and 8% of boys) (WHO, 2018).

Riskesdas data in 2013 showed the prevalence of overweight and obesity in children under five was 11.9%, which means there was a decrease compared to 14% in 2010. In children aged 5-12 years, the prevalence of overweight and obesity reached 10.8% and 8.8%. Meanwhile, for adolescents aged 13-15 years, the prevalence of overweight reached 8.3% and obesity 2.5%. In adolescents 16-18 years the prevalence of overweight is 5.7% and obesity is 1.6%. Overall, there is

still an increase in the prevalence of overweight and obesity from 1.4% in 2007 to 7.3% in 2013 (Health Research and Development Agency, 2013).

2.2.4 Etiology

Obesity is caused by a combination of genetic, behavioral, environmental, physiological, social, and cultural variables, all of which contribute to energy imbalance and fat deposition. The proportional contributions of each of these components have been widely examined, and while genes play an essential part in body weight regulation, the World Health Organization Consultation on Obesity concluded that (Racette S et al, 2003).

Obesity in children is a consequence of the interaction between environmental and genetic factors, where environmental factors are influenced by circumstances in the family, community and school. Environmental factors that play a role in the etiology of obesity include psychosocial and emotional factors, which can increase children's weight through maladaptation strategies by using eating activities to suppress negative emotions. Several other environmental factors that contribute to obesity include the increased use of sugary drinks, sweet snacks, fast food with high fat content and large portions, and foods with a high glycemic index. Consumption of sugar-sweetened beverages, including fruit juices, has been suggested as an important factor in causing obesity in children, accounting for 10-15% of daily caloric needs. Environmental changes in increasing calorie intake are accompanied by predisposing factors in the form of a decrease in calorie use with a decrease in daily physical activity in children due to the use of electronic goods (Kumar and Kelly, 2017).

Genetic factors play a role in 30%-50% of obesity variations, most of which are caused by polygenetics, but currently there are several single genetic defects and syndromes associated with obesity. Children with genetic syndromes associated with obesity usually manifest as early onset obesity and have characteristic features on physical examination such as stunted, facial dysmorphic, developmental delay, mental retardation, retinal changes, or hearing loss. For example, Prader-Willi syndrome is the most common syndrome associated with obesity, with manifestations of hypotonia, difficulty eating in infancy, followed by hyperphagia that progresses to obesity. Meanwhile, single gene defects that play a role in obesity include mutations in melanocortin receptor 4 (Speiser et al., 2005).

Epigenetic factors also found that play a role in the development of obesity. These epigenetic factors can influence the interaction between individuals with the environment and nutrition in increasing body weight (Chang and Neu, 2015). Another cause of obesity is endocrine disorders, which are generally accompanied by stunted and/or hypogonadism. Endocrine disorders cause weight gain accompanied by an increase in endogenous and exogenous glucocorticoids, hypothyroidism, and growth hormone deficiency. epilepsy (Hamed, 2014).

2.3 Metabolic Syndrome

2.3.1 Definition

Hypertension, central obesity, and atherogenic dyslipidemia are among the metabolic disorders that make up the metabolic syndrome (MetS). MetS is

significantly linked to an elevated risk of cardiovascular disease (CVD) caused by atherosclerosis (Rochlani et al., 2017).

Metabolic syndrome is defined as a constellation of physiological, biochemical, clinical processes and metabolic factors that can directly increase the risk of atherosclerosis and type 2 diabetes mellitus (Al-Hamad et al, 2017). Obesity with metabolic syndrome can increase the risk of death twofold compared to without metabolic syndrome (Kaur et al, 2014).

The definition of metabolic syndrome for adults has been recommended by various organizations, including the World Health Organization (WHO), NCEP III, the International Diabetes Foundation (IDF) and The National Heart, Lung and Blood Institute (NHLBI). From these recommendations, it was agreed that based on the Join Task Force, the criteria for adult metabolic syndrome were as follows:

1. Elevation of waist circumference based on a specific definition of a country's population
2. Systolic blood pressure of 130 mmHg or more and/or diastolic of 85 mmHg or more or with hypertension treatment
3. Fasting blood sugar 100 mg/dL or more or in hyperglycemic therapy
4. Triglycerides 150 mg/dL or more or on therapy
5. HDL cholesterol less than 40 mg/dL in men and less than 50 mg/dL in women or on treatment for lowering HDL cholesterol.

2.3.2 Pathophysiology

The pathogenic mechanism of MetS is very complicated and needs to be fully elucidated. Whether the various components of MetS represent different

pathologies or the performance of common pathogenic mechanisms is still under debate. The wide disparity in the geographic distribution of MetS and the recent "catch-up" trend in the developing world highlights the importance of environmental and lifestyle factors, such as excessive calorie consumption and lack of physical activity as the main contributors. Visceral obesity has been shown to be the main cause of most ways of causing MetS, thus stressing the importance of high caloric intake as the main cause. Among all the proposed mechanisms, insulin resistance, neurohormonal activation, and chronic inflammation appear to be the main players in the initiation, progression, and transition of MetS to CVD (Rochlani Y et al., 2017).

2.3.2.1 Obesity

Obesity is defined as an abnormal or excessive buildup of fat or adipose tissue in the body that can be harmful to one's health. Leptin is an adipocyte hormone that helps people lose weight and eat less. Obesity is linked to cellular leptin resistance. Adipokines and free fatty acids secreted by adipose tissue generate systemic inflammation, which leads to insulin resistance and elevated triglyceride levels, which contributes to obesity (Panuganti K et al, 2020).

Excess fat buildup (regionally, worldwide, or both) increases health risks, resulting in overweight and obesity. Because, as shown below, body weights and fat distributions that lead to the expression of comorbid

disorders occur at varying thresholds depending on the population, it is the point at which health risk increases that is most relevant (Purnell J, 2018)

2.3.2.2 Dyslipidaemia

Dyslipidemia is a significant risk factor for heart disease and stroke (Kopin, L. and Lownstein, C., 2017). Dyslipidemia has long been recognized as a separate risk factor for cardiovascular disease. In patients with MetS, low HDL cholesterol and hypertriglyceridemia have been proven to be independently and strongly linked to myocardial infarction/stroke (Kolovou, Anagnostopoulou, Cokkinos., 2005).

2.3.2.3 Glucose Intolerance

Glucose intolerance refers to a range of glucose metabolic problems such as impaired fasting glucose (IFG), impaired glucose tolerance (IGT), and type 2 diabetes, and is a serious public health concern (T2D) (Stull A., 2016). The defects of insulin action in glucose metabolism include the inability to inhibit gluconeogenesis in the liver and the inability to mediate glucose uptake in insulin-sensitive tissues (ie, muscle and adipose tissue). In order to compensate for the defect of insulin action, insulin secretion must be increased to maintain normal blood sugar. If this compensation fails, insulin secretion defects will dominate and hyperglycemia will occur. (Aganović I and Dušek T, 2007)

2.3.2.4 Hypertension

The relationship between insulin resistance and hypertension has been well established. Several different mechanisms have been proposed. First, when insulin is injected into a person of normal weight, it is a vasodilator and has a secondary effect on the reabsorption of sodium in the kidneys. In the case of insulin resistance, the vasodilation effect of insulin can be lost, but the kidney's role in sodium reabsorption is retained. Fatty acids themselves can mediate relative vasoconstriction. Hyperinsulinemia may cause increased sympathetic nervous system (SNS) activity and lead to the development of high blood pressure (Aganović I and Dušek T, 2007).

