UNIVERSITY OF INDONESIA

RELATIONSHIP BETWEEN INTAKES OF DIFFERENT FATTY ACIDS AND INSULIN LEVEL IN ABDOMINAL OBESE ADULT MEN IN JAKARTA

THESIS
in partial fulfillment of the requirements for the degree of
Master of Science in Community Nutrition

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STUDY PROGRAM IN NUTRITION
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AUTHOR'S DECLARATION OF ORIGINALITY PAGE

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PREFACE

The increasing prevalence of diabetes melitus in Indonesia, emphasize the importance of early detection of insulin resistance. Insulin resistance is a silent condition that increases the chances of developing diabetes and heart disease. Obesity is epidemic now in Indonesia. Based on RISKESDAS 2007, there were 22.7% obese men aged ≥ 15 years with 27.9% abdominal obesity in both gender. Prevalence of abdominal obesity in Jakarta is higher than the national figure (27.9% vs 18.8%). Abdominal obese individuals have greater risk for having insulin resistance than non-abdominal obese counterparts.

Intake of different types of fatty acid has various influence to insulin resistance. While in Indonesia intake of different types of fatty acid is unfavorable with total fat intake about 29.5%-35.8% of total calories comprising of SFA (20% of total calories), MUFA (2.6%-6.1% of total calories) and PUFA (2.6%-4.6% of total calories).

Limited studies are available describing the association between intakes of various fatty acids and insulin level among abdominal obese adult men. Therefore, this study aimed to determine the association between intake of different fatty acids and insulin level in abdominal obese adult men in Jakarta.

This thesis is divided into six parts which consisted of Introduction (Part 1), Literature review on insulin profiles, fatty acid intakes and influencing factors (Part 2), Methods used in this study (Part 3), Description of results (Part 4), Discussions (Part 5) and Conclusions and recommendations (Part 6). Two manuscripts are prepared for submission to Journal of Nutrition and European Journal of Clinical Nutrition. These draft manuscripts are included in the appendix 1 .

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Jakarta, 06 July 2009

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PUBLICATION APPROVAL FOR ACADEMIC PURPOSES

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ABSTRAK

Nama : Andi Yasmin Syauki
Program studi : Ilmu Gizi
Judul : Hubungan antara asupan berbagai jenis lemak dan insulin pada laki-laki dewasa dengan obesitas sentral di Jakarta

Obesitas merupakan masalah kesehatan yang sudah mendunia, termasuk di Indonesia. Situasi ini erat kaitannya dengan terjadinya perubahan asupan gizi ditandai dengan perubahan pola makan dimana hal ini dapat menyebabkan penyakit degenerasi. Lingkar pinggang merupakan salah satu faktor prediksi yang kuat pada resistensi insulin, yang merupakan fase dini perkembangan penyakit diabetes melitus. Asupan asam lemak jenuh yang tinggi dapat menyebabkan terjadinya resistensi insulin. Data mengenai hubungan antara asam lemak dan resistensi insulin di Indonesia sangat terbatas. Untuk melihat hubungan antara berbagai asupan lemak dengan insulin pada laki-laki dewasa dengan obesitas sentral di Jakarta, maka diadakanlah penelitian dengan metode potongan lintang.

Kuesioner semi kuantitaf-frekuensi makanan yang telah divalidasi digunakan untuk memperoleh data asupan lemak pada 126 laki-laki usia 30-50 tahun dengan obesitas sentral di Jakarta yang sebelumnya telah mengikuti prosedur skrining melalui pemeriksaan klinis dan pengambilan darah. Pengukuran antropometrik dilakukan untuk mendapatkan data berat badan, tinggi badan dan lingkar pinggang. Data plasma insulin puasa, plasma glukosa puasa, plasma asam lemak bebas dan profil lemak darah diperoleh melalui pemeriksaan biokimia. Kuesioner global aktivitas fisik dan surveilens penyakit kronik digunakan untuk memperoleh data aktivitas fisik, kebiasaan merokok, konsumsi alkohol, sayuran dan buah.

Asupan lemak total, lemak jenuh, lemak tidak jenuh tunggal maupun ganda (% dari total kalori) diperoleh lebih tinggi dibandingkan rekomendasi PERKENI/NCEP/AHA/ADA (41.23%, 21.51% and 9.32%), kecuali asupan lemak tidak jenuh ganda berdasarkan PERKENI (6.87%). Asupan omega-3 dan omega-6 tidak memenuhi rekomendasi berdasarkan IOM. Hiperkolesterolemia dan hipertrigliseridemia ditemukan pada penelitian ini. Sementara itu, insulin puasa berada dalam nilai normal (7.63 u/L).

Tidak ditemukan hubungan antara asupan berbagai jenis lemak dengan insulin pada laki-laki dewasa dengan obesitas sentral, tetapi plasma asam lemak bebas memiliki hubungan positif dengan asupan lemak tidak jenuh ganda (% dari total kalori) (r_p=0.190, $p<0.05$), dan plasma glukosa puasa (r=0.193, $p<0.05$). Penelitian kasus-kontrol perlu dilakukan untuk dapat melihat secara jelas hubungan antara asupan berbagai jenis lemak dengan insulin pada seseorang dengan dan tanpa obesitas sentral atau pada seseorang dengan dan tanpa resistensi insulin.

Kata kunci : asupan berbagai jenis lemak, insulin, obesitas sentral

ABSTRACT

Name : Andi Yasmin Syauki
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Title : Relationship between intake of different fatty acids and insulin levels in abdominal obese adult men in Jakarta

Obesity is known as the major global health problems, including in Indonesia. This situation is associated with nutritional transitional characterized by changing in dietary patterns, leading to the prevailing degenerative diseases. Waist circumference is strong predictor of insulin resistance, an initial phase for development of type 2 diabetes melitus. High intake of SFA is contributed to insulin resistance. Data on the relations between intake of fatty acids and insulin resistance in Indonesia are very limited. A cross-sectional study was undertaken to examine the association between intake of different fatty acids and insulin level in abdominal obese adult men in Jakarta.

Dietary fatty acids was obtained through validated fat SQ-FFQ to 126 men with abdominal obesity aged 30-50, who pass the screening procedure through clinical and blood asssessment. Anthropomethric assessments were done to obtain body weight, height and waist circumference. Biochemichal assessments were undertaken to obtain fasting plasma insulin, glucose, FFA and profile lipid. Global Physical Activity Questionnaire and STEPS questionnaire were used to obtain data on physical activity, smoking habit, alcohol use, fruit and vegetable consumption.

Intake of total fat, SFA, MUFA and PUFA (% of total calories) were found higher than that of the PERKENI/NCEP/AHA/ADA recommendations (41.23% , 21.51% and 9.32%), except PUFA intake based on PERKENI (6.87%). Intake of omega-3 and omega-6 PUFA did not meet the requirement suggested by IOM. Hypercholesterolemia and hypertrigliseridemia were found among subjects. Mean fasting plasma insulin was found within desirable range (7.63 u/L).

There is no correlation between intakes of different fatty acids and insulin levels in abdominal obese adult men, but FFA plasma were positively correlated with PUFA intake (% of total calories) (r_p=0.190, $p<0.05$) and fasting plasma glucose (r_p=0.193, $p<0.05$). Further study need to be conducted to have clearly understanding of the relationship between intake of different fatty acids and insulin level between abdominal obese and non-abdominal obese or insulin resistance and non insulin resistance using case-control study.

Keywords : fatty acids intake, insulin, abdominal obese

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

AA	Arachidonic Acid
ADA	American Diabetes Association
ADMA	Asymetric Dimethyl Arginine
AFABP	Adipocyte Fatty Acid binding Protein
AHA	American Heart Association
ALA	Alpha-Linolenic Acid
ANP	Atrial Natriuretic Peptide
ARIC	Atherosclerosis Risk in Communities
BMI	Body Mass Index
BP	Blood pressure
CE	Cholesterol Ester
CHO	Carbohydrate
CHD	Coronary Heart Disease
CoA	Coenzym A
CVD	Cardio Vascular Disease
DHA	Docosahexaenoic Acid
DM	Diabetes Mellitus
EI/BMR	Energy Intake/Basal Metabolic Rate
EPA	Eicosapentaenoic Acid
FA	Fatty Acid
FaHM	Fact Hypotheses Matrix
FFA	Free Fatty Acid
FFAs	Free Fatty Acids
FFQ	Food Frequency Questionnaire
FGD	Focus Group Discussion
FPG	Fasting Plasma Glucose
fsIVGTT	frequent sampling intravenous glucose tolerance
GPAQ	Global Physical Activity Questionnaire
HDL	High Density Lipoprotein
HDLC	High Density Lipoprotein Cholesterol
HOMA-IR	Homeostasis Model Assessment-Insulin Resistance
HUFA	High Unsaturated Fatty Acid
IDF	International Diabetes Federation
IDR	Indonesian Dollar Rupiah
IFIC	International Food Information Council
IMGU	Insulin Mediated Glucose Uptake
IOM	The Institute of Medicine

IR	Insulin Resistance
IRAS	Insulin Resistance Atherosclerosis Study
JNC-VII	Joint National Conference-VII
LA	Linoleic Acid
LDL	Low Density Lipoprotein
LDLC	Low Density Lipoprotein Cholesterol
MET	Metabolic Equivalent
MUFA	Monounsaturated Fatty Acid
NA	Not Available
NCEP	National Cholesterol Education Panel
NIDDM	Non-Insulin Dependent Diabetes Mellitus
NIH	National Institutes of Health
n-3 PUFA	Omega-3 Polyunsaturated Fatty Acid
n-6 PUFA	Omega-6 Polyunsaturated Fatty Acid
PERKENI	*Perkumpulan Endokrinologi Indonesia* (Indonesian Endocrinology Society)
PL	Phospholipids
P(M)S	Poly-monounsaturated saturated
P/S	Polyunsaturated/Saturated
PUFA	Polyunsaturated Fatty Acid
RBP4	Retinol binding Protein 4
RISKESDAS	*Riset Kesehatan Dasar* (Baseline Health Research)
REE	Resting Energy Expenditure
SEAMEO	South East Asian Ministers of Education Organization
SFA	Saturated Fatty Acid
SGPT	Serum Glutamic Piruvat Transaminase
SPSS	Stastical Package for Social Science
STEPS	STEPwise apprach to surveillance
SQ-FFQ	Semi Quantitative-Food Frequency Questionnaire
TG	Triglyceride
TNF-α	Tumor Necrosis Factor-alpha
T2DM	Type 2 Diabetes Melitus
U	Unit
US	United States
USA	United States of America
VF	Visceral Fat
VLDL	Very Low Density Lipoprotein
VIM	Variable Indicator Matrix
WC	Waist Circumference
WFR	Weighed Food Record

WHO	World Health Organization
WHR	Waist Hip Ratio
Y/N	Yes/No

OPERATIONAL DEFINITIONS

ADULT MEN :

Men aged 30-50 years old

ABDOMINAL OBESE MAN :

Is defined as respondents who had waist circumference ≥ 90 cm (South Asian cutt-off for male)

OBESITY :

Obesity corresponds to body mass index (BMI). For classification, used both WHO and Asian Adult Population classification.

Table A. WHO Classification of Overweight in Adult according to BMI

Classification	BMI (kg/m^2)	Risk of co-morbidities
Overweight	≥ 25	
Pre-obese	25.00-29.99	Increased
Obese class I	30.00-34.99	Moderate
Obese class II	35.00-39.99	Severe
Obese class III	≥ 40	Very severe

Source : WHO Expert Consultation, 2004

Table B. Proposed Classification of Overweight by BMI in Adult Asians Population

Classification	BMI (kg/m^2)	Risk of co-morbidities
Overweight	23.00-27.49	Increased risk
Obese I	≥ 27.50	High risk

Source : WHO Expert Consultation, 2004

INTAKE OF DIFFERENT FATTY ACIDS :

Intake of total fat, SFA, MUFA, PUFA , omega 3 and omega 6 in within one month using validated fat SQ-FFQ and used classification based on recommendation from PERKENI, NCEP, AHA and ADA.

Table C. Favorable Fatty Acids based on PERKENI, NCEP, AHA, ADA Recommendation

Dietary intake (% of total E)	PERKENI, 2006	NCEP, 2001	AHA, 2006^	ADA, 2005^
Total fat	20-25	≤ 25-35	25-35	≤ 25-35
SFA	< 7	< 7	< 7	< 7
MUFA	≥ 10	> 20	NA	> 20
PUFA	< 10	> 10	NA	> 10
n-6	NA	NA	NA	NA
n-3	NA	NA	NA	NA

LIPID PROFILES :

Is defined as plasma total cholesterol, LDL cholesterol, HDL cholesterol, triglyceride and FFA plasma.

HYPERCHOLESTEROLEMIA :

Is measured by the classification of cholesterol from NCEP (National Cholesterol Education Program), 2001 :

Table D. Classification of Total, LDL, HDL Cholesterol and Tryiglyceride

Classification	Cholesterol total (mg/dl)	LDL-cholesterol (mg/dl	HDL-cholesterol (mg/dl)	Triglyceride (mg/dl)
• Low	-	-	undesirable< 40	-
• Optimal /desirable	< 200	< 100	-	< 150
• Near optimal/above optimal	-	100-129	-	-
• Borderline high	200-239	130-159	-	150-199
• High	≥ 240	160-189	(desirable) ≥ 60	200-499
• Very high	-	≥ 190	-	≥ 500

HYPERTENSION

Is defined as respondents with hypertension on systolic and diastolic blood pressure. It is measured by the classification from JNC VII, systolic blood pressure ≥ 140 mmHg and diastolic blood pressure ≥ 90 mmHg

HYPOLIPIDEMIA MEDICINE :

Medicine used for reducing cholesterol and triglycerides, such as simvastatin and fibrat at least one month before blood collection.

SMOKING HABIT :

Is measured using STEPS instruments developed by WHO. Respondents who smoke tobacco products such as cigarrete, cigars or pipes daily currently.

ALCOHOL USE :

Is measured by STEPS instruments developed by WHO. Respondents who drinks alcohol products (beer, wine, fermented drinks) within the past 12 months.

FRUIT CONSUMPTION :

Is measured by STEPS instruments developed by WHO. Respondents who usually consume fruit in days per week within the past 12 months.

VEGETABLE CONSUMPTION :

Is measured using STEPS instruments developed by WHO. Respondents who usually consume vegetable in days per week within the past 12 months.

PHYSICAL ACTIVITY :

Is measured by The Global Physical Activity Questionnaire developed by WHO. It collect information on physical activity participation in three settings (or domains) as well as sedentary behaviour, i.e activity at work, travel to and from places, and recreational activities. Total physical activity as MET-minutes/week and categorized based on physical activity cutoff value as high, moderate, and low. (Detailed description on methods).

PART 1
INTRODUCTION

1.1 Background

There is 230 million people world wide diagnosed as diabetes melitus which is one of the most common chronic disease worldwide and the fourth or the fifth leading cause of death in developed world [1]. Based on RISKESDAS 2007, diabetes melitus is the second major cause of death for age ≥ 5 year in Indonesia [2]. The main cause of death of diabetic patient is coronary heart disease (CHD)[3]. Diabetes Melitus cases mostly is type 2 diabetes melitus (T2DM), which is generally have insulin resistance [4].

