The Effect of Duration of Ramadan Fasting on Food Intake, Lipid Profiles, and Pro-inflammatory Cytokines (TNF-α and IL-6) in Overweight Male Subjects in Malang

Thesis

To fulfill the requirements of Master Degree in

Agriculture Product of Technology

Submitted by

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DEDICATION

I dedicate this thesis

The spirit of my father the loving memory

To my Father, who first to teach me

MOHAMMED ZEKRI

And

To my beloved Mother, for her silent prayers

SASIA SAEED ALMOKADMY

To my Husband, for care and support all the time and how the Guide for me.

To my brothers and sisterfor their love, endless support and encouragement.,

And to my Children Lamis, Malk, Lamar and Mohammed

You have successfully made me the person I am becoming

You will always be remembered

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In the name of Allah, the Most Gracious and the Most Merciful.

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Thesis

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Ringkasan

Penelitian ini bertujuan untuk melihat pengaruh durasi puasa Ramadan terhadap asupan makanan, BMI, profil lipida plasma (TG, TC, HDL dan LDL), dan penanda inflamasi (TNF α dan IL-6) pada pria dengan kelebihan berat badan. Metode penelitian yang digunakan dalam penelitian ini adalah metode penelitian eksperimen dengan desain penelitian satu kelompok pre-test-post-test. Populasi dalam penelitian ini adalah pria Indonesia yang berumur 20 sampai 30 tahun, tidak memiliki riwayat diabetes dan hipertensi, serta bukan atlet dan memiliki indeks masa tubuh berkisar 25- 30 kg/m^2. Jumlah Responden yang diteliti adalah 23 orang. Penelitian dilakukan pada bulan Ramadan. Pengamatan terhadap Responden dilakukan 7 hari sebelum puasa, 14 hari selama berpuasa Ramadan dan 21 hari selama berpuasa Ramadan atau menjelang berakhirnya puasa Ramadan. Parameter yang diamati adalah asupan makanan, IMT analisis darah berupa profil lipid (TG, TC, HDL, dan LDL), dan penanda inflamasiyaitu IL6 dan TNF-α. Data asupan kalori, IMT dan profil lipid dianalisis secara statistika menggunakan metode Repeated Anova, sedangkan data IL-6 dan TNF-αmenggunakan *pairedt test*, korelasi antar parameter dihitung dengan metode Rank Spearman.

Hasil penelitian menunjukkan bahwa puasa Ramadan mampu menurunkan asupan makanandan IMT secarasignifikan.Kolesterol total dan LDL berbeda nyata. Sedangkan TG dan HDL tidak berbeda nyata.Selanjutnya, puasa Ramadantidak menyebabkan perubahan yang nyata padaTNF-αdan IL6.

Kata Kunci : puasa Ramadan, asupan makanan, BMI, lipid plasma, penanda inflamasi

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Summary

This study aims to investigate duration of Ramadan fasting on the daily intake of total foods, BMI, plasma lipid profiles (TG, TC, HDL and LDL)before Ramadan fasting, 14 days of Ramadan fasting, and 21 days of Ramadan fasting, and inflammatory marker (TNF α and IL-6)before Ramadan fasting, and 21 days of Ramadan fasting of overweight male subjects. The research method used in this study is an experimental research method with one group pre-test-post-test design. The respondent for this research is Indonesian male between 20 to 30 years old, do not have a history of diabetes and of hypertension, not an athlete and having body mass index of 25-30 kg/m^2.

The number of Respondents is 23 men. The study was conducted in the month of Ramadan. Observations of the respondents were done on 7 day before fasting, 14 days during fasting and 21 days during Ramadan or before the end of the Ramadan fast. Parameters measured were food intake, anthropometric, blood analysis in the form of a lipid profile (TG, TC, HDL and LDL), and inflammatory markers of IL6 and TNF-α. Data food intake, anthropometric and lipid profiles were analyzed statistically using repeated anova, while data IL6 and TNF-αusing paired t.The correlation between the parameters is done by Spearman Rank method.

The results showed that the fasting of Ramadan is able to lower the food intake significantly. Moreover, the BMI value was significant different. Total Cholesterol, and LDL were significant different at 14 days of Ramadan fasting. The TG and HDL were not significant different. Moreover,TNF-α and IL-6 were not significant different..

Keywords : Ramadan fasting, Food Intake, BMI, lipid profile, pro-inflammatory cytokines

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LIST OF ABBREVIATIONS

AHI : Apnoea-Hypopnoea Index
ANOVA : Analysis of Variance
Apo A : Apolipoprotein A
Apo B : Apolipoprotein B
ASP : Aspartate
BDNF : Brain-derived Neurotropic Factor
BMI : Body Mass Index
BW : Body Weight
CETP : Triglyceride Ester Transfer Protein
CHOD-PAP : Cholesterol Oxidase - phenol + aminophenazone
CI : Convidence Interval
CRP : C-Reactive Protein
DM : Diabetes Mellitus
DM : Diabetes Mellitus
ELISA : Enzyme-linked Immunosorbent Assay
FPG : Fasting Plasma Glucose
GLUT-4 : Glucose Transporter type 4
GPO-PAP : Gliserolphosphat Oksidase-phenol + aminophenazone
HDL : High Density Lipoprotein
HDL-C : High Density Lipoprotein Cholesterol
HGH : Human Growth Hormone
Response
IFG : Impaired Fasting Glucose
IGF-1 : Insulin-like Growth Factor-1
IL-6 : Interleukin-6
LDL : Low Density Lipoprotein
LDL-C : Low Density Lipoprotein Cholesterol
LPL : Lipoprotein Lipase
LSD : Least Significant Difference
Mg : Magnesium
mRNA : messenger Ribonucleic Acid
OR : Odd Ratio
OSA : Obstructive Sleep Apnea
p : Probability
PAF : Population Attributable Fraction
PAI-1 : Plasminogen Activator Inhibitor type 1
PPAR-gamma : Peroxisome proliferator-activated receptor gamma
SuRFNCD : Surveillance of Risk Factors of Non-communicable Diseases
SUSENAS : The National Socioeconomic Survey
TC : Total Cholesterol
TG : Triglycerides
TG-rich : Triglycerides-rich
TIARP : TNF(alpha)-included adipose-related protein
TNF-a : Tumor Necrosis Factor alpha
TNF-R1 : Tumor Necrosis Factor Receptor 1
TNF-R2 : Tumor Necrosis Factor Receptor 2
VLDL : Very Low Density Lipoprotein
WHO : World Health Organization

CHAPTER I
INTRODUCTION

1.1. Background

Obesity remains a global health problem in both developed and developing countries. According to WHO (World Health Organization), until the 20th century, overweight is common, and become a global epidemic problem. Currently, 1.6billionadultsin worldwide has a problem of overweight, and at least 400 million of them are obese. By 2015, an estimated 2.3 billion adults will be overweight and 700 million are obese. Incidence of obesity in developed countries such as in the countries of Europe, America, and Australia has reached epidemic levels. This incident not only in developed countries alone, obesity in developing countries has become even more serious health problems. For example, 70% of the adult population Polynesia and Samoa in the category of obesity. Prevalence of overweight and obesity also increased significantly in the Asia-Pacific region. For example, 20.5% of South Korea's population classified as overweight and 1.5% classified as obese. In Thailand, 16% of the population were overweight and 4% are obese. In urban areas of China, the prevalence of overweight was 12% in men and 14.4% in women, while in rural areas the prevalence of overweight in men and women respectively 5.3% and 9.8% (Chen, 2008).

In Indonesia, the prevalence of obesity also shows a figure quite alarming. Based on SUSENAS 2004, the prevalence of obesity in children has reached 11%. Based on data from the Health Research (Kemenkes) in 2007, the national prevalence of obesity is common in people aged ≥ 15 years were 10.3% consisted of 13.9% males and 23.8% of women, while the prevalence of overweight in children 6-14 years of age in males 9.5% and 6.4% in women. This figure is almost the same as the WHO estimates by 10% in children aged 15-17 years. According to the Indonesian Ministry of Health (Kemenkes, 2009), 9.8 million people from 210 million (4.7%) of people in Indonesia are obese, where 23.0% in men and 43.0% in women in the age group 40-49 years in 12 major cities in Indonesia with increasing age, changes occur in the body, decreasing metabolism, and increased fat in the body that cause an increase in the prevalence of cardiovascular diseases such as coronary heart disease,

hypertension and stroke, causing fat to accumulate in the blood vessel walls (pangkahila, 2007).

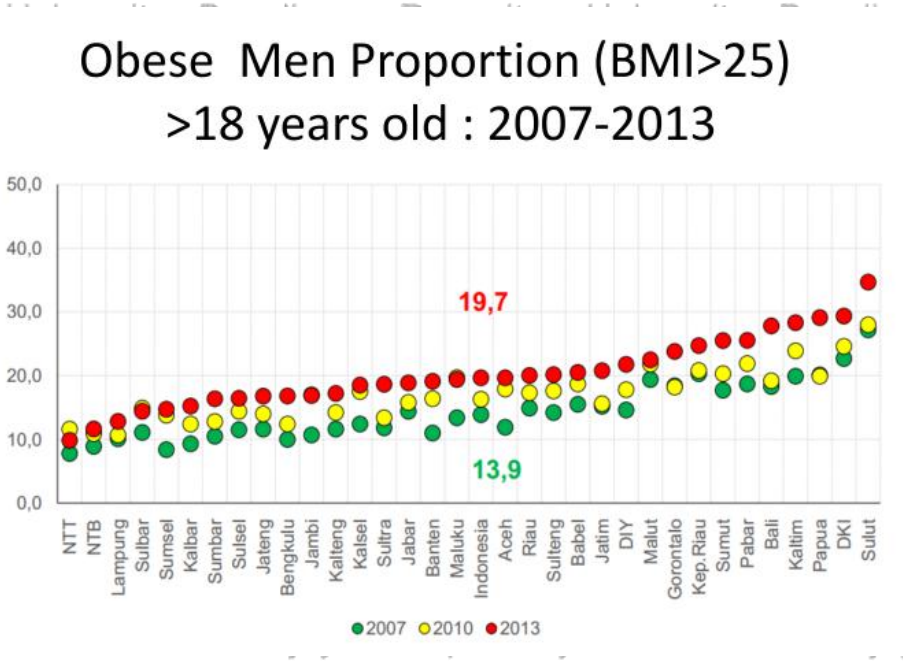

Figure 1.1. Obese Men Proportion (BMI>25) > 18 years old : 2007-2013 (Kemenkes, 2013)

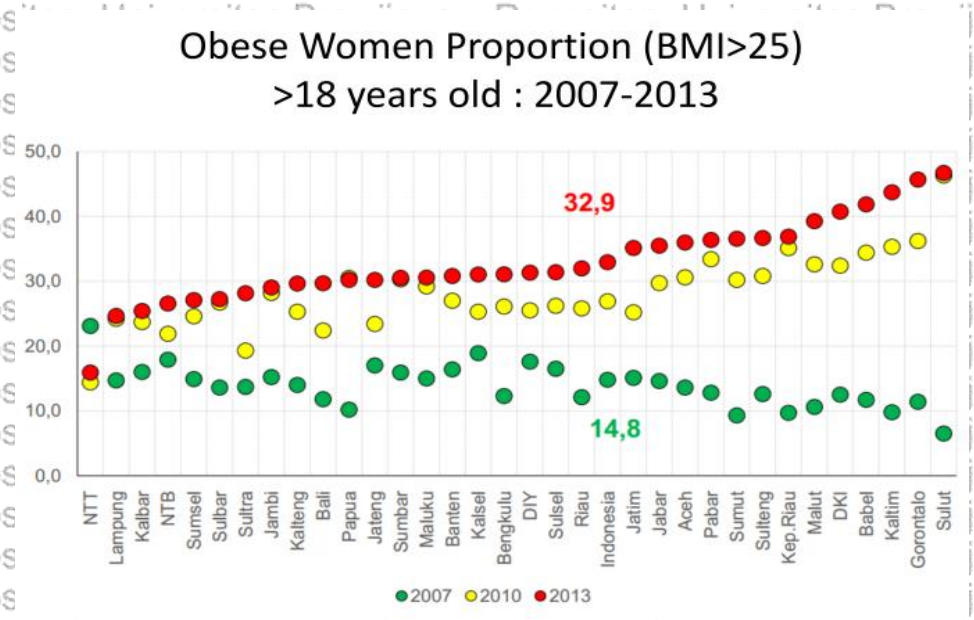

Figure 1.2. Obese Women Proportion (BMI>25) > 18 years old : 2007-2013 (Kemenkes, 2013)

The obesity cases spread in all regions in Indonesia. In adults over the age of 18 years, the percentage of obesity cases experienced a significant increase from 13.9% (2007) to 19.7% (2013) in men and 14.8% (2007) to 32.9% (2013) in women (Kemenkes, 2013). An increasing of unhealthy lifestyle is considered as one of the causes of the increasing number of obesity. In

developing countries such as Indonesia and Saudi Arabia, obesity became a national problem. Most of the people in the developing countries perceive being overweight or obese as an indicator of prosperous life (WHO, 2005). This is very different than in the states of the West that has a very high population growth, they acknowledge that being overweight or obese was dangerous because it is related to the negative effects on health.

Whilst increasing number and prevalence of overweight and obesity can be seen across the developing countries, the levels differ markedly by country and region. Within the developing countries, rates are high in parts of Latin America, Near East and North Africa, and the Pacific, but notably low in other parts of Africa and some Asia. For example, in 1980, 36% of Mexican adults were overweight : that had increased to 68% by 2008. In the Republic of Korea, the corresponding rates were 14% and 32%(Stevens *et al.,* 2012).

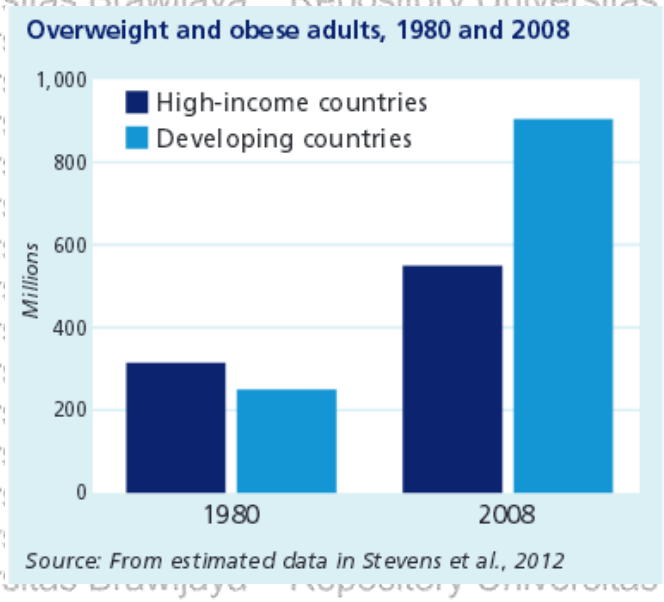

Figure 1.3. Overweight and obese adults, 1980 and 2008 (Stevens *et al.,* 2012)

Overweight or obesity is a medical condition marked by excess body which causes health adverse effect (WHO, 2005). Excessive food intake, physical inactivity, and genetic susceptibility simultaneously trigger obesity. Obese people have a slow metabolism, so they are only able to eat a little food (Kushner, 2007). Someone obese have a body mass index (BMI) over 30 kg / m2. Obesity triggers various diseases, particularly heart disease, type 2 diabetes, obstructive sleep apnea, cancer, osteoarthritis and asthma (Kushner, 2007). However, Kushner (2007) describes not only the diet and physical activity is a major factor of obesity, but also influenced by genes, endocrine disorders,

medications or psychiatric illness. Reducing the consumption of energy-dense foods such as foods high in fat and sugar and increasing fiber intake, the quality of the intake be increased.

However, obesity is a cause of death was preventable, although the prevalence of adults and children increases (Pollack, 2013). Obesity is generally a major disease in the modern world (especially in the western world). an unhealthy lifestyle which showed that reduces the high fat diet, regular exercise for 30 minutes every day to maintain a healthy weight and not smoking be a long-term effort of the whole community of physicians, patients, and government policies relating to reducing the risk of atherosclerosis.(Weinstock, 2013).

Fasting gives the digestive chance to rest, improve the regeneration of cells of the gastrointestinal tract instruments, as well as reducing the workload of digestion (Kim, 2010). Those are occurred due to a diet change from three meals a day to two times and reducing habits of snacks consumption during fasting (Ziaee, 2006). Furthermore, fasting can reduce blood sugar levels, triglyceride and blood pressure and accidental change of low density lipoprotein (LDL), without the addition of HDL and increasing human growth hormone response (HGH) which protects muscle and metabolic balance (Kaplan*et al.,* (2008); Watson (2014),Benjamin*et al.,* (2009), Aksungar*et al.,(*2005). Ziaee (2006) said that when fasting, a change in diet of three meals a day to two times. When not fasting, some people often eat snacks that contain lots of triglyceride. So that by fasting, we can reduce the habit. But of course, these benefits will be achieved when we apply a good diet when breaking and dawn.

Adipose tissue is essentially a network of energy perverts, called endocrine organ. Adipose tissue produces bioactive peptides called adipokines, besides the effect on adipocyte function as an endocrine and paracrine organ, also has an effect on more than one metabolic pathway in blood vessels. Adipose tissue is the largest organ in the human body that produces a small adipocytokine, and provide the total impact on the functioning of the body. Adipocytokine adipose tissue has a large number coming from the vein and removed from adipocytes to the systemic circulation. Adipocytokine secreted into leptin, adiponectin, angiotensinogen, resistin, TNF-alpha, IL-6, ASP and PAI-1 (Ronti*et al.*.., 2006).