2.3.3 Criteria International Diabetes Federation

The criteria for metabolic syndrome in children and adolescents are more varied than the criteria for adults. These criteria are taken from various studies and consensus so that there are no definite criteria (Al Hamad et al, 2017; Magge et al, 2017). Some investigators used criteria from the National Health and Nutrition Examination Survey III, but the estimated prevalence results were deviant, ranging from 4.2% to 9.2%, a difference that doubled (Cooks et al, 2003; Ferranti et al. , 2004). In 2007 the International Diabetes Federation (IDF) recommended that the definition of metabolic syndrome in children be the same as adults, but only for ages 10-16 years. For waist circumference, the 90th percentile or adult cut of point

is used to express abdominal obesity. According to the IDF, if the age is more than 16 years, the definition of adult is used.

According to the modification of The National Cholesterol Education Program (NCEP), metabolic syndrome is found if 3 signs of the following are found: waist circumference >90 cm (men) and >80 cm (women), triglyceride levels > 150 mg/dL, cholesterol levels HDL < 40 mg/dL (men) and < 50 mg/dL (women), systolic blood pressure 130 mmHg or diastolic blood pressure 85 mg/dL, and fasting plasma glucose > 100 mg/dL. Meanwhile, according to The International Diabetes Federation (IDF), metabolic syndrome consists of: central obesity (waist circumference >90 cm (men) and >80 cm (women), accompanied by 2 criteria based on the NCEP criteria (Zimmet et al, 2007) .

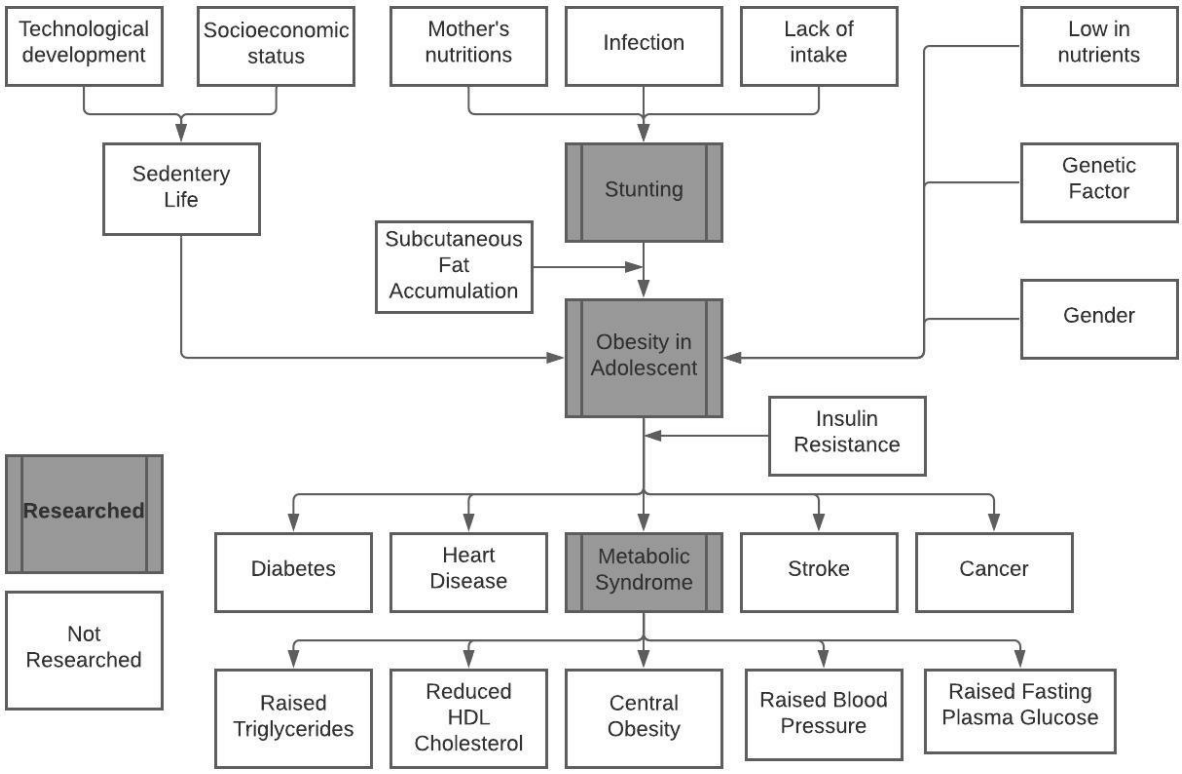
According to the new IDF definition, for a person to be defined as having the metabolic syndrome they must have central obesity. It is called metabolic syndrome if central obesity is found (aged 10-16 years, abdominal circumference >90th percentile, age >16 years if abdominal circumference is 94 cm for male and 80 cm for female accompanied by 2 or 4 of the following criteria.

Table 2.1 IDF Criteria (Translational Pediatrics, 2017)

Variables	**IDF definition ages 10–16 years**
Defining Criteria	Central obesity plus at least 2 out of 4 criteria
Central Obesity	WC ≥ 90^{th} percentile or adult cut-off if lower
Hypertension	SBP ≥ 130 mmHg or DBP ≥ 85 mmHg or treatment with anti-hypertensive medication
Hypertriglyceridemia	TG ≥ 150 mg/dL
Low HDL	HDL < 40 mg/dL
Impaired Glucose	FPG ≥ 100 mg/dL or known T2DM

CHAPTER 3
RESEARCH CONSEPTUAL FRAMEWORK

3.1 Conceptual Framework



3.2 Explanation of Conceptual Framework

Obesity has been linked to sedentary lifestyle patterns in children and adolescents, such as playing digital games, using computers, and notably watching television (Rey-López et al, 2008). Sedentary behavior is correlated with socioeconomic status (SES), and children and adolescents from lower socioeconomic backgrounds engage in more sedentary behavior in both screen-based and non-screen-based activities. Sedentary

behavior among school-aged children, on the other hand, was found to be correlated with greater SES (Mielke G et al, 2016).

Lower IQ, mother's height, male sex, mother and father's educational level, poverty, socioeconomic status, residence, child care behavior (inadequate complementary feeding and breastfeeding), cultural beliefs, access to health care, and environmental ecosystems have all been linked to stunting in children in previous studies (Ramli et al, 2009). Infectious infections, along with poor food, are now widely accepted as the potential causes of a major proportion of stunting. Stunting syndrome is a term that describes how starvation and infection interact throughout the maternal, baby, and child lifecycle to prevent growth (Millward D, 2017). A shortage of energy-protein consumption has been linked to an increased risk of stunting in children under the age of five. The amount of essential amino acids (EAAs) consumed and other risk factors for stunting in children under the age of five were lower in stunted children than in non-stunted children (Maulidiana A et Sutjiati E, 2021).