Insulin resistance is a state in which a given concentration of insulin produces less than normal biologic response from fat, muscle and liver cells[5]. Abnormalities of insulin action can trigger a number of important clinical and pathophysiologic states because dysfunction of insulin to promote overall glucose metabolism[5]. There is consideration from case-control studies that insulin resistance is a common finding in several metabolic disorders, such as glucose intolerance, dyslipidemia, hyperuricemia and hypertension[6]. Insulin resistance is antedates to the development of diabetes and it is also a major metabolic defect that can increase the chances of developing heart disease [7,8].

The cause of the vast majority of insulin resistance cases remains unknown. Insulin resistance is a heterogenous syndrome which is genetic and environment factors play an important role for development of this syndrome. Overweight, physically inactive and intake of excessive amount of fat and high-glycemic-index carbohydrate are modifiable (nutritional) determinants of insulin resistance and hyperinsulinemia [9]. Several studies suggest that obese subjects with a predominatly central (abdominal/truncal) distribution of body fat are more insulin resistant and have higher circulating insulin levels (hyperinsulinemia) than those with peripheral obesity [5]. Excess of visceral adipose tissue accumulation in

the presence or absence of obesity, is associated with insulin resistance, hyperinsulinemia, glucose intolerance [10]. Women and men are different in their distribution of fat, which is men are likely to have abdominal fat [11]. This pattern are associated with higher risk of diabetes mellitus, cardiovascular disease and hypertension [12].

The main driving forces for the increased prevalence of insulin resistance are modern Westernized diets and patterns of eating associated with the dramatic rises in obesity. Insulin resistance is often linked to the macronutrient content in the diet [13]. Modern diet is usually high in fat, especially saturated fatty acids (SFA) and processed meats, which is promoted to excessive energy intake. The accumulation of increased body fat stores and as body fat stores expand beyond a certain point, which varies between people and with age, it leads to the development of insulin resistance [14]. It is shown in a large prospective study of men with 12 years of follow-up, the dietary intake of saturated fat and total fat were related to the risk of developing diabetes primarily through its association with greater body mass index (BMI) [15]. Epidemiologic studies also demonstrated that high intake of total fat (>40% of total calories) is associated with insulin resistance [7]. Clinical studies have paralel finding that there were significant correlations between plasma insulin and glucose and total dietary fat in grams, dietary fat, saturated and monounsaturated fatty acid and as a percentage of total calories and dietary cholesterol. It is also showed that saturated fat and the percentage of body fat had the strongest correlations with fasting insulin [15]. Other clinical trials showed there was positive effect on long-chain omega 3 PUFA consumption to insulin resistance in young overweight individuals [16].

WHO predicts there is an increasing of patient T2DM from 8.4 million in 2000 to 21.3 million in 2030 in Indonesia. The prevalence of DM in Jakarta had risen from 1.7% in 1982 to 5.7% in 1993 (urban area) then in 2001 become 12.8% (sub-urban area). According to Bureau Statistics of Indonesia with growth population prediction, there will be 12 million person with T2DM age above 20

years in urban area (prevalence 14.7%) and 8.1 million in sub-urban area (prevalence 7.2%) [17] in 2030. Derived from RISKESDAS 2007, prevalence of diabetes melitus is 5.7% in urbanized city in Indonesia [2].

Nowadays, there are one billion overweight adults around the world and at least 300 million of them obese. This situation usually happen in developing countries with under nutrition problems [18]. Based on RISKESDAS 2007, there are 22.7% obese men aged ≥ 15 years with 27.9% have abdominal obesity in both sexes. Prevalence of abdominal obesity in Jakarta is higher than national figure (27.9% vs 18.8%). Prevalence of abdominal obesity starts to increase in the 35-44 year age group (24.4%) and was the highest is in 45-54 year age group (26.1%) [2].

Recently, there are changing of lifestyle such as westernization food, for example, the growing of fast food restaurant and the increasing of economic life for medium until high economic class, which influence the increasing of the prevalence of obesity in Indonesia [19]. One of the dominant cooking methods in Indonesian food culture is frying and this may compromise the cardio-protective effect of foods [20]. Study of nutrient intake and their relations to lipid profiles in four ethnic in Indonesia showed that the mean of the contribution of total fat to calories ranged from 29.5% to 35.8%. The percentage of SFA to total calories also high around 20%. Meanwhile, the percentage of PUFA to total energy ranged from 2.6% to 4.6%. The percentage of MUFA to total calories among four ethnic groups were still inadequate based on the recommended value (2.6%-6.1%). The P/S ratio also showed lower value (0.15-0.25). The dietary pattern of all these four ethnic groups might be associated with having the risk of dyslipidemia or hypercholesterolemia [21].

Biomarkers and biochemichal of fatty acid intake would give an objective alternative to dietary assessment[22]. In order to obtain valid data of biomarkers and biochemichal of fatty acids, there were a criteria for respondents such as there is no liver function disorder and kidney function disorder for controlling lipid metabolism among abdominal obese person.

The increasing prevalence of diabetes mellitus and obesity in Indonesia also followed by unfavorable intake of different fatty acids prevailing to insulin resistance. Still in hypothesis, whether insulin resistance leading to lipolysis or lypogenesis. This study was a part of a bigger study among abdominal obese adult men, in particular this study want to see dietary fat intake and the association. In order to obtain data about associaton between various intake of different fatty acids with the high prevalence of T2DM and obesity in Indonesia, we are conduct a cross sectional study to examine the relationship between intake of different fatty acids and insulin level in abdominal obese adult men in Jakarta as capital city of Indonesia.

1.2 Problem Statement and Rationale of the Study

1.2.1 Problem Statements

a. With increasing prevalence of diabetes mellitus in Indonesia, the importance of early detection of insulin resistance is emphasized. Insulin resistance is a silent condition that increases the chances of developing diabetes and heart disease.

b. Obesity is epidemic now in Indonesia. Based on RISKESDAS 2007, there is 22.7% obese men aged ≥ 15 years with 27.9% have abdominal obesity in both sexes. Prevalence of abdominal obesity in Jakarta is higher than national figure (27.9% vs 18.8%)

c. Abdominal obese person has greater risk for having insulin resistance than non-abdominal counterparts .

d. Intake of different type fatty acid give various influence to insulin resistance. While in Indonesia intake of different type of fatty acid is unfavorable i.e total fat intake (29.5%-35.8% of total energy), SFA (20% of total calories), MUFA (2.6%-6.1% of total calories) and PUFA (2.6%-4.6% of total calories).

e. In Indonesia, the association between intakes of various of fatty acids and insulin level among abdominal obese adult men are still unclear.

1.2.2 Rationale of the Study

a. Until now, the existence of the background dietary intake in a different of fatty acids as far as its association with insulin level among Indonesian people especially in abdominal obese adult men are still indefinite.

b. Insulin resistance in abdominal obese is influenced by their fat intake profile. However, it is only described in Western population and in Western diet. Therefore, the study should be conducted to provide information Asian population and Asian diet.

c. Nutritional counselling can be given to abdominal obese adult men group in the hospital to reduce the risk from abnormal insulin levels that they might have.

1.3 General and Specific Objectives

1.3.1 General Objective

To determine the association between intake of different fatty acids and insulin level in abdominal obese adult men in Jakarta

1.3.2 Specific Objectives

To assess, among abdominal obese adult men :

- The intake patterns of different fatty acids such as total fat, PUFA, MUFA, SFA and lipid profiles
- Fasting plasma insulin and fasting plasma glucose profiles
- Other potential determinants of insulin level, including physical activity, smoking habit, alcohol use, fruit and vegetable consumption and fiber intake
- Association between intake of total fat, SFA, PUFA, MUFA with insulin level

1.4 Research Question

Is there any association between intakes of different fatty acids and insulin level in abdominal obese adult men in Jakarta?

1.5 Hypotheses of the Study

a. Higher total fat intake is associated with increased fasting plasma insulin in abdominal obese adult men

b. Higher SFA intake is associated with increased fasting plasma insulin in abdominal obese adult men

c. Higher MUFA intake is associated with decreased fasting plasma insulin in abdominal obese adult men

d. Higher PUFA intake is associated with decreased fasting plasma insulin in abdominal obese adult men

e. Higher ratio P(M)S is associated with decreased fasting plasma insulin in abdominal obese adult men

f. Higher ratio of (n-6) and (n-3) PUFA is associated with increased fasting plasma insulin in abdominal obese adult men

1.6 Conceptual Framework

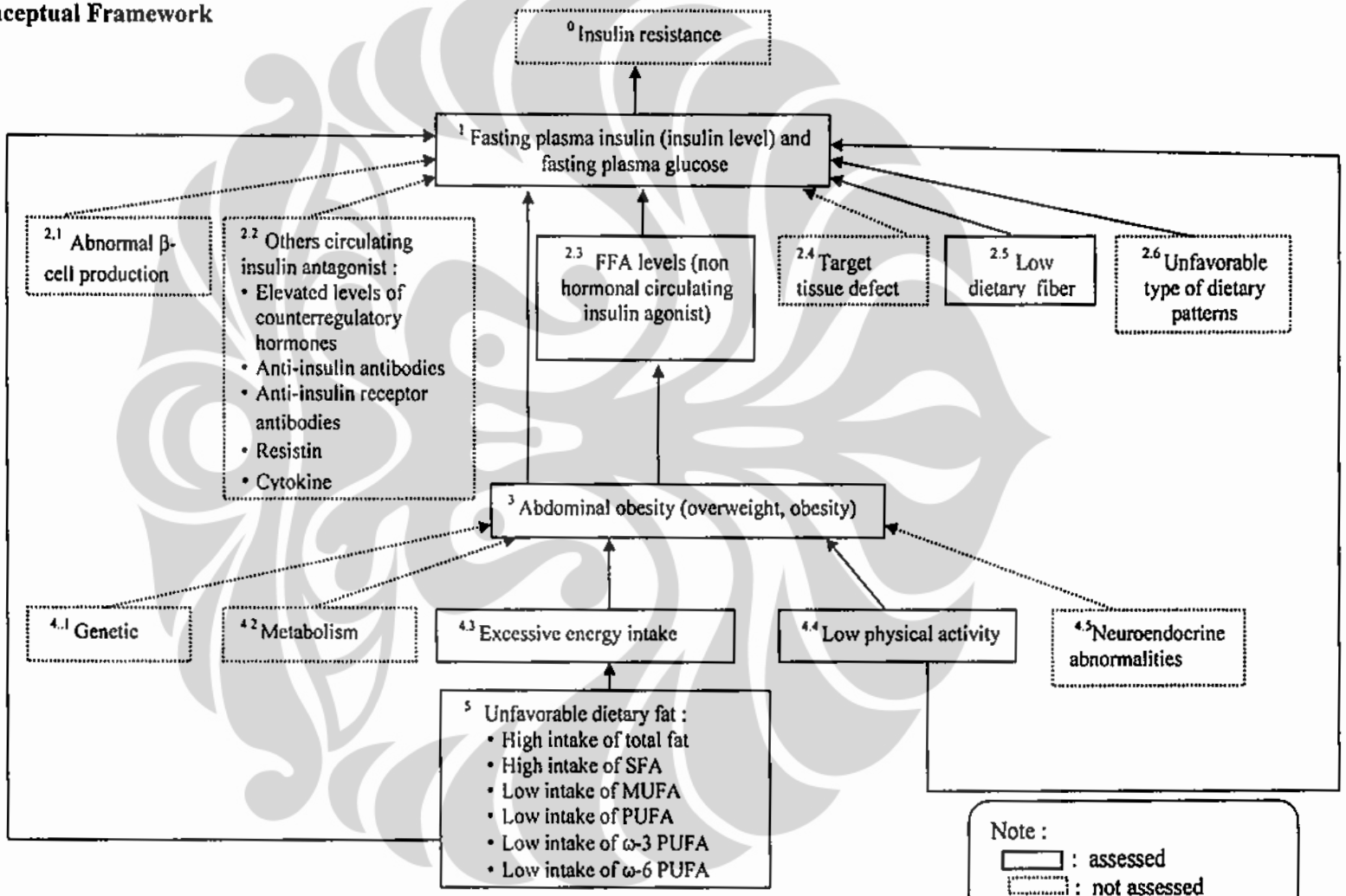

Figure 1. Conceptual Framework

1.7 Fact and Hypotheses Matrix

Table 1 presents the list of fact and hypotheses matrix of this study

Table 1. Fact and Hypotheses Matrix (FaHM)

Variable 1	Variable 2	References
Insulin resistance	HOMA IR > 2	Bonora et al, 1998
Fasting plasma insulin (insulin level)	Abnormal β-cell secretory product	Olefsky et al, 2003
	Circulating insulin antagonists : • Hormonal (glucocorticoids, GH, glucagon, cathecolamine, placental lactogen) • Non-hormonal (elevated FFA, anti-insulin antibodies, anti-insulin receptor antibodies, resistin, cytokines)	McAuley, 2006
	Target tissue defects	
	Excess adiposity	
	Unfavorable dietary fat	
	Decline of physical activity	
	Low dietary fiber	
	Unfavorable dietary pattern	
FFA levels (non-hormonal circulating insulin antagonists)	Abdominal obesity (overweight, obesity)	Tataranni et al, 2003
Abdominal obesity (overweight, obesity)	Genetic	Ard, 2006
	Metabolism	Saltzman, 2006
	Excessive energy intake	
	Low physical activity	
	Neuroendocrine abnormalities	
Excessive energy intake	Unfavorable dietary fat : • High intake of total fat • High intake of SFA • Low intake of MUFA • Low intake of PUFA	Anderson, 1999 Kenney, 2003 McAuley, 2006 Rizerus, 2006 Shikany, 2006 Lean et al, 2000

PART 2
LITERATURE REVIEW

2.1 Diabetes Melitus

Prevalence of diabetes melitus globally is predicted to more than double between 2000 and 2030, from 171 million to 366 million. This condition is influenced by industrialization, a concomitant rise in obesity and sedentary lifestyles in developing countries [23]. Diabetes melitus is a syndrome of disordered metabolism, usually due to a combination of hereditary and environmental causes, resulting in abnormally high blood sugar levels (hyperglicemia). Glucose and insulin had been suggested play important role in the pathophysiology of diabetes disease [24]. There are four classification of DM such as type 1 (insulin-dependent or juvenile DM), type 2 (non-insulin-dependent DM or adult-onset diabetes), gestational DM (hyperglycemia identified during pregnancy), and secondary diabetes (due to pancreatic damage or insulin resistance caused by other diseases or treatments). Type 2 DM accounts for 90% to 95% of all cases of diabetes. This type is characterized by insulin resistance and inadequate in compensatory insulin secretion [3,23].