TNF-a is a cytokine with multiple multi-potential immunological function was first produced from molecules associated with adipose tissue between

obesity and insulin resistance, and plays a role in energy balance. One way to prevent any transfer of fat and obesity are increasing the concentration of the TNF-a, which can slow down the lipoprotein lipase (LPL) in 3T3L1 cells negatively correlated with LPL mRNA, and activates hormone-sensitive lipase via paracrine and autocrine. LPL decline will reduce fat intake by adipose tissue and increases triacylglycerol. The high triacylglycerol will stimulate adipose tissue lipolysis is accompanied by increased secretion of VLDL from the liver. (Fain*et al.,* 2008).

Trayhurn (2005) explained, increased levels of IL-6 were detected in the blood, in addition to originate from endothelial cells, fibroblasts and macrophages, are also derived from adipose cells, which act as an autocrine and parakrine. In basal conditions, the secretion of IL-6 and TNF-α as a result of stimulation increased 60 times in the form of 3T3-L1 adiposite. Through the expression of IL-6 adipose and non-adipose cells. Another modulator role in the expression of IL-6 in the adipose cells are glucocorticoids and catecholamines that cause a decrease in LPL activity of adipose tissue that has implications in the reduction of fat, protein synthesis stimulation in the acute phase, the increased activity of the hypothalamic pituitary axis and thermo genesis. Interleukin 6 is a member of pro-inflamantory cytokine secreted by monocytes, macrophages, and fat tissue) Trayhurn, 2005).

In humans, IL-6 can stimulate an inflammatory reaction, which led to protection against obesity, and induces the production of TNF-α that mediate inflammatory reactions in vitro as shown by the administration of IL-6 in adipose cells. This reaction is mediated by protein TIARP / TNF-α in individuals who suffer from obesity. Generally, IL-6 can cause hemostasis disorders, diabetes mellitus type 2 and causes obesity, so that IL-6 can be used as targets for research (Permana, 2009).

1.2. Problem Statements

Based on the background that has been described, then can be prepared the problem statements as follows:

1. Are there any differences in the average (daily) or total of food intake of overweight male subjects due to Ramadan fasting?
2. Are there any differences in body weight of overweight male subjects due to Ramadan fasting?

3. Are there any differences in lipid profile (LDL, HDL, TG, TC) of overweight male subjects due to Ramadan fasting?
4. Are there any differences in inflammatory markers of overweight male subjects due to of Ramadan fasting?

1.3. Objective of The Research

The objectives of the study are as follows:

1. To examine the effect of the duration of Ramadan fasting on the average or total food intake ofoverweight male subjects.
2. To examine the effect of the duration of Ramadan fasting on the body weight of overweight male subjects.
3. To examine the effect of duration of Ramadan fasting on the blood lipid profilesin the of overweight male subjects
4. To examine the effect of duration of Ramadan fasting on the inflammatory marker of overweight male subjects

1.4. Benefits of The Research

The results of this study are expected to provide information of the effect of duration of Ramadan fasting on the body weight, blood lipid profiles, and inflammatory markers on overweight people.

CHAPTER II
REVIEW

2.1. Obesity

2.1.1. Definition of Obesity

Obesity is a condition in which found the presence of excess fat in the body. To categorized the obesity level, the body mass index (BMI) calculated as weight (kilograms) divided by height squared (square meter) expressed as (kg/ m^2 is used (WHO, 2015).Table 2.1 shows a classification of BMI, obesity level and risk of morbidity for European or Americans and for Asian. It shows that obesity was indicated by BMI of ≥25 for Europeans or Americans which is higher than those for Asians.

Table 2.1.BMI and Morbidity Risk Classificationsfor Europeans and for Asians (WHO, 2015).

	For Europeans		**For Asians**	
Classification	**BMI (kg/m^2)**	**Morbidity Risk**	**BMI (kg/m^2)**	**Morbidity Risk**
Skinny	< 18.5	Low	< 18.5	Low
Normal	18.5 – 24.9	Medium	18.5 – 22.9	Medium
Obesity	≥ 25		≥ 23	
Pre Obesity	25 – 29.9	Increased	23 – 24.9	Increased
Obesity I	30 – 34.9	Medium	25 – 29.9	Medium
Obesity II	≥ 35 – 39.9	Heavy	≥ 30	Heavy
Obesity III	≥ 40			

Based on BMI, obesity is divided into three categories, namely : obesity level I with BMI 30.00-34.99; level II obesity with BMI 35.00-39.99; and obesity level III with BMI over 40.00. Cut off point of obesity in the Asia Pacific region has lower criteria than the WHO criteria in general. Cut off point of obesity in the population of Asia Pacific is a BMI ≥ 25.00. Based on the cut-off point of obesity in the population of Asia Pacific, obesity is divided into two categories, namely: level I obesity with BMI 25.00-29.99 and level II obesity with BMI ≥ 30.00. Based on fat distribution, obesity can be divided into two types, ie, central obesity and general obesity (Qiao, 2012).There are two types of fat accumulation in human body, namely: gynecoid and android. Gynecoid shape is the accumulation of fat, especially in the lower body (buttocks) while the

accumulation of fat in the abdomen is called android shape or better known as central obesity / visceral obesity. The research proves that there is a close relationship between central obesity and cardiovascular disease risk factors in the metabolic syndrome is classified as type 2 diabetes mellitus, impaired glucose tolerance, hypertension and dyslipidaemia. Weight loss by diet, exercise and medication can improve lipid profiles and better control *glycaemic*(Alshehri, 2010).

WHO (2015) proposed a criterion for classifying body fat based on BMI, according to which individuals with BMI≥30kg/m are considered obese. However, in Western populations, obesity-related health problems are already observed in individuals with BMI < 30kg/m and some studies in Caucasians have shown increased risk of cardiovascular diseases with BMI values below 25kg/m.

The American Diabetes Association (2005) explained that the obesity is one of the risk factors in diabetes mellitus type II. Obesity is a strong risk factor in causing diabetes mellitus type II and more than two-thirds of patients with type II diabetes are obese. Barnett, (2009) explains that in women with a BMI of 25 kg / m2 had a five-fold risk of developing diabetes than those with a BMI of 22 kg / m2. Risk becomes higher as 28-fold with a BMI of 30 kg / m2 and 93-fold with a BMI> 35 kg / m2.

Shuster*et al.,* (2012) explained that abdominal fat contained in two main compartments subcutaneous and visceral. Fatty acids are released by visceral fat into the portal circulation. Some studies suggest that excess visceral fat is associated with metabolic risk factors than the other compartment.

Several studies have shown that the collection of the abdominal fatty acids will cause a variety of metabolic abnormalities that can lead to lipoprotein atherogenic dyslipidemia characterized by increased concentrations of triglycerides and small dense LDL particles, and decreased HDL triglyceride concentrations. Increasing the number of particles very low density lipoprotein (VLDL) and LDL apo B illustrates the observed increase in atherogenic dyslipidemia. Influence of any abnormality atherogenic lipoprotein is an interesting topic, but has not been fully resolved (Shuster*et al.,* 2012).

Furthermore (kolovou*et al.,* 2005) explains that the enlargement of adipocytes which showed an increase in lipolytic activity, plays a role in the increased release of free fatty acids via the portal circulation to the liver. Free fatty acid levels are high portal will stimulate the synthesis of TG in the liver,

which will be secreted in VLDL and production of apolipoprotein B which is the main protein in the liver. In circumstances *normolipidemic*, VLDL secretion is affected by TG and triglyceride, and there is a relationship between triglyceride synthesis and production of VLDL particles smaller. Hyper tigliseridemia on central obesity and insulin resistance associated with the secretion of VLDL particles rich in TG.

Obesity is associated with insulin resistance. In these conditions, the levels of LDL in normal or there was a slight increase, but a change in the composition of LDL particles. Abnormalities of LDL particles are the result of hypertriglyceridemia. Small dense LDL will not appear until the TG content of more than 1.5 mmol / L. In these circumstances, the accumulation of large VLDL rich in TG (VLDL 1). At the time of VLDL 1 in lipolysis by lipoprotein lipase, will produce a conformational change in the apo B and LDL particles. These particles can't be related to the LDL receptor and can cause long levels in the circulation. The enzyme triglyceride ester transfer protein (CETP) cause exchange of TG in LDL and HDL. TG-rich LDL forming small dense LDL are associated with increased risk of cardiovascular disease. Some studies suggest that small dense LDL particles atherogenic because it causes a decrease in LDL receptor that mediates clearance, then more easily into the arterial wall, so it tends to be related to the arterial wall proteoglycans, then high oxidation sensitivity, play an important role against macrophage uptake. LDL heterogeneity based on the variable content of cholesterol ester molecules that are at the core of LDL, while the absolute number of LDL apo B on the surface does not change (kolovou*et al.,* 2005).

Low HDL triglyceride in individual atherogenicdyslipidemia is often considered a secondary consequence of the increase in TG. An increase in plasma TG will cause CETP mediates the exchange of TG-triglyceride ester between LDL and VLDL. This exchange will form a TG-rich HDL. TG-rich HDL triglyceride but not easier catabolized. TG components undergo hydrolysis and dissociate with apolipoprotein A which is the major protein of HDL. Other mechanisms that contribute to a decrease in HDL triglyceride levels is a change fat into the liver as a result of insulin resistance, which will decrease the production of apo A by the liver (Kolovou*et al.*, 2005).

2.1.2. Causes of Obesity

Obesity is caused by many genes (polygenes). Some studies resulted that 127 from 135 candidates genes linked with obesity have been identified (Rankined*et al.,* 2006). Those identified genes were obtained from studies of association of BW, BMI, overweight and obesity; association of body composition and fat distribution phenotype and association of BW and body composition (Rankined*et al.,* 2006).

Abnormalities in these genes will lead to abnormalities in nutrients. Mutations in the gene PPAR-γ causes PPAR-γ is not active. In single gene that is known of them is the presence of a mutation in the leptin gene, melenocortin-4 receptor, pro-opiomelanocortin, and the PPAR-γ gene. The presence of mutations in multigene, the cause of obesity is known that individuals who come from families with obesity, obesity likely 2-8 times larger than the family who are not obese (Rankined, 2006).

a. Physical Activity

Physical activity is any body movement produced by skeletal muscles that requires energy expenditure. No physical activity (physical inactivity) is an independent risk factor for chronic diseases, and overall is estimated to cause of death globally (WHO, 2010). Exercise increases circulation, improve insulin sensitivity, lower blood pressure and triglyceride while increasing HDL. Through regular exercise and isotonic (aerobic physical activity approximately 30 min / day) can decrease peripheral resistance which lowers blood pressure. Regular exercise should ideally be done three to five times a week and at least a half hour each session of moderate intensity. Someone who tends to not do physical activity balance will be obese (WHO, 2010).

b. Excessive food intake

Women who are obese are more responsive to the taste and smell of food compared with women with normal weight. They will eat when they feel full, and do not eat when hungry. Pattern of overeating can lead to obesity (Emilia and Freitag, 2010).

2.1.3. Effect of Obesity on Health

a. Diabetes Mellitus

Obesity is closely linked to the pathphysiology of diabetes mellitus on the incidence of metabolic syndrome related to fat and glucose metabolism. adipolysis (substances from adipose tissue that is pro-inflammatory, trigger insulin resistance, hypertension, and thrombosis) have increased. Under normal circumstances adipolysis maintained by the hormone from adipocytes cells that act as anti-inflammatory cytokines of the tumor necrosis factor-α (TNF-α), which destroy the fat in the liver and disrupt the release of insulin in the pancreas. Free fatty acids also reduce the use of muscle glucose-stimulated insulin, which also contribute to the hyperglycemia. Lipotoxicitydue to excess free fatty acids contribute to a reduction of insulin secretion from pancreatic β cells, which eventually will experience fatigue β cells, which encourages the onset of diabetes than women with obesity (NattayaLakshita, 2012).

Esteghamati*et al.*, (2014) studied about "*Trends in the prevalence of diabetes and impaired fasting glucose in association with obesity in Iran : 2005-2011*". The aims in this research to estimate the prevalence and trends of diabetes mellitus (DM) and impaired fasting glucose (IFG), 2005-2011, and to determine the contribution of obesity to DM prevalence. Data from Surveillance of Risk Factors of Non-communicable Diseases (SuRFNCD) conducted in 2005, 2007, and 2011 were gathered. DM was defined as presence of self-reported previous diagnosis or a fasting plasma glucose (FPG)≥7 mmol/L. IFG was diagnosed with FPG levels between 5.6 and 6.9 mmol/L. Prevalence rates for 2011 and trends for 2005-2011 were determined by extrapolating survey results to Iran's adult population. Population attributable fraction (PAF) of obesity was also calculated. In 2011, IFG and total DM prevalence rates were 14.60% (95%CI : 12.41-16.78) and 11.37% (95%CI : 9.86-12.89) among 25-70 years, respectively. DM was more common in older age ($p < 0.0001$), in women ($p = 0.0216$), and in urban-dwellers ($p = 0.0001$). In 2005-2011, trend analysis revealed a 35.1% increase in DM prevalence (OR: 1.04, 95%CI : 1.01-1.07, $p = 0.011$); albeit, IFG prevalence remained relatively unchanged (OR: 0.98, 95%CI: 0.95-1.00, $p = 0.167$). In this period, DM awareness improved; undiagnosed DM prevalence decreased from 45.7% to 24.7% ($p < 0.001$). PAF analysis demonstrated that 33.78%, 10.25%, and 30.56% of the prevalent DM can be attributed to overweight (BMI≥25kg/m(2)), general obesity (BMI≥30 kg/m(2)), and

central obesity (waist circumference≥90 cm), respectively. Additionally, the DM increase rate in 2005-2011, was 20 times higher in morbidly obese compared with lean individuals. More than four million Iranian adults have DM which has increased by 35% over the past seven years, owing in large part, to expanding obesity epidemic.

b. Hypertension

Obesity may increase the incidence of hypertension. This is due to the fat can cause blockage in the blood vessels, thereby increasing blood pressure. Based on the Framingham Heart Study, hypertension cases occur in women is directly related to obesity. Hypertension originated from renal sodium re-absorption in the body which causes an increase in extracellular fluid volume and blood volume. High levels of sodium increases blood pressure. Obese women require high blood pressure to maintain sodium and fluid balance. Several potential mechanisms capable of mediating the sodium retention and hypertension in obesity, including activation of the sympathetic nervous system, activation of the renin-angiotensin-aldosterone and renal compression. Hyperinsulinemia, or insulin excess due to the chaotic blood sugar regulation, obesity plays an important role in the development of hypertension (Emilia and Freitag, 2010).

c. Obstructive Sleep Apnea

Obstructive Sleep Apnea (OSA) is associated with obstructions of the upper airway during sleep, which is caused by collapse of the dilator muscles and soft tissues of the pharyngeal wall. OSA may be diagnosed based on the presence of 5 or more of these respiratory events (Apnoea-Hypopnoea Index > 5) with concurrent evidence of OSA symptoms (daytime sleepiness, snoring and choking arousals from sleep). Alternatively, OSA is also diagnosed when patients display an AHI of greater than 15 events per hour with no subjective report of these additional symptoms (ICSD-2, 2005).

The two main indications of OSA are the respiratory disturbances, and the arousals from sleep needed to reinstate breathing. These two indications lead to the two main clinical consequences of the disorder, hypoxia of the brain and the heart, and sleep fragmentation. Daytime sleepiness and hypoxia of the brain are associated with cognitive deficits, such as impaired working memory and

attention (Gagnon *et al.,* 2014). Hypoxia of the heart increases the risk of heart problems including hypertension (Gruber*et al.,* 2006). Patients with OSA are also at higher risk for Type II diabetes and stroke. Daytime sleepiness effects the patients' and their partner's quality of life and there is a high rate of comorbid anxiety and depression in this sample (ICSD-2, 2005). The effects of sleepiness and impaired concentration is most obvious in the car accident risk odds-ratio of 7.3 over five years compared to individuals of similar demographic status without OSA (Gruber*et al.,* 2006).

d. Inflammation

TNF-α is a multi-potential cytokine with several immunological functions. TNF-α is the first product that has the adipose tissue molecular relationship between obesity and insulin resistance, in which the addition of TNF-α may increase insulin resistance individual. Serum concentration will increase in obese individuals and high concentrations associated with insulin resistance, endothelial dysfunction, increased C reactive protein (CRP) and interleukin-6 (IL-6) (Ronti*et al.,*2006).

Ronti*et al.,*(2006) said that TNF-α in obesity associated with insulin resistance, increased free fatty acids by adipocytes, decrease the synthesis of adiponectin and insulin signalling disorders. Reduced TNF-α will reduce obesity that cause hyperlipidemia which contributes to glucose transporter-4 (GLUT-4) in the other molecules involved in the role of insulin.

Wang*et al*. (2006) explained TNF-α plays a role in energy balance. At high concentrations of TNF-α inhibits lipoprotein lipase (LPL) in 3T3L1 cells and activate hormone sensitive lipase through antocrine and paracrine pathways. The decline in LPL activity will suppress exogenous fat intake by adipose tissue and increases triacylglycerol in circulation. High triglyceridelipolysis will stimulate adipose tissue accompanied by increased secretion of VLDL from the liver. This is one method to prevent fat storage and obesities. In addition, TNF-α increases apoptosis adiposity.

Wang*et al.* (2006) explained that TNF-α synthesis production can be shown to be responsive to both nutritional and immunological regulators. Additionally, there are regional effects such that subcutaneous adipose tissue expresses mRNA for TNF-α and TNF-R1 and TNF-R2 to a greater extent than omental tissue.