To promote optimal health for both the individual and future children, good nutrition and a healthy lifestyle are required throughout the life cycle. If a youngster does not get them, he or she may become stunted (Branca F et Ferrari M, 2002). The link between maternal stature and children's linear growth is most likely owing to genetic aspects controlled by mothers, such as sanitation, appropriate nourishment, and reproductive health (Manggala et al, 2018). Another factor linked to stunting was gender, with males being shown to be at a higher risk of being stunted than females (Vonaesch P et al, 2017).

The weight and skinfold thickness of stunted children are significantly lower than those of non-stunted children. The relatively large amount of subcutaneous fat and large waist circumference in stunted children may be related to the development of obesity (Kruger H et al., 2004). Fat increase in children after peaking the height speed has been described and may be due to hormonal changes during menarche, which may increase the child's susceptibility to fat deposits (Kruger H, 2005).

The concentrated accumulation of body fat in adolescent obesity is related to insulin resistance, while the distribution of peripheral body fat is not important in metabolism. Compared with most metabolically "unhealthy" obese patients, up to 30% of obese patients are metabolically healthy, have insulin sensitivity similar to healthy normal-weight individuals, have lower visceral fat content, and lower carotid intima-media thickness (Engin A, 2017).

Obesity in teenagers can lead to a variety of problems, including diabetes, heart disease, metabolic syndrome, stroke, and cancer. Obesity-related diabetes is a major crisis in modern society. In this society, food is sufficient and exercise is optional. Mechanism studies have revealed the link between many fat-derived molecules and inflammation, which together are hypothesized to be the basis of the obesity-diabetes link, thus representing the expected goal of therapeutic intervention (Lazar M, 2005). In young adulthood, there is expected to be an increase in obesity, as well as an increase in the incidence of CHD (Coronary Heart Disease) and the overall number of CHD events and fatalities. As the population enters middle age, the trend is expected to continue in both absolute and relative terms (Bibbins-Domingo K et al, 2007). The metabolic syndrome's incidence rose with the severity of obesity, reaching 50% in very obese children. Each half-

unit rise in the body mass index (converted to a z score) was linked to a higher risk of metabolic syndrome in overweight and obese people (Weiss et al, 2004). Increased BMI throughout puberty and adolescent, but not childhood BMI, was linked to an increased risk of adult stroke (Ohlsson C, 2017). Before the age of 55, late adolescent is a strong predictor of coronary heart disease and stroke in males (Falkstedt D, et al, 2007). Specific epigenetic marks in children with severe obesity that were not found in children with mild obesity were linked to the "IRS1 target genes" pathway and various cancer traits, including pancreatic cancer, breast tumors, hepatocellular carcinoma, and colon cancer, according to genome-wide methylation analysis (Bendor C et al, 2020).

According to the new IDF definition, for a person to be defined as having the metabolic syndrome they must have Central obesity (defined as waist circumference with ethnicity specific values). It is called metabolic syndrome if abdominal obesity is found (aged 10-16 years, abdominal circumference >90th percentile, age >16 years if abdominal circumference is 94 cm for male and 80 cm for female accompanied by 2 or 4 of the following criteria (IDF, 2018).

3.3 Research Hypothesis

1. There is an association between stunted and obese adolescents
2. There is an association between normal stature (non stunted) and obese adolescent
3. There is an association between stunted in obese adolescents and the risk of metabolic syndrome
4. There is an association between stunted in obese adolescents and the risk of metabolic syndrome

5. There is an association between normal stature in obese adolescents and the risk of metabolic syndrome

CHAPTER 4
RESEARCH METHODS

4.1 Type & Design of the Research

4.1.1 Type of the Research

The type of the research is an analytic observational study.

4.1.2 Design of the Research

The design of the research is a cross sectional study using the medical records from the inpatient of the Child Health Department of Dr. Soetomo Hospital between October 2019 - December 2019.

4.2 Research Population, Sample, Technique and Sample Size

4.2.1 Research population

The population in this study were obese adolescents in Junior High School (SMP) and Senior High School (SMA) in Sidoarjo and Surabaya.

4.2.2 Research sample

The sample is obese adolescents aged 13-18 years who meet the criteria for metabolic syndrome.

4.2.2.1 Inclusion Criteria

1. Obese adolescents aged 13-18 years.
2. Meets the criteria for metabolic syndrome.
3. Children whose parents have agreed to participate in the study sample and have signed an informed consent.

4.2.2.2 Exclusion Criteria

Children who have been diagnosed with diabetes mellitus and are taking anti-diabetic drugs.

4.2.3 Sampling Technique and Sample Size

The sample size is calculated by the formula:

$$n = \frac{(Z_1 - \frac{\alpha}{2}\sqrt{[2P2*(1-P2)]} + Z_1 - \beta\sqrt{[P1*(1-P1)+P2(1-P2)]})^2}{(P1-P2)^2}$$

Information :

n : Sample size

α : Type one error (specified 0.05)

β : Values in the standard normal distribution of the same power (power) = 0.10 is 1.28

P1 : The proportion of polymorphisms in the at-risk group, namely obesity with metabolic syndrome (0.49)

P2 : The proportion of polymorphisms in the non-risk group is obesity without metabolic syndrome (0.32)

$Z_{1-\alpha/2}$: Alpha standard value (based on z table normal curve = 1.96)

$Z_{1-\beta}$: Beta standard value (based on z table normal curve = 0.84)

$$n = \frac{\left[(1{,}96\sqrt{[2(0{,}32)*(1-0{,}32)]} + 0{,}84\sqrt{[0{,}49*(1-0{,}49)+0{,}32(1-0{,}32)]}\right]^2}{(0{,}49-0{,}32)^2}$$

$$n = \frac{(1{,}3606 + 0{,}5743)^2}{0{,}0289}$$

$$n = \frac{(1{,}9349)^2}{0{,}0289} = \frac{3{,}7438}{0{,}0289} = 129{,}5 = 130$$

The calculation of the size of the research subjects based on Lemeshow 1997, the number of research subjects obtained was 130 samples for obesity. So the total sample size is 2n by 260 people because it is estimated that 50% are obese with metabolic syndrome.

4.3 Research Variables and Variable Operational Definition

4.3.1 Research Variables

A. Independent Variables :

- Total cholesterol
- HDL
- LDL
- Triglycerides
- Fasting blood sugar

B. Dependent variable

- Obesity with a BMI over the 95th percentile
- Stunted adolescents with height for age < 2 SD
- Metabolic syndrome using anthropometry based on IDF criteria

4.3.2 Operational Definitions

4.3.2.1 Obesity in Adolescent

Obese adolescents are boys and girls aged 13 to 18 years old who have a BMI over the 95th percentile (P>95) based on an anthropometric evaluation using the CDC (Centers for Disease Control) curve from 2000. The BMI is calculated by multiplying a person's weight in kilograms by their height in meters squared. A brand digital tread scale with a 0.1 kg precision was used to determine body weight. Body weight was determined using a brand digital tread scale with a 0.1 kg precision. Adolescents aged 13-18 years and from anthropometric examination based on the 2000 CDC curve, BMI based on age was obtained with a P value> 95.