2.2 Insulin, Insulin Resistance and Influencing Factors

Insulin is an anabolic essential hormone produced in the pancreatic β-cell in the pancreas which is primarily responsible for the metabolism and storage of ingested body fuels [5,24,25]. Insulin is released after a person with normal metabolism had meal ("postprandial") as a response to increase blood glucose after a meal (extrinsic rhythm) [24]. In contrast with normal metabolic people, an insulin-resistant person has different level of insulin on muscle and adipose cells with the result that glucose level stay higher than normal (hyperglycemia). To compensate it, the pancreas in an insulin-resistant individual is stimulated to release more insulin causing the elevated insulin levels in the body (hyperinsulinemia) and blood glucose still at normal level or slightly increases. At

the end, there is decompensation of β- cell pancreas to produce insulin and then clinically diabetes melitus will be present, characterized by the increasing of blood glucose [1]. At study of Genuth, there is a difference on insulin secretion rates between non-diabetic and diabetic subjects. In a healthy lean adults, insulin is secreted approximately 31 U daily, while in obese non-diabetic adults it is secreted about 114 U daily because of peripheral insulin resistance. In lean type 2 diabetic individuals with diabetes for more than 10 years, insulin is produced only about 14 U daily [25].

Insulin resistance is condition in which a given concentration of insulin produces less than normal biologic responses [5]. Insulin resistance was first described in the 1970s and in 1988 Reaven suggested that it was the underlying cause of a syndrome characterized by hyperinsulinemia, hypertension, increased triglyceride, reduced HDL cholesterol, hyperglycemia and an increased risk of coronary heart disease [26]. Insulin resistance is a silent condition that increases the chances of developing diabetes and heart disease [8].

2.2.1 Cellular Determinants of Insulin Resistance

The underlying causes of insulin resistance still remain unclear. Based on the circulation of insulin from β-cell to the target tissue, insulin resistance according to the known etiologic mechanism can be due : [5]

2.2.1.1 Abnormal β-cell Secretory Product

Abnormal β-cell secretory product can be caused by abnormal insulin molecule and incomplete conversion of proinsulin to insulin.This condition was found in several patients who secreted a structurally abnormal, biologically defective insulin molecule as a result of a mutation in the structural gene for insulin. Although genetic mutations account for a minor role in the large part of insulin resistance, an alteration of insulin transduction, which may be due to genetic mutations, could contribute to the impairment of insulin secretory profile and insulin resistance. Others were described with familial hyperinsulinemia,

caused by incomplete conversion of proinsulin to insulin within the β-cell secretory granule as a result of structural abnormalities at the proteolytic cleavage sites of the proinsulin molecule. These conditions make the patient only resistant to their endogenous insulin and not to exogenous insulin [5,24].

2.2.1.2 Circulating Insulin Antagonist

Circulating insulin antagonist may be hormonal or non-hormonal.

(a) hormonal

Insulin is one of principal hormonal messenger on energy reservoir in humans. Multiple hormones counter the effects of insulin. Elevated level of counterregulatory hormones such as cortisol, growth hormone, glucagon, and cathecolamine can act as insulin antagonist. Excessive level of cortisol or glucocorticoids impair carbohydrate metabolism, for example, increase the activity of key hepatic gluconeogenesis enzymes and the release of gluconeogenic substrates (amino acids, lactate, and glycerol) from peripheral tissues. This can cause increasing of hepatic glucose production. Additionally, some glucocorticoids can initiate a decrease in insulin binding to receptor, both in vivo and in vitro, which is mediated through a decrease in both receptor affinity and number. In chronic excessive of growth hormone, it can lead to carbohydrate intolerance and most studies have shown that growth hormone leads to an impairment of stimulated glucose uptake and may also impair insulin's ability to suppress hepatic glucose output [5,27].

Excessive level of cathecolamine can stimulate glucagon secretion (β2-effect) and increase hepatic glucose production by direct stimulation of glycogenolysis and gluconeogenesis. In combination, these effects tend to cause hyperglycemia and are opposite to the actions of insulin. Additionally, cathecolamines directly inhibit peripheral glucose uptake. Glucagon influences glucose metabolism by augmenting hepatic glycogenolysis and gluconeogenesis. Glucagon can counteract some of insulin's effects but has no influence on

insulin's ability to promote peripheral glucose metabolism and does not lead to a true state of insulin resistance. Cushing's syndrome, pheocromacytoma, glucagonoma, acromegaly is well-known clinical syndrome in which elevated levels of these hormones can induce an insulin-resistant. However, in the usual case of obesity or T2DM, excessive level of counterregulatory hormones is not an important contributory factor to insulin resistance [5].

In general, the counterinsulin hormones (also called counterregulatory hormones) liberate the energy from fuel reservoirs by action opposite to those of insulin [27].

(b). Non-hormonal

Elevated free fatty acids (FFA), anti-insulin antibodies, anti-insulin receptor antibodies, resistin and cytokines will affect insulin activity. The excessive fat intake causes an increased influx of triglycerides into the blood and an excess of plasma levels of FFAs, which induces insulin resistance, with consequent hyperglycemia. The increased levels of glucose stimulate pancreatic β cells to secrete more and more insulin, generating hyperinsulinemia, which further triggers the elevation of triglycerides and close to vicious circle. FFAs are also involved in modulating insulin production by pancreatic β cells and cytokine secretion by hepatocytes, adipocytes, muscle cells and inflamatory cells [5,24].

Insulin resistance was initially recognized as an "allergy" to insulin, with the production of antibodies anti-insulin. This condition was found in all patients who received animal-derived insulin for a long enough period of time. Although anti-insulin antibodies do not usually lead to a clinically significant insulin-resistant state, the presence of these antibodies alters the pharmacokinetics of insulin. High titers of high-affinity antibodies can act as a reservoir for insulin by binding the hormone when it initially enters the circulation and later releasing it. This condition will increase the half-life of circulating insulin and prolongs the time course of insulin action. Circulating endogeneous immunoglobulins directed

against the insulin receptor have been described in insulin-resistant diabetic patients. Patients will be discovered with antibodies against other critical proteins scheme, such as glucose transporter [5,24].

Resistin is a new adipocyte hormone that play important role as a link between increased fat mass and insulin resistance. Resistin decreases insulin-dependent glucose transport in vitro and increases fasting blood glucose concentrations and hepatic glucose production in vivo [24].

2.2.1.3 Target Tissue Defect

Defect of insulin action can be in the insulin receptor or post-receptor. Decreased cellular insulin receptors can be found in a variety of pathophysiologic situations, commonly in obesity and T2DM. The first step in insulin action is binding to the receptor, it is apparent that a decrease in cellular insulin receptors could lead to insulin resistance. Other insulin receptor defect is the decreasing of insulin receptor's sensitivity. Post-receptor defect can caused by abnormality of phosphorylation and transduction signal in muscle cell especially in the skeletal and liver. This condition initiate compensation such as increasing levels of insulin secretion resulting hyperinsulinemia in the fasting state also in postprandial state [4,5].

2.2.2 Nutritional Determinants of Insulin Resistance

Insulin resistance and its associated metabolic abnormalities are considered to be important determinants of coronary heart disease. The nutritional determinants of insulin resistance can be translated into non-pharmacological interventions that aimed at contributing to coronary heart disease risk reduction. There are several factors related to lifestyle regarded as a surrogate measure of insulin resistance such as excess adiposity, inadequate physical activity, unfavorable dietary fat, low dietary fiber, unfavorable dietary patterns [26].

2.2.2.1 Excess Adiposity

The secretion of insulin by the pancreas is directly proportional to adiposity. Chronic hyperinsulinemia is responsible for increased hunger, elevated food intake and weight gain in obese individuals. Although hyperinsulinemia- a compensatory response to peripheral insulin resistance- is one of the most prevalent hormonal abnormalities associated with obesity, it is not known whether changes in insulin release are involved in the etiology of obesity. Most data indicate that hyperinsulinemia and insulin resistance are likely to be secondary to established obesity. Other data, however, suggest that obesity and insulin resistance may result from independent mechanism [28].

The importance of regional adiposity in determining insulin action has been widely assumed but the findings have not been entirely consistent. BMI and WC are highly correlated as a predictor of insulin-mediated glucose uptake (IMGU). Meanwhile, not all overweight or obese person are insulin resistant. On a priori basis that WC is a superior predictor of the adverse effects of excess adiposity. This is also supported by result from several studies indicated that the correlation coefficients between visceral fat (VF) and IMGU are usually < 0.6 values that are no greater than the relation between IMGU and either BMI or WC (r=0.9). Based on cross sectional and longitudinal epidemiological studies in which waist circumference has been used to assess abdominal obesity and surrogate measures (e.g. fasting insulin) to determine insulin sensitivity showed that abdominal or truncal adiposity has been implicated as having a key role in the pathogenesis of insulin resistance, which is in obese subjects with a predominantly central (abdominal/truncal) distribution of body fat are more insulin resistant and have higher circulating insulin levels than those with peripheral obesity [26,29].

2.2.2.2 Inadequate of Physical Activity

A higher lean body mass is associated with enhanced glucose disposal. Physical activity contributes to increased lean body mass, enhanced insulin sensitivity and favorably influences many of the other metabolic abnormalities associated with obesity and the metabolic syndrome. Several mechanism have been proposed to explain how exercise enhances insulin action. These mechanism can be classified as those that exert their effect on muscle indirectly through the hormone's action at another side and those that act directly at the muscle. The exercise induces increase in muscle blood flow and will be augmented the effect of insulin on glucose uptake by increasing insulin transport across the endothelium. Exercise may also increase insulin-stimulated glucose utilization by a mechanism secondary to insulin's suppresive effect on nonesterified FFA availability. The absolute magnitude of the insulin-induced suppression of plasma FFA levels and fat oxidation mechanism by which exercise increases insulin action [26,30].

2.2.2.3 Unfavorable Dietary Fat

Diets high in total fat are energy dense, which is may be less satiating than carbohydrates as least in some individuals. As a result, they tend to promote excess energy intake and are associated with an increased risk of obesity and insulin resistance. High fat diets may promote insulin resistance through their obesogenic potential, for example, the contribution of fat to total calories, the nature of dietary fat influence insulin resistance and other features of the metabolic abnormalities in the context of energy balance. Current dietary recommendations focus not only on the total fat intake but on the types of fat consumed, especially of the importance on differing roles of specific fatty acids in health and disease [26,31].

Diets high in SFA consistently impair insulin sensitivity and blood lipids. Garg's meta analysis of 10 randomized crossover trials comparing isoenergetic

high-MUFA and high-CHO diets in patients with type 2 diabetes concluded that consumption of high-MUFA diets improved fasting and postprandial blood glucose and 24-h glucose and insulin profiles while have no effect on fasting insulin and insulin sensitivity. In the study of Luscombe et al (1999), 2 low-fat diets (21% and 23% energy from fat) and high-MUFA diet (35% energy from fat) provided similiar glycemic control, as assessed by fasting glucose and insulin levels (Ros, 2003, p. 619S).. This finding also similiar with the result of Vessby et al (2001) in a parallel-arm feeding trial in 162 healthy subjects who were given diets with 37% energy from fat, either a high-SFA diet (17% SFA, 14%MUFA) or a high-MUFA diet (8% SFA, 23% MUFA) (Ros, 2003, p.619S). Insulin sensitivity was impaired on the SFA diet but did not change on the MUFA diet, except for subjects whose relative intake of total fat was above the median of 37% energy [32].

Eleven studies compared the effects on insulin sensitivity of isoenergetic diets high (typically 40-50% total energy) and low (typically 15-25% total energy) in total fat intake. The comparisons were made under isoenergetic conditions with the bulk of the remaining energy provided by varying carbohydrate, protein remaining fairly constant, and insulin sensitivity assessed by means of a clamp or frequent sampling intravenous glucose tolerance (fsIVGTT). In six of the eleven studies, there were no significant differences in insulin sensitivity on the high and low fat diets. Four studies suggested reduced sensitivity on the high fat diets but in these the percentage of energy from fat was extremely higher as well as intake of saturated fat. In one study, insulin sensitivity appeared to be slightly enhanced on the high fat diet compared with the high carbohydrate diet but the study was carried out in people with diabetes in whom the sudden substantial increase in carbohydrate may have resulted in a deterioration in glycemic control. These finding still need support from other studies [26].

2.2.2.4 Low Dietary Fiber

Diets rich in low energy-dense foods, including wholegrain cereals and cereal products and other foods rich in dietary fiber promote satiety and may, as a consequence, facilitate appropriate energy intake which is can reduce the risk of obesity as well as reducing the risk of insulin resistance. In the context of energy balance and weight maintenance there is evidence that type of carbohydrate may influence insulin sensitivity and associated metabolic features [26].

Most carbohydrate containing, fiber rich foods also have a low glycemic index. Two large cross sectional studies using validated food frequency questionnaires to assess nutrient intakes and either the frequently sampled intravenous glucose tolerance test or homeostasis model assessment for insulin resistance found that intake of dietary fiber was inversely associated with the probability of having insulin resistance. In the Insulin Resistance Atherosclerosis Study (IRAS), it is demonstrated that fiber increased insulin sensitivity even after adjustment for body mass index (BMI). However neither study found any relationship between insulin sensitivity and glycemic index or glycemic load. [26]

2.2.2.5 Unfavorable Type of Dietary Patterns

Dietary factors have affected the evolutionary development of human energy needs and what these imply in the terms of the types of food which, ideally, we should eat. Particular interest has been taken in the idea that, as Homo sapiens evolved in Africa, early humans moved from a vegetarian existence, seen in other primates, to an omnivorus lifestyle characterized by the need for the hunting of animals as well as the gathering of fruits and roots from the jungle [33].

Mediterranean diet has the potential to protect against insulin resistance, type 2 diabetes and coronary heart disease. An Italian study (Esposito et al, 2004) had reported on a randomized trial in which patients with the metabolic syndrome received advice either regarding a Mediterranean dietary pattern or a 'prudent' diet, relatively low in fat and high in fiber rich carbohydrate. Those randomized to

the Mediterranean diet were advised to increase their daily consumption of wholegrains, fruits, vegetables, nuts and olive oil, while those given advice regarding the 'prudent' diet received information about healthy food choices. From eight cross-sectional and six cohort studies, there are two studies have shown a negative association between BMI and the Mediterranean diet. Meanwhile, three cohort studies reported a positive association between BMI and MUFA or Mediterranean diet and SFA. Another cohort study has shown a negative relationship between BMI and the Mediterranean diet. The Mediterranean diet was associated with lower levels of high-sensitivity C-reactive protein and interleukins 6, 7 and 18, reduced insulin resistance and improved endothelial function score compared with the 'control' prudent diet group amongst whom these measures showed little change. After two years, 40 of the 90 subjects in the intervention group still had features of the metabolic syndrome as compared with 78 of the 90 patients in the control group. These findings do indeed suggest that the Mediterranean style dietary pattern reduces insulin resistance and associated abnormalities. However because the Mediterranean diet was more vigorously implemented and weight loss was greater, the findings do not provide evidence for superiority over the so-called prudent diet [26,34].