Trayhurn (2005) explained, increased levels of IL-6 were detected in the blood, in addition to originate from endothelial cells, fibroblasts and macrophages, are also derived from adipose cells, which act as an autocrine and parakrine. In basal conditions, the secretion of IL-6 and TNF-α as a result of stimulation increased 60 times in the form of 3T3-L1 adiposite. Through the expression of IL-6 adipose and non-adipose cells. Another modulator role in the expression of IL-6 in the adipose cells are glucocorticoids and catecholamines that cause a decrease in LPL activity of adipose tissue that has implications in the reduction of fat, protein synthesis stimulation in the acute phase, the increased activity of the hypothalamic pituitary axis and thermo genesis. Interleukin 6 is a member of pro-inflammatory cytokine secreted by monocytes, macrophages, and fat tissue) Trayhurn, 2005).

IL-6 can stimulate an inflammatory reaction, which led to protection against obesity, and induces the production of TNF-α that mediate inflammatory reactions in vitro as shown by the administration of IL-6 in adipose cells. This reaction is mediated by protein TIARP / TNF-α in individuals who suffer from obesity. Generally, IL-6 can cause hemostasis disorders, diabetes mellitus type 2 and causes obesity, so that IL-6 can be used as targets for research (Permana, 2009)

2.2. Ramadan Fasting

2.2.1. Definition of Ramadan Fasting

Ramadan is the holy month in which Muslims devoted to refrain from eating, drinking, smoking, and sexual intercourse from dawn to sunset. Fasting in Ramadan is the fourth pillar of Islam and every Muslim healthy concern since he reached puberty. While adult Muslims who are sick, travelling (distance is determined by the rules of Islam), pregnancy, diabetes (according to the doctor's advice) or through menstrual bleeding is not allowed to fast.

Ramadan lasts 29-30 days based on the detection of a crescent moon. Every day before dawn, Muslims observe the pre-fast meal called Sahour and fast until sunset. The fasting meal known as if tar. When fasting, every adult Muslim will have limited food intake and hydration until dawn. Obligation to eat in just a short period of time overnight caused some behaviour changes in sleep, feeding schedule, and mealtimes (Aloui *et al.,* 2012).

2.2.2. Health Benefits Of Fasting Ramadan

Medical world describes some of the benefits of fasting for Muslims, among which are (Hewitt, 2014):

1. Fasting Helps Reduce Weight

   Fasting can be a safe and effective way to lose weight according to numerous studies showing that fasting is controlled within a set number of hours, allowing the body to burn fat and fat cells more effectively than usual dietfasting) Hewitt, 2014).

2. Fasting Help Repairing Genes

   And reduce circulating levels of IGF-1, fasting is also beneficial to the improvement of a number of genes. As long as we have plenty of food, the human body will become more productive. The body which breaks down, by itself will recycles old and red cells. Fasted 4 consecutive days was also able to reduce IGF-1, so as to reduce the risk of cancer (Mosley and Spencer, 2013).

3. Fasting Improves Insulin Sensitivity

   Fasting has been shown to have positive effect son insulin sensitivity, which allows you to tolerate carbohydrates (sugars) that are better than if you did not fast. A study showed that after a period of fasting, insulin becomes more effective in telling the cells to take up glucose from the blood (Hewitt, 2014).

4. Fasting Accelerate Metabolism

   Intermittent Fasting gives your digestive system a rest, and it can string then your metabolism to burn calories more efficiently through. If your digestion is poor, it can affect your ability to metabolize food and burn fat. Intermittent fasting can regulate digestion and promote healthy intestinal function, thus increasing your metabolic function (Hewitt, 2014).

5. Fasting Improves Longevity

   One of the main effects of aging is slower metabolism, faster and more efficient metabolism of younger body. The less we eat, the less it takes a toll on our digestive system (Hewitt, 2014).

6. Fasting Improves Hunger and Healthy Heart

   Fasting helps regulate the hormone in our bodies so that we experience what true hunger obese individuals do not receive the proper signals to let them know they are full because of excessive eating. The longer we are fasting; your body is unable to regulate itself to release hormones that right, so that

we can experience what real hunger. When our hormones are working properly, we get the full Quicker (Hewitt, 2014).Over the past 20 years, the number of diabetics increased almost tenfold and there are no clear signs of slowing trend. Diabetes is associated with an increased risk of heart attacks, strokes, impotence, blindness and loss of your extremities due to poor circulation. It is also associated with brain shrinkage and dementia. Not picture prey. One way to prevent diabetes is spiralling into reducing carbohydrates and replace it by eating more vegetables and fat, because these foods do not cause such a huge spike in blood glucose. They also do not have a dramatic effect on insulin levels. Another way is to try intermittent fasting.

7. Fasting Improves The Immune System and Reduce the Spread of Cancer Cells Intermittent fasting improves the immune system because it reduces free radical damage, regulates inflammatory conditions in the body and starves off cancer cell formation (Hewitt, 2014).Intermittent fasting can help reduce the risk of cancer disease. Surgery is used to remove the tumour, chemotherapy or radiotherapy may even damage healthy cells. The treatment tends to damage the cells that divide rapidly which causes hair loss, whereas the fasting protects cancer cells from damage caused by normal spread of cancer.
8. Fasting Improves The Brain Function (Mosley and Spencer, 2013).

   Fasting has shown to improve brain function, because it boosts the production of a protein called brain-derived neurotropic factor (BDNF). BDNF activates brain stem cells to convert into new neurons, and triggers numerous other chemicals that promote neural health. This protein also protects your brain cells from changes associated with Alzheimer's and Parkinson's disease (Hewitt, 2014).
9. Fasting Improves The Mood

   Fasting has helped many people feel more connected to life during the practices reading, meditation, yoga and martial arts etc. With no food in the digestive system, this makes room for more energy in the body – the digestive is one of the most energy absorbing systems in the body. Fasting for self-enlightenment, allows us to feel better both consciously and physically. With a lighter body and a clearer mind we become more aware and grateful for the things around us (Mosley and Spencer, 2013).

CHAPTER III
FRAMEWORK

3.1. Conceptual Framework

Excessive accumulation of fat in the chest wall and below the diaphragm indirectly cause shortness of breath and other respiratory disorders with an emphasis on the lungs, even when a mild activity (Kemenkes, 2008). Respiratory problems causing temporary cessation of breathing during sleep (sleep apnea), so that during the day people often feel sleepy. Obesity can lead to orthopedic problems, including osteoarthritis worsen (especially in the hip, knee and ankle) and lower back pain (Kemenkes, 2008). A person with excess weight has a weight that is relatively larger than the surface of the body, resulting in inefficiency in heat dissipation body, consequently the body becomes excess sweating (Kemenkes, 2008).

Obesity is directly harmful to one's health. Obesity and overweight can start at any age. Some periods of age showed a great possibility for the occurrence of overweight and obesity (Nurmalina, 2011). One of the causes of Coronary heart disease is because foods high in fat, especially saturated fat, and occurs continuously, so it is easily absorbed by the body through the bloodstream. Therefore, the fat should be transformed into glycerol by the enzyme lipase (Permadhi *et al.,* 2008). Storage of residual fat in the metabolism of triglycerides and liver form bile acids function as digesting fat. Eat more fat, it also means an increase in blood triglyceride levels (Permadhi *et al.,* 2008). Increased levels of triglycerides cause arteriosclerosis or thickening of the coronary arteries (Purnamasari, 2010) resulting in reduced flexibility of the arteries, causing the occurrence of coronary heart disease. Similarly, when the flow of blood that carries oxygen to the heart wall tissue is stopped (Purnamasari, 2010).

Then, diabetes mellitus (DM) occurred because pancreatic not be able to produce more insulin to normal carbohydrate metabolism, resulting largely seingga glucose cannot be converted into glycogen. Therefore, high blood sugar (hyperglycemia) be increased, while the excess glucose is excreted through the urine (glycosuria) (Price and Wilson, 2006).

Symptoms of this disease is indicated by the frequent sufferers feel thirsty and tired with weight loss even though the appetite has not changed. In addition, obesity can stimulate the growth of cells that cause cancer. Then obesity can

suppress the lungs which causes respiratory distress and asphyxiation, even if the patient only a mild activity. Respiratory disorders can cause oxygen levels in the blood during sleep terjadu be reduced later causes breathing to a standstill for a while (sleep apnea) (Gruber*et al.,* 2006).

Metabolic syndrome is a combination of several interrelated abnormalities, which consists of central obesity, hypertension, hyperglycemia, and dyslipidemia. Simona (2005) describes the visceral fat that accumulates resulting in impaired glucose metabolism that cannot meet the energy needs due to insulin resistance. Insulin resistance also causes conditions of hyperglycemia and increased activity of the sympathetic nervous system that provide a substantial contribution to the occurrence of hypertension. Basically this mechanism shows the role of insulin resistance, inflammation, oxidative stress, endothelial dysfunction, kidney, sympathetic nervous system and renal hemodynamic changes will cause impaired renal (Simona (2005)).

A person identified as having a BMI of at least 30 have an increased risk of death besaar 50-100% more than someone with a BMI of 20-25. Type obesity apple has about three times the risk of heart disease compared with normal weight. Increased fat in the abdominal area causing aortic stiffness of the blood vessel, which is the main artery that delivers blood to the organs of the body (Kemenkes, 2008).

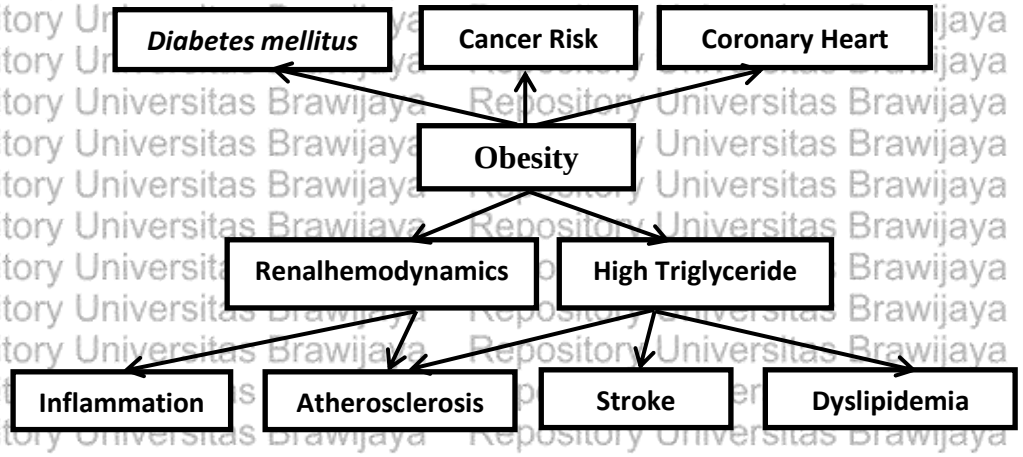

Figure 3.1.The Scheme of Diseases Caused by Obesity

3.2. Framework

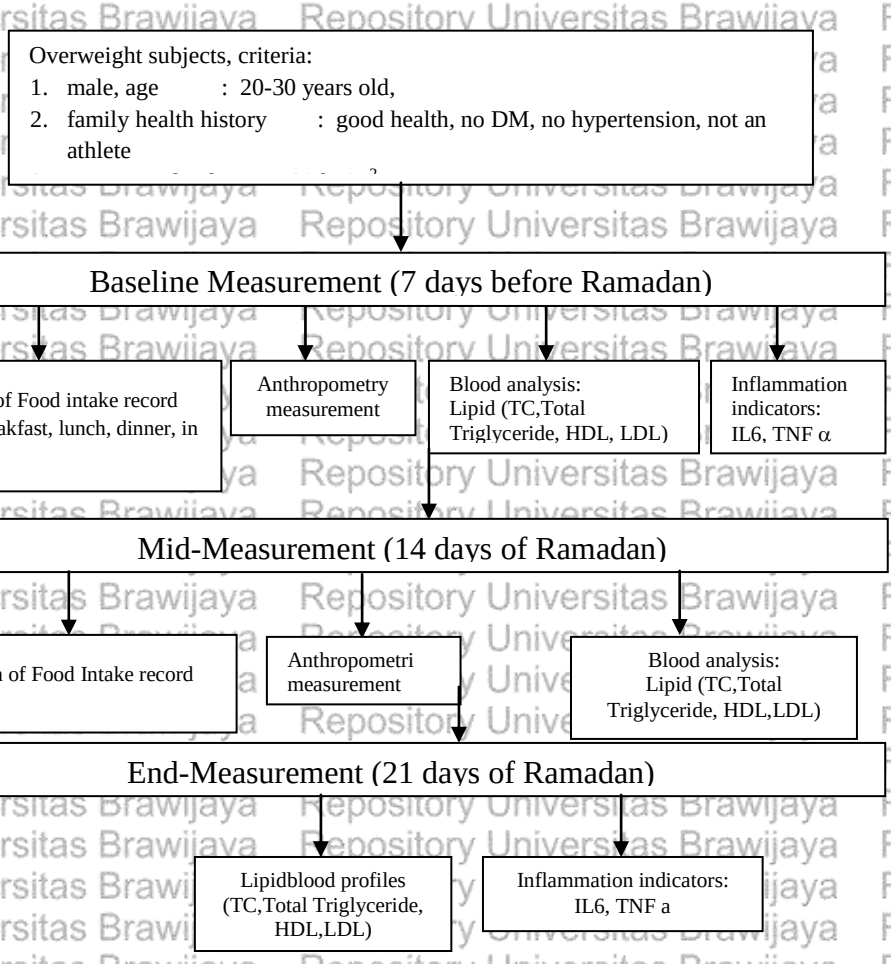

Figure 3.2.The Scheme of The Research

The scheme of this research is presented in Figure 3.2.Subject requirements are men aged 20-30 years, BMI 25-30 kg/m^2, do not have a history of diabetes, do not have hypertension and not an athlete. Before Ramadan fasting, precisely at before fasting, subjects are asked to record their food intake of breakfast, lunch, dinner and other foods for a week. Blood will be taken to measure total cholesterol (TC), high-density lipoprotein (HDL), low density lipoprotein (LDL), TG, and inflammatory indicators.

After all the initial examinations and measurements, subjects are ordered Ramadan fasting. At 14th day of Ramadan fasting, blood was taken to be analysed of total cholesterol (TC), high-density lipoprotein (HDL), low density lipoprotein (LDL), TG, and inflammatory indicators. During the fasting, subjects are asked to record their food intake of breakfast and dinner. End Measurement was performed at 21th day of Ramadan fasting. At this measurement, blood was

taken to be analysed of total cholesterol) TC), high-density lipoprotein (HDL), low density lipoprotein (LDL), TG and inflammatory indicators. In this study male is used as subjects because they can do full time of Ramadan fasting.

3.3. Hypothesis

Hypothesis is a relationship which is predicted by logic between two or more variable which is expressed in a statement that can be tested (Hair *et al.*, 2007). The hypothesis of this research are as follows:

1. There are some differences in the average (daily) or total of food intake of overweight male subjects due to Ramadan fasting.
2. There are some differences in body weight of overweight male subjects due to Ramadan fasting.
3. There are some differences in lipid profile (LDL, HDL, TG, TC) of overweight male subjects due to Ramadan fasting.
4. There are some differences in inflammatory markers of overweight male subjects due to Ramadan fasting.

CHAPTER IV
RESEARCH METHODOLOGY

4.1. Methods and Research Design

The research method used in this study is an experimental research method. The general objective of this experimental study is to investigate the influence of a particular treatment of a particular group (subjects) before and after treatments (311 / EC / KEPK / 05 / 2015).

The research design used in this study is a the one group pre-test-post-test design. In this design, subjects are given a pre-test before treatment, then the subjects are given treatments and after that the post-test are performed to see the results. The results of treatment can be determined more accurately, because it can be compared the condition before and after treatments within the same subjects. Chart of the one group pre-test- post-test design is as follows:

O_1	X	O_2
Pre-test	Treatment	Post-test

4.2. Research Location

The location of the research is on Malang –East Java - Indonesia. The subjects involved in this research were people who live in Malang.

4.3. Population and Sample

4.3.1. Population

Population is an area consisting of object: / subject that possesses the qualities and certain characteristics set by researchers to be studied and extracted in conclusion (Hair *et al.,* 2007). In this research populations referred to men in Malang city aged between 20 and 30 years, do not have a history of diabetes and of hypertension and not an athlete.

4.3.2. Sample

The sample is part of a number of characteristic and owned by a population. The sampling technique used in this study is a purposive sampling. Sampling criteria are men aged 20-30 years and they do not have a history of diabetes mellitus and BMI of 25-27 kg/m2. There are 30 males representing the number of subject for a pilot study. Based Kamal *et al.,* (2012), 23 (twenty three)

subjects who had Ramadan fasting gave statistically significant different in body weight, triglyceride, HDL and LDL profiles ($p<0.0001$) before Ramadan fasting and 21 days of Ramadan fasting.

4.4. Data Collecting Method

In order to necessary data can provide information, picture, notification, and accurate facts about an incident / circumstances, need to specify the data collection techniques appropriate to the characteristics of the observations will be investigated. Collecting data method in this research is:

1) Participant recruitment

Researchers are looking for male participants who are preferably Moslem. The candidates who are interested to participate in this research then be asked to provide information about his age, body weight, height and family health backgrounds. The criteria in accordance with the requirement criteria for this study are (men aged 20-30 years, good health, the BMI is around 25- 27 kg / m^2,do not have a background of diabetes and hypertension and not an athlete).

