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Submission date: 19-Oct-2021 11:21AM (UTC+0800)
Submission ID: 1677730063
File name: th_Hormone_Status_In_Obese_Subjects_and_Correlation_With_Age.pdf (154.32K)
Word count: 3068
Character count: 16464

STRADA Jurnal Ilmiah Kesehatan
DOI: 10.30994/sjik.v9i2.300
ISSN: 2252-3847 (print); 2614-350X (online) Vol.9 No.2 November 2020 Page.368-373

Growth Hormone Status In Obese Subjects and Correlation With Age

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ABSTRACT
Obesity is a global problem that is very dangerous for health, because obesity increases the risk of disability, complications and premature death. In addition, obesity also increases the risk of decreased Growth Hormone (GH) secretion. Decreased GH secretions have an impact on increasing body fat and decreasing lean body mass. This study aims to analyze the status of Growth Hormone (GH) in obese subjects and the correlation of GH with age. This study used a cross sectional study method using 30 obese female adolescent Body Mass Index (BMI) subjects 25-35 kg/m2, Percentage of Body Fat (PBF) above 30% and fasting blood glucose (FBG) below 100 mg/dL. Measurement of GH levels used the Enzym Link Immunosorbent Assay (ELISA) method. Data analysis techniques used the Pearson Correlation test with the Statistical Package for Social Science (SPSS). Results obtained mean GH levels (761.119±504.627) pg/mL and average age (20.863±1.082) years (r = -0.483) and (p=0.023). Based on the results of the study, it can be concluded that there is a negative correlation between GH status and age in obese subjects.

Keywords: Age, Growth hormone status, Obesity

Received May, 26, 2020; Revised June17, 2020; Accepted June 30, 2020

STRADA Jurnal Ilmiah Kesehatan
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BACKGROUND

Obesity is one of the main problems in public health, which occurs in BMI reaching 25-35 kg/m^2 (Budipitojo *et al*., 2016). It is estimated that more than 15% of the population in European countries such as Britain, Germany and Poland are obese (Ostman *et al*., 2004). The prevalence rate of obesity in the United States is estimated at 31% (Hedley *et al*., 2002). In Indonesia, the prevalence of obesity shows a tendency to increase from year to year (Budipitojo *et al*., 2016). Based on the results of the Basic Health Research (Riskesdas) in 2018, it's showed that the prevalence of obesity at the age of 18 years old reached 21.8%. This amount is higher than in 2013 (14.8%) and 2007 (10.5%) (Riskesdas, 2018). The high prevalence of obesity is a global problem that will threaten public health (Nimptsch *et al*., 2019).

Obesity is a global health problem that must be considered because obesity increases the risk of disability (Kibria, 2019), complications (Akter *et al*., 2014) and premature death (Rosella *et al*., 2019). In addition, obesity also increases the risk of chronic non-communicable diseases, such as type 2 diabetes mellitus, several types of cancer (Nimptsch *et al*., 2019), hypertension and cardiovascular disease (CVD) (Gadde *et al*., 2018). The cause of obesity is multifactorial but a common factor that contributes to weight gain in adolescents is an imbalance between energy intake and energy expenditure (Fan and Evans, 2017; Kurdanti *et al*, 2015). Obesity in adolescents is associated with adverse effects on health quality including metabolic complications involving many cytokines and hormones (Rambhojan *et al*., 2015). According to Thomas *et al*. (2013) explained that obesity interferes with the physiological and pathological functions that regulate and suppress the secretion of Growth Hormone (GH). Decreased GH secretion has an impact on increasing body fat and decreasing lean body mass (Kreitschmann-Andermahr *et al*., 2009).