A new range of dietary patterns has now emerged and several are being widely recommended and adapted for weight loss and cardiovascular risk reduction. Amongst the best known of these are the high fat-low carbohydrate diets (e.g. Atkins) and the high protein dietary approach (e.g. Zone, South Beach diet). There is no doubt those alternative dietary approaches can facilitate weight loss, lower insulin levels and improve many of the metabolic derangements associated with insulin resistance in the short term. However, long term data are limited. Two 12-month randomized trials by Stern et al (2004) and Foster et al (2003) suggest that any initial benefit achieved by the high fat Atkins approach compared with the high carbohydrate-high fiber-low fat approach (the 'prudent' diet) is lost after one year. Studies by McAuley et al (2005, 2006) comparing the high fat, high protein, and high carbohydrate-high fiber approaches in insulin

resistant women provided that the early benefits of the high fat diet compared with the high carbohydrate diet in terms of weight loss, fasting insulin and triglyceride are not significant after one year [26].

2.3 Overweight, Obesity, Abdominal Obesity and Factors Affecting

Throughout evolution, animals and humans have developed redundant mechanisms promoting accumulation of fat tissue during periods of abundance to survive periods of famine. However, obesity has reached epidemic proportions in both industrialized countries and in urbanized populations around the world after the liability of obesogenic environment, such as readily available high-fat foods and reduced physical activity [35].

Obesity is a complex phenotype, resulting from the direct effects and interactions of genetic factors, the environment, and behavior. It has been clearly established that genetic factors contribute to one's predisposition to or protection from obesity. Current estimates are that 25% to 40% of variation in BMI is likely due to genetically determined factors. These factors include determinations of resting metabolic rate, where fat is stored (visceral vs. extremities), physiologic response to overfeeding and perhaps to some extent eating behaviors [12]. Genetics contribute significantly to obesity which is in the population studies using a variety of family data designs have found that the heritability of body mass or body fat, that is, the proportion that can be explained by genetic transmission, is in the range of 24-70%. It is hypothesized that this effect is polygenic (caused by several genes). The phenotypical expression of these genes may differ depending on a person's age and sex. In addition, the genes may only increase susceptibility to obesity through obesity-encouraging environment, which is includes an abundant food supply and minimal physical activity requirements, may also be necessary for their expression [11]. Neuroendocrine abnormalities cause obesity in fewer than 1% cases, such as hypothyroidism, Cushing's syndrome and polycystic ovary syndrome, but these abnormalities are rarely cause severe degrees of

obesity. Thus, markedly obese person are least likely to have an underlying neuroendocrine disorders [12].

To predispose a person to weight gain, an abnormality in energy metabolism would have to cause a reduction in maintenance energy requirements. Thus, assuming that energy intake and voluntary physical activity remain the same, the metabolic disorder would have to reduce daily energy expenditure. There are three major components of energy expenditure, i.e resting energy expenditure (REE), thermic effect of food and activity-related energy expenditure. Although the mechanisms causing obesity are not completely understood, their net effect is an imbalance of energy intake and expenditure, creating an energy excess that is stored as fat [12]. Two behaviours are thought to contribute to energy imbalance, such as excessive energy intake and inadequate physical activity. Sedentary lifestyles are common in developed countries. Technological advances have brought about limited opportunities for physical activity in work and home environments. Even in the leisure time, many individuals are not active [11] Decreased in energy expended on activities daily living, in the workplace and with recreational activities have been proposed as contributors to increases in obesity. Weight gain has been associated with time spent watching television or participating insedentary behaviors in adults [36]. Eventhough obesity is likely a disease with multiple genetic and molecular causes, the impact of an obesogenic environment on this disease is substantial. Therefore research should be conducted to uncover the major environmental determinants of obesity and most importantly, to understand which are realistically modifiable [35].

Body fat distribution is a better predictor of the health hazards of obesity than is the absolute amount of body fat [12]. Vague (1956) first described the association with diabetes of 'android' (upper body or truncal) obesity characterized by distribution of subcutaneous and visceral fat around the abdomen (Dewan and Wilding, 2005, p. 253). Central adiposity is an important clue to the presence of insulin resistance and hyperinsulinemia. By contrast, 'gynoid' obesity

in the gluteofemoral region is shown weakly associated with insulin resistance. Although most epidemiological surveys only used BMI as a predictor of diabetes risk, there is also good evidence that subjects with central adiposity, as assessed by measurement of waist circumference or waist-hip ratio (WHR), are at greater risk of T2DM [37]. Obesity, particularly visceral adiposity, contributes to the clustering of many other risk factors, such as hypertension, insulin resistance/type 2 diabetes melitus and dyslipidemia within individual patients. The molecular mechanisms underlying the metabolic abnormalities induced by visceral adiposity have yet to be fully elucidated, however, adipocytokines, such as adiponectin, tumor necrosis factor-α and resistin seem to play an important role in this process [38].

There are multiple factors involved in the mechanism of the increasing adipose tissue mass results in a reduction in insulin action in adipose tissue and at other sites, especially skeletal muscle and liver, such as metabolic factors, principally free fatty acids, local production of adipocytes hormones, collectively known as adipokines and effects mediated by altered secretions and action of systemic hormones and neurotransmitters, such as cortisol and noradrenaline [38].

After years of controversy, there is now very strong evidence that acute elevation of plasma FFA levels result in insulin resistance. Lipolytic activity in abdominal adipocytes is considerably higher than in subcutaneous adipocytes. The possible mechanism is the releasing FFA from the abdominal fat deposits directly to the liver through the portal vein causing hepatic insulin resistance, which is accompanied by increased gluconeogenesis and increased lipoprotein and triglyceride export to other tissue [35]. High concentrations of FFA have been shown to inhibit glucose metabolism in muscle in vitro and in vivo, but the precise mechanism have not been fully elucidated. Randle et al (1963) is trying to explain the concept of substrate competition between FFA and glucose (Dewan and Wilding, 2005, p.257). There are many possible mechanism of this situation. One possibility is that reduced sensitivity to insulin's effects on skeletal muscle is

due to inhibition of glucose transport or its phosphorylation by hexokinase, thus reducing the amount of glucose being metabolized by the glycolytic pathway, and also reducing glycogen synthesis [37].

Recent years, theory of the adipocyte as an endocrine organ had been develop. Many of these hormonal products have been found to influence insulin sensitivity in experimental animal models, and there is increasing evidence that some may also be important in humans. Tumour necrosis factor alpha (TNF-α) was one of the first adipokines to be identified that also caused insulin resistance. Adiponectin has also attracted recent interest, this peptide is inversely related to insulin resistance-concentrations fall as fat mass increases and it has also been found to improve insulin action experimentally [37].

2.4 Fat Intake

Before the 1920s, it was belived that fat did not play an essential dietary role if sufficient vitamins ans minerals were in the diet. However, in 1972, Evans and Burr demonstrated that animals fed semipurified fat-free diets had impaired growth and reproductive failure (Jones and Papamandjani, 2001, p. 104). This result indicated that fat are required for health. Subsequently Burr and Burr (1929) documented the nutritional essentially of a specific component of fat, linoleic acid (Jones and Papamandjani, 2001, p. 104). In the absence of this essential nutrient, symptoms developed, including scaliness of the skin, water retention, impaired fertility and growth retardation [39].

The traditional Mediterranean, Chinese and Japanese diets consumed until about 50 years ago contained very modest amounts of fat (15-25% of total calories). Traditonally, dietary fat had been seen as a rare commodity that amplifies the quality of the diet However, humans enjoy the 'mouth feel' and melting qualities of fats because it usually contains volatile fatty acids, and other odours, which are readily sensed by the 300 or more human olfactory receptors and perceived as extremely attractive. Butter was a traditionally expensive food,

available only to the rich. With the French invention of margarine in the nineteenth century, with its better keeping qualities, it became possible to obtain cheaper fat. Fat contains more than twice the energy per gram as does carbohydrate or protein, which explains why humans preferentially store fat as the primary energy reservoir [33,39].

In the twentieth century-particularly after the Second World War, there was a universal governmental initiative, with huge subsidies of agricultural research, buildings and equipment, as well as direct payments for crop and animal production. This led to a remarkable increase in animal protein and crop production and substantial increases in the availability of butter and vegetable oils. These prized commodities could then be eaten even by the poor. Thus, as countries became more affluent and went through the so-called "nutrition transition", there was a marked increase in sugars, fats, oils and animal protein. This 'cheap food' policy allowed even the poor to eat plenty of meat, butter/fats and sugars and thereby obtain the animal protein conducive to their children's growth, as well as plenty of energy from fats and sugars to maintain physical workload. Unfortunately, this food policy was associated with a huge epidemic of coronary heart disease in the 1940-60s affecting first the more affluent and agriculturally most innovative countries [33].

CVD especially coronary heart disease have become the biggest causes of death and disability in the world and particularly now in the developing countries. For certain diseases common to Western civilization, the evidence for reducing risk by adopting certain fat intake profiles is compelling. Western countries responded from 1970s by removing the overt fat from carcasses and manipulating the fatty acid composition of vegetable oils to first lower the saturated and later the trans fatty acid content. Quantitative fat intake is undergoing renewed scrutiny as a scientist debate the precentage of daily energy from fat that is necessary for health. Current recommendations relating to quantitative fat intake call for a reduction in total fat intake as well as a reduction in satureted fat. However, they

maintained the production of less metabolically adverse fats to fill cheap but desirable foods, e.g. fried and baked products. These are sold in huge amounts on the basis of 'consumer choice', with incomprehensible, although compositionally accurate, food labels. Heart disease rates fell as the saturated fatty acid intakes came down, but obesity and diabetes rates escalated as total fat and sugar intakes went up further [33,39].

2.4.1 Fat Intake in Relation to Insulin Resistance, Obesity and Abdominal Obesity

The majority of person with T2DM are overweight or obese. A high proportion of excess body fat in these persons is intra-abdominal (visceral), which have been associated with multiple adverse metabolic and physiologic outcomes, including insulin resistance and impaired glucose tolerance. Therefore, weight loss is imperative in most persons with diabetes [3]. Weight loss leads to a reduction in insulin resistance and has long-term effects on the maintenance of reduced blood glucose levels. Weight management also should include behavioral modification to encourage healthy eating behaviors, together with increased physical activity [40].

The role of dietary composition in modifying the risk of excess weight gain has become a subject of intense interest since the issue of obesity gained prominence as a new public health problem. There are several reasons to concentrate on reducing fat to achieve and maintain weight loss. A high-fat diet is associated with an increase in total energy intake. Fat is the most energy-dense macronutrient and does not seem to regulate its oxidation, or to regulate appetite [41].

Energy density has an important impact on daily food intake, for example, if volunteers are offered a variety of foods that have either their fat or their sugar content manipulated then the normal response of adults is to eat the same volume of food as usual, i.e there is no immediate adaptation in intake to reduce overall consumption when the fat or the sugar content is surreptitiously raised. This

means that normal adults who are provided (unwittingly) with an array of high-fat food providing 60% rather than 20% energy as fat, automatically continue to eat the same volume of food for several days and can accumulate 2 extra days'energy intake [33]. As a consequence that high fat diets may promote insulin resistance via their obesogenic potential, therefore it is important to give appropriate dietary advice which is facilitate weight managament and ensures the greatest degree of insulin sensitivity [26].

2.4.2 Saturated Fatty Acids

Fatty acids can be saturated and unsaturated, depending on double bonds. Saturated fatty acids do not contain any double bonds or other functional groups along the chain [42] Table 2.1 lists the most common of saturated fatty acids.

Table 2.1 : The Most Common Saturated Fatty Acids

Common name	Chemical name	Chemical structure	Code
Butyric	Butanoic acid	$CH_3(CH_2)_2COOH$	C4:0
Caproic	Hexanoic acid	$CH_3(CH_2)_4COOH$	C6:0
Caprylic	Octanoic acid	$CH_3(CH_2)_6COOH$	C8:0
Capric	Decanoic acid	$CH_3(CH_2)_8COOH$	C10:0
Lauric	Dodecanoic acid	$CH_3(CH_2)_{10}COOH$	C12:0
Myristic	Tetradecanoic acid	$CH_3(CH_2)_{12}COOH$	C14:0
Palmitic	Hexadecanoic acid	$CH_3(CH_2)_{14}COOH$	C16:0
Stearic	Octadecanoic acid	$CH_3(CH_2)_{16}COOH$	C18:0

(Source from : Alice H. Linchestein and Peter J.H. Jones, 2001[43])

Epidemiological data from US women and men have found a diet with more saturated fat from animal products are associated with the development of insulin resistance [14]. There is a consistent positive association between intake of saturated fatty acids (SFA) and hyperinsulinemia (as an indicator of insulin resistance) which appears to be independent of adiposity [26]. Saturated fat may

affect insulin sensitivity. In NIDDM, subjects a stepwise increase in saturated fat from 5 to 45 g had no impact on the glycaemic response to carbohydrate, although insulin increased after consumption of 15 g [41]. Meat contains longer-chain saturates and compiled data further indicate that the intake of saturated fat should be limited to < 8% of total energy [14,39].

2.4.3 Unsaturated Fatty Acids

Fatty acids containing double bonds in their carbon chains are described as unsaturated fatty acids [42]. More commonly used is identification of the position of the first carbon of a double bond relative to the methyl terminus of the fatty acid. Double bonds identified relative to the methyl end use the terms "n" or "ω" to indicate distance of the first bond along the carbon chain [44].

2.4.4 Monounsaturated Fatty Acids

If only one double bond is present, the fatty acid described as monounsaturated fatty acids. To contain a single double bond, a fatty acid must be at least 12 carbon atoms in length. These monounsaturated fatty acids (MUFA) typically possess a double bond at the n-9 or n-7 position [44]. Detailed of the most common monounsaturated fatty acids can be seen in table 2.2

Table 2.2 : The Most Common Monounsaturated Fatty Acids

Common name	Chemical name	Chemical structure	Code
Palmitoleic acid	9-hexadecaenoic acid	$CH_3(CH_2)_5CH=CH(CH_2)_7COOH$	C16:1, n-7 cis
Oleic acid	9-octadecaenoic acid	$CH_3(CH_2)_7CH=CH(CH_2)_7COOH$	C18:1, n-9 cis
Elaidic acid	9-octadecaenoic acid	$CH_3(CH_2)_7CH=CH(CH_2)_7COOH$	C18:1, n-9 trans

(Source from : Alice H. Linchestein and Peter J.H. Jones, 2001[43])

There is a growing consensus that diets for diabetic individuals should include only modest amounts of saturated fat but could include moderate-to-high levels of MUFA. Partial replacement of complex carbohydrates with MUFA in NIDDM patients may improve glycemic control [25].

A study by Pérez-Jiménez et a (2001). examining the effects of a high monounsaturated fatty acid (MUFA) (McAuley, 2006, p.7). Mediterranean diet also suggests an improvement in insulin sensitivity when compared with a diet richer in SFA [26]. High-MUFA diets result in improved insulin sensitivity and glycemic control compared to low-saturated fat, high carbohydrate diets [3].