2) Physical and metabolic data

Participants completed an anthropometric measurement (body weight, height and BMI) by trained research staff from nutrition program medical faculty. General physical exam and medical history were collected by care provider in Medical Faculty of Brawijaya University.

3) Blood sampling

During this study, blood sampling were taken three times before Ramadan, at 14thRamadan fasting and at 21stRamadan fasting. Blood sampling were performed by professional plebhotomist from SIMA Laboratory. Blood would be analyzed. Blood was analyzed for lipid profiles (total triglyceride, HDL, LDL, TG), and inflammatory indicators. Blood sampling were taken on specific time after fasting overnight, or 8-10 hours before blood sampling. To take blood samples, cleaning taken place that the fossa vein with 70% alcohol swab and let it dry. Then put a hedge bond at the upper arm and ask the patient to clench and open hands several times in order veins clearly visible. After that, tense skin with the fingers of the left hand so that the vein is not moving. Furthermore, puncture vein slowly until the tip of the needle into the lumen of the vein, the needle hole facing

up. Remove or loosen damming slowly, then pull the suction syringe to obtain the desired amount of blood. Remove the hedge. Then put cotton in the top of the needle, and the needle then narrow revoked. Subjects were asked to press puncture sites last for a few minutes with cotton or plaster. Then lift the needle and syringe and blood stream into the tube provided through the wall (Berger, 2010).

a. Lipid Profile Analysis

1. Total Cholesterol

Paramesh (2011) explained that the total cholesterol were analyzed with CHOD-PAP method. Ester hydrolysis of the cholesterol and lipoprotein through-esterase cholesterol, cholesterol and oxidized by oxidase be hydrogen peroxide which then reacts with 4-aminoantipyrine and phenol mediated by peroxidase. Absorbance was measured with a spectrophotometer.

2. HDLCholesterol.

Paramesh (2011) showed HDL cholesterolwere analyzed using method CHOD-PAP by way of precipitating LDL and VLDL use phosphotungstat acid and Mg ions. After sentrifugated, the enzyme is added to the supernatant containing HDL, then measure the absorbance.

3. Triglyceride

Triglycerides measurements using GPO-PAP method (Paramesh, 2011). Triglycerides hydrolyzed and produce glycerol which then became quinoneimine, then color produced measured absorbance.

4. LDL Cholesterol

LDL cholesterol is measured by different7 as follows:

LDL cholesterol= total triglyceride –HDL cholesterol– Triglyceride/5

b. Plasma inflammatory marker

Plasma inflammatory marker (IL-6 and TNF-α) were measured by Enzyme-linked Immunosorbent Assay (ELISA) (Sies & Packer, 2005).

4) Food intake

Food intake assessed by trained research staff from Nutrition Study Programme Medical Faculty Brawijaya University using the 24 h repeated food recall combined with a 2-day food record. Dietary assessment was performed using paper format and visual aids (models of food and food photos) directly to the participant. The 24 h repeated food recall combined with a 2-day food record

were collected from all participants before fasting, at 14th and at 21st Ramadan fasting. Data is processed using Nutrisurvey (Erhardt 2007)

4.5 Research Variable

Variable in this study is divided into two, namely the independent variable and the dependent variable. The independent variable is a variable whose value can be determined but not controlled by other variables. The independent variable in this study is the duration of Ramadan. The dependent variable is a variable whose value can be determined and controlled by the other variables. The dependent variable in this research is food intake, BMI, plasma lipid profile (TG, TC, HDL and LDL), and inflammatory markers (TNF-α and IL-6).

1. Food intake

Food intake is the total number of calories in a daily di*et*. Daily food intake is one of the largest predictors of overweight and obesity (Stedman, 2006).To provide a complete record of all food and drink consumed on the previous day, a 24-hour recall is used. A research assistant helped to make a record of subject's food intake. Subjects are asked by the nutritionist, who has been trained in interviewing techniques, to recall the subject's exact food intake during the previous 24-H period or preceding day. Thus the method assesses the actual food intake of individuals. However, a single 24-H recall is not sufficient to describe an individual's usual intake of food and nutrients; multiple 24-h recall on the same individual over several days are required to achieve this object. Nevertheless, multiple single-day recalls on different individuals can give a valid measure of the intake of a group or population.

There are four stages of the interview often used. First, the list of foods consumed on the previous day was obtained. The second, a detailed description of each food consumed, including cooking methods and brand names (if any). Standard questions used to obtain specific details for each food item, for example, for dairy products, the identification should include the type of dairy products, brand names, and the percentage of fat (butter fat or milk fat). Third, the approximate number of each food item consumed, generally in household measures, and entered in the data sheet or computer-based data entry forms. Photos, a set of measuring cups, spoons, and authorities, local equipment household (calibrated before use), or the model of food of various types can be

used as memory aids or to assist patients in assessing the size of food portions consumed (Stedman, 2006). Information about the ingredients should also be collected at the same time. Fourth, make sure all the items, (including the use of vitamin and mineral supplements) have been recorded correctly.

2. Plasma Lipid

Blood plasma is a yellowish clear liquid that the alkaline reaction. Plasma Blood Plasma is a yellowish clear liquid which is an alkaline reaction. Plasma contains a complex mixture of organic and inorganic substances which consist of 92% water, (Gladine, 2007). Fat or lipid is a substance that is rich in energy, serves as the main energy source for the body's metabolic processes that circulate in the body and obtained through two sources, which comes from food, and the production of the liver, which can be stored in fat cells as energy reserves.

3. Plasma Inflammation Marker

TNF-α is a multi-potential cytokine with several immunological functions. Wang*et al.,* (2006) explain that TNF-α synthesis production can be shown to be responsive to both nutritional and immunological regulators. Trayhurn (2005) explained, IL-6 is a cytokine that has some influence secreted by immune cells, endothelial cells, fibroblasts, adipose tissue and skeletal muscle. Pro-inflammatory cytokines is increased in subjects with insulin resistance and obesity and can be expressed as a predictive factor for type 2 diabetes and myocardial infarction. Levels of IL-6 are increased levels of IL-6 were detected in the blood are derived from endothelial cells, fibroblasts, and macrophages.

4. Ramadan Fasting

Ramadan is the holy month in which Muslims devoted to refrain from eating, drinking, smoking, and sexual intercourse from dawn to sunset, and lasts for 30 days. Fasting in Ramadan is the fourth pillar of Islam and every Muslim healthy concern since he reached puberty. While adult Muslims who are sick, travelling (distance is determined by the rules of Islam), pregnancy, diabetes (according to the doctor's advice) or through menstrual bleeding is not allowed to fast (Aloui *et al.*, 2012).

4.6 Data Analysis Method

4.6.1 Paired t test

Mean comparison test for 2 dependent populations to determine the mean difference between the two sets of data are dependent. To test the hypothesis mean comparison paired t-test statistics were used (Walpole *et al.*, 2011):

$$t_{test} = \frac{\bar{X}_d}{s_d/\sqrt{n_d}}, \quad df = n - 1$$

Explanation:

$\bar{x}_d$ = Mean of difference

s_d = Standard deviation of difference

n = The number of sample of difference

d = Difference before treatment and after treatment

Hypothesis:

H_0 : d = 0 (there is no mean difference between before treatment and after treatment)

H_1 : d ≠ 0 (there is mean difference between before treatment and after treatment)

Critical Value:

$$t\_table = t_{(\alpha/2;df)}$$

Testing criteria:

There is a difference significant between before and after treatment, if the |t test| ≥ t table or probability (significance) ≤ alpha (α) then H0 are rejected. So, otherwise there is no difference significant between before and after treatment if the |t test| < t table or probability (significance) ≤ alpha (α).

Before analyzing the data using paired t test, then tested the normality using kolomogorov Smirnov test. The normality test aims to determine whether the data examined normal distribution or not. If data have normal distribution, then paired t test can be proceed, but if the data is not normally distributed then the method of analysis that can be used is the Paired Wilcoxon Test (Walpole *et al.*, 2011).If the probability (significance)> alpha (5%), The data have a normally

distribution, otherwise if the probability (significance)≤ alpha (5%), The data doesn't have a normally distribution.

The data will be analyzed using paired t are inflammatory marker between before Ramadan fasting and 21 days of Ramadan fasting.

4.6.2 Repeated Anova

The data obtained is repeated anova were used to analyze the significant difference of more than two categories (treatment or factor) of the parameters investigated. To examine the differences between the before, during, and after treatment using the test statistic F. (Walpole *et al.,* 2011).

If the test statistic F ≥ F tables (alpha = 5%) or probability (significance) ≤ alpha, H0 is rejected. So there is a difference between before, during, and after treatment, otherwise if the test statistic F <F table (alpha = 5%) or probability (significance)> alpha there is no relationship between the before, during, and after treatment. If there is one pair of different treatments significantly, then we did multiple comparison test using LSD (Walpole *et al.,* 2011).

Before analyzing the data using ANOVA repeated, then tested the normality using kolomogorov Smirnov test. The normality test aims to determine whether the data examined normal distribution or not. If data have normal distribution, then repeated ANOVA can be proceed, but if the data is not normally distributed then the method of analysis that can be used is the Kruskal Walis Test (Walpole *et al.,* 2011).If the probability (significance)> alpha (5%), The data have a normally distribution, otherwise if the probability (significance)≤ alpha (5%), The data doesn't have a normally distribution.

The data will be analyzed using repeated anova like : food intake, body mass index, and plasma lipids (TC, TG,HDL,LDL) between before Ramadan fasting, 14 days of Ramadan fasting, and 21 days of Ramadan fasting.

4.6.3 Rank Spearman Correlation

Spearman rank correlation analysis is a method used to determine whether there is a relationship and how strong degree of correlation between two variables regardless of the dependent variable and the independent variables were ordinal scale. To determine the relationship between two variables, namely by looking at the correlation coefficient with the following criteria:

a. 0 – 0.05 : There is no correlation
b. 0.05 – 0.25 : Very weak correlation
c. 0.25 – 0.5 : Strong enough correlation
d. 0.5 – 0.75 : Strong correlation
e. 0.75 – 0.99 : Very strong correlation
f. 1 : Perfect correlation

Pearson correlation coefficients can be searched using the following formula

$$r_{x_1x_2} = \frac{n\sum x_1x_2 - (\sum x_1)(\sum x_2)}{\sqrt{[n\sum x_1^2 - (\sum x_1)^2][n\sum x_2^2 - (\sum x_2)^2]}}$$

Explanation:

r_{x1x2} = correlation coefficient between X_1 and X_2
n = The number of sample

The data will be analyzed using rank spearman correlation is: the obesitywith inflammatory marker between before Ramadan fasting and 21 days of Ramadan fasting.

CHAPTER V
RESULT AND DISCUSSION

5.1 Identity of Respondents

The number of respondents participated in this study is 23 males. (Are) They are all Javanese? The characteristic of age, weight and height the respondents are presented below.

5.1.1 Identity of Respondents by Ages

Distribution of respondent's age is presented in Figure 5.1

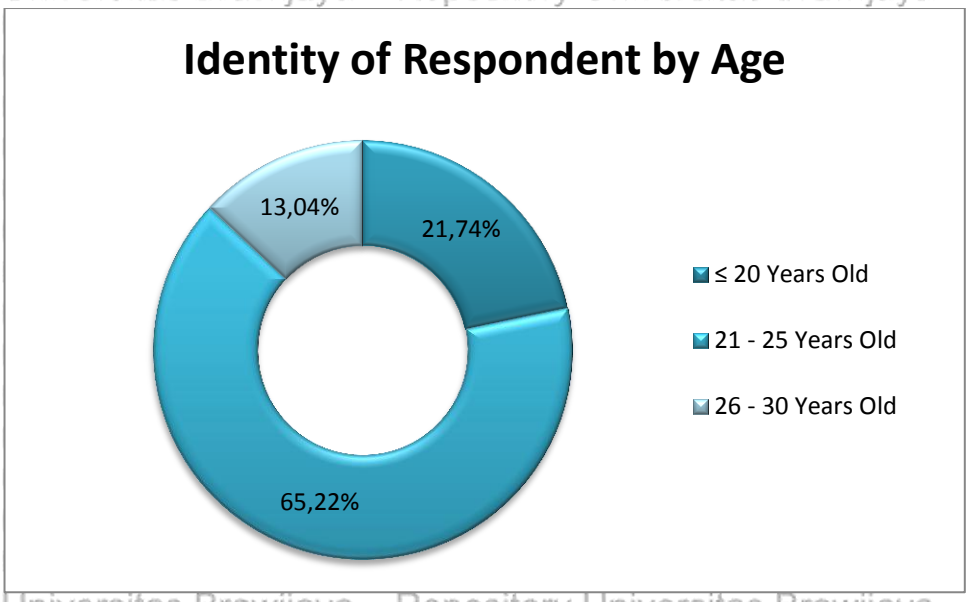

Figure 5.1. The distribution of respondent's age

The age of respondents were grouped in to three bands. The respondents with age of ≤ 20 years old are 21.74%Then 13.04% of respondents aged between 21-25 years old, and 65.22% of the respondents aged between 26-30 years old. This indicates that most respondents in this study were aged between 21-25 years old.

5.1.2 Identity of Respondents by Weight

The body weight of respondents are Figure 5.2.

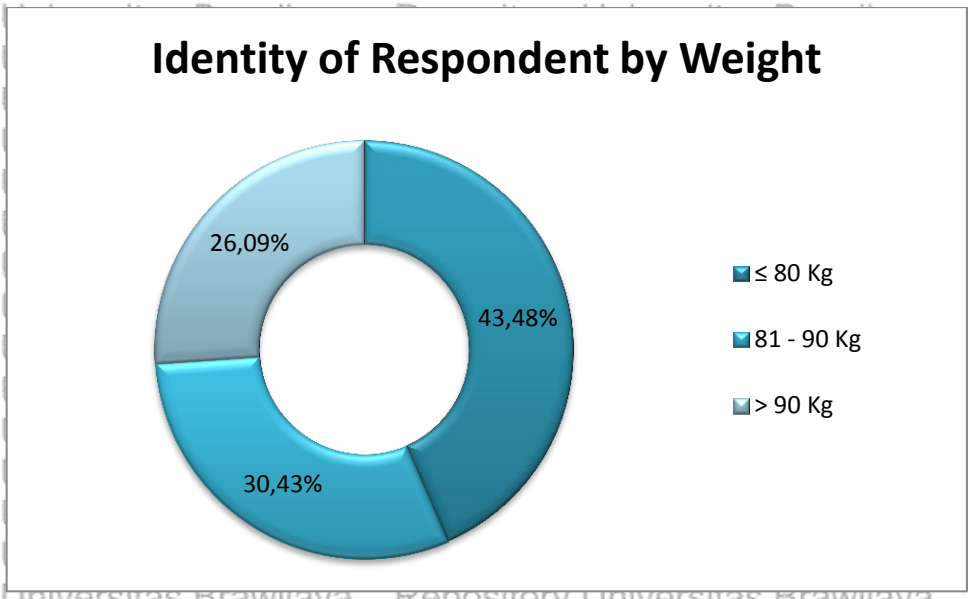

Figure 5.2. The Percentage of Respondent by Weight

The results of the identity of respondents by weight, it can be seen that the percentage of respondents who weigh less than 80 kg, 81-90 kg and higher than 90 kg are 43.5%,30.4% and26.1%, respectively. This suggests most of respondents have a weight of less than 80 Kg.

5.1.3 Identity of Respondents by Height

Distribution of respondent's height are presented inFigure 5.3.

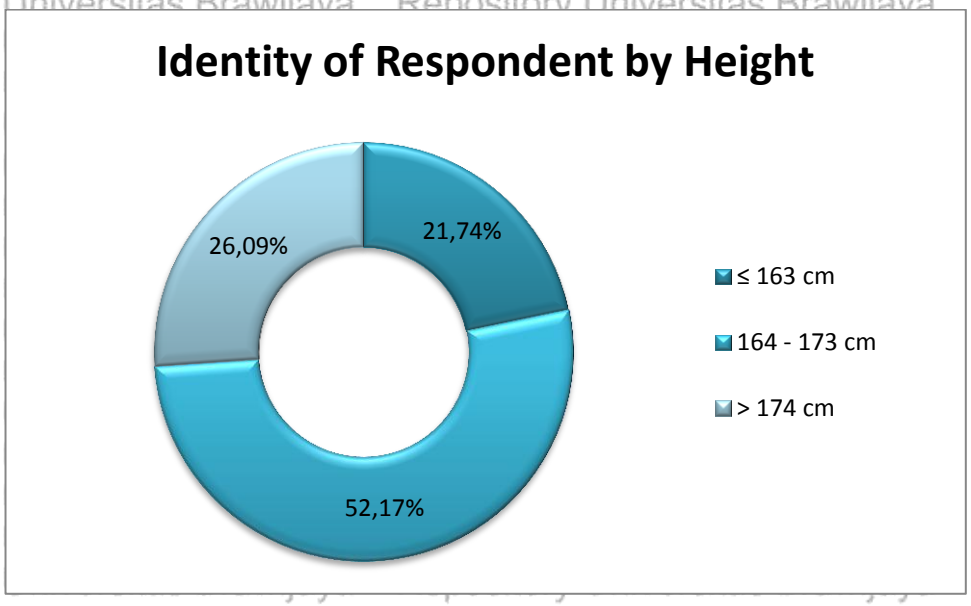

Figure 5.3. Distribution of respondent height

The percentage of the respondents with the height of <163 cm,164-173 cm and >174 cm are 21.7%, 52.2% and 26.1% respectively This suggests that the majority of respondents have a height between 164-173 cm.