Anthropometry is one way of diagnosing obesity by measuring weight in kilograms (kg), height in centimeters (cm), waist circumference (WC) in cm, Waist Hip Ratio (WHR) by measuring hip circumference (Hip). Circumference (HC) in cm Central obesity can be determined based on the measurement of waist circumference and waist hip ratio.

- Body weight was measured standing up using a digital tread scale with the Seca brand with an accuracy of 0.1 kg

• Height was measured by standing using a brand microtoise with an accuracy of 0.1 cm

• Waist circumference (waist circumference) was measured with a metline using a brand seca by measuring the length of the body circumference on the abdomen parallel to the navel

• Hip circumference (hip circumference) was measured with a metline using a brand seca by measuring the widest part of the hips BMI (Body Mass Index) which is calculated from weight in kilograms divided by TB in meters squared

4.3.2.2 Blood Pressure

Arterial pressure when blood is pumped by the heart throughout the body. It was measured using a digital sphygmomanometer (OMRON HealthCare Co. Kyoto Japan) by measuring systolic and diastolic pressure. Hypertension if the results of diastolic and systolic measurements are 90th percentile based on age and sex.

4.3.2.3 Metabolic Syndrome

Metabolic syndrome is a group of health disorders that occur together. The IDF (International Diabetes Foundation) criteria have been met by adolescents with metabolic syndrome. Based on the IDF (The International Diabetic Federation) criteria, central obesity is obtained plus at least 2 or 4 of the following criteria:

For ages 10-16 years:

1. Central obesity with waist circumference 90th percentile according to the WHO waist circumference table for children, male is 90 cm in diameter and female is 80 cm in Asia (WHO waist circumference) accompanied by:
2. Blood pressure 90th percentile (systolic 130/diastolic 85 mmHg)
3. Hypertriglyceridemia ≥ 150 mg/dl
4. HDL levels < 40 mg/dl
5. Fasting blood sugar ≥ 100 mg/dl

For ages over 16 years:

1. Central obesity with waist circumference 90th percentile accompanied by:
2. Blood pressure 90th percentile (systolic 130/diastolic 85 mmHg)
3. Hypertriglyceridemia ≥ 150 mg/dl
4. HDL levels < 40 mg/dl for men

   HDL levels < 50 mg/dl for women
5. Fasting blood sugar ≥ 100 mg/dl

4.3.2.4 Blood Sugar Level

The level of sugar in the serum of research subjects is assessed by extracting venous blood and measuring it in milligrams per deciliter (mg/dl)

using the spectrophotometric method. The fasting blood sugar levels in the serum of research participants who had fasted for 10-12 hours were measured. Both a ratio and an ordinal scale are used to present the data. The cutoff value for fasting blood sugar is 100mg/dL.

4.3.2.5 Total cholesterol level

Total cholesterol level is the total cholesterol level in serum checked by Cholesterol Kit PUREAUTO S CHO-N Sekisui Medical Co., LTD.

4.3.2.6 HDL (High Density Lipoprotein) levels

High Density Lipoprotein (HDL) levels in serum measured by HDL-Cholesterol Kit PUREAUTO S CHO-N Sekisui Medical Co., LTD.

4.3.2.7 LDL (Low Density Lipoprotein) levels

Low Density Lipoprotein (LDL) levels in serum measured by LDL-Cholesterol Kit PUREAUTO S CHO-N Sekisui Medical Co., LTD.

4.3.2.8 Triglyceride level

Triglyceride (TG) level is the level of triglycerides in serum checked with Autosera S TG-N Kit PUREAUTO S CHO-N Sekisui Medical Co., LTD.

4.4 Instrument

The research instrument used for this research is a medical record in the October 2019 to December 2019 meeting the inclusion criteria.

4.5 Research Location and Time

4.5.1 Research location

1. Sampling was carried out in SMP or SMA in Sidoarjo and Surabaya.
2. Blood clinical chemistry examination was carried out at the Kedungdoro Laboratory Surabaya.

4.5.2 Research time

September 2021 to December 2021.

4.6 Data Collection and Processing

4.6.1 Data Collection

The data used is primary data collected based on screening to schools from October 2019 to December 2019.

4.6.2 Data Processing

The data was classified based on the variables as explained in the operational definition and divided into age groups based on the IDF.

4.7 Data Processing and Analysis

4.7.1 Data Processing

The Collected data is then carried out the stages of data processing which includes coding, entry, and cleaning.

1) Coding

This stage is a special code assignment for each variable to facilitate data entry and analysis.

2) Enter Date

Stages of entering data from medical records into a laptop/computer using software

3) Data Cleaning

The process of cleaning the data from possible errors during the analysis so that corrections can be made immediately

Furthermore, the data is grouped based on research variables

4.7.2 Data Analysis

4.7.2.1 Bivariate Analysis

Each of the two variables will be analyzed using bivariate analysis so that the distribution of each variable is known. The result of the data analysis will be displayed in the form of a table containing sums and percentages.

4.7.2.2 Chi-Square Analysis

Chi-Square analysis was carried out on those suspected to be related or correlated. Chi-Square analysis to find the relationship between the independent and dependent variable on a nominal scale.

4.7.2.3 Prevalence Ratio

The prevalence ratio indicates the role of risk factors in the occurrence of effect in cross-sectional studies. Prevalence ratio is a relative risk estimate which is a comparison between disease prevalence (effect) in the group with risk, with prevalence effect in the group without risk, namely by using the help of a table 2x2.

In this study, PR calculation using computerized system assistance. The interpretation of the result is as follows:

1) PR = 1, means that the variable that is suspected as a risk factor does not affect the occurrence of the effect.

2) PR > 1 and the confidence interval range does not include the number 1, meaning that the variable is a risk factor for the onset of the disease.

3) PR < 1 and the confidence interval range does not include the number 1, meaning the factors studied are protective factors, not risk factors.

4.8 Research Ethics

The approval of the research ethics is obtained from the Health Research Ethics Committee of Dr. Soetomo Hospital.

CHAPTER 5
ANALYSIS OF RESEARCH RESULTS

The sample from this study was taken based on previous research with the title "Adiponectin Gene Polymorphism ADIPOQ +45 T>G, ADIPOQ – 11377 C>G and Adiponectin Plasma in Obese Adolescent and The Risk of Metabolic Syndrome". By collecting subjects the research was carried out in 12 junior and senior high schools in Surabaya and Sidoarjo from March 2020 to June 2020.

The table showed in 5.1, from a total of 36 obese adolescents who were willing to be the research sample, 11 obese adolescents met the criteria for metabolic syndrome and 25 other obese adolescents who did not meet the criteria for the metabolic syndrome. There are 11 adolescents with metabolic syndrome, there are 3 adolescents who are stunted and 8 adolescents who are non-stunted. Meanwhile, of the 25 adolescents who did not have metabolic syndrome, 13 were stunted and 12 were non-stunted.