Accordingly, MUFAs should compose 15 to 20% of total energy intake, with carbohydrates and MUFAs together composing 60% to 70% of total calories in patient with T2DM. Cis-MUFA found in olive oil, canola oil and nuts, which have several properties that may be beneficial in persons with diabetes [3]. Dietary sources of MUFA include nuts and vegetable oils such as olive, canola, high oleic safflower and sunflower oils that are liquid at room temperature [7].

2.4.5 Polyunsaturated Fatty Acids

Fatty acids with more than one double bond are described as polyunsaturated fatty acid [42]. Each subsequent double bond invariably occurs three carbon atoms further along the carbon chain from the bond preceding it. Therefore, the number of double bonds within a fatty acid is restricted by its chain length. Fatty acid with 18 carbon atoms or more that possess more than a single double bond will contain the first bond of their series only at the n-9, n-6, or n-3 position [44]. Table 2.3 presents the most common polyunsaturated fatty acids

Table 2.3 : The Most Common Polyunsaturated Fatty Acids

Common name	Chemichal name	Chemical structure	Code
Linoleic acid	9,12-octadecadienoic acid	$CH_3(CH_2)_4CH=CHCH_2CH=CH(CH_2)_7COOH$	C18:2, n-6,9 all cis
γ-linolenic acid	6,9,12-octadecatrienoic acid	$CH_3(CH_2)_4CH=CHCH_2CH=HCH_2CH=CH(CH_2)_4COOH$	C18:3, n-6,9,12 all cis
Arachidonic acid acid	5,8,11,14-eicosatetraenoic acid	$CH_3(CH_2)_4CH=CHCH_2CH=HCH_2CH=CHCH_2CH=CH(CH_2)_3COOH$	C20:4, n-6,9,12,15 all cis
α-linolenic acid	9,12,15-octadecatrienoic acid	$CH_3CH_2CH=CHCH_2CH=CHCH_2CH=CH(CH_2)_7COOH$	C18:3, n-3,6,9 all cis
Eicosapentaenoic acid	5,8,11,14,17-eicosapentaenoic acid	$CH_3(CH_2CH=CH)_5(CH_2)_3COOH$	C20:5, n-3,6,9,12,15 all cis
Docosahexaenoic acid	4,7,10,13,16,19-docosahexaenoic acid	$CH_3(CH_2CH=CH)_6(CH_2)_2COOH$	C22:6, n-3,6,9,12,15,18 all cis

(Source from : Alice H. Linchestein and Peter J.H. Jones, 2001[43])

Artificially high intakes and supplemental PUFA are not advised and the ADA recommends restriction to below 8% of calories The WHO recommendation for the general population is 3 to 7% of calories from PUFA. The effects of fish or omega-3 fatty acids intakes among the glucose-intolerant population seem smaller, suggesting that the beneficial effects of fish on coronary heart disease may be less in glucose-intolerant people. Intakes of omega-3 fatty acids above 3-4 g/day have been found to increase fasting plasma glucose and HbA1c in NIDDM patients [25].

Some epidemiological studies have found polyunsaturated fatty acids to be inversely associated with insulin levels, while others have reported a positive association. Serum or muscle fatty acid composition, a biomarker for dietary intake of some fatty acids, has also been related to insulin sensitivity measured by clamp studies. The studies have shown an association between insulin sensitivity

and various fatty acids, a positive association for linoleic acid and negative associations for palmitic, palmitoleic, di-homo-γ-linolenic acids [26].

Vegetable oils are major dietary sources of essential fatty acids and other unsaturated fatty acids. The fatty acid profile of vegetable oils varies widely and therefore different proportions of linoleic acid (LA) and α-linolenic acid. Safflower, sunflower, corn and soybean oils are more high in LA, yet of these, only soybean oil is a significant source of acid (ALA). High fat fish and marine mammals contain larger amounts of long chain n-3 fatty acid, EPA and DHA. The longer-chain n-6 fatty acids, such as arachidonic acid are found in foods of animal origin, including organ meats [44].

2.4.6 Polyunsaturated-Saturated Fatty Acid Ratio (PS ratio)

For nutritional purposes, it is useful to know how much polyunsaturated fat and how much saturated fat there is in food. This is called P/S ratio. With a high P/S ratio the fat at room temperature will be soft or even liquid; with a low P/S ratio the fat will be hard. Generally, animal fat have a low P/S ratio and plant fats have a high P/S ratio. The fats of poultry and fish have a higher P/S ratio than the fats of cows, pigs and sheep [45].

2.4.7 Polyunsaturated-Monounsaturated-Saturated Ratio (PMS ratio)

Concerning of the risk of heart disease , limit the total fat intake to 30% or less of total energy. From this 30%, about one third should come from SFA, one third should come from PUFA and one third should come from MUFA. It was recommended that the ratio of PUFA : MUFA : SFA should be 1 : 1 : 1 [21].

2.4.8 Omega-3 to Omega-6 Polyunsaturated Fatty Acid Ratio

Ongoing study of optimal amounts of PUFA in the diet has led to the premise that a balance of omega-6 and omega-3 PUFA is necessary for maximing the benefits of these fats. Some scientist believe that diets high in omega-6 PUFA relative to omega-3 PUFA may be associated with the increased prevalence of

chronic diseases, including heart disease. This has led to the proposed use of a target intake ratio of omega-6/omega-3 PUFA for assessing health risk and making dietary recommendations. The ratio of omega-6/omega-3 fatty acids in today's diet is estimated to be more than 10:1, with some estimates as high as 30:1. A few studies suggest that a much lower ratio of omega-6 to omega-3 fatty acids, ranging between 2:1 and 5:1, is desirable to in reducing the risk of disease [46].

2.5 Studies Assessing Fat Intake Profile and their Validation

Due to the growing knowledge about the role of specific fatty acids in health and disease, dietary intake measurement of individual fatty acids or classes of fatty acids are becoming important. There are several methods assessing dietary of individual fatty acids with their validation in different design study with the different aims and findings (Table 2.4).

Table 2.4 : Studies of Assessing Fat Intake Profile and their Validation

Author (year)	Study design	Subjects	Outcome	Dietary determinant	Finding
S.A. McNaughton,et al (2007)	Cohort study	115 adults who participate in the Nambour Skin Cancer Study, Australia	Plasma phospholipids fatty acids (saturated, MUFA, trans, PUFA)	FFQ of fatty acids (self – administered, over preceding 6 months and WFR (weighed food record) on two-consecutive days, every 2 months for 12 months	- Median intakes of FFQ were generally higher than the WFR, except for the ω-3 PUFA groups - Correlations between the FFQ and WFR were moderate (r:0.32-0.59), except for trans FA (r:0.03) - Correlations between each dietary assessment methods and the plasma phospholipids were poor for all FA othere than PUFA - The FFQ validity coefficients for total ω-3 PUFA, total long chain ω-3 PUFA, EPA, AA, and DHA were 0.50,0.63,0.45, 0.62 and 0.62
Kiyonuri Kuriki, et al (2003)	Cross-sectional study	A total 106 middle-aged Japanese dietitians from the membership of the Aichi Prefectural Dietetic Association	Plasma FA (total FA, SFA, MUFA, ω-3 PUFA, and ω-6 PUFA)	7-d weighed diet records and lifestyle assessment	- Plasma concentrations of EPA, DHA, and ω-3 HUFA (marine foods, not linoleic and ALA from plant origins) have positive correlations with dietary intakes (r: 0.303-0.602, p<.05) in both genders - Multiple linear regression adjusted for age, BMI, total E intake, fat consumption and lifestyle factors showed that dietary intakes of EPA, DHA and HUFA were positively associated with age in men (p<.05) - Lifestyle factors were not associated with dietary FA intakes and plasma concentrations

Table 2.4 (continued)

Author (year)	Study design	Subjects	Outcome	Dietary determinant	Finding
Qi Sun, et al (2007)	Cohort study	306 participants in The Nurses' Health Study who are female registered nurses aged 30-55 years living in 1 of 11 US states	Plasma and erythrocytes FA	SQ-FFQ, 61 items, during the previous year	- DHA in erythrocytes and plasma provided the strongest correlations with its intake, but erythrocytes DHA concentrations)were better than plasma DHA concentrations (r:0.56 vs r:0.48) - Total trans FA in erythrocytes was more strongly correlated with intake than were those in plasma (r:0.43 vs r:0.30)
Jing Ma et al (1995)	Cross-sectional study	4009 men and women aged 45-64 years from The Atherosclerosis Risk in Communities (ARIC) in four US communities	Plasma PL*, plasma CE*,	- FFQ for FA (interviewer-administered) with 66 item FFQ, during the past year which a slightly modified instrument developed by Willet	- Correlations between dietary and plasma FA for PL and CE, respectively, were as follows:SFA (r:0.15 and 0.23),MUFA (r:0.05 and 0.01),PUFA (r:0.25 and 0.21), linoleic acid (r:0.22 and 0.28), linolenic acid (r:0.15 and 0.21), EPA* (r:0.20 and 0.23) and DHA* (r:0.42 and 0.42) - The correlations between diet and plasma FAs held relatively constant regardless of whether participants were overeweight, had chronic disease, were alchol drinkers, or were cigarette smokers

* PL: phosphplipid, CE:cholesterol ester,
*EPA:cicosapentaenoic acid, DHA:docosahexaenoic acid

PART 3
METHODS

3.1 Variable and Indicator Matrix (VIM)

To organize the relationship between variables and the potential indicators of the variables, a variable - indicator matrix (VIM) table is needed (Table 3.1).

Table 3.1 Variable-Indicator Matrix

No	Variables	Indicators	Methods	References
1	Fasting plasma insulin	Insulin levels	Blood analysis	Tjokroprawiro, 2002
2.3	Plasma FFA	Plasma FFA levels	Blood analysis	Durrington, 1989
2.5	Low dietary fiber	<20 g/day consumption of fiber	3-day non consecutive 24 hr-recall	• PERKENI, 2006 • NCEP, 2001
3	Abdominal obesity (overweight, obesity)	• Waist circumference ≥ 90 cm • BMI ≥ 25 kg/m^2	Anthropomethic assessment	• WHO, 2004 • IDF ,2006
4.3	Excessive energy intake	Total fat > 30-35% of total calories	Fat SQ-FFQ	• PERKENI, 2006 • NCEP, 2001
4.4	Low physical activity	Categorical MET value (high, moderate, low)	GPAQ questionnaire	WHO , 2005
5	Unfavorable dietary fat :		Fat SQ-FFQ	• Mc Auley,2006 • Jones et al,2006 • Kenney, 2006 • Shikany, 2006 • Anderson,1999 • PERKENI, 2006 • NCEP, 2001 • IOM, 2002
	• High intake of total fat	• Intake of total fat >20-35% of total calories		
	• High intake of SFA	• Intake of SFA > 7% of total calories		
	• Low intake of MUFA •	• Intake of MUFA < 10% of total calories		
	• Low intake of PUFA	• Intake of PUFA <10% of total calories		
	• Low intake of ω-3 PUFA	• Intake of ω-3 PUFA < 1.6 g/d		
	• Low intake of ω-6 PUFA	• Intake of ω-6 PUFA < 17 g/d		

3.2 Study Design

The study design of this study was cross-sectional. Furthormore, this study was a part of a doctorate project titled "Relations of Lipogenesis, Lipolysis and Protein Transports with Elevated FFA Concentration in Centrally Obese Men. A Study on Asymetric Dimethyl Arginine (ADMA), Atrial Natriuretic Peptide (ANP), Leptin, Insulin, Retinol binding Protein 4 (RBP 4) and Adipocyte Fatty Acid binding Protein (AFABP)" with Mrs. Indriyanti Rafi Sukmawati, M.Si, of Faculty of Medicine, Hasanuddin University, as the Principle Investigator.

3.3 Study Population

The criteria of adult man to be included in this study were men aged 30 to 50 years with abdominal obesity (waist circumference ≥ 90 cm).

The exclusion criteria of this study were:

- Subject with :
  - FPG ≥ 126 mg/dl (have diabetes)
  - SGPT ≥ 110 U/L (have liver function disorder)
  - Blood pressure systolic ≥ 140 mmHg and diastolic ≥ 90 mmHg (JNC-VII) (have hypertension)
  - Creatinin serum ≥ 1.6 mg/dl (have kidney function disorder)
  - Consume hypolipidemia medicine within one month

For the reliable result of biochemichal assessment, subject should not have fever at that time of blood collection (indicated by body temperature ≤ 37.5 °C)

3.4 Study Site

This study was carried out in five government institutions and three private offices in Central Jakarta and East Jakarta district .

3.5 Sampling Method and Sample Size

3.5.1 Sampling Method

Central Jakarta district is the center of several government institutions. More than half of the subject work in Central Jakarta in government institution and only a few of them work in East Jakarta district. Subjects who worked in private office mostly came from East Jakarta district. Eventhough this sampling method was purposive, but the proportion of age was still inline with data from Bureau Statistics of Indonesia. All eligible subjects did the screening procedure first before blood samples (Table 3.1).