GH is one of the hormones that regulate the body's metabolism, including lipid metabolism. GH can regulate fat levels in tissues and blood lipid profiles. Decreased GH levels are associated with increased body fat, which is characterized by the occurrence of obesity (Ratnayanti, 2012). Obesity induces hyper-insulinemia, hypo-adiponectinemia, and hyper-leptinemia, reduced serum ghrelin and increased levels of free fatty acids (FFA), this causes inhibition of GH secretion from the pituitary (Savastano *et al*., 2014). GH secretion is regulated centrally by the hypothalamus hormone, which is Growth Hormone Releasing Hormone (GhRH) and somatostatin. GhRH functions to stimulate GH production, while somatostatin inhibits GH secretion (Ratnayanti, 2012). The biological activity of GH indicates that it is an important precursor for the benefit of changes in body fat and lean tissue mass in obesity. GH is influenced by physiological and pathological factors such as age, sex, body composition, sleep quality, nutritional status and changes in respiratory patterns such as hyperventilation. These factors can significantly change GH secretion (Thomas *et al*., 2013). Research by (Savastano *et al*., 2006) results in a negative correlation between GH status and age in obese subjects (Savastano *et al*., 2006). Based on this background, it's concluded that the correlation between GH levels with age still needs more complete discussion. Therefore we need an analysis of the correlation of GH status with age in obese women subjects.

METHOD

This study used a cross sectional study method using 30 obese female adolescent BMI 25-35 kg/m^2, PBF above 30% and FBG below 100 mg/dL. All of these research procedures

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were approved by the Health Research Ethics Commission of the Faculty of Medicine, Airlangga University, Surabaya number 309/EC/KEPK/FKUA/2019.

Blood sampling was carried out in 4 ml cubital veins. The blood was centrifuged for 15 minutes at 3000 rpm. The serum was separated and stored at -80 ° C for analysis of GH levels the following day. Blood sampling was done at 07.00-09.00 WIB. Measurement of GH levels used ELISA kit (Catalog No. E-EL-H0177; Elabscience, Inc., China, 2019) with a unit of concentration of pg/mL. The standard curve range is 78.13-5000 pg/mL and the level of GH sensitivity in the kit is 46.88 pg/mL. Height measurements used a stadiometer (SECA, Chino, CA). Measurement of body weight, BMI and PBF used TANITA (Body Composition Analyzer DC3607601 (2) -1604 FA, TANITA Corporation of America, Inc.).

Subject taking techniques used consecutive sampling techniques. Statistical analysis using statistical software packages for social science (SPSS). The normality test uses the Shapiro-Wilk test. Data that were normally distributed were tested using Pearson correlation with a significant level (P<0.05). All data are displayed with mean ± SD.

RESULT

The results of descriptive analysis of the research subjects' characteristics can be seen in table 1 below.

Table 1. Research Subjects' Characteristics

Variable	n	Mean	SD
Height (m)	30	1.572	0.047
Weight (kg)	30	72.413	7.271
BMI (kg/m^2)	30	29.113	1.719
PBF (%)	30	44.731	2.999
FBG (mg/dL)	30	90.090	6.989

Based on table 1, it can be seen that the average height of the subjects was 1.572 m with a body weight of 72.413 kg, BMI of 29.113 kg/m^2, PBF of 44.731% and an average FBG of 90.090 mg/dL. The results of the correlation analysis of GH status with age in obese subjects can be seen in table 2.

Table 2. Correlation between GH and Age

Variable	n	Mean±SD	Pearson r	*P*-value
GH (pg/mL)	30	761.119±504.627	-0.483	0.023*
Age (years)	30	20.863±1.082		

Based on table 1 Pearson correlation test results indicate that there is a correlation of GH status with age in obese subjects with a negative correlation direction (r = -0.483) and (p=0.023).

DISCUSSION

Based on Table 2, it can be seen that there is a correlation between GH status and age. The correlation between GH status and age shows the direction of the negative correlation. In line with previous studies it was produced that there was a negative correlation between GH status and age in obese subjects (Savastano *et al*., 2006). As we get older, the prevalence of obesity increases. Increasing age will increase total body fat content, especially the distribution of central fat. The prevalence of obesity was found to be higher