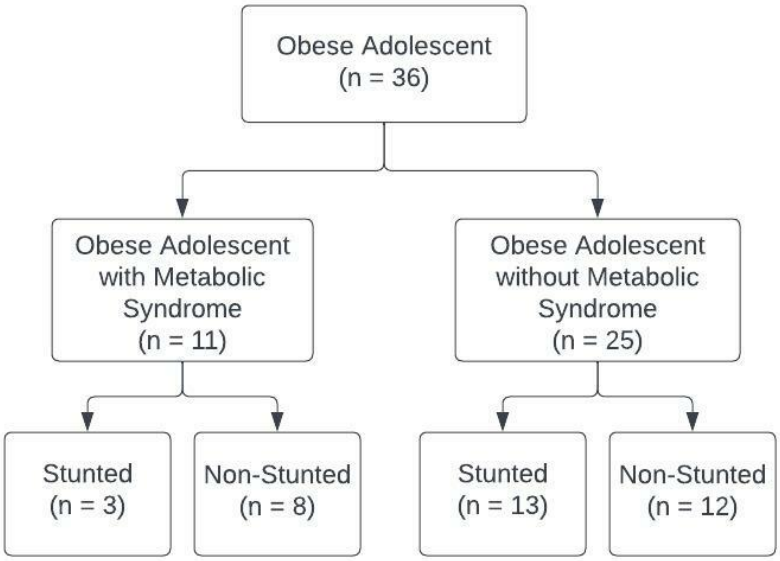


Figure 5.1 Research Subject Flow

5.1 Demographic Data on Stunted Obese Adolescents and Non-stunted Obese Adolescents

Demography of obese adolescents by age and gender are presented in table 5.1.

Table 5.1 Demographic of Research Sample Characteristic

Demographic	Stunted Obese	Non-stunted Obese	P value
	n (%) = 16	n (%) = 20	
Demographics			
Age			
13 - 15 years old	10 (62,5%)	10 (50%)	0.741
16 - 18 years old	6 (37,5%)	10 (50%)	
Gender			
Male	9 (56,25%)	11 (55%)	1.000
Female	7 (43,75%)	9 (45%)	

chi-square test

Demographic data are presented in table 5.1. Age is divided into 13-15 and 16-18 years. For ages 13-15 years, as many as 20 adolescents were obese, with 10 adolescents (62,5%) experiencing stunted and 10 adolescents (50%) experiencing non-stunted. For the age of 16-18 years, as many as 16 adolescents are obese, with 6 adolescents (37,5%) experiencing stunted and 10 adolescents (50%) experiencing non-stunted.

Adolescents with obesity male sex as many as 20 adolescents with 9 adolescents (56,25%) experiencing stunted and 11 adolescents (55%) experiencing non-stunted.

Meanwhile, there were 16 obese adolescent girls with 7 adolescents (43,75%) experiencing stunted and 9 adolescents (45%) experiencing non-stunted.

Table 5.2 Demographic of Research Sample Antrhopometry

Anthropometry	Stunted Obese x ± SD	Non-stunted Obese x ± SD	p-value
BMI	34.5969 ± 5.82403	32.0725 ± 2.50513	0.000
Weight	76.7813 ± 13.51267	86,5300 ± 9.24976	0.042
Height	148.9063 ± 3.90392	163.0750 ± 7.15298	0.010
Blood Glucose	87.5625 ± 10.56389	88.0000 ± 7.34847	0.754
HDL	43.5625 ± 7.11776	42.8500 ± 5.83343	0.599
LDL	113.1250 ± 25.62778	114.1500 ± 38.18690	0.369
Total Cholesterol	168.6875 ± 30.22740	172.3000 ± 39.50896	0.535
TG	98.6250 ± 39.36983	101.6500 ± 64.35124	0.297
Sistole	124.3750 ± 9.46485	121.000 ± 13.72665	0.476
Diastole	81.8750 ± 7.50000	80.2500 ± 12.19092	0.226

one way anova test

Table 5.3 Demographic of Research Sample BMI Normality

Anthropometry	Stunted Obese x ± SD	Non-stunted Obese x ± SD	p-value
BMI	34.5969 ± 5.82403	32.0725 ± 2.50513	0.445

mann-whitney test

Based on the demographic data in table 5.3. Due to the significance of the BMI found *(p=0.000*), it is necessary to test for normality using the Mann Whitney test, when this test is carried out, the relationship between BMI can be determined (*p=0.445*). The average of obese adolescents who are stunted is 34.57 kilograms and adolescents who are not stunted are 32.07 kilograms.

It was found that the average weight of obese adolescents who were stunted was 76.78 kilograms and non-stunted was 86.53 kilograms. Meanwhile, the average height of obese adolescents who were stunted was 148.91 centimeters and non-stunted was 163.08 centimeters. The sugar in the blood of obese adolescents who are stunted is on average 87.56 mg/dL while for obese adolescents who are not stunted it is 88 mg/dL.

The results of this study also found that the mean of obese adolescents had an HDL level of 43.56 mg/dL while for adolescents who were not stunted it was 42.85 mg/dL. For the results of the adolescent LDL levels, the average was 113.13 mg/dL in stunted adolescents and 114.15 mg/dL in non-stunted adolescents. In the data obtained from the total cholesterol of obese adolescents with stunted has an average of 168.69 mg/dL while for obese adolescents without stunted has an average of 172.3 mg/dL.

Triglycerides in obese adolescents were found from the study to have an average of 98.63 mg/dL in stunted adolescents and 101.65 mg/dL for non-stunted adolescents. Obese adolescents also have a mean systolic blood pressure of 124.38 mmHg in stunted adolescents and 121 mmHg in non-stunted adolescents. For diastolic blood pressure alone, the average of obese adolescents who are stunted is 81.88 mmHg and for obese adolescents without stunted it is 80.25 mmHg.

5.2 Association of stunted in Obese Adolescents and Component of Metabolic Syndrome

From 36 adolescents who are obese, the components of the metabolic syndrome were classified according to stunted and non-stunted and presented in table 5.4.

Table 5.4 Association of stunted in obese adolescent and MetS Component

Mets Parameters	Stunted obese n=16	Non-stunted obese n=20	r	p-value
Central obesity	14 (87,5%)	19 (95%)	-0.135	0.418
Hyperglicaemia	1 (6,25%)	1 (5%)	0.027	0.871
Hypertriglycaeride	2 (12,5%)	5 (5%)	-0.157	0.346
Low level HDL	6 (37,5%)	11 (55%)	0.739	0.543
Hypertension	5 (31,25%)	7 (35%)	-0.040	0.813

phi-coefficient

From table 5.4 we can see that adolescent obesity which is classified as central obesity found 36 adolescents, with 14 adolescents (87,5%) experiencing stunted and 19 adolescents (95%) experiencing non-stunted. When viewed from glucose in the blood or fasting blood glucose, there were 2 adolescents, with 1 adolescent (6,25%) stunted and 1 adolescent (5%) non-stunted. Then for excess triglycerides, there were 7 adolescents, with 2 adolescents (12,5%) of whom were stunted and 5 adolescents (25%) were non-stunted. For HDL levels that are still below normal, there are 17 adolescents with 6 adolescents (37,5%) experiencing stunted and 11 adolescents (55%) experiencing non-stunted. The latter is the high blood pressure experienced by obese adolescents based on systolic and diastolic values, it was found that 12 adolescents had hypertension with 5 adolescents (31,25%) stunted and 7 adolescents (35%) experiencing non-stunted.