3.5.2 Sample Size

Number of minimum sample size was calculated using the following coefficient correlation hypothesis test (Ariawan, 1998 [52]) :

$$n = \{z_{1-\alpha/2} + z_{1-\beta}/\zeta\}^2 + 3$$

n = 125 abdominal obese adult men, with 10% for rejection rate

where,

$\zeta = 0.5 \ln \{ 1+r/1-r\}$

$Z_{1-\alpha/2}$ = 95% confidence level

$Z_{1-\beta}$ = 80% power of the test

ζ = transformasi Fisher = 0.266

r = 0.26 correlation coefficient of SFA with fasting insulin among non-diabetic men aged 32-74 years (Maron et al, 1991 [53])

n = sample size

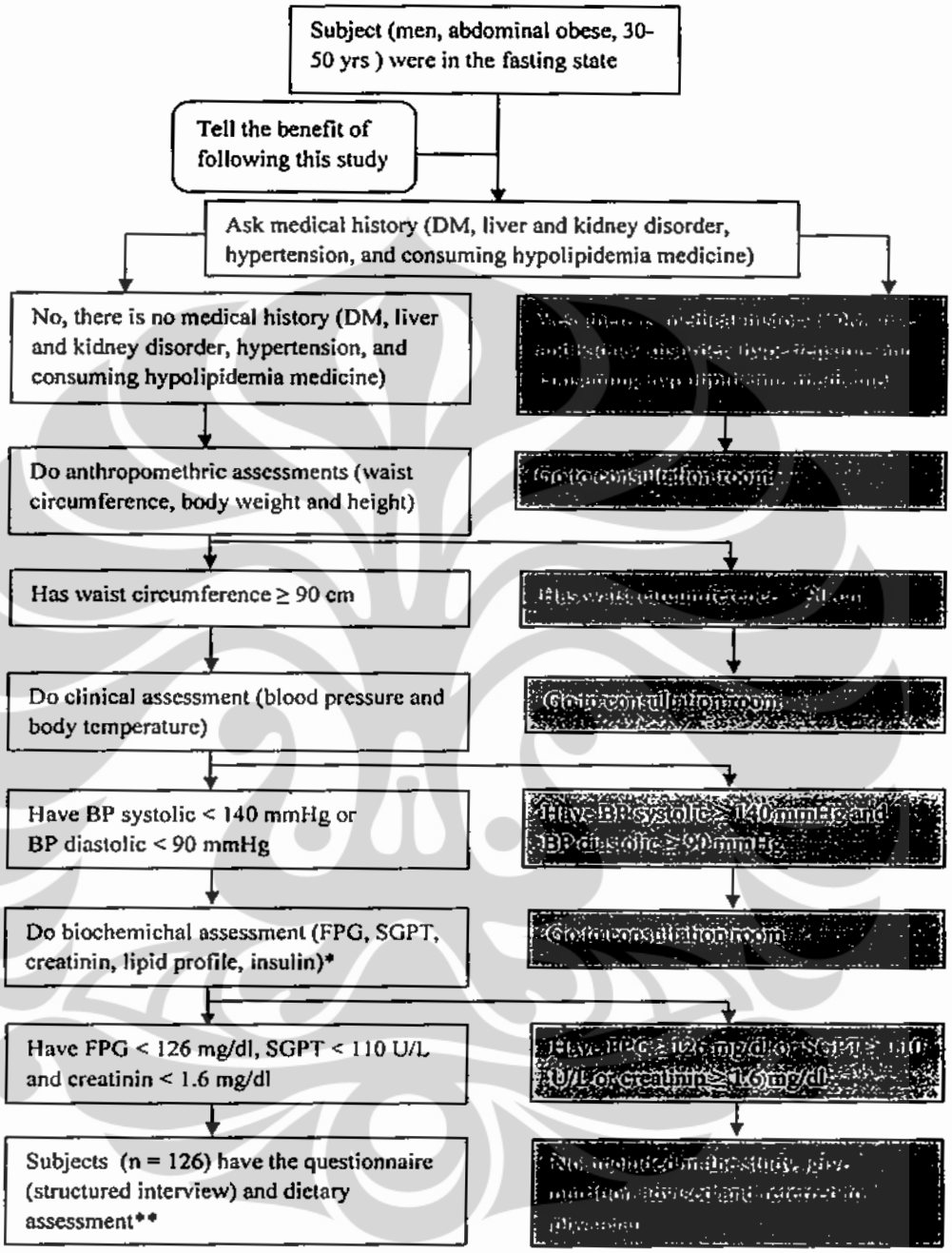

Figure 3 Screening Procedure of Subject

NB:

* : result of biochemichal assessment was received two days later

** : SQ-FFQ of fat and 3-day non-consecutive 24 hr-recall

▭ : fulfill the criteria (included)

▭ : not fulfill the criteria (excluded)

University of Indonesia

3.6 Data Collection Procedures

Data collection was done through several methods such as structured interview, anthropomethric assessments (body weight, height, waist circumference), biochemichal assessments (fasting plasma glucose, fasting plasma insulin, fasting plasma FFA and profile lipid), clinical assessments (blood pressure and body temperature), dietary assessment (SQ-FFQ of fat and 3-day non-consecutive 24 hr-recall). The data collection was conducted between March and May 2009.

3.6.1 Structured Interview

Structured interview was used to obtain the information on the subject characteristics, such as socio-economic-demographic like name, address, age, ethnicity, occupation, income, expediture; medical history (DM, hypertension, liver disorder, kidney disorder and the use of hypolipidemia medicine within one month) and lifestyle using GPAQ and STEPS questionnaires, which was designed for surveillance for chronic disease in USA (smoking habit, alcohol use, fruit and vegetables consumption, physical activity) [36,40,51,55].

3.6.2 Anthropometric Assessments

Anthropometric assessments were conducted by measuring the body weight, height, and waist circumference of subjects.

Body weight. Body weight of subjects was measured using the electric SECA Alpha Model 872 platform scale. The scale was positioned in flat surface and the starting point was set on zero. When measuring the weight, the subject did not have meal before. The electric SECA Alpha Model 872 platform scale was placed on a hard level surface and checked for zero-balance before each assessment. The subject was stand unassisted in the centre of the platform and straight ahead, standing relaxed but still and clothing minimally one piece with mobile-phone, wallet and anythings heavier were put off. The body weight was recorded to the

nearest 0,01 kg [54]. The assessment was taken account twice for every subject, and the average was taken.

Height. Height of subject was measured using microtoise. When the microtoise tape is used, it is first placed on the floor. The tape was then pulled out to its fullest extent and released and the end was fixed with a hard banded to a door or hard wall. When measuring the height, subject was asked to take off their slippers/shoes also all hair accesories. Subject was standing erect directly below the point of attachment with head correctly in the Frankfurt plane (horizontal position), feet together, knees straight, heels, buttock and shoulder blades were in contact with the vertical surface of the wall. Arms were hanging loosely at the sides with palms facing the thighs, shoulders were relaxed. The moveable headboard was then gently lowered until it touched the crown of the head and compressed the hair. The height of subjects were recorded by the antropometrist after their eye were at the same level with the headboard. [54,55]. The assessment was taken account twice for every subject and the average was taken.

Waist circumference. Waist circumference was measured by a waist-watcher tape measure with the fasting condition or one hour after have breakfast and wear little clothing to allow the tape to be correctly positioned. Subject was standing erect with the abdomen relaxed, arms at the sides, feet together and their weight equally divided over both legs and asked to lift it their cloth and waist circumference was seen clearly. To perform the assessment at the natural waist, the lowest rib margin (the tenth rib) was first located then palpated the iliac crest in the midaxillary line. A waist-watcher tape was applied horizontally midway between the lowest rib margin and the lateral iliac crest made into a complete loop and fitted firmly around the waist by a spring mechanism, which controlled by a pushbutton. This enables the subjects to have both hands free for adjusting the position of the tape. The tape was then read while in place. Removal was particularly helpful to overweight subject who may find it hard to read the tape while it is in position [54].

The assessment was taken account twice for every subject and the average was taken.

3.6.3 Biochemical Assessments

Biochemical assessments were done by Prodia Laboratory

Fasting plasma glucose. Fasting plasma glucose gives more precise information of insulin production. Fasting plasma glucose was quantitatively analyzed by using standard procedure utilizing The ADVIA Chemistry Systems Glucose Hexokinase II (GLUH).

Fasting plasma insulin. Fasting plasma insulin helps to determine whether a high blood glucose reading is the result of insufficient insulin or poor use of insulin. Fasting plasma glucose was quantitatively analyzed by using standard procedure utilizing IMMULITE/IMMULITE 1000 Insulin.

Fasting plasma FFA. Fasting plasma FFA is a biomarker of dietary fat. This was quantitatively analyzed by using standard procedure based on calorimetri.

3.6.4 Clinical Assessments

Blood pressure Blood pressure assessment was measured by auscultatory methods using sphygmomanometer and stethoscope. This was comprising an inflatable (Riva-Rocci) cuff placed around the upper arm at roughly the same vertical height as the heart, attached to a mercury manometer. The mercury manometer is the gold standard for arterial pressure assessment, measures the height of a column of mercury, giving an absolute result without need for calibration and consequently not subject to the errors and drift of calibration which affect other methods with no sound interrupt while measuring. Uncover the elbow to measure blood pressure and let the subject sit as convenience before start the assessments. Place sphygmomanometer with distal side of cuff about 1.5 cm from brachial artery. Lock the cuff and then inflated by repeatedly squeezing a rubber bulb when doing palpation on the radial artery. Pulsation of radial artery was decreased untill can

not be palpate. After that, open the cuff untill 3 torr/second and palpation systolic blood pressure was obtained if radial artery can be palpate. Released the cuff pressure and hold on for 15 seconds. Meanwhile, put diafragma of stethoscope in the brachial artery. Lock the cuff and inflated by repeatedly squeezing a rubber bulb untill 20 torr above palpation systolic blood pressure. Start to listen with the sthetoscope to the brachial artery at the elbow. Open the cuff untill 3 torr/second. When blood just starts to flow in the artery, the turbulent flow created a "whoosing" or pounding (first Korotkoff sound). The pressure at which this sound first heard was the auscultation systolic blood pressure. Intensity of Korotkoff sound will be increased when manset pressure going down. The cuff pressure further released until no sound can be heard (fifth Korotkoff sound), this called the auscultation diastolic blood pressure [56].

Body temperature. Body temperature was measured using mercury thermometer. The temperature of thermometer at 35°C or below before the assessment was done. The thermometer was put in the axilla at ipsilateral of the elbow. Elbow in the immobilized condition for 5-10 minutes. Then read the value in the thermometer [56].

3.6.5 Dietary Assessments

Dietary assessment was conducted in two stages :

A. Development and validation of SQ-FFQ of fat [57]

The development and validation of fat SQ-FFQ were done on December 2008 – February 2009, with the following sequence :

1. A long list of foods (single and composite dishes), which were potentially important fat sources, was selected using Indonesian food composition tables, existing fat questionnaires (from other studies) and results of existing surveys on adult men consumption.

2. Three series of focus group discussion (FGD) among 8 adult men were conducted to elicit foods, focusing on fat rich foods that are usually (typically) consumed by adult men in the study area.

3. The selected food list (step 1) was compared to the results of the FGD (step 2); the food items that were never consumed (or not commonly eaten) by adult men under study were excluded. On the other hand, the food items obtained exclusively from the FGD, which may generally be consumed by adult men were added to the list.

4. Subsequently, a rough questionnaire was designed using the food list derived from step 3 and asked in the last FGD to see the response, which was identified as potentially major sources of dietary fat, incorporating the frequency response and the portion size questions. The response of specific food sources of fat ranging from monthly, weekly, daily and never for the frequency of consumption of specified serving sizes based on household measures (e.g cups, spoons) and natural unit (e.g. 1 slice bread). Additional questions were added to collect information on the trimming of visible fat from meat, frequency of fried at home and fried takeaway food and specific types of fats and oils used on bread, vegetables and in cooking. Questions on the use of self-prescribed nutritional supplements were also included. Subject were asked to recall their frequency of consumption of food items over the preceding 1 month.

5. After run and analyze the last FGD then the definitive form of fat SQ-FFQ were ready, the data collection for measuring its relative validity with the 7-day dietary records was conducted to cover "true intake" of different type of fatty acids especially SFA, MUFA, PUFA. Ultimately, a validation of fat SQ-FFQ was done on 51 subjects include obese and non obese adult men. After analysis the relative validation, this SQ-FFQ can be used to estimate the usual intake of fat with several statistical analyses such as mean difference in Paired t-test, Spearman's correlation and cross-classification.

B. Dietary Intake Assessment

- Fat Intake

Fat SQ-FFQ (interviewer-administered), which had been validated before, was administered to assess the dietary fat intake of abdominal obese adult men. Data was collected in March - May 2009 by two trained enumerators using food models and calibrated households utensils. Subjects were asked to recall their frequency of consumption of food items over the preceding 1 month.

- Other nutrient intake

Intake of carbohydrate especially fiber and energy were measured using 3-day non-consecutive 24 hr recall.

3.7 Data Analysis

3.7.1 Nutrient Analysis:

All items consumed assessed by both fat SQ-FFQ and dietary record were converted to daily nutrients with the use Nutri-Survey 4.0 program. Recipes were created for foods that could not be found within the available database. Intake omega-3 and omega-6 PUFA were analyzed based on data of Wiranda G. Piliang (2006) and Burhan Bahar.(2006) For further analysis, nutrient intakes were transferred to the SPSS software program (version 15.0 for Windows). A univariate analysis such as mean, standard deviation, median and percentiles 25 and 75 was presented to show the intake of energy and nutrient per day while percentage was used to show the general characteristics of the subjects.

3.7.2 Statistical Analysis:

Data entry and statistical analysis were done using SPSS program version 15. Total physical activity was analyzed as MET value/week and categorized as high, moderate, and low based on physical activity cut-off value (Table 3.2)..

Total physical activity MET-minutes/week = the sum of the total MET minutes of activity computed for each setting.

Equation : Total physical activity = {(P2*P3*8) + (P5*P6*4) + (P11*P12*8) + (P14*P15*4)}

Table 3.2 Classification of Total Physical Activity

Level of total physical activity	Physical activity cutoff value
High	• IF : (P2+P11) ≥ 3 days AND total physical activity MET minutes per week is ≥ 1500 OR • IF : (P2+P5+P8+P11+P14) ≥ 7 days AND total physical activity MET minutes per week is ≥ 3000
Moderate	• IF : level of physical activity does not reach criteria for high levels of physical activity AND at least one of the following : • IF : (P2+P11) ≥ 3 days AND ((P2*P3) + (P11*P12)) ≥ 3*20 minutes OR • IF : (P5+P8+P14) ≥ 5 days AND ((P5*P6)+(P8*P9)+(P14*P15) ≥ 150 minutes • IF : (P2+P5+P8+P11+P14) ≥ 5 days AND total physical activity MET minutes per week ≥ 600
Low	IF level of physical activity does not reach the criteria for either high or moderate levels of physical activity

Descriptive statistics were done to describe frequency, mean, median, standard deviation and range value. Kolmogorov-Smirnov test was used to test the normality of sample distribution. Pearson correlation test to find the correlation between intakes of different fatty acids, FFA plasma, FPG, insulin level and insulin resistance. Partial correlation was used to find the correlation between intakes of different of fatty acids and insulin level controlling for age, waist circumference, FFA plasma, physical activity (MET value/week), smoking habit

(Y/N), alcohol use (Y/N) and fibre intake. Multiple logistic regression was peformed to assess the likelihood of having insulin resistance. Fatty acids in the model was put as individual due to highly multicollinearity. This analysis used multiple categorization, i.e unfavorable intake of individual fatty acid intake (0 = met the standard, 1 = did not meet the standard), unfavorable intake of fiber intake (0 = met the standard, 1 = did not meet the standard), habitual of every day smoking (0=no, 1=yes), habitual of alcohol use (0=no, 1=yes), adequacy of physical activity (0 = did not meet the standard (low physical activity), 1 = met the standard (moderate and high pysical activity)).

3.8 Ethical Consideration

The data was collected after obtaining the ethical approval from the Committee of The Medical Research Ethics of the Faculty of Medicine, University of Indonesia. Informed consent was obtained from all the subjects prior data collection and their participation were voluntary. Comprehensive and thorough explanation of the study's purpose and procedures were given to the subjects prior to interviewing and at any inquires. Subject's identities and the result of the study were used strictly for the purpose of the study and treated confidentially. Permission for recording the FGDs using tape recorder were obtained prior to conducting each FGD. At the end of data collection, all subjects were given counselling for their condition and metabolic disorder that they might have and given information on the result of biochemichal assessment.