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in subjects with older age. At an older age there is a decrease in muscle mass and changes in several types of hormones that trigger the accumulation of abdominal fat. Obesity can cause health problems, such as type 2 diabetes mellitus, dyslipidemia, cardiovascular disease, hypertension, several types of cancer, sleep apnea and metabolic syndrome (Tchernof and Despres, 2013). In addition, obesity can also cause insulin resistance (Boden, 2011). Increased insulin resistance occurs simultaneously with increased levels of fat in the body (Puspitasari, 2018). This explanation shows that the prevalence of obesity was found to be higher in samples with older age, but in this study subjects aged 19-23 were already obese. This indicates that obesity does not recognize age. So obesity at the age of 19-23 years old also has the same risk as the age above it. In addition, Thomas *et al*. (2013) explained that obesity affects GH secretion. According to Pangkahila (2011) in normal people aged 25-30 years old called the subclinical stage of aging there is a decrease in GH levels in addition to estrogen and testosterone. As we age, the amplitude of the GH pulse also decreases. GH secretions are reduced by 50% every seven years after the age of 18-25 years old. At the age of 35-45 years old hormone levels decrease to 25%. In the next stage, that is at the age of 45 years old and over or the clinical stage, the reduction in GH levels continues (Pangkahila, 2011).

GH secretion in obesity is also influenced by ghrelin secretion. Ghrelin is a peptide hormone that regulates food intake and stimulates GH secretion (Budipitojo *et al*., 2016). Ghrelin is a very important factor in energy homeostasis, a molecule that is oxygenated (increases appetite) and adipogenic (increases fat formation) strong (Ueno *et al*., 2005). Giving this hormone will stimulate appetite, food intake and increase body weight. Circulating levels of ghrelin decrease after eating and increase before meals (Cummings, 2006). This protein stimulates appetite through the hypothalamic arcuate nucleus (Kojima *et al*., 2004). The arcuate nucleus is also a target site for leptin, an appetite suppressant hormone from fat tissue (Cummings, 2006). Ghrelin circulating in the circulation can activate neurons that express neuropeptide Y (NPY) and agouti-related peptide (AGRP) in the arcuate nucleus (Ueno *et al*., 2005; Kojima *et al*., 2004). NPY and AGRP stimulate food intake (Kojima *et al*., 2004). Giving ghrelin continuously can increase body weight (Cummings, 2006). In obesity there is a reduction in the activity of ghrelin production possibly due to increased insulin secretion as a result of the positive energy balance process. Therefore, obese people experience a disruption in ghrelin production. If ghrelin is disrupted, it will affect the stimulation of GH secretion, so GH secretion is also disturbed in obese subjects.

In obese subjects, GH secretion and stimulation are impaired; includes responses to all traditional pharmacological stimuli acting on the hypothalamus, such as insulin-induced hypoglycemia or arginine (ARG), and to direct the stimulation of somatotropin by exogenous GhRH (Savastano *et al*., 2014). The highest GH secretion levels occur 1-4 hours after starting to sleep (for 3 and 4). Peak secretions at night, when sleeping reaches 70% of GH secretions a day, the amount is greater in children and decreases in the elderly. Glucose input will not inhibit this expenditure. Stress, emotional, physical and chemical, trauma, physical exercise, therapy and administration of pyrogen can stimulate GH expenditure (Rusli, 2010). GH secretion is associated with a state of insulin resistance, such as obesity, non-alcoholic fatty liver disease (NAFLD) and type-2 diabetes, associated with low GH secretion. Because GH can contribute to insulin resistance and develop when calorie supply exceeds demand, a reduction in GH secretion that occurs with obesity can be an adaptive phenomenon to prevent insulin resistance. However, the reduction in GH secretion can further increase fat accumulation by reducing lipolysis thereby worsening the

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condition of obesity (Savastano *et al*., 2014). Other interactions related to GH and adipose tissue are related to the activity of the steroid 11b-hydroxy enzyme type 1 dehydrogenase (11b-HSD-1), which catalyzes the conversion of inactive cortisone to active cortisol. Because GH inhibits 11β-Hydroxysteroid dehydrogenase type-1 (11β-HSD-1), express in adipose and liver tissue (Agha and Monson, 2007).

CONCLUSION

Based on the results of the study, it can be concluded that there is a negative correlation between GH levels with age in obese adolescent girls. Based on the results of the study it is recommended to do further research by using more subjects to strengthen the findings of this study. Future research is recommended to correlate GH status with age in underweight, normal weight, overweight and obesity subjects.

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