5.1.4 Identity of Respondents by Body Mass Index (BMI)

Distribution of respondent's Body Mass Index (BMI)are presented in Figure5.4.

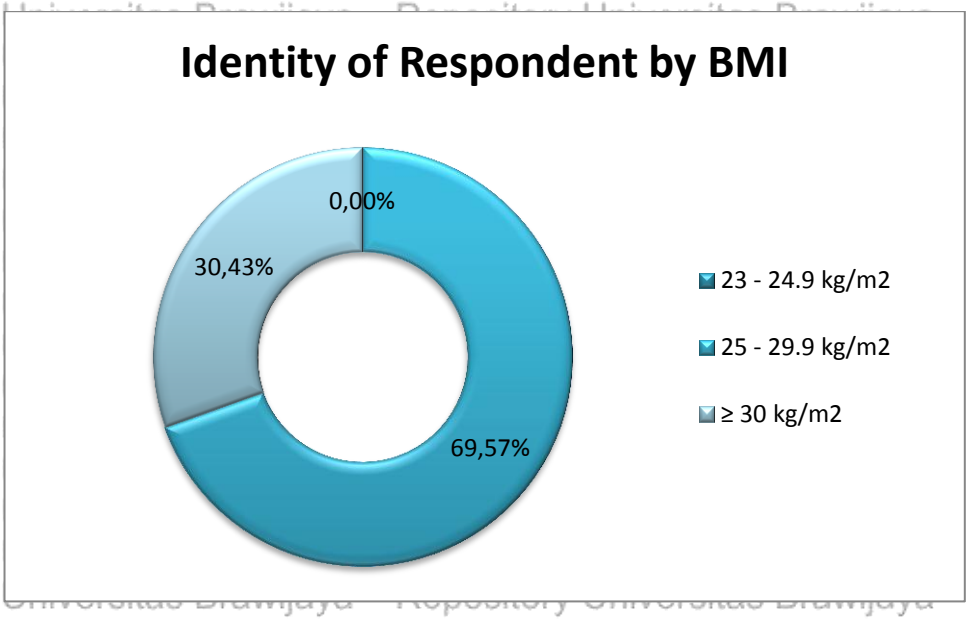

Figure 5.4. Distribution of respondent Body Mass Index (BMI)

The percentage of the respondents with the Body Mass Index (BMI)of 23–24.9kg/m^2, 25–29.9kg/m^2 and >30kg/m^2 are 69.6%, 0.00% and 30.4% respectively This suggests that the majority of respondents have a Body Mass Index (BMI)between 23 – 24.9kg/m^2.

5.2 Analysis of Food Intake

Figure 5.5 shows that food intake before Ramadan fasting was higher than 14 days of Ramadan fasting and 21 days of Ramadan fasting, while food intake 21 days of Ramadan fasting lower than before Ramadan fasting and 14 days of Ramadan fasting. Therefore, Ramadan fasting can reduce food intake. This result is consistent with research conducted by Alquraishi (2014), Shariatpanahi (2012), Abdurrahman (2015), and Chennaoui (2009)that there was significantly decrease food intake 21 days of Ramadan fasting.

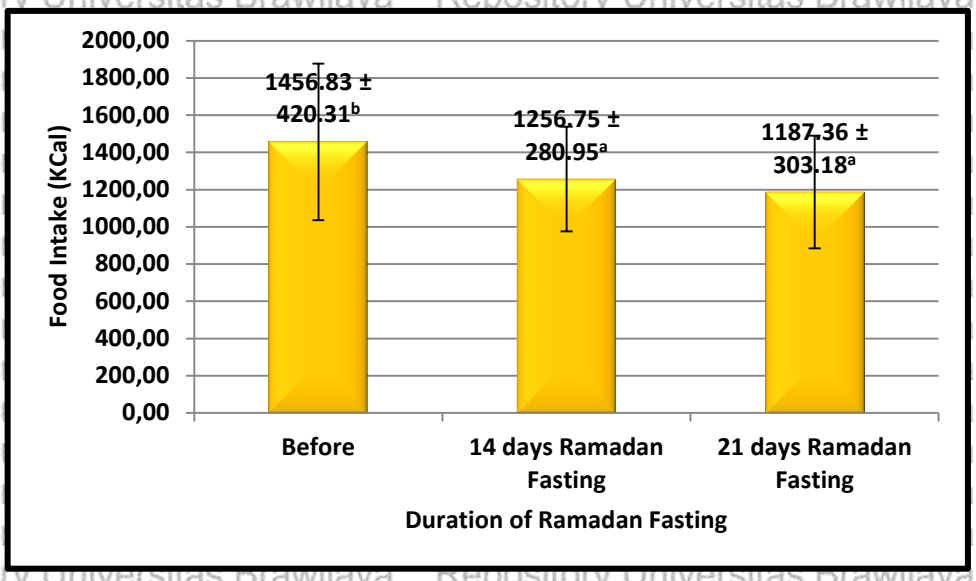

Figure 5.5. The Average of Food Intake Before, 14 daysand 21 days of Ramadan Fasting

The Results of testing difference with Repeated anova in food intake before Ramadan fasting, 14 days ofRamadan fasting, and 21 days ofRamadan fasting can be seen in Appendix1. From the Appendix 1 it shows that it was significant difference in food intake before Ramadan fasting, 14 days of Ramadan fasting, and 21 days of Ramadan fasting. The result of testing difference with LSD showed the average of food intake 21 days of Ramadan fastingis lower and significantly different than food intake before Ramadan fasting.but not significantly different with food intake 14 days of Ramadan fasting

When fasting, the body organs can rest, while other cells gather themselves to survive. If we do not consume enough food, the body will respond negatively, ie by converting fat into an energy source, thus causing weight loss. Every time the body metabolizing the energy, but converting the energy contained in the nutrients into energy potential, while the remaining will be stored in the body, skin cells, kidney cells, fat, eyelids and glycogen. At the time of fast changes in eating patterns initially 3 times to 2 times. An overweight subject (male or female?) who has fasting will eat fewer after14 daysand 21 days of Ramadan fasting (Unalacak*et al.*2010 ,).

5.3 Analysis of Body Mass Index

Figure 5.6 shows that body mass index of the subject before Ramadan fasting was higher than that 14 days of Ramadan fasting and 21 days ofRamadan fasting, while body mass index14 days of Ramadan fastingwas lower than before Ramadan fasting and 21 days ofRamadan fasting. Therefore based on repeated anova and LSD, Ramadan fasting can reduce body mass index. This result is consistent with research conducted by Nermat*yet al.,* (2009) that there was significant improvement in weight and BMI. Norouzy*et al.,* (2014) also showed that Ramadan fasting can improve significantly on body mass index after Ramadan. and Shariatpanahi (2012)and Alquraishi (2014) showed Ramadan fasting can reduce BMI significantly. In Memari, et al., (2011) and Sadiya, et al., (2011) study were informed Ramadan fasting was effectively reduce body mass index.

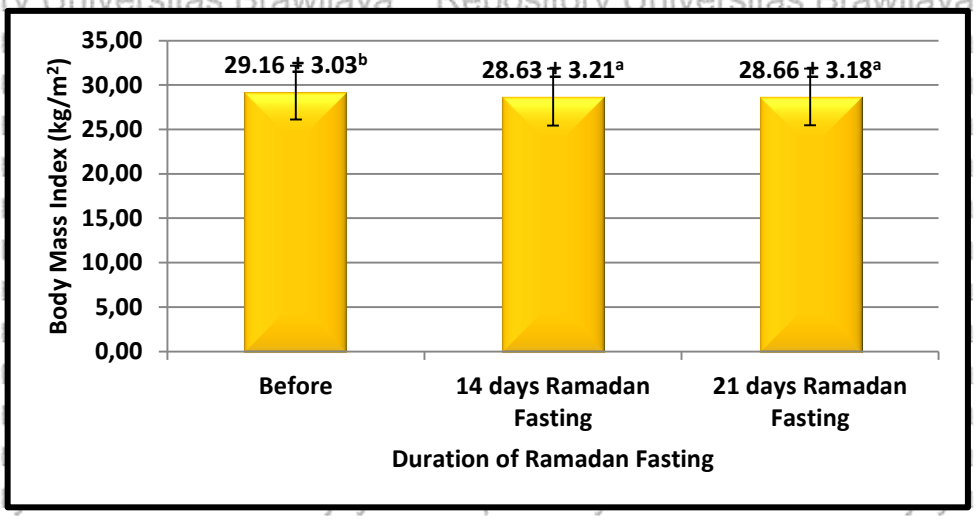

Figure 5.6. The Average of Body Mass IndexBefore, 14 days , and 21 days of Ramadan Fasting

The Results of testing difference with Repeated anova in body mass index before, 14 days of Ramadan fasting, and 21 days of Ramadan fasting can be seen in Appendix2. From the Appendix 2 it shows that it was significant difference in body mass index before, 14 days of Ramadan fasting, and 21 days of Ramadan fasting. The result of testing difference with LSD showed the the average body mass index before fasting Ramadan is higher than and significantly different with body mass index at14 days and at 21 days of Ramadan fasting.The body mass index of respondent at 14 daysand 21 days of Ramadan fasting were not significant different..

Azizi (2010) describes a decrease in the amount of intake of food or drink between dawn and break their time; patients also control or limit the amount or type of food intake of the night after breaking; also due to the restriction of daily activities of the fasting causes weight loss.

5.4 Analysis of Lipid Profile

Analysis of the testing difference lipid profile of overweight male subjects before, of and after fasting Ramadan was based on five indicators, including total cholesterol, triglyceride, HDL cholesterol, and LDL cholesterol.

5.4.1 Analysis of Total Cholesterol

Figure 5.7 shows that total cholesterol21 days of Ramadan fasting was higher than before and 14 days of Ramadan fasting, while total cholesterolbefore fasting Ramadan lower than 14 days of Ramadan fastingand 21 days of Ramadan fasting.Therefore, we can conclude Ramadan fasting can increase cholesteroltotal.This result is consistent with research conducted by Barkia *et al.,* (2011). His study informed that Ramadan fasting could significant increasing total cholesterol. Then Nematy, *et al.,* (2012) in their research show Ramadan fasting significantly improves total cholesterol, and Khaled *et al.*, (2006) also showed that there was increase total cholesterol significantly in overweight.

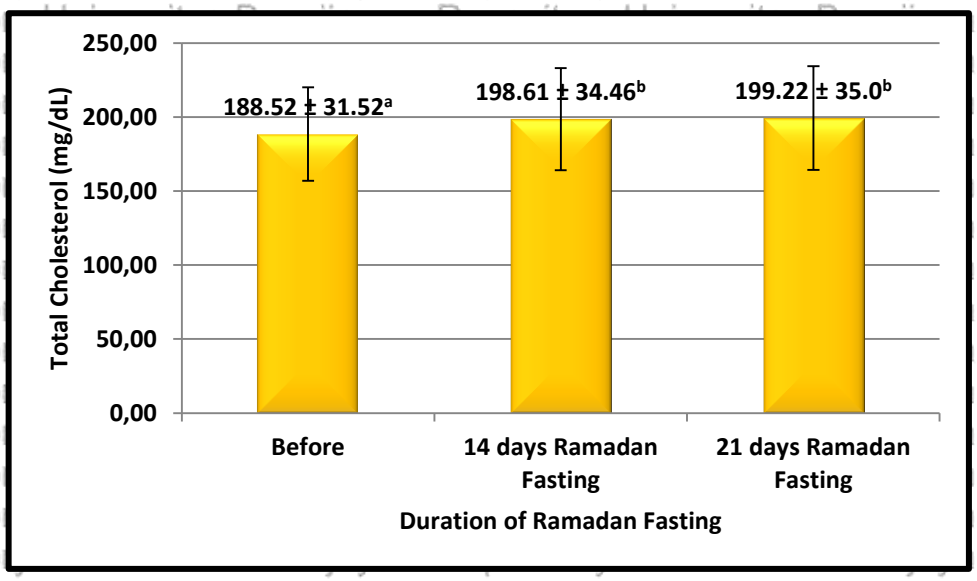

Figure 5.7. The Average of Total cholesterol Before, 14 days of Ramadan fasting, and 21 days of Ramadan fasting

The results of testing difference with Repeated anova in total cholesterol before, 14 days of Ramadan fasting, and 21 days of Ramadan fasting can be seen in Appendix3. From the Appendix 3 it shows that it was significant difference in total cholesterol before, 14 days and 21 days of Ramadan fasting. The result of testing difference with LSD showed the average of total cholesterol before fasting Ramadan is lower than and significantly different with total cholesterol14 days and 21 days of Ramadan fasting. Total cholesterol after fasting Ramadan is the highest and significantly different with before fasting. Than total cholesterol before fasting Ramadan significantly different with total cholesterol21 days of Ramadan fasting.

5.4.2 Analysis of LDL

Figure 5.8 shows that LDL21 days of Ramadan fasting was higher than before and 14 days of Ramadan fasting, while LDL before fasting Ramadan is lower than 14 daysand21 days of Ramadan fasting.Therefore, we can conclude Ramadan fasting can increase LDL. This conclusion is consistent with research conducted by Bouguerra, (2006) and Norouzy *et al.,* (20`1) in their research show Ramadan Fasting was significant improvement in LDL Level. Chaouachi *et al.,* (2008) explained LDL–C increased by 0.20 mmol after Ramadan fasting, than Ziaee *et al.,* (2006) and Shehab *et al.,* (2012) also explained that Ramadan fasting significant improvements in LDL-C level even after 4 weeks post Ramadan.

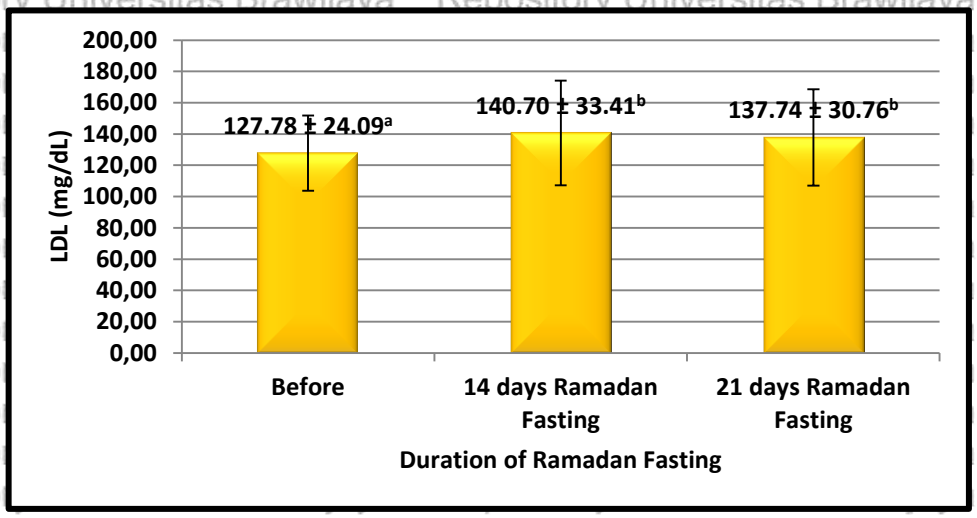

Figure 5.8. The Average of LDL Before, 14 days of Ramadan fasting, and 14 days of Ramadan fasting

The Results of testing difference with repeated anova in LDL before, 14 days of Ramadan fastingand21 days of Ramadan fastingcan be seen in Appendix6. From the Appendix 6 it shows that it was significant difference in LDL before, 14 days of Ramadan fastingand21 days of Ramadan fasting. The result of testing difference with LSD showed the there are significant different between before, 14 days of Ramadan fastingand21 days of Ramadan fasting. The LDL of 21 days of Ramadan fasting is higher and significantly different with LDL before Ramadan fasting, but not significantly different with LDL 14 days of Ramadan fasting..

Because of decreasing Body weight, adipose tissue become shrink thus the fat and cholesterol that normally stored in fatty tissue have nowhere to go but the bloodstream, then IT causing a rise in cholesterol. This effect is not permanent and cholesterol levels will drop as the weight stabilizes. Medications used to to treat high cholesterol, such as Z-hydroxy-Z-Coa reductase inhibitors, are not effective in controlling cholesterol when it comes from fatty tissue stores (Kamal *et al*., 2012; and Ziaee *et al*., 2016).

5.4.3 Analysis of HDL

Figure 5.9showsno significantly different of HDL among before, 14 days of Ramadan fastingand21 days of Ramadan fasting, It was different result with research conducted by Salhamoud*et al.,*(2005)that Serum triglycerides, VLDL cholesterol and HDL cholesterol were not significantly increased 21 days of Ramadan fasting. Then Abdelgadir*et al.,* (2015) showed that there was no significant difference in fasting total cholesterol, triglyceride, HDL and LDL observed in Sudanese and Emiratis group in 2015. Huda*et al.*, (2009) explained a slight but not significant increase (p=0.073) in HDL– C was observed. No significant changes were observed on total cholesterol and LDL – C, and Hagdoost and Pooranjbar (2009) also explained that Ramadan fasting reduced but not significant in HDL cholesterol.