PART 4
RESULTS

4.1 Development and Validation of Fat SQ-FFQ

After the development of fat SQ-FFQ, validation of fat SQ-FFQ was done to 51 subjects (men, 30-50 years, obese and non-obese person). Four subjects were excluded due to unfinished 7-day dietary records (n=2), surrogate interview of SQ-FFQ (n=1) and non Indonesian man (n=1). The 6-day records were used for analysis in order to allow, for each subject, one day when the record was poor (tend to be under-estimated). This 6-day records was the number required to estimate true average fat intake for groups of individuals. The cut-off for underreporting energy intake with 99.7% confidence limit was 0.92 for individual (n=1) with 6-day records. According to this criteria, data from 45 subjects can be used for validation analysis. One subject was excluded due to the highest ratio of EI/BMR (over-reporting). With all the criteria, 44 subjects were eligible to have validation analysis.

In validation study, (Table 4.1) the majority of subjects (77.3 %) were in younger age group (30-39 year). Most of the subjects were bachelor degree (45.5%). More subjects were private employee (40.9%) than government employee (22.7%).

Table 4.1 Socio-Demographic Characteristics of Subjects for Validation (n=44)

Characteristics	n (%)
Age :	
• 30-39 year	34 (77.3)
• 40-50 year	10 (22.7)

Table 4.1 (continued)

Characteristics	n (%)
Education :	
• 9-year schooling	8 (18.2)
• senior high school	11 (25)
• bachelor degree	20 (45.5)
• master degree	5 (11.4)
Occupation :	
• private employee	18 (40.9)
• government employee	10 (22.7)
• medical doctors	5 (11.4)
• others	11 (25)

Table 4.2 showed that most of subjects (61.4%) had normal waist circumference. According to WHO classification on BMI, most (50%) of subjects were in normal range, but fourteen (31.8%) of the subjects were in pre-obese stage. Meanwhile, based on Asian Adult Population classification on BMI, distribution of subjects into normal range, overweight and obese-I was more comparable.

Table 4.2 Antropomethric Characteristics of Subjects for Validation (n=44)

Characteristics	n (%)
Waist circumference :	
• < 90 cm (normal)	27 (61.4)
• ≥ 90 cm (abdominal obese)	17 (38.6)
BMI :	
Based on WHO classification :	
• < 18.50 kg/m^2 (mild underweight)	3 (6.8)
• 18.50-24.99 kg/m^2 (normal)	22 (50)
• ≥ 25.00 kg/m^2 (overweight)	
o 25.00 -29.99 kg/m^2 (pre-obese)	14 (31.8)
o 30.00-34.99 kg/m^2 (obese class I)	4 (9.1)
o 35.00-39.99 kg/m^2 (obese class II)	0 (0)
≥ 40 kg/m^2 (obese class III)	1 (2.3)

Table 4.2 (continued)

Characteristics	n (%)
BMI :	
Based on Asian Adult Population classification :	
• < 18.50 kg/m^2 (underweight)	3 (6.8)
• 18.50-22.99 kg/m^2 (normal range)	12 (27.3)
• 23.00-27.49 kg/m^2 (overweight)	17 (38.6)
• ≥ 27.50 kg/m^2 (obese I)	12 (27.3)

Analysis of relative validation used three types of statistical analysis i.e mean differences with Paired T-test, Spearman's correlation test and cross-classification test. The result of relative validity analysis showed that this fat SQ-FFQ was valid. There is no significance difference between energy, total fat, SFA, MUFA and PUFA intake of 6-day records and SQ-FFQ (Table 4.3) and coefficient correlation were in moderate correlation (r: 0.183-0.304) with one significant coefficient ($p<0.05$) (Table 4.4). There were 27.3%-36.3% correct classification of individual fatty acids intake and <10% of gross misclassification except for MUFA which was slightly over 10% (11%) (Table 4.5).

Table 4.3 Mean Intake of Total Fat, SFA, MUFA, PUFA in 6d-Records and in Fat SQ-FFQ (n=44)

	Fat SQ-FFQ	6 day- records
Energy (Kcal)	1878.16 ± 811.31	2047.55 ± 357.88
Total fat (g)	85.64 ± 43.44	79.77 ± 19.76
SFA (g)	39.36 ±19.45	33,41 ± 10.15
MUFA (g)	21.59 ± 12.06	20.82 ± 5.50
PUFA (g)	16.11 ± 12.06	17,57 ± 4.54

Table 4.4 Correlation Coefficient between 6d-Records and Fat SQ-FFQ (n=44)

	Correlation between 6d-records and fat SQ-FFQ
Total fat	0,185
SFA	0,304*
MUFA	0,183
PUFA	0,222

*significant correlation at p<0.05, Spearman correlation test

Table 4.5 Cross-Classification of Fat SQ-FFQ and 6d-Records (n=44)

	Correctly classified into the same quartile(%)	Classified into one adjacent quartile(%)	Classified into extreme quartiles (%)
Total fat	27.2	40.8	4,6
SFA	36.3	38.6	6.8
MUFA	27.2	43.1	11.3
PUFA	27.3	36.2	9

4.2. Relationship Between Intake of Different Fatty Acids and Insulin Level in Abdominal Obese Adult Men in Jakarta

4.2.1 General Characteristics of the Study Population

4.2.1.1 Socio-Economic-Demographic Characteristics

Table 4.6 lists the characteristics in socio-economic-demographic of the subjects. This study involved 126 subjects, in whom Javanese (63.5%) was dominant ethnicity. Most of them (46.8%) were diploma/bachelor degree and majority of them (68.3%) worked as government employee.

Half (51.2%) of this population had income between IDR 2.000.000 - IDR <5.000.000 monthly followed (19.2%) by IDR 5.000.000 - IDR 10.000.000 monthly. Expenditure between food and non-food per month were almost equal as

individual (IDR 400.000 vs IDR 500.000) but as family expenditure, food was almost twice than non-food expenditure (IDR 1.000.000 vs IDR 550.000).

Table 4.6 Socio-Economic-Demographic Characteristics of the Study Population

Characteristics	30-39 years		40-50 years		Total	
	n	%	n	%	n	%
Subjects	65	51.6	61	48.4	126	100
Ethinicity						
▪ Javanese	37	56.9	43	70.5	80	63.5
▪ Betawinese	2	3.1	5	8.2	7	5.6
▪ Sundanese	10	15.4	3	4.9	13	10.3
▪ Sumatran	13	20	7	11.5	20	15.9
▪ Celebesian	1	1.5	3	4.9	4	3.2
▪ Balinese	2	3.1	0	0	2	1.6
Marital status						
▪ married	56	86.2	60	98.4	116	92.1
▪ single	9	13.8	1	1.6	10	7.9
Education						
▪ 9-year schooling^	1	1.5	2	3.3	3	2.4
▪ Senior high school	12	18.5	18	29.5	30	23.8
▪ Diploma/bachelor degree	31	47.7	28	45.9	59	46.8
▪ Master degree	21	32.3	13	21.3	34	27
Occupation						
▪ Civil workers	43	66.2	43	70.5	86	68.3
▪ Private employee	14	21.5	4	6.6	18	14.3
▪ Policeman	4	6.2	14	23	18	14.3
▪ Others^^	4	6.2	0	0	4	3.2
Income (IDR/month)	n=65		n=60		n=125	
▪ >10.000.000	14	21.5	5	8.2	19	15.2
▪ 5.000.000-10.000.000	15	23.1	9	15	24	19.2
▪ 2.000.000-<5.000.000	24	36.9	40	66.7	64	51.2
▪ 1.000.000-<2.000.000	10	15.4	6	10	16	12.8
▪ <1.000.000	2	3.1	0	0	2	1.6

Table 4.6 (continued)

Characteristics	30-39 years		40-50 years		Total	
	n	%	n	%	n	%
Subjects	65	51.6	61	48.4	126	100
Expenditure (IDR/month) Individual						
▪ Food	n=64 450.000 (300.000-862.500)		n=59 300.000 (250.000-500.000)		n=123 400.000 (300.000-600.000)	
▪ Non food	n=61 500.000 (300.000-1.000.000)		n=59 500.000 (250.000-800.000)		n=120 500.000 (300.000-1.000.000)	
Family						
▪ Food	n= 60 1.250.000 (200.000-1.500.000)		n=51 1.000.000 (600.000-1.500.000)		n=111 1.000.000 (450.000-1.500.000)	
▪ Non food	n=61 500.000 (0 -1.500.000)		n=51 700.000 (300.000-1.500.000)		n=112 550.000 (202.500-1.500.000)	

^ : elementary school + junior high school

4.2.1.2 Anthropometric-Health Related Characteristics

In this study, (Table 4.7) the mean waist circumference of the subjects were comparable between younger age group (30-39 year) and older age group (40-50 year) about 97 cm. Using WHO classification on BMI (WHO, 2004), pre-obese was the highest percentage (61.1%) while based on Asian adult population classification (WHO, 2004), the highest percentage (72.2%) was under obese I category.

Table 4.7 Anthropomethric-Health Related Characteristics of the Study Population

Characteristics	30-39 year		40-50 year		Total	
	n	%	n	%	n	%
Subjects	65	51.6	61	48.4	126	100
Median (25th–75th percentile)						
Waist circumference (cm)	97 (92.5-101)		97 (92.5-101)		97 (92.75-101)	
BMI						
WHO classification,2004						
• Overweight (kg/m^2) :						
• 25.00-29.99 (pre-obese)	37	56.9	40	65.6	77	61.1
• 30.00-34.99 (obese class I)	22	33.8	18	29.5	40	31.7
• 35.00-39.99 (obese class II)	4	6.2	3	4.9	7	5.6
• ≥ 40 (obese class III)	2	3.1	0	0	2	1.0
Adult Asian population classification,WHO, 2004						
• 23.00-27.49 (overweight)	19	29.2	16	26.2	35	27.8
• ≥27.5 (obese I)	46	70.8	45	73.8	91	72.2

4.2.1.3 Lifestyle-Health Related Characteristics

Table 4.8 presents lifestyle-health related characteristics. Most of subjects (60.3%) in this study did not smoke every day. Twelve cigarrettes was median for those subjects who smoked every day. Majority of subjects (79.4%) did not drink alcohol in past one year. Consumption on fruit was relatively low as less than half (42.1%) consumed fruit every day. Contrastly, most of these subjects (57.1%) eat vegetable every day. Most of subjects (59.3%) were in moderate physical activity. A few of subjects (11.9% and 13.5%) had daily walking of 15 minutes and medium intensity of sport of 3-7 days/week.

Table 4.8 Lifestyle-Health Related Characteristics of the Study Population

Characteristics	30-39 years		40-50 years		Total	
	n	%	n	%	n	%
Subjects	65	51.6	61	48.4	126	100
Smoking every day	26	40	24	39.3	50	39.7
Drink alcohol past one year	11	16.9	15	24.6	26	20.6
Fruit consumption (days/week)						
▪ 7 (every day)	24	36.9	29	47.5	53	42.1
▪ 0-6 (not every day)	41	63.1	32	52.5	73	57.9
Vegetable consumption (days/week)						
▪ 7 (every day)	30	46.2	42	68.9	72	57.1
▪ 0-6 (not every day)	35	53.8	19	31.1	54	42.9
Physical activity minutes per week (MET value) :						
▪ High	4	6.2	4	6.6	8	6.3
▪ Moderate	43	66.2	32	52.5	75	59.3
▪ Low	18	27.7	25	41	43	34.1

4.2.2 Dietary Intake of the Study Population

Dietary fat intake profile (total fat, SFA, MUFA, PUFA, and omega 6) from fat SQ-FFQ and selected nutrients (energy, protein, fat, and carbohydrate) from 3 day-recall were expressed as mean, while dietary omega 3 PUFA from fat SQ-FFQ and dietary fiber were expressed as a median, which is presented in table 4.9

Intake of individual fatty acids (% of total calories) of this study were higher than previous study figure (Djuwita-Hatma, 2001) (total fat: 41.23% vs 29.5-35.8%, SFA: 21.51% vs 20%, MUFA: 9.32% vs 2.6-6.1%, PUFA: 6.87% vs 2.6-4.6%). Ratio of P(M)S also showed higher figure than previous study (0.75 vs 0.15-0.25). Intake of omega-3 and omega-6 PUFA did not meet the requirement based on IOM, 2002 (1.0 g vs 1.6 g and 16.77 g vs 17 g).

Data from 3-day recall showed that both fat and fiber intakes were less favorable; percentage of total fat was higher than the recommendation (33.57% vs

< 30%). while fiber intake was lower than the recommendation (PERKENI and NCEP) (10 g vs 25 g and 30 g).

Our study found that peanut was the main source of total PUFA and MUFA intake among the subjects. Meanwhile coconut milk in vegetable dish *(lodeh)*, was the main source of SFA intake. Omega-3 and omega-6 PUFA intake were found in peanut and in several marine fish. (Table in appendix 8).

Inter-correlations among individual fatty acids (in gram) were high and significant (Table in appendix 8)

Table 4.9 Dietary Intake Profile of the Study Population

Dietary intake	30-39 years		40-50 years		Total	
	n	% of total calories	n	% of total calories	n	% of total calories
Subjects	65		61		126	
Fat intake (SQ-FFQ)						
▪ Total fat (g)	89.14 ± 37.90	40.81 ± 4.01	84.94 ± 41.87	41.68 ± 7.00	87.10 ± 39.77	41.23 ± 5.66
▪ SFA (g)	46.63 ± 21.30	21.22 ± 3.37	44.58 ± 24.39	21.82 ± 5.45	45.64 ± 22.77	21.51 ± 4.50
▪ MUFA (g)	20.11 ± 8.53	9.30 ± 1.62	18.94 ± 9.40	9.34 ± 1.82	19.54 ± 8.95	9.32 ± 1.71
▪ PUFA (g)	14.52 ± 7.60	6.61 ± 2.00	14.50 ± 7.52	7.15 ± 2.02	14.51 ± 7.52	6.87 ± 2.02
▪ ω-6 (g)^a	17.77 ± 8.70	8.30 ± 3.00	15.70 ± 10.00	7.68 ± 3.09	16.77 ± 9.30	8.00± 3.01
▪ ω-3 (g)^b	1.10 (0.80-2.00)	0.60 (0.42 - 0.80)	0.90 (0.50-1.40)	0.46 (0.33 -0.73)	1.00 (0.60-1.70)	0.54 (0.35 - 0.76)
Ratio						
▪ P(M)S (%)	0.73 (0.63 - 0.83)		0.78 (0.67 - 0.91)		0.75 (0.64 - 0.88)	
▪ ω-6 :ω-3 (%)	13.20 (10.90 - 19.14)		16.08 (10.82 - 23.00)		14.27 (10.90 - 21.42)	
Other intakes (3d-recall)						
▪ Energy intake (Kcal)	1907.12 ± 417.92		1850.44 ± 405.42		1879, 68 ± 411.25	
▪ Protein intake (g)	67.37 ± 16.77	14.18 ± 2.07	65.57 ± 16.4	14.23 ± 2.13	66.5 ± 16.55	14.21 ± 2.1
▪ Fat intake (g)	73.2 ± 26.61	33.97 ± 6.63	68.69 ± 21.8	33.14 ± 6.7	71.02 ± 24.41	33.57 ± 6.65
▪ Carbohydrate intake (g)	242.37 ± 50.22	51.3 ± 6.5	240.9 ± 57.6	52.2 ± 6.44	241.65 ± 53.73	51.75 ± 6.46
▪ Fiber intake (g)	9.8 ± 3.14		10.71 ± 4.1		10 (8-12)	

^a : source from *Fisiologi Nutrisi*, IPB Press, 2006 [58]
^b: source from *Fisiologi Nutrisi*, IPB Press, 2006 [58] dan *Panduan Praktis Memilih dan Menangani Produk Perikanan*, Burhan Bahar,Jakarta, 2006 [59]

4.2.3 Laboratory Profile of the Study Population

Laboratory profile (Table 4.10) showed that the average fasting plasma insulin were in desirable range (<27 u/L), but the lipid profile indicated hypercholesterolemia and hypertrigliseridemia, i.e. cholesterol total was 210.33 mg/dl, cholesterol LDL was 142.46 mg/dl, trigliserida was 152.27 mg/dl (normal value, cholesterol total < 200 mg/dl, cholesterol LDL < 100 mg/dl, trigliserida <150 mg/dl). HDL cholesterol in this study did not meet the higher (desirable) criteria set by NCEP (2001) of 41.53 mg/dl (normal value, cholesterol HDL ≥ 60 mg/dl).