5.3 Association of stunted in Obese Adolescents and Metabolic Syndrome

Of the 36 samples of obese adolescents obtained, they were further divided into 4 categories, namely metabolic syndrome with stunted, metabolic syndrome without stunted, non-metabolic syndrome with stunted and non-metabolic syndrome without stunted.

Table 5.5 Association of stunted in obese adolescent and MetS

Metabolic Syndrome Status	Stunted obese n=16	Non-stunted obese n=20	r	p-value
Metabolic Syndrome	3 (18,75 %)	8 (40%)	0.229	0.169
Non-Metabolic Syndrome	13 (81,25%)	12 (60%)		

chi-square

Tabel 5.5 identifies differences between the metabolic syndromes of the two groups. Results of the chi-square test for metabolic disorder using three or more risk factors obtained did not show any differences that were statistically significant in the two groups (p=0.169). However, this is different from other research findings that were published by (Shibrina et al., 2017), which concluded that metabolic syndrome episodes occurred more frequently in stunted obesity groups than in nonstunted obesity groups. In the group of people with stunted obesity, 14 adolescents (70%) had metabolic syndrome (three risk factors), while 6 adolescents (30%) did not. In contrast, in the non-stunted obese group, metabolic syndrome was absent in 9 participants (45%) compared to 11 subjects (55%) who had it.

Based on data from table 5.5 there are categories of metabolic syndrome as many as 3 adolescents (18,75%) were stunted and as many as 8 adolescents (40%) were non-

stunted. Meanwhile, for the non-metabolic syndrome category, there were 13 adolescents (81,25%) who were stunted while 12 adolescents (60%) were non-stunted.

5.4 Interaction of stunted in Obese Adolescents and Metabolic Syndrome with Component of Metabolic Syndrome

From all samples obtained, table 5.6 determined the interaction between the components of the metabolic syndrome with 4 categories of metabolic syndrome each mean with stunted, metabolic syndrome with non-stunted, non-metabolic syndrome with stunted and non-metabolic syndrome with non-stunted.

Table 5.6 Interaction of stunted in obese adolescent and MetS with Component

Component of Metabolic Syndrome	Metabolic Syndrome		Non-Metabolic Syndrome		p-value
	Stunted n = 3	Non-stunted n = 8	Stunted n = 13	Non-stunted n = 12	
BMI	43.0300	32.4150	32.6508	31.8442	0.000
Waist circumference	95.8333	96.6250	93.3462	94.5417	0.867
Fasting Plasma Glucose	98.6667	91.7500	85.0000	85.5000	0.031
Triglyceride	147.0000	119.7500	87.4615	89.5833	0.216
HDL	39.3333	40.2500	44.5385	44.5833	0.269
LDL	132.6667	109.1250	108.6154	117.5000	0.670
Systole	131.6667	130.0000	122.6923	115.0000	0.015
Diastole	86.6667	88.7500	80.7692	74.5833	0.011

one way anova

Data table 5.6 shows that each component of the metabolic syndrome is classified into several categories then all nominal data are calculated to find the average of all variables. Then from these data it is possible to determine the average of each component as well as its significance to the metabolic syndrome and stunted categories.

CHAPTER 6
DISCUSSION

6.1 Demographic Data on Stunted Obese Adolescents

In this study, the age of the sample was categorized into two groups, namely the age of 13-15 years and 16-18 years. This is because HDL levels have different minimum thresholds for women aged 16-18 years (<50mg/dL) and women aged 13-15 years (<40mg/dL) based on IDF criteria. The age of the sample in this study was younger than similar studies. In comparison to individuals in the age group of 25 to 34 years, participants in the age group of 35 to 44 years had a risk of Metabolic Syndrome of 1.84 times (Sihombing and Tjandraini, 2015).

From this study, there were more male adolescents (55.56%) than girls (44.44%). According to the findings of several studies on the condition, women are more likely than males to have metabolic syndrome, and this prevalence is quickly rising in the United States. But men are more likely than women to have metabolic syndrome in Europe. According to the findings of a related study, syndrome metabolic was 17.5% common, with women being more likely to have it (21.3%) than men (12.9%), although there has never been any published research on the prevalence of syndrome metabolic in Indonesia (Bantas et al., 2012).

Based on this study, the average weight of stunted and non-stunted obese adolescents is 81.65 Kilograms. According to research conducted to check the content of obese adolescents as much as 37% of the energy consumed by obese, stunted adolescents comes from foods high in calories but poor in micronutrients (Puspitasari et al., 2018).

From this study, it was also found that the average height of obese adolescents was 155.99 Centimeters, this could have an effect on merache in female adolescents. Every adolescent girl experiences menarche at a different age, which is a marker of the onset of puberty (10 to 15 years). Adolescent girls with stunted growth experience menarche on average much later than girls with normal growth (Amaliah et al., 2012). By slowing down the period of menacrhe, it can affect the slowing down of the growth process in these adolescent girl.

6.2 Association of stunted in Obese Adolescents and Component of Metabolic Syndrome

Based on this study showed by table 5.3, almost all samples of obese adolescents had waist circumferences that had been determined as components of the metabolic syndrome, namely 14 of 16 obese adolescents with metabolic syndrome and 19 of 20 obese adolescents with non-metabolic syndrome. Waist circumference is a requirement that must be possessed by someone to be called a metabolic syndrome in male (> 90 Cm) and female (> 80 Cm) (WHO, 2018). Greater relative weight loss in patients participating in a behavioral, intensive weight management program was associated with greater improvement in the metabolic syndrome component. Assessing the effect of weight loss on metabolic risk may benefit from tracking changes in WC during a weight loss program (Rothberg et al., 2017).

Based on the research shown there are 16 obese adolescents who experience stunted, where 1 out of 16 obese adolescents (6,25%) with stunting have high FPG. Meanwhile, 15 out of 16 obese adolescents (93.75%) experienced stunting with normal FPG. Research conducted in Africa was 184 obese adolescents, 18 adolescents (9.78%) of

the participants had a fasting plasma glucose level 100 mg/dL, while 166 adolescents (90.21%) had normal fasting plasma levels < 100 mg/dL (Kruger et al., 2013). While for obese adolescents who are not stunted but have low FPG as many as 1 out of 20 adolescents (5%) and for obese adolescents of non stunted with normal FPG as many as 19 adolescents (95%) This study is consistent with studies that was done in Bitung. According to the research, out of 95 obese adolescents, 6 (6.3%) had high FPG levels l, whereas 89 (93.7%) had normal FPG levels (Dieny et al., 2020).