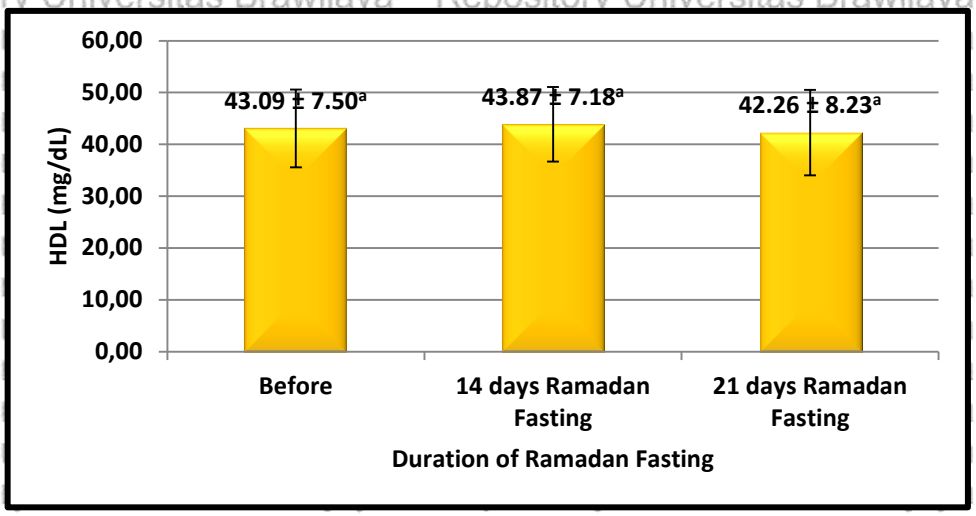

Figure 5.9. The Average of HDL Before, 14 days, and 21 days of Ramadan fasting

The Results of testing difference in HDL before, 14 days of Ramadan fasting, and 21 days of Ramadan fasting can be seen in Appendix5. Statistical difference test with repeated anova shows there was no significant difference before in HDL before, 14 days of Ramadan fasting, and 21 days of Ramadan fasting.

The absence of differences in levels of HDL cholesterol can be explained by the mechanism of Reverse Cholesterol Transport, HDL cholesterol is released as tiny particles of poor cholesterol, containing Apo A, C, and E, and so-called nascent HDL. Nascent HDL comes from the small intestine and liver, flattened shape and containing Apo A-1. Nascent HDL will approach the macrophages to take up cholesterol from macrophages, nascent HDL turn into mature HDL cholesterol, which is round (Dowod, 2005).

In Addition HDL decrease and not significant because the activity that undertaken by the respondent before the fasting of Ramadan is less. The food that consumed by the respondent is not saturated. And the dietary fiber that consumed respondent is quite low.

5.4.4 Analysis of Triglyceride

Figure 5.10 shows no significantly different of triglyceride among before, 14 days of Ramadan fasting, and 21 days of Ramadan fasting, even though it seems the triglyceride 21 days of Ramadan fasting was lower than before Ramadan fasting. It was different result with research conducted by Aksungar, et. Al (2005) and Aksungkar *et al.,* (2007) in their research show Ramadan Fasting

no significantly changed in total cholesterol, triglyceride, and LDL Level, and Khafaji *et al.*, also showed that there were no significant difference in Total cholesterol, triglyceride, serum leptin, or hs-CRP in overweight.

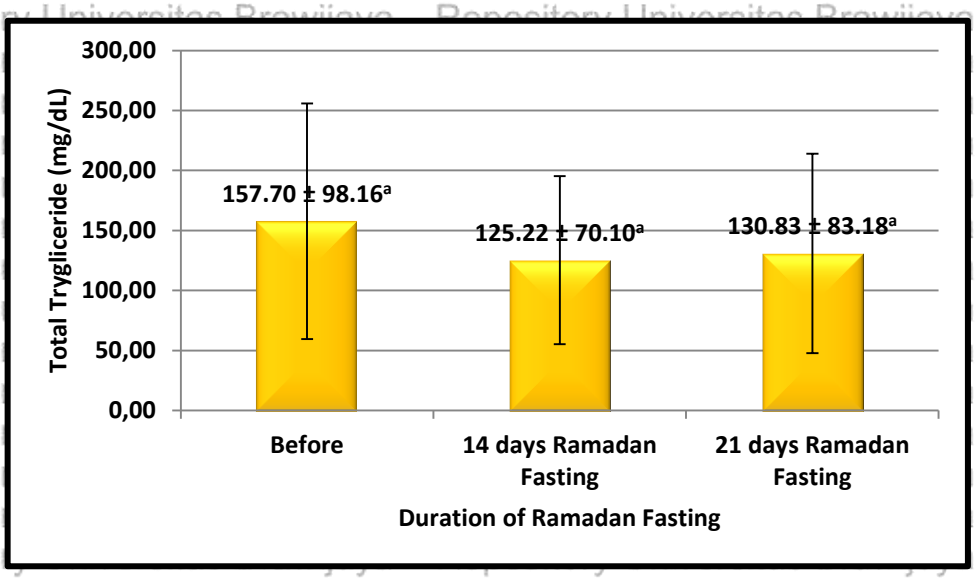

Figure 5.10. The Average of Triglyceride Before, 14 days, and 21 days of Ramadan fasting

The Results of testing difference in triglyceride before, 14 days of Ramadan fasting, and 21 days of Ramadan fasting can be seen in Appendix 4. Statistical difference test with repeated anova shows there was no significant difference before in triglyceride before, 14 days of Ramadan fasting, and 21 days of Ramadan fasting.

The effect of Ramadan fasting on lipid profile, vary in many studies, possibly due to changes in diet and reduced activity. Ziaee et al. (2006) found no difference in levels of triglycerides (TG) were significant before and after Ramadan despite TG levels decreased of Ramadan. This condition is thought to result from the consumption of a diet that is high in carbohydrates, especially sugars. Another cause is a change in the pattern of consumption of complex carbohydrates, such as cereals, fruit and vegetables, into simple carbohydrates such as sugary drinks or with artificial sweeteners of Ramadan (Ziaee et al., 2006).

Guyton and Hall (2006), explain that carbohydrates are a source of energy for the first time in the use of energy by the body, but the amount of carbohydrate reserves stored by the body which is usually only a few hundred grams, especially in the form of glycogen in the liver and muscles. This reserve can provide the energy needed for body functions perhaps only for a half day.

Therefore, there will be a progressive shrinkage of adipose tissue, resulting in free fatty acid levels in plasma increases during fasting and heavy exercise that shows fatty acid needs enormous as an energy source (Guyton and Hall, 2006). This state is achieved by hydrolyzing TG back into fatty acids and glycerol, then both of these compounds are transported to active tissues where both can be oxidized to produce energy (Guyton and Hall, 2006).

5.5 Analysis of Inflammatory Marker

Inflammatory markers of overweight male subjects before and 21 days of Ramadan fasting were based on two indicators, including TNF-α and IL-6.

5.5.1 Analysis of TNF-α

The average TNF-α can be seen in the Figure 5.11.

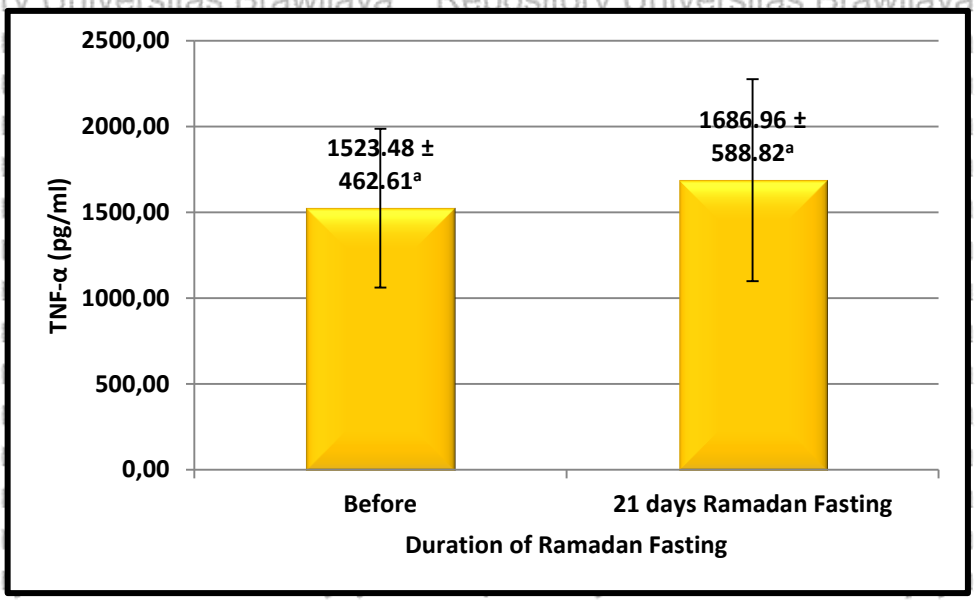

Figure 5.11. The Average of TNF-α Before and 21 days of Ramadan fasting

The results (Figure 5.11) shows that TNF-α 21 days of Ramadan fasting was higher than before fasting Ramadan, while TNF-α before fasting Ramadan lower than 21 days of Ramadan fasting. Therefore, we can conclude Ramadan fasting can increase TNF-α. This result is consistent with research conducted by Lahdimawan*et al.,* (2013). That Ramadan fasting can make TNF-α increased insignificantly. Then Feizollahzadeh*et al.,* (2014) explained that Ramadan fasting did not decrease serum TNF-α levels (P= 0.100), Halberg*et al.,* (2005) also explained that Ramadan fasting did not changes in IL-6 and TNF-α.

The Results of testing difference in TNF-α before and 21 days of Ramadan fasting can be seen in Appendix7. Statistical difference test with paired t-test shows there was no significant difference before in TNF-α before and 21 days of Ramadan fasting.

5.5.2 Analysis of IL-6

The average IL-6 is presented in Figure 5.12 :

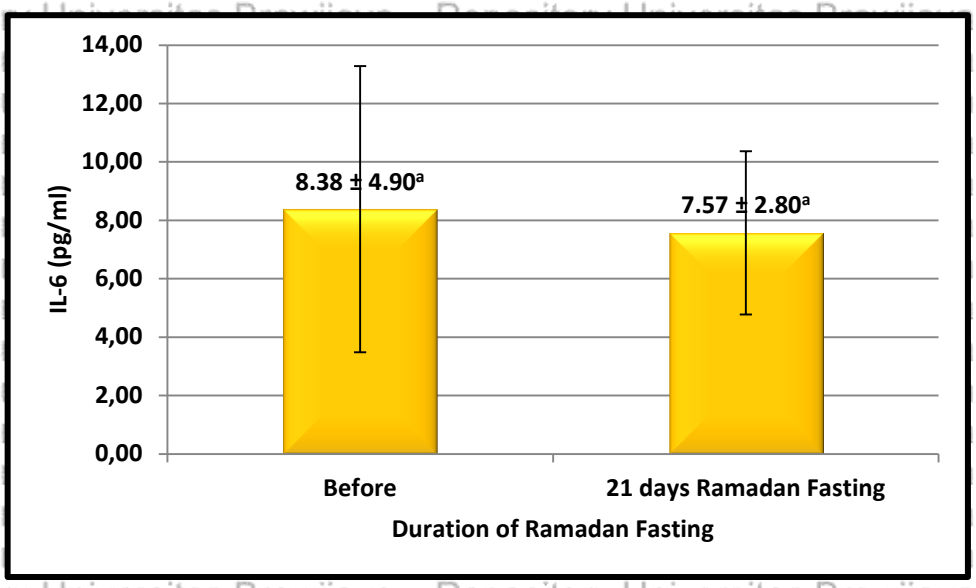

Figure 5.12. The Average of IL-6 Before and 21 Days Of Ramadan Fasting

Statistical analysis of the data IL-6 before and 21 days of Ramadan fasting can be seen in Appendix9. Statistical difference test with paired t-test shows there was no significant difference in IL-6 before and 21 days of Ramadan fasting.

Gustafson *et al*., (2007) explain adipose tissue is fatty tissue that hold the fat reserves are ready for use in the body. Adipose tissue as an active cell, in addition to a role in the backup and energy use also acts as an endocrine organ. Adipose cells actively produce several hormones and cytokines, such as Adiponectin, Leptin, Angiotensin, Resitin, PAI-1, TNF-α, and IL-6. Thus adipose cells play a role in the development of insulin resistance and obesity occur. Cytokine, IL-6 and TNF-α than as an inflammatory reaction in the body's defence mechanisms also have an important role as a hormone in the metabolism of glucose and lipids. But of Ramadan fasting, IL-6 and TNF-α do not decresed properly. This is because BMI is quite low (less than 30), so that the body cannot

decrease the adipose tissue is not much, causing IL-6 and TNF-α produced also becomes insignificant (Gustafson *et al*., 2007).

5.6 Correlation Analysis Between Food Intake and Inflammatory Marker

The results of correlation analysis between food intake and inflammatory marker can be seen in Appendix9. The correlation coefficient between the food intake of the TNF-α = 0.020 with probability = 0.896. The results indicate that the coefficient is positive and the probability >α (5%), so that H0 is accepted. Thus it can be interpreted that there is a positive relationship and that is not significant between the food intake on the TNF-α. This means that the higher the food intake, the higher the TNF-α.

The correlation coefficient between the food intake of the IL-6 = 0.532 with probability of 0.176. The results indicate that the coefficient is positive and the probability >α (5%), so that H0 is accepted. Thus it can be interpreted that there is a positive relationship and that is not significant between the food intake on the IL-6. This means that the higher the food intake, the higher the IL-6.

5.7 Correlation Analysis Between BMI and Inflammatory Marker

The results of correlation analysis between BMI and inflammatory marker can be seen in Appendix10. The correlation coefficient between the BMI of the TNF-α = -0.148 with probability = 0.327. The results indicate that the coefficient is negative and the probability >α (5%), so that H0 is accepted. Thus it can be interpreted that there is a negative relationship and that is not significant between the BMI on the TNF-α. This means that the higher the BMI, the lower the TNF-α.

The correlation coefficient between the BMI of the IL-6 =0.067 with probability of0.657. The results indicate that the coefficient is negative and the probability >α (5%), so that H0 is accepted. Thus it can be interpreted that there is a negative relationship and that is not significant between the BMI on the IL-6. This means that the higher the BMI, the lower the IL-6.

No significant correlation between food intake with inflammatory markers, inflammatory markers and BMI with the BMI because most of the subjects is quite low, below 25 kg / m2, precisely between 23-24.9 kg / m2,so that the body cannot decrease the adipose tissue is not much(Gustafson *et al*., 2007).

CHAPTER VI
CONCLUSION AND RECOMMENDATION

1.1. Conclusions

Based on the research results, it can be concluded that:

1. This study showed food intake of the subjects before, 14 daysand 21 days of Ramadan fasting was significantly different. The average of food intake after Ramadan is lower than those before Ramadan fasting. Therefore food intake of the subjects significantly decrease after 21 days Ramadan fasting.
2. This study showed the body mass index of the subjects before, 14 daysand 21 days of Ramadan fasting was significantly different. The average of body mass index after 21 days of Ramadan fasting is lower than those before Ramadan fasting. Therefore, fasting Ramadan can decrease the body mass index significantly of the subject.
3. This study showed total cholesterol and LDL increase significantly. The TG and HDL of the subjects are not significant difference.
4. TNF-α and IL-6after 21 days fasting are not significant difference.

1.2. Recommendations

Based on the research results, it can be recommended that:

1. Adding parameter observed (carbohydrates, protein, total fat, age, gender, physical activity, psychological factors, etc.) during Ramadan fasting for next study, so that it might be seen clearly the difference influence.
2. Extending the time of observation til the end of Ramadan (30 days)so the expected effect of fasting might be observed
3. The people who are overweight should reduce their weight by fasting, in addition to reducing weight, although can increase cholesterol but the effect is not permanent.

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APPENDIX

Appendix1. Data Observation Food Intake and Analysis of Repeated Anova

a. Observed Data

No	Name	Age (Years)	Food Intake		
			Before	During	After
1	Adi	29	1549.71	1358.74	1294.70
2	Bambang	28	1358.36	1391.31	1117.69
3	Sumantri	23	2025.79	1385.83	1730.87
4	Pungky Prio A.	22	2274.60	1311.20	1680.09
5	Neola Hestu Prayogo	23	1220.51	1073.30	1223.97
6	Mada Maulana Aulia	22	1399.21	1138.07	918.44
7	Yohanes	23	1319.20	915.47	1086.77
8	Noranobel P.B	22	1370.89	1548.73	1286.81
9	Arbi	23	822.14	811.07	959.50
10	Andran D.P	19	1009.21	1327.04	1186.40
11	Adit	22	1447.30	1379.51	1420.39
12	A. Hilmi	25	1967.16	1317.57	909.32
13	A. Razaq	25	1858.60	1554.81	1255.89
14	Virma	24	2164.03	1527.40	1480.41
15	Hakim	24	1571.73	1417.30	904.96
16	RoniNurdianto	19	1235.97	1017.39	1091.93
17	Julian Deni	25	1221.61	1353.67	1204.28
18	Anggadha	23	2159.36	1663.40	1746.10
19	Rudi	20	981.36	906.27	902.69
20	Shelby	20	1199.59	794.17	696.20
21	Dhityo	21	1149.76	1352.27	1212.77
22	Fajar Hani Priyandhika	20	1182.99	1648.89	1390.13
23	Yayan Arifianto	28	1018.00	711.79	609.04

Name	Ages	Duration	Protein	Fat
Adi	29	Before	49.20	68.67
		During	39.24	57.94
		After	61.70	72.70
Bambang	28	Before	61.66	54.94
		During	47.87	47.57
		After	50.80	76.20

Sumantri	23	Before	44.50	47.14
		During	64.87	60.26
		After	35.80	34.80
Pungky Prio A.	22	Before	51.80	47.74
		During	68.67	74.09
		After	71.60	76.80
Neola Hestu Prayogo	23	Before	48.24	50.01
		During	46.16	46.69
		After	56.80	41.90
Mada Maulana Aulia	22	Before	41.01	51.41
		During	37.54	47.34
		After	52.70	60.80
Yohanes	23	Before	35.31	27.59
		During	44.44	34.71
		After	155.70	119.60
Noranobel P.B	22	Before	58.63	58.99
		During	48.57	52.04
		After	23.30	73.10
Arbi	23	Before	34.63	37.44
		During	46.14	44.83
		After	53.60	63.50
Andran D.P	19	Before	52.09	70.96
		During	49.50	61.33
		After	18.00	18.50
Adit	22	Before	56.99	59.19
		During	59.27	59.66
		After	113.30	90.50
A. Hilmi	25	Before	65.40	51.91
		During	37.46	36.98
		After	57.80	60.10
A. Razaq	25	Before	50.10	57.83
		During	51.67	42.56
		After	53.30	38.00
Virma	24	Before	65.44	43.07
		During	58.54	54.09
		After	88.90	97.70
Hakim	24	Before	56.33	47.14
		During	33.91	36.56
		After	21.90	27.70
RoniNurdianto	19	Before	45.44	43.24
		During	42.93	47.45
		After	28.90	40.50
Julian Deni	25	Before	50.80	48.20

		During	49.08	42.08
		After	131.50	80.50
Anggadha	23	Before	62.07	58.81
		During	66.66	57.83
		After	75.70	55.50
Rudi	20	Before	43.96	43.66
		During	45.33	44.93
		After	36.50	94.30
Shelby	20	Before	30.96	31.83
		During	28.50	31.25
		After	30.00	34.10
Dhityo	21	Before	56.63	55.44
		During	49.80	47.64
		After	95.00	110.90
Fajar Hani Priyandhika	20	Before	75.53	88.97
		During	63.85	56.73
		After	85.80	102.30
Yayan Arifianto	28	Before	29.19	28.16
		During	27.14	19.90
		After	50.90	39.80

Name	Ages	Duration	Carbohydrate	Dietary Fiber
Adi	29	Before	145.97	11.51
		During	165.50	13.27
		After	156.80	4.90
Bambang	28	Before	169.83	9.46
		During	129.66	7.80
		After	93.40	12.10
Sumantri	23	Before	199.84	13.53
		During	233.47	7.93
		After	130.50	3.70
Pungky Prio A.	22	Before	168.77	6.17
		During	187.67	7.90
		After	276.80	12.50
Neola Hestu Prayogo	23	Before	109.14	3.81
		During	159.14	8.21
		After	180.00	6.50
Mada Maulana Aulia	22	Before	126.10	3.51
		During	84.50	4.06
		After	69.90	5.00
Yohanes	23	Before	132.40	4.73
		During	149.61	5.51

		After	273.50	7.30
Noranobel P.B	22	Before	199.14	7.30
		During	155.33	3.76
		After	162.20	4.30
Arbi	23	Before	85.01	4.04
		During	94.24	5.64
		After	148.90	6.30
Andran D.P	19	Before	121.54	4.39
		During	110.95	4.38
		After	79.40	2.70
Adit	22	Before	159.17	7.69
		During	164.00	6.69
		After	243.70	8.00
A. Hilmi	25	Before	147.29	4.89
		During	104.88	2.48
		After	146.60	7.50
A. Razaq	25	Before	214.31	10.03
		During	168.83	9.49
		After	214.30	13.40
Virma	24	Before	224.79	11.71
		During	194.83	11.67
		After	243.20	13.20
Hakim	24	Before	191.01	5.79
		During	110.41	2.57
		After	75.30	1.20
RoniNurdianto	19	Before	112.53	4.17
		During	124.00	4.22
		After	78.00	3.70
Julian Deni	25	Before	177.46	7.31
		During	154.27	5.18
		After	456.00	9.50
Anggadha	23	Before	224.16	11.10
		During	243.34	13.54
		After	310.00	14.60
Rudi	20	Before	84.97	2.77
		During	78.71	3.49
		After	218.10	2.20
Shelby	20	Before	95.66	4.21
		During	74.18	2.40
		After	120.10	4.30
Dhityo	21	Before	155.24	6.00
		During	147.13	5.11
		After	149.80	5.70

Fajar Hani Priyandhika	20	Before	146.00	11.60
		During	156.58	8.20
		After	200.40	12.70
Yayan Arifianto	28	Before	85.91	3.73
		During	83.93	6.01
		After	142.20	5.40

b. Descriptive Analysis

Food intake	Minimum	Maximum	Average	Standard Deviation
Before	822.14	2274.60	1456.83	420.31
During	711.79	1663.40	1256.75	280.95
After	609.04	1746.10	1187.36	303.18

c. Normality Test

One-Sample Kolmogorov-Smirnov Test

		Food_Intake
N		69
Normal Parameters[a,b]	Mean	1300.3136
	Std. Deviation	354.50298
Most Extreme Differences	Absolute	.107
	Positive	.107
	Negative	-.044
Kolmogorov-Smirnov Z		.885
Asymp. Sig. (2-tailed)		.414

a. Test distribution is Normal.

b. Calculated from data.

d. Repeated Anova

Tests of Within-Subjects Contrasts

Measure:MEASURE_1

Source	Food_Intakes	Type III Sum of Squares	df	Mean Square	F	Sig.
Food_Intakes	Linear	835036.523	1	835036.523	16.001	.001
	Quadratic	65478.459	1	65478.459	1.681	.208
Error(Food_Intakes)	Linear	1148136.283	22	52188.013		
	Quadratic	856925.000	22	38951.136		

e. Multiple Comparison (LSD Test)

Pairwise Comparisons

Measure:MEASURE_1

(I) Food_Intakes	(J) Food_Intakes	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Before	During	200.081*	71.973	.011	50.818	349.344
	After	269.466*	67.365	.001	129.759	409.173
During	Before	-200.081*	71.973	.011	-349.344	-50.818
	After	69.385	46.578	.151	-27.211	165.982
After	Before	-269.466*	67.365	.001	-409.173	-129.759
	During	-69.385	46.578	.151	-165.982	27.211

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons : Least Significant Difference (equivalent to no adjustments).

Food intake	Mean	Probability			Notation
		After	During	Before	
After	1187.363		0.151	0.001	a
During	1256.748	0.151		0.011	a
Before	1456.829	0.001	0.011		b

Appendix2. Data Observation Body Mass Index and Analysis of Repeated Anova

a. Observed Data

No	Name	Age (Years)	BMI		
			Before	During	After
1	Adi	29	32.7	31.5	31.4
2	Bambang	28	26.3	25.8	25.6
3	Sumantri	23	28.5	28	27.3
4	Pungky Prio A.	22	29.3	28.5	28.7
5	Neola Hestu Prayogo	23	29	29.5	28.8
6	Mada Maulana Aulia	22	26.7	27.1	26.8
7	Yohanes	23	26.1	25.3	25.3
8	Noranobel P.B	22	26.7	26.7	27
9	Arbi	23	27.05	27.2	27.2
10	Andran D.P	19	38.8	38.9	39.1
11	Adit	22	28	28.2	28.1
12	A. Hilmi	25	28.5	28.1	28
13	A. Razaq	25	25.5	24.7	25.1
14	Virma	24	27.2	27	27
15	Hakim	24	28.5	24.2	28.1
16	RoniNurdianto	19	29.8	28.8	28.7
17	Julian Deni	25	31.7	31.1	30.9
18	Anggadha	23	30.1	29.4	27.7
19	Rudi	20	31.9	31.8	31.7
20	Shelby	20	30.05	29.3	29.2
21	Dhityo	21	28.8	29.1	29
22	Fajar Hani Priyandhika	20	33.2	32.9	33.9
23	Yayan Arifianto	28	27.2	26.9	26.6

Name	Ages	Duration	Waist Circum-ference	Hip Circum-ference	Waist/Hip Circumference Ratio
Adi	29	Before	100.45	102.95	0.97
		During	100.05	104.05	0.96
		After	100	104.5	0.95
Bambang	28	Before	97.4	103.25	0.94
		During	97.8	103.35	0.94
		After	97.5	103.25	0.94
Sumantri	23	Before	85.55	96.5	0.88
		During	85	96.25	0.88
		After	85	96.8	0.87

Pungky Prio A.	22	Before	97.5	104.2	0.93
		During	94	101.5	0.92
		After	94	102	0.92
Neola Hestu Prayogo	23	Before	88.6	94.6	0.93
		During	89	94.6	0.94
		After	88	94	0.93
Mada Maulana Aulia	22	Before	89.8	100.4	0.89
		During	92	99.65	0.92
		After	92.1	100	0.92
Yohanes	23	Before	80.55	100	0.8
		During	80.1	98.3	0.8
		After	80	100.1	0.79
Noranobel P.B	22	Before	90.5	108	0.835
		During	90.75	108	0.835
		After	90	101.5	0.83
Arbi	23	Before	92.75	101.55	0.91
		During	92.05	100.05	0.91
		After	92	101	0.91
Andran D.P	19	Before	118.25	117.4	1
		During	117	117.4	0.99
		After	117	117.8	0.99
Adit	22	Before	99.65	111.5	0.89
		During	100.3	111.1	0.9
		After	99	110.9	0.89
A. Hilmi	25	Before	103.15	111.95	0.92
		During	102.05	110	0.92
		After	101.55	109.5	0.92
A. Razaq	25	Before	91.5	101.5	0.9
		During	90.5	101	0.89
		After	90	101.1	0.885
Virma	24	Before	98.9	106.4	0.925
		During	97	106.5	0.91
		After	97	106.55	0.91
Hakim	24	Before	87.5	101.1	0.86
		During	87.3	101.2	0.86
		After	87.05	101	0.86
RoniNurdianto	19	Before	91.25	105.95	0.86
		During	91	105.2	0.86
		After	91	105.5	0.91
Julian Deni	25	Before	102.05	107.5	0.945
		During	101.8	107.5	0.94
		After	101.5	107.5	0.49
Anggadha	23	Before	104.5	105.1	0.99
		During	104.05	104.25	0.995
		After	104	104.5	0.99

Rudi	20	Before	99.9	102.9	0.965
		During	99.9	102	0.965
		After	100	103	0.97
Shelby	20	Before	101.1	101.65	0.99
		During	100.1	102.2	0.97
		After	100.3	103	0.97
Dhityo	21	Before	93.5	108.9	0.86
		During	93.5	108	0.86
		After	93.5	107	0.87
Fajar Hani Priyandhika	20	Before	107	108.8	0.98
		During	108	109	0.99
		After	108.5	109.5	0.99
Yayan Arifianto	28	Before	90.25	100.45	0.895
		During	88.65	99.9	0.88
		After	88	100.1	0.87

Name	**Ages**	**Duration**	**Weight**	**Percent Body Fat**	**Visceral Fat**
Adi	29	Before	94.5	29.2	18
		During	91.7	29.2	17
		After	90.7	28.6	16
Bambang	28	Before	76.35	25.2	10
		During	74.9	26.5	10
		After	74.1	27.3	10
Sumantri	23	Before	72	22	12
		During	70.1	21.4	12
		After	69.1	20.9	11
Pungky Prio A.	22	Before	88.8	26.6	12
		During	86.4	25.9	12
		After	86.8	27	12
Neola Hestu Prayogo	23	Before	76.7	24.9	13
		During	77.3	23.3	13
		After	75.5	22.7	12
Mada Maulana Aulia	22	Before	75.8	21.8	18
		During	77	22.5	10
		After	26.2	21.2	10
Yohanes	23	Before	67.7	23.55	10
		During	65.55	21.1	9
		After	65.6	21.3	9
Noranobel P.B	22	Before	79.5	24.3	10
		During	79.5	22.9	10
		After	80.3	22.8	10
Arbi	23	Before	70.15	28.15	11
		During	70.4	27.4	11
		After	70.4	27.3	11

Andran D.P	19	Before	111.6	35.7	23
		During	111	35.1	23
		After	111.6	34.4	23
Adit	22	Before	94.3	26.2	11
		During	94.8	24.7	11
		After	94.2	25.5	11
A. Hilmi	25	Before	89.8	26.9	12
		During	88.5	24.9	12
		After	88.1	22.1	11
A. Razaq	25	Before	75	27.7	9
		During	73.1	18.1	7
		After	74.2	19.6	7
Virma	24	Before	86.05	26.7	11
		During	84.6	27.6	11
		After	85.7	26.7	11
Hakim	24	Before	75.7	28	13
		During	75	29.2	12
		After	74.6	26.4	12
RoniNurdianto	19	Before	87.15	26.7	13
		During	84.3	26	12
		After	84	25.9	12
Julian Deni	25	Before	90.6	27.3	16
		During	88.7	25.7	15
		After	88.2	26.3	15
Anggadha	23	Before	93.7	28.2	13
		During	92	26.5	12
		After	92.6	28.4	13
Rudi	20	Before	84.8	32.8	16
		During	84.5	32.1	16
		After	84.1	32	15
Shelby	20	Before	86.65	30.35	14
		During	76.8	29.2	13
		After	76.6	28.7	12
Dhityo	21	Before	83.6	26.6	12
		During	84.1	26.1	12
		After	84.4	26.9	12
Fajar Hani Priyandhika	20	Before	98.9	30.6	17
		During	100.2	30.1	18
		After	100.4	30.3	18
Yayan Arifianto	28	Before	74.4	26.7	12
		During	73.2	25.9	11
		After	72.5	25.3	11

Name	Ages	Duration	Resting Metabolism	Triceps	Height
Adi	29	Before	1962	19	170
		During	1907	18	170
		After	1906	18	170
Bambang	28	Before	1696.5	15	170.6
		During	1666	15	170.6
		After	1649	15	170.6
Sumantri	23	Before	1639	15	159.15
		During	1658	15.05	159
		After	1597	15	159
Pungky Prio A.	22	Before	1894	16	173.95
		During	1859	15	174
		After	1858	15	174
Neola Hestu Prayogo	23	Before	1701	14.5	162.2
		During	1721	15	162.2
		After	1695	14	162.2
Mada Maulana Aulia	22	Before	1709	15	168.5
		During	1725	15	168.5
		After	1719	15	168.5
Yohanes	23	Before	1561.5	16	161
		During	1539	15	161
		After	1539	15	161
Noranobel P.B	22	Before	1757	15	172.5
		During	1766	15	172.5
		After	1779	15	172.5
Arbi	23	Before	1579	17	161.15
		During	1586	17	161.2
		After	1587	17	161.1
Andran D.P	19	Before	2178	25	169.4
		During	2175	24	169.4
		After	2191	24	169.4
Adit	22	Before	1990	17	183.3
		During	2011	17	183.3
		After	1995	17	183.3
A. Hilmi	25	Before	1908	16	177.65
		During	1902	16	177.5
		After	1902	16	177.5
A. Razaq	25	Before	1672	15.5	172.05
		During	1687	15	172
		After	1738	15	172
Virma	24	Before	1851	15	177.8
		During	1820	15	177.8
		After	1844	15	177.8
Hakim	24	Before	1667	18	163.1

		During	1676	18	163.1
		After	1659	18	163.1
RoniNurdianto	19	Before	1867	16	171.15
		During	1824	15	171.1
		After	1826	15	171.1
Julian Deni	25	Before	1915	15	168.8
		During	1897	14	168.9
		After	1884	14	168.9
Anggadha	23	Before	1962	16	176.85
		During	1948	16	176.8
		After	1943	16	176.8
Rudi	20	Before	1784	20	163.2
		During	1783	20	163.2
		After	1777	20	163.2
Shelby	20	Before	1704.5	17	162
		During	1679	16.15	162
		After	1679	16.1	162
Dhityo	21	Before	1808	17	170.6
		During	1819	17	170.6
		After	1819	17	170.6
Fajar Hani Priyandhika	20	Before	2026	20	177.4
		During	2050	21	172
		After	2051	21	172
Yayan Arifianto	28	Before	1654	16	165.25
		During	1642	16	165
		After	1631	15	165

b. Descriptive Analysis

Body Mass Index	Minimum	Maximum	Average	Standard Deviation
Before	25.50	38.80	29.05	3.00
During	24.20	38.90	28.55	3.16
After	24.90	39.10	28.59	3.13

c. Normality Test

One-Sample Kolmogorov-Smirnov Test

		BMI
N		69
Normal Parameters[a,b]	Mean	28.8159
	Std. Deviation	3.10267
Most Extreme Differences	Absolute	.152
	Positive	.152
	Negative	-.078
Kolmogorov-Smirnov Z		1.262
Asymp. Sig. (2-tailed)		.083

a. Test distribution is Normal.

b. Calculated from data.

d. Repeated anova

Tests of Within-Subjects Contrasts

Measure:MEASURE_1

Source	BMI	Type III Sum of Squares	df	Mean Square	F	Sig.
BMI	Linear	2.875	1	2.875	13.239	.001
	Quadratic	1.132	1	1.132	2.096	.162
Error(BMI)	Linear	4.778	22	.217		
	Quadratic	11.882	22	.540		

e. Multiple Comparison (LSD Test)

Pair wise Comparisons

Measure:MEASURE_1

(I) BMI	(J) BMI	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Before	During	.522*	.197	.015	.113	.930
	After	.500*	.137	.001	.215	.785
During	Before	-.522*	.197	.015	-.930	-.113
	After	-.022	.203	.916	-.442	.399
After	Before	-.500*	.137	.001	-.785	-.215
	During	.022	.203	.916	-.399	.442

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons : Least Significant Difference (equivalent to no adjustments).