In this study, there were 16 obese adolescents who were stunted, of which 2 out of 16 obese adolescents (12.5%) with stunted had high triglycerides. Meanwhile, 14 out of 16 obese adolescents (87.5%) were stunted with normal triglycerides. In another study (Afifah Y N, 2016) which discussed triglycerides in obese adolescents with stunted, 8 adolescents (30.77%) had high triglyceride levels while 18 adolescents (69.23%) had normal triglyceride levels. This research is still classified as relevant to the data that has been obtained. While for obese adolescents who are not stunted but have high trygliceride as many as 5 out of 20 adolescents (25%) and for obese adolescents of normal stature without metabolic syndrome as many as 15 adolescents (75%). This study is similar to the results of a study conducted in Jepara, specifically in junior high and high school children, 40 samples of obese adolescents were taken, 15 adolescents (37.5%) had high triglyceride levels while 25 adolescents (62.5%) others had high triglyceride levels. triglyceride levels under normal conditions (Putri LP 2015).

There are 16 obese adolescents who experience stunting, where 6 out of 16 obese adolescents (37.5%) with stunting have low HDL. Meanwhile, 10 out of 16 obese adolescents (62.5%) experienced stunting with normal normal. In another study discussing

HDL in obese adolescents with stunted, 19 adolescents (73.08%) had HDL levels (≤40 mg/dL) while 7 adolescents (26.92%) had normal levels of triglycerides or (>40 mg/dL) (Afifah Y N, 2016). This has different results due to the different measurement bases with the IDF criteria measures where HDL levels are low (defined as < 1.04 mmol/L [40 mg/dL] in men or < 1.29 mmol/L [50 mg/dL] in women) (IDF 2006). While for obese adolescents who are not stunted but have low HDL as many as 11 out of 20 adolescents (55%) and for obese adolescents of non stunted with normal HDL as many as 9 adolescents (45%). This study is consistent with studies that was done in Bitung. According to the research, out of 50 obese adolescents, 31 (62%) had HDL levels below normal, whereas 19 (38%) had normal HDL levels (Senduk et al., 2016).

Based on the data, There were 16 obese adolescents who were stunted, of which 5 out of 16 obese adolescents (31,25%) with stunted had hypertension. Meanwhile, 11 out of 16 obese adolescents (68,75%) were stunted with normal blood pressure. Another study stated that of 11 obese adolescents with metabolic syndrome, 9 (81.8%) of them were included in hypertension, while 2 other adolescents (18.2%) did not include hypertension (Rahman L. H. 2016). While for obese adolescents who are not stunted but have hypertension as many as 7 out of 20 adolescents (35%) and for obese adolescents of normal stature with normal blood pressure as many as 13 out of 20 adolescents (65%). Another study stated that from 57 samples, 46 obese adolescents (71.9%) had hypertension, while 11 obese adolescents (17.2%) had normal blood pressure (Fitriana et al., 2012). Meanwhile in another study, there was no difference between adolescents who were stunted and those who had grown to a healthy height as young children in terms of the prevalence of high systolic/diastolic blood pressure. Adolescents hypertension and stunting are not related in

any way, according to research written by (Rachmi et al., 2016). Regarding the potential link between childhood stunting and high blood pressure in adolescence, regardless of weight status, there is scant data.

6.3 Association of stunted in Obese Adolescents and Metabolic Syndrome

Tabel 5.4 identifies differences between the metabolic syndromes of the two kelompoks. Results of the chi-square test for metabolic disorder using three or more risk factors obtained did not show any differences that were statistically significant in the two groups (p=0.169). However, this is different from other research findings that were published by (Shibrina et al., 2017), which concluded that metabolic syndrome episodes occurred more frequently in stunted obesity groups than in nonstunted obesity groups. In the group of people with stunted obesity, 14 adolescents (70%) had metabolic syndrome (three risk factors), while 6 adolescents (30%) did not. In contrast, in the non-stunted obese group, metabolic syndrome was absent in 9 participants (45%) compared to 11 subjects (55%) who had it.

6.4 Interaction of stunted in Obese Adolescents and Metabolic Syndrome with Component of Metabolic Syndrome

In table 5.5, it can be seen that the average obese adolescent with metabolic syndrome and stunted is the largest in the components of BMI, FPG, TG, HDL, LDL, and Systole. It can also determined that the significance of BMI (p=0.000), fasting plasma glucose (p=0.031), systole (p=0.015), diastole (p=0.011) showed significant and significant results.

BMI is one of the factors that can determine a person is obese or not. In this study, the average BMI of the entire sample of obese adolescents was 34,99 kg/m^2, with an average of of metabolic syndrome adolescents experiencing stunted 43,03 kg/m^2 and for metabolic syndrome without stunting as much as 32,65 kg/m^2. Severe obesity has become more prevalent over time, especially among younger people. This may be explained by the fact that fast food restaurants, which serve unhealthy items like hamburgers, fried chicken, and pizza, have been rapidly increasing in popularity since the mid-1980s (Oh SW, 2011).

CHAPTER 7
CLOSING

7.1 Conclusion

1. In this study, it can be said that there is an insignificant association between obese adolescents who are stunted and the risk of metabolic syndrome.
2. From this study conducted on 36 samples of obese adolescents from the age of 13 to 18 years, it was found that 11 adolescents had metabolic syndrome according to IDF criteria. From 36 samples, 16 of them were stunted (<-2SD) according to the CDC's growth chart.
3. Obese adolescents with stunted cannot be used as a predictor of metabolic syndrome risk.
4. Blood Pressure, BMI, and FPG are parameters that have significant p-value results in stunted obese adolescents compared to obese adolescents without stunted.

7.2 Suggestion

1. The public is advised to be more aware of child malnutrition, especially during the growth and development period where the amount of nutrients that enter must be adjusted to the needs of sufficient calories in each phase of growth so as not to be stunted and also obese.
2. The results of the study of obese adolescents who experience stunted can be used as evaluation material so that in the future it can be a concern for all health workers and the government to pay attention to nutritional adequacy in children before adolescence.
3. Further research needs to be done with a larger number of samples to better understand the incidence of stunted adolescents and the risk of metabolic syndrome.

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ATTACHMENT

1. Schedule of Activities

Activities	2021									2022	
	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Jan	Feb
Proposal Preparation											
Proposal Seminar											
Proposal Revision											
Licensing Research											
Data Collection											
Data Processing											
Preparation of Research Report Manuscript											

Result Seminar											

2. Data Collection Sheet

01. No Rekam Medik :		**02. Nama Pasien :**	
03. Kelamin : ▫ **Laki laki** ▫ **Perempuan**	**04. Umur ……. thn**	**05. Berat Badan ……. Kg**	**06. Tinggi Badan ……. cm**
07. Diagnosa MRS/ Diagnosa Kerja	**08. Tanggal Keluar Rumah Sakit**	**09. Lama Perawatan**	
10. Diagnosa MRS/Diagnosa Kerja			
11. Alasan Masuk Rumah Sakit **1. Observasi** **4. Terapi Initial** **2. Skreening** **5. Terapi Komplementari** **3. Diagnosis** **6. Terapi Sekunder** **7. …….** **8. …….**		**12. Alasan Keluar Rumah Sakit** **1. Tujuan MRS telah selesai** **2. Dipindahkan ke RS lain** **3. Pulang paksa** **4. Pulang berobat jalan** **5. Meninggal** **6. …….**	
13. Diagnosa Akhir ● **Utama :** ● **Komplikasi : 1.** **2.** **3.** ● **Sekunder :**			

3. SPSS output Age and gender demographics

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.206[a]	1	.650		
Continuity Correction[b]	.013	1	.910		
Likelihood Ratio	.206	1	.650		
Fisher's Exact Test				.741	.456
Linear-by-Linear Association	.200	1	.655		
N of Valid Cases	36				

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 6.67.

b. Computed only for a 2x2 table

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.006[a]	1	.940		
Continuity Correction[b]	.000	1	1.000		
Likelihood Ratio	.006	1	.940		
Fisher's Exact Test				1.000	.604
Linear-by-Linear Association	.005	1	.941		
N of Valid Cases	36				

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 7.11.

b. Computed only for a 2x2 table

4. SPSS output Anthropometry and the metabolic component of the syndrome

Group Statistics

	Stunted	N	Mean	Std. Deviation	Std. Error Mean
IMT	stunted	16	34.5969	5.82403	1.45601
	tidak stunted	20	32.0725	2.50513	.56016
BB	stunted	16	76.7813	13.51267	3.37817
	tidak stunted	20	86.5300	9.24976	2.06831
TB	stunted	16	148.9063	3.90392	.97598
	tidak stunted	20	163.0750	7.15298	1.59946
Hiperglikemia	stunted	16	1.9375	.25000	.06250
	tidak stunted	20	1.9500	.22361	.05000
HDL	stunted	16	43.5625	7.11776	1.77944
	tidak stunted	20	42.8500	5.83343	1.30440
LDL	stunted	16	113.1250	25.62778	6.40695
	tidak stunted	20	114.1500	38.18690	8.53885
Kolesterol_Total	stunted	16	168.6875	30.22740	7.55685
	tidak stunted	20	172.3000	39.50896	8.83447
TekDarah_Sistole	stunted	16	124.3750	9.46485	2.36621
	tidak stunted	20	121.0000	13.72665	3.06937
TekDarah_Distole	stunted	16	81.8750	7.50000	1.87500
	tidak stunted	20	80.2500	12.19092	2.72597

5. SPSS output Interaction of stunted in obese adolescent and MetS with Componen

Descriptives

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
						Lower Bound	Upper Bound		
IMT	MetS stunted	3	43.0300	1.83510	1.05950	38.4714	47.5886	41.57	45.09
	MetS non-stunted	8	32.4150	1.73611	.61381	30.9636	33.8664	30.30	35.15
	Non-MetS stunted	13	32.6508	4.46717	1.23897	29.9513	35.3503	26.80	40.06
	Non-MetS Non-Stunted	12	31.8442	2.96303	.85535	29.9615	33.7268	29.20	40.13
	Total	36	33.1944	4.42290	.73715	31.6979	34.6909	26.80	45.09
Waist_Circum	MetS stunted	3	95.8333	4.07226	2.35112	85.7173	105.9494	93.00	100.50
	MetS non-stunted	8	96.6250	10.32248	3.64955	87.9952	105.2548	80.00	111.00
	Non-MetS stunted	13	93.3462	10.03408	2.78295	87.2826	99.4097	80.50	112.00
	Non-MetS Non-Stunted	12	94.5417	7.13970	2.06106	90.0053	99.0780	86.00	107.00
	Total	36	94.6806	8.62815	1.43803	91.7612	97.5999	80.00	112.00
Glukosa_Puasa	MetS stunted	3	98.6667	23.45918	13.54417	40.3908	156.9425	80.00	125.00
	MetS non-stunted	8	91.7500	9.23889	3.26644	84.0261	99.4739	83.00	112.00
	Non-MetS stunted	13	85.0000	3.13581	.86972	83.1050	86.8950	81.00	91.00
	Non-MetS Non-Stunted	12	85.5000	4.68071	1.35121	82.5260	88.4740	80.00	93.00
	Total	36	87.8056	8.78577	1.46429	84.8329	90.7782	80.00	125.00
Trigliserida	MetS stunted	3	147.0000	24.75884	14.29452	85.4956	208.5044	128.00	175.00
	MetS non-stunted	8	119.7500	83.37823	29.47865	50.0441	189.4559	17.00	260.00
	Non-MetS stunted	13	87.4615	33.39565	9.26229	67.2808	107.6423	52.00	171.00
	Non-MetS Non-Stunted	12	89.5833	48.28788	13.93951	58.9027	120.2640	32.00	222.00
	Total	36	100.3056	53.98720	8.99787	82.0389	118.5722	17.00	260.00
HDL	MetS stunted	3	39.3333	13.20353	7.62306	6.5339	72.1327	25.00	51.00
	MetS non-stunted	8	40.2500	5.62520	1.98881	35.5472	44.9528	33.00	47.00
	Non-MetS stunted	13	44.5385	5.36370	1.48762	41.2972	47.7797	37.00	57.00
	Non-MetS Non-Stunted	12	44.5833	5.51788	1.59287	41.0774	48.0892	37.00	54.00
	Total	36	43.1667	6.34935	1.05823	41.0184	45.3150	25.00	57.00
LDL	MetS stunted	3	132.6667	11.54701	6.66667	103.9823	161.3510	126.00	146.00
	MetS non-stunted	8	109.1250	33.54075	11.85845	81.0842	137.1658	47.00	163.00
	Non-MetS stunted	13	108.6154	26.10089	7.23908	92.8428	124.3880	76.00	145.00
	Non-MetS Non-Stunted	12	117.5000	42.09837	12.15275	90.7520	144.2480	68.00	212.00
	Total	36	113.6944	32.76219	5.46037	102.6093	124.7796	47.00	212.00
TekDarah_Sistole	MetS stunted	3	131.6667	7.63763	4.40959	112.6938	150.6396	125.00	140.00
	MetS non-stunted	8	130.0000	14.14214	5.00000	118.1769	141.8231	120.00	160.00
	Non-MetS stunted	13	122.6923	9.26809	2.57050	117.0917	128.2930	110.00	140.00
	Non-MetS Non-Stunted	12	115.0000	10.00000	2.88675	108.6463	121.3537	100.00	130.00
	Total	36	122.5000	11.98213	1.99702	118.4458	126.5542	100.00	160.00
TekDarah_Distole	MetS stunted	3	86.6667	5.77350	3.33333	72.3245	101.0088	80.00	90.00
	MetS non-stunted	8	88.7500	13.56203	4.79490	77.4119	100.0881	60.00	100.00
	Non-MetS stunted	13	80.7692	7.59555	2.10663	76.1793	85.3592	70.00	90.00
	Non-MetS Non-Stunted	12	74.5833	7.21688	2.08333	69.9979	79.1687	60.00	80.00
	Total	36	80.9722	10.26919	1.71153	77.4976	84.4468	60.00	100.00

6. Demographic of Research Sample Characteristic of BMI with mann-whitney test

Test Statistics[a]

	IMT
Mann-Whitney U	136.000
Wilcoxon W	346.000
Z	-.764
Asymp. Sig. (2-tailed)	.445
Exact Sig. [2*(1-tailed Sig.)]	.459[b]

a. Grouping Variable: Stunted

b. Not corrected for ties.