BMI	Mean	Probability			Notation
		During	After	Before	
During	28.696		0.916	0.015	A
After	28.748	0.916		0.001	A
Before	29.200	0.015	0.001		B

Appendix3. Data Observation Total Cholesterol and Analysis of Repeated Anova

a. Observed Data

No	Name	Age (Years)	Cholesterol (mg/dL)		
			Before	During	After
1	Adi	29	172	206	213
2	Bambang	28	174	190	180
3	Sumantri	23	209	234	227
4	Pungky Prio A.	22	185	224	243
5	Neola Hestu Prayogo	23	162	166	173
6	Mada Maulana Aulia	22	221	227	258
7	Yohanes	23	174	177	186
8	Noranobel P.B	22	184	181	196
9	Arbi	23	229	239	238
10	Andran D.P	19	130	139	136
11	Adit	22	179	190	197
12	A. Hilmi	25	230	222	234
13	A. Razaq	25	171	156	166
14	Virma	24	163	157	163
15	Hakim	24	207	214	219
16	RoniNurdianto	19	148	202	142
17	Julian Deni	25	205	224	209
18	Anggadha	23	139	130	130
19	Rudi	20	212	230	223
20	Shelby	20	221	229	222
21	Dhityo	21	173	194	193
22	Fajar Hani Priyandhika	20	192	175	220
23	Yayan Arifianto	28	256	262	214

b. Descriptive Analysis

Total cholesterol	Minimum	Maximum	Average	Standard Deviation
Before	130.00	256.00	188.67	30.84
During	130.00	262.00	197.96	33.86
After	130.00	258.00	198.83	34.33

c. Normality Test

One-Sample Kolmogorov-Smirnov Test

		TC
N		69
Normal Parameters[a,b]	Mean	195.4493
	Std. Deviation	33.58069
Most Extreme Differences	Absolute	.092
	Positive	.048
	Negative	-.092
Kolmogorov-Smirnov Z		.762
Asymp. Sig. (2-tailed)		.606

a. Test distribution is Normal.

b. Calculated from data.

d. Repeated anova

Tests of Within-Subjects Contrasts

Measure:MEASURE_1

Source	Cholesterol	Type III Sum of Squares	df	Mean Square	F	Sig.
Cholesterol	Linear	1315.565	1	1315.565	6.907	.015
	Quadratic	344.377	1	344.377	1.780	.196
Error(Cholesterol)	Linear	4190.435	22	190.474		
	Quadratic	4256.290	22	193.468		

e. Multiple Comparison (LSD Test)

Pairwise Comparisons

Measure:MEASURE_1

(I) Cholesterol	(J) Cholesterol	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Before	During	-10.087*	3.577	.010	-17.506	-2.668
	After	-10.696*	4.070	.015	-19.136	-2.255
During	Before	10.087*	3.577	.010	2.668	17.506
	After	-.609	4.552	.895	-10.048	8.831
After	Before	10.696*	4.070	.015	2.255	19.136
	During	.609	4.552	.895	-8.831	10.048

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons : Least Significant Difference (equivalent to no adjustments).

Total cholesterol	Mean	Probability			Notation
		before	During	After	
Before	188.6667		0.010	0.015	A
During	197.9583	0.010		0.895	B
After	198.8333	0.015	0.895		B

Appendix4. Data Observation Triglyceride and Analysis of Repeated Anova

a. Observed Data

No	Name	Age (Years)	Triglyceride (mg/dL)		
			Before	During	After
1	Adi	29	93	152	103
2	Bambang	28	132	91	107
3	Sumantri	23	109	53	42
4	Pungky Prio A.	22	124	72	170
5	Neola Hestu Prayogo	23	84	157	78
6	Mada Maulana Aulia	22	211	137	163
7	Yohanes	23	83	61	87
8	Noranobel P.B	22	148	126	98
9	Arbi	23	467	147	142
10	Andran D.P	19	103	82	77
11	Adit	22	160	145	224
12	A. Hilmi	25	302	139	129
13	A. Razaq	25	79	44	53
14	Virma	24	46	33	54
15	Hakim	24	256	256	295
16	RoniNurdianto	19	88	96	62
17	Julian Deni	25	153	163	134
18	Anggadha	23	42	35	32
19	Rudi	20	88	61	59
20	Shelby	20	230	148	171
21	Dhityo	21	153	164	212
22	Fajar Hani Priyandhika	20	255	311	367
23	Yayan Arifianto	28	221	207	150

b. Descriptive Analysis

Triglyceride	Minimum	Maximum	Average	Standard Deviation
Before	42.00	467.00	157.46	96.01
During	33.00	311.00	124.00	68.82
After	32.00	367.00	128.54	82.12

c. Normality Test

One-Sample Kolmogorov-Smirnov Test

		TG
N		69
Normal Parameters[a,b]	Mean	137.9130
	Std. Deviation	84.55519
Most Extreme Differences	Absolute	.147
	Positive	.147
	Negative	-.105
Kolmogorov-Smirnov Z		1.221
Asymp. Sig. (2-tailed)		.102

a. Test distribution is Normal.

b. Calculated from data.

d. Repeated anova

Tests of Within-Subjects Contrasts

Measure:MEASURE_1

Source	Trygliseride	Type III Sum of Squares	df	Mean Square	F	Sig.
Trygliseride	Linear	8302.696	1	8302.696	2.218	.151
	Quadratic	5560.696	1	5560.696	3.744	.066
Error(Trygliseride)	Linear	82346.304	22	3743.014		
	Quadratic	32670.971	22	1485.044		

e. Multiple Comparison (LSD Test)

Pairwise Comparisons

Measure:MEASURE_1

(I) Trygliseride	(J) Trygliseride	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Before	During	32.478	16.673	.064	-2.099	67.056
	After	26.870	18.041	.151	-10.545	64.284
During	Before	-32.478	16.673	.064	-67.056	2.099
	After	-5.609	8.857	.533	-23.978	12.760
After	Before	-26.870	18.041	.151	-64.284	10.545
	During	5.609	8.857	.533	-12.760	23.978

Based on estimated marginal means

a. Adjustment for multiple comparisons : Least Significant Difference (equivalent to no adjustments).

Triglyceride	Mean	Probability			Notation
		During	After	Before	
During	124.0000		0.533	0.064	A
After	128.5417	0.533		0.151	A
Before	157.4583	0.064	0.151		A

Appendix5. Data Observation HDL and Analysis of Repeated Anova

a. Observed Data

No	Name	Age (Years)	HDL Cholesterol (mg/dL)		
			before	During	After
1	Adi	29	43	45	41
2	Bambang	28	37	40	34
3	Sumantri	23	42	48	46
4	Pungky Prio A.	22	37	45	44
5	Neola Hestu Prayogo	23	54	51	52
6	Mada Maulana Aulia	22	50	53	56
7	Yohanes	23	46	43	43
8	Noranobel P.B	22	48	52	58
9	Arbi	23	39	43	41
10	Andran D.P	19	38	38	40
11	Adit	22	49	46	47
12	A. Hilmi	25	37	37	39
13	A. Razaq	25	47	41	41
14	Virma	24	53	45	45
15	Hakim	24	35	31	29
16	RoniNurdianto	19	38	45	31
17	Julian Deni	25	41	42	38
18	Anggadha	23	55	52	48
19	Rudi	20	50	56	48
20	Shelby	20	40	40	38
21	Dhityo	21	33	34	31
22	Fajar Hani Priyandhika	20	27	29	29
23	Yayan Arifianto	28	52	53	53

b. Descriptive Analysis

HDL	Minimum	Maximum	Average	Standard Deviation
Before	27.00	55.00	43.04	7.34
During	29.00	56.00	43.67	7.09
After	29.00	58.00	42.04	8.12

c. Normality Test

One-Sample Kolmogorov-Smirnov Test

		HDL
N		69
Normal Parameters[a,b]	Mean	43.0725
	Std. Deviation	7.56408
Most Extreme Differences	Absolute	.069
	Positive	.057
	Negative	-.069
Kolmogorov-Smirnov Z		.577
Asymp. Sig. (2-tailed)		.893

a. Test distribution is Normal.

b. Calculated from data.

d. Repeated anova

Tests of Within-Subjects Contrasts

Measure:MEASURE_1

Source	HDL	Type III Sum of Squares	df	Mean Square	F	Sig.
HDL	Linear	7.848	1	7.848	.709	.409
	Quadratic	21.920	1	21.920	2.861	.105
Error(HDL)	Linear	243.652	22	11.075		
	Quadratic	168.580	22	7.663		

e. Multiple Comparison (LSD Test)

Pair wise Comparisons

Measure:MEASURE_1

(I) HDL	(J) HDL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Before	During	-.783	.871	.379	-2.589	1.024
	After	.826	.981	.409	-1.209	2.861
During	Before	.783	.871	.379	-1.024	2.589
	After	1.609	.850	.072	-.154	3.371
After	Before	-.826	.981	.409	-2.861	1.209
	During	-1.609	.850	.072	-3.371	.154

Based on estimated marginal means

a. Adjustment for multiple comparisons : Least Significant Difference (equivalent to no adjustments).

HDL	Mean	Probability			Notation
		After	Before	During	
After	42.0417		0.409	0.072	A
Before	43.0417	0.409		0.379	A
During	43.6667	0.072	0.379		A

Appendix6. Data Observation LDL and Analysis of Repeated Anova

a. Observed Data

No	Name	Age (Years)	LDL Cholesterol (mg/dL)		
			Before	During	After
1	Adi	29	124	153	161
2	Bambang	28	125	147	135
3	Sumantri	23	138	171	167
4	Pungky Prio A.	22	133	178	173
5	Neola Hestu Prayogo	23	98	100	109
6	Mada Maulana Aulia	22	154	166	183
7	Yohanes	23	122	129	131
8	Noranobel P.B	22	118	115	123
9	Arbi	23	135	178	179
10	Andran D.P	19	86	96	89
11	Adit	22	120	129	124
12	A. Hilmi	25	151	166	178
13	A. Razaq	25	120	117	119
14	Virma	24	109	105	110
15	Hakim	24	148	143	147
16	RoniNurdianto	19	104	149	97
17	Julian Deni	25	154	168	153
18	Anggadha	23	79	70	69
19	Rudi	20	159	172	164
20	Shelby	20	149	174	157
21	Dhityo	21	121	134	128
22	Fajar Hani Priyandhika	20	115	88	127
23	Yayan Arifianto	28	177	188	145

b. Descriptive Analysis

LDL	Minimum	Maximum	Average	Standard Deviation
Before	79.00	177.00	128.21	23.65
During	70.00	188.00	140.42	32.71
After	69.00	183.00	137.88	30.09

c. Normality Test

One-Sample Kolmogorov-Smirnov Test

		LDL
N		69
Normal Parameters[a,b]	Mean	135.4058
	Std. Deviation	29.76739
Most Extreme Differences	Absolute	.072
	Positive	.049
	Negative	-.072
Kolmogorov-Smirnov Z		.597
Asymp. Sig. (2-tailed)		.869

a. Test distribution is Normal.

b. Calculated from data.

d. Repeated anova

Tests of Within-Subjects Contrasts

Measure:MEASURE_1

Source	LDL	Type III Sum of Squares	df	Mean Square	F	Sig.
LDL	Linear	1140.022	1	1140.022	7.326	.013
	Quadratic	965.399	1	965.399	5.666	.026
Error(LDL)	Linear	3423.478	22	155.613		
	Quadratic	3748.435	22	170.383		

e. Multiple Comparison (LSD Test)

Pairwise Comparisons

Measure:MEASURE_1

(I) LDL	(J) LDL	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
Before	During	-12.913*	3.780	.002	-20.752	-5.074
	After	-9.957*	3.679	.013	-17.585	-2.328
During	Before	12.913*	3.780	.002	5.074	20.752
	After	2.957	3.834	.449	-4.995	10.908
After	Before	9.957*	3.679	.013	2.328	17.585
	During	-2.957	3.834	.449	-10.908	4.995

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons : Least Significant Difference (equivalent to no adjustments).

LDL	Mean	Probability			Notation
		Before	After	During	
Before	128.2083		0.013	0.002	A
After	137.8750	0.013		0.449	B
During	140.4167	0.002	0.449		B

Appendix7. Data Observation TNF-α and Analysis of Repeated Anova

a. Observed Data

No	Name	Age (Years)	Level TNF alfa (pg/mL)	
			Before	After
1	Adi	29	1800	800
2	Bambang	28	1000	1720
3	Sumantri	23	1010	1830
4	Pungky Prio A.	22	1490	1930
5	Neola Hestu Prayogo	23	1040	2160
6	Mada Maulana Aulia	22	1120	2110
7	Yohanes	23	750	1670
8	Noranobel P.B	22	1270	1750
9	Arbi	23	1980	3630
10	Andran D.P	19	1760	1130
11	Adit	22	1930	1220
12	A. Hilmi	25	2100	1400
13	A. Razaq	25	2250	1670
14	Virma	24	2520	1260
15	Hakim	24	2020	1170
16	RoniNurdianto	19	1500	720
17	Julian Deni	25	1100	1210
18	Anggadha	23	1510	1850
19	Rudi	20	1280	1870
20	Shelby	20	1240	1980
21	Dhityo	21	1780	1700
22	Fajar Hani Priyandhika	20	1210	2000
23	Yayan Arifianto	28	1380	2020

b. Descriptive Analysis

TNF-α	Minimum	Maximum	Average	Standard Deviation
Before	750	2520	1523.48	462.61
After	720	3630	1686.96	588.82

c. Normality Test

One-Sample Kolmogorov-Smirnov Test

		IL_6
N		46
Normal Parameters[a,b]	Mean	7.9747
	Std. Deviation	3.96743
Most Extreme Differences	Absolute	.112
	Positive	.112
	Negative	-.065
Kolmogorov-Smirnov Z		.757
Asymp. Sig. (2-tailed)		.615

a. Test distribution is Normal.

b. Calculated from data.

d. Paired T Test

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	IL-6Before	8.3805	23	4.90120	1.02197
	IL-6 After	7.5688	23	2.79829	.58348

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	IL-6Before& IL-6 After	23	-.497	.016

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	IL-6Before-IL-6 After	.81165	6.74378	1.40618	-2.10458	3.72788	.577	22	.570

Appendix8. Data Observation IL-6 and Analysis of Repeated Anova

a. Observed Data

No	Name	Age (Years)	Level IL-6 (pg/mL)	
			Before	After
1	Adi	29	10.750	3.000
2	Bambang	28	5.500	4.917
3	Sumantri	23	6.500	5.250
4	Pungky Prio A.	22	4.417	7.833
5	Neola Hestu Prayogo	23	8.417	9.250
6	Mada Maulana Aulia	22	12.167	9.833
7	Yohanes	23	3.250	9.750
8	Noranobel P.B	22	2.833	11.583
9	Arbi	23	10.417	6.667
10	Andran D.P	19	6.917	6.167
11	Adit	22	9.167	7.167
12	A. Hilmi	25	9.917	2.583
13	A. Razaq	25	10.250	10.250
14	Virma	24	10.750	7.583
15	Hakim	24	22.417	5.500
16	RoniNurdianto	19	18.833	3.750
17	Julian Deni	25	9.833	6.000
18	Anggadha	23	7.583	9.000
19	Rudi	20	2.750	12.917
20	Shelby	20	1.750	7.667
21	Dhityo	21	7.333	8.000
22	Fajar Hani Priyandhika	20	4.250	12.333
23	Yayan Arifianto	28	6.750	7.083

b. Descriptive Analysis

IL-6	Minimum	Maximum	Average	Standard Deviation
Before	1.75	22.42	8.38	4.90
After	2.58	12.92	7.57	2.80

e. Normality Test

One-Sample Kolmogorov-Smirnov Test

		TNF_Alpha
N		46
Normal Parameters[a,b]	Mean	1605.2174
	Std. Deviation	530.05551
Most Extreme Differences	Absolute	.100
	Positive	.100
	Negative	-.070
Kolmogorov-Smirnov Z		.677
Asymp. Sig. (2-tailed)		.749

a. Test distribution is Normal.

b. Calculated from data.

f. Paired T Test

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	TNFBefore	1523.4783	23	462.60732	96.46029
	TNF After	1686.9565	23	588.82344	122.77817

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	TNFBefore& TNF After	23	-.182	.407

Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	TNFBefore - TNF After	-163.47826	812.20580	169.35662	-514.70239	187.74586	-.965	22	.345

Appendix9. Correlation between Food Intake on Inflammatory Marker

Correlations

			Food	TNF_Alpha	IL_6
Spearman's rho	Food	Correlation Coefficient	1.000	.020	.203
		Sig. (2-tailed)	.	.896	.176
		N	46	46	46
	TNF_Alpha	Correlation Coefficient	.020	1.000	.532**
		Sig. (2-tailed)	.896	.	.000
		N	46	46	46
	IL_6	Correlation Coefficient	.203	.532**	1.000
		Sig. (2-tailed)	.176	.000	.
		N	46	46	46

**. Correlation is significant at the 0.01 level (2-tailed).

Appendix10. Correlation between BMI on Inflammatory Marker

Correlations

			BMI	TNF_Alpha	IL_6
Spearman's rho	BMI	Correlation Coefficient	1.000	-.148	-.067
		Sig. (2-tailed)	.	.327	.657
		N	46	46	46
	TNF_Alpha	Correlation Coefficient	-.148	1.000	.532**
		Sig. (2-tailed)	.327	.	.000
		N	46	46	46
	IL_6	Correlation Coefficient	-.067	.532**	1.000
		Sig. (2-tailed)	.657	.000	.
		N	46	46	46

**. Correlation is significant at the 0.01 level (2-tailed).