Table 4.10 Laboratory Profile of the Study Population

Characteristics	30-39 years	40-50 years	Total
	n	n	n
Subjects	65	61	126
Fasting plasma insulin (u/L)	7.53 ± 3.97	7.73 ± 5.17	7.63 ± 4.57
Fasting plasma glucose (mg/dl)	90.23 ± 8.67	95.18 ± 9.02	92.63 ± 9.15
Fasting FFA (mM/l)	0.54 ± 0.19^1	0.57 ± 0.15	0.55 ± 0.17^2
Cholesterol total (mg/dl)	205.6 ± 34.38	215.38 ± 31.44	210.33 ± 33.22
Cholesterol LDL direk (mg/dl)	137.58 ± 33.81	147.66 ± 28.43	142.46 ± 31.6
Cholesterol HDL (mg/dl)	42.17 ± 6.19	40.86 ± 8.3	41.53 ± 7.29
Trigliserida (mg/dl)	150.32 ± 64.54	154.34 ± 55.7	152.27 ± 60.18
Total chol/HDLC	5.00 ± 1.06	5.42 ± 1.07	5.19 ± 1.08
LDLC/HDLC	3.33 ± 0.93	3.70 ± 0.83	3.51 ± 0.90
TG/HDLC	3.32 (2.10-4.90)	3.30 (2.70-5.20)	3.31 (2.54-4.81)

^1 : n = 63
^2 : n=124

Fasting plasma glucose were positively correlated with fasting plasma insulin and fasting plasma FFA (r=0.243, p<0.01 and r=0.215, p<0.05, respectively, Pearson correlation test) as seen in table 4.11.

Table 4.11 Correlation Coefficient (r) and Significant Level (p) between Fasting Plasma Insulin, Fasting Plasma Glucose, Fasting Plasma FFA in Abdominal Obese Adult Men

	Coefficient correlation (r)		
	Fasting plasma insulin	Fasting plasma glucose	Fasting plasma FFA
Fasting plasma insulin	1.000		
Fasting plasma glucose	0.243 (0.006)**	1.0000	
Fasting plasma FFA	-0.075 (0,402)	0.215 (0.016)*	1.000

* significant correlation at $p<0.05$, Pearson correlation test
**significant correlation at $p<0.01$, Pearson correlation test

4.2.4 Correlation between Fatty Acids Intake with Fasting Plasma Insulin, Fasting Plasma Glucose and Fasting Plasma FFA

Results in this study showed that there were no correlations between intake of individual fatty acids and fasting plasma insulin (Table 4.12). PUFA intake was positively correlated with fasting plasma glucose as gram and with fasting plasma FFA as % of total calories ($r=0.175$, $p<0.05$ and $r=0.180$, $p<0.05$, Pearson correlation test).

A multiple logistic regression analyses were performed to examine the odds ratio having insulin resistance. This odds ratio should not be regarded as conclusive in this study, rather they are showing trend of having insulin resistance. Prevalence of insulin resistance (IR), with cut-off point about 2, among subjects was 32.5%. Results of multiple logistic regression showed three determinants were significantly associated with insulin resistance, i.e waist circumference, fasting plasma glucose and MUFA intake (Table in appendix 8)

Table 4.12 Correlation Coefficient between Fatty Acid Intake, Fasting Plasma Insulin, Fasting Plasma Glucose, and Lipid Profile in Abdominal Obese Adult Men

	Correlation coefficient (r)					
	Fasting plasma insulin		Fasting plasma glucose		Fasting plasma FFA	
	Unadjusted	Adjusted^	Unadjusted	Adjusted^	Unadjusted	Adjusted
Total fat intake (g)	0.117	0.129	0.153	0.167	-0.063	-0.076
SFA intake (g)	0.104	0.131	0.119	0.141	-0.084	-0.082
MUFA intake (g)	0.130	0.136	0.136	0.145	-0.062	-0.081
PUFA intake (g)	0.075	0.053	0.175*	0.164	0.035	0.009
Total fat intake (% of total calories)	-0.064	0.009	0.051	0.037	-0.022	-0.020
SFA intake (% of total calories)	-0.025	0.052	0.000	0.005	-0.080	-0.063
MUFA intake (% of total calories)	-0.049	-0.004	-0.026	-0.024	-0.010	0.006
PUFA intake (% of total calories)	-0.076	-0.094	0.101	0.046	0.180*	0.161
Ratio P(M)S (%)	-0.021	-0.051	0.048	0.026	0.120	0.108
Ratio ω-6 : ω-3 (g)	0.092	0.155	0.008	-0.030	0.015	0.014

*significant correlation at $p<0.05$, Pearson correlation test

^adjusted with age, WC, physical activity, smoking habit, alcohol use, fiber intake

PART 5
DISCUSSION

5.1 Fatty Acid and Other Intake Profiles

This study showed that total fat intake was up to 40% of the total calories among abdominal obese adult men and almost all the subject were in pre-obese (WHO classification, WHO 2004) or in obese I (Adult Asian Population classification, WHO 2004). Socio-economic of subjects showed that most came from middle class (income : IDR. 2.000.000-5.000.000/month). Unfavorable fat intake (based on Mc. Auley, 2006) were found among subjects for SFA (>8% of total calories intake) and MUFA (<10% of total calories). PUFA intake was 6,87% of total calories which meets Mc. Auley's (2006) recommendation (5%) but was still below the standard set by NCEP (2001) [60](>10%). Both this and previous study (Djuwita-Hatma, 2001) found that fatty acid intakes of Indonesian adults were not yet favorable compared to standard. The dietary fat results were higher than the previous study of lipid among four ethnic groups in Indonesia (Djuwita-Hatma, 2001) and study of determinants of blood pressure (Kamso, 2007 [61]) which may be due to more obese subjects in this study. Serum lipid profiles in this study were characterized by hypercholestrolemia, low HDL-cholesterol and hypertrigliceridemia among these abdominal obese adult men. This finding was similar with other study, except for HDL-cholesterol which did not met the criteria for high (desirable) and tryglyceride value which was not in normal range (Weta, 1999[62]). Other study (Djuwita-Hatma, 2001) found that while cholesterol total was in normal range, LDL-cholesterol and HDL-cholesterol did not meet the recommendation; and there was no available data on tryglyceride profile. Our study was more informative (available information on dietary intake and biochemichal indicators) and more specific on sample population (abdominal obese adult men) compared with those three previous studies. (Table 5.1).

Table 5.1 Profiles of Energy Intake, Macronutrient Intakes, Individual Fatty Acids Intake and Biochemichal Indicators in Indonesian Subjects with Different Characteristics

Characteristics	Weta et al, 1999	Kamso et al, 2007	Djuwita, 2000	Syauki et al, 2009
Sample size	222	556	1430	126
Study of population	elderly men and women aged 60 - ≥ 80 years old	elderly men and women aged 55-80 years old	adults aged 18->55 years old	abdominal obese men aged 30-50 years old
Dietary intakes :		Men :	Men :	
▪ Total calories (Kcal)	NA	1257	1151.5-1791.8	1879, 68 ± 411.25
▪ Carbohydrate (g)	NA	170	152.6-237.2	241.65 ± 53.73
▪ Protein (g)	NA	51.6	41.9-68.6	66.5 ± 16.55
▪ Fat :				
• Total fat (g)	NA	42.9	39.1-65.2	87.10 ± 39.77
• SFA (g)	NA	25.1	25.7-39.2	45.64 ± 22.77
• MUFA (g)	NA	7.8	6.1-12.0	19.54 ± 8.95
• PUFA :	NA	6.2	3.7-9.3	14.51 ± 7.52
▪ n-6 (g)	NA	NA	NA	16.77 ± 9.30 ^
▪ n-3 (g)	NA	NA	NA	1.00 (0.60-1.70) ^
Biochemichal profiles :				
▪ Cholesterol total (mg/dl)	233.62 ± 50.47	NA	170.84 - 194.15	210.33 ± 33.22
▪ LDL-cholesterol (mg/dl)	124.14 ± 40.66	NA	114.78 - 134.37	142.46 ± 31.6
▪ HDL-cholesterol (mg/dl)	62.00 (50.00- 84.40)	NA	33.77 - 45.85	41.53 ± 7.29
▪ Triglyceride (mg/dl)	133.13 ± 69.09	NA	NA	152.27 ± 60.18
▪ Fasting plasma glucose (mg/dl)	90.20 (79.6-107.2)	NA	NA	92.63 ± 9.15
▪ Fasting plasma insulin (u/L)	NA	NA	NA	7.63 ± 4.57
▪ FFA (mM)	NA	NA	NA	0.55 ± 0.17
▪ TC/HDLC	3.70 ± 0.56	NA	4.21 - 5.35	5.19 ± 1.08
▪ LDL/HDLC	2.08 ± 0.27	NA	2.67 -3.61	3.51 ± 0.90
▪ TG/HDLC	2.12 ± 0.37	NA	NA	3.31 (2.54-4.81)

Table 5.2 presents dietary fatty acids intake of the subjects in comparison with recommendation by PERKENI, NCEP, AHA, and ADA. Based on the recommendations of PERKENI, NCEP, AHA, and ADA, there were unfavorable fattty acid intake profiles in this study, except for PUFA in which majority of them met the criteria according to PERKENI standard. Based on PERKENI, 2006 and NCEP, 2001, it is confirmed that abdominal obese adult men had the same pattern of fatty acids profile. Only a few of them had total fat and SFA intake within the recommended level. Different results were shown in MUFA, PUFA, and fiber intakes. According to PERKENI, 2006 one third (31%) met the criteria for MUFA intake, 90.5% for PUFA intake and none for fiber intake. Meanwhile based on NCEP, 2001 none of them met the criteria for MUFA intake and only a few of them met the standard for PUFA and fiber intakes.

Table 5.2 Dietary Fatty Acids Intake of the Subjects in Comparison with Recommendation by PERKENI, NCEP, AHA and ADA

Dietary intake (% of total E)	Syauki et al, 2009	PERKENI, 2006		NCEP, 2001^		AHA, 2006^	ADA, 2005^
		standard	n (%)	standard	n (%)		
Total fat	41.23 ± 5.66	20-25	1 (0.8)	≤ 25-35	13 (10.3)	25-35	≤ 25-35
SFA	21.51 ± 4.50	< 7	1 (0.8)	< 7	1 (0.8)	< 7	< 7
MUFA	9.32 ± 1.71	≥ 10	39 (31)	> 20	0 (0)	NA	> 20
PUFA	6.87 ± 2.02	< 10	114 (90.5)	> 10	12 (9.5)	NA	> 10
n-6	8.00± 3.01	NA	NA	NA	NA	NA	NA
n-3	0.54 (0.35 - 0.76)	NA	NA	NA	NA	NA	NA
Fiber (g)	10 (8-12)	25	0 (0)	20-30	3 (2.4)	NA	20-30

^NCEP : National Cholesterol Education Program
AHA[65] : American Heart Association
ADA[66] : American Diabetes Association

Fat intake generally should not exceed 30% of energy. Most importantly, saturated fats, because of their atherogenic potential, should be held at a maximum of 10% of energy needs. Polyunsaturates, with their tendency to lower HDL-cholesterol values and their susceptibility to oxidation, should also be held under 10%. However, up to 35% of energy from fat can be used for non obese individuals with acceptable serum triglyceride values if the additional fat comes from monounsaturated sources such as canola or olive oils [25]. Diets in affluents countries, however, are loaded with fat (up to 150 gr per day), mostly saturated, giving up to 40 per cent of the daily total calories. Much of this fat is of animal origin or is partially hydrogenated plant. Its essential fatty acid content is nevertheless satisfactory [45]. In this study, fried food, coconut milk and peanut were food commonly eaten among the subject as total fat intake. Almost two third of them were come from MUFA and PUFA intake. Monounsaturated (MUFA) and polyunsaturated fatty acids (PUFA) act as protective factors for heart health [46]. Short-term intervention studies in healthy volunteers have shown that the isocaloric substitution of MUFA for SFA, or even substituting MUFA for carbohydrates, can have positive effects on insulin sensitivity [7]. Limited experimental evidence suggests that MUFA diets favorably influence blood pressure, coagulation, endothelial activation, inflammation, and thermogenic capacity. Energy-controlled high-MUFA diets do not promote weight gain and more acceptable than low-fat diets for weight loss in obese subjects [32]. Short-term intervention studies in healthy adults on PUFA as isocaloric subsitution of saturated fatty acids showed the effect of lowering of LDL cholesterol when compared to diet rich in saturated fatty acids. Long-term intervention trials of PUFA have not been conducted, and also diets enriched in PUFA have not consistently been shown to improve metabolic disorders, such as insulin sensitivity; thus intake of PUFA should be limited to ≤ 10% of total calories. This study found that only a few of food from total fat intake were came from omega-6 PUFA and none of them were came from omega-3 MUFA. Omega-6 and omega-3 as in the family of PUFA, well known as "good fats" were two major types of

PUFA in the diet. Evidence suggests that omega-3 fatty acids may help prevent heart disease with moderate consumption to replace saturated fatty acids, omega-6 and omega-3 fatty acids were well recognized as fats that can help reduce both blood cholesterol levels and risk of heart disease [7,46].

Figure of P(M)S ratio in this study was better than previous study (Djuwita-Hatma, 2001) (0.75 vs 0.15 - 0.25). P(M)S ratio should be about 1.0 to lowering the saturated fat in the diet then it will reduce the cholesterol levels, which will reduce the risk of coronary heart disease [45]. In this study, the ratio of omega-6/omega-3 fatty acids was 14:1 and is suitable with estimation of today's diet (10:1). This profile was higher than the suggested ratio, ranging between 2:1 and 5:1 [46]. In this study, 28% of subjects had 1x/week of meat intake and 23% of them consumed fish 3x/week. The dietary fats rich in polyunsaturated fatty acids are the ones likely to be rich in essential fatty acids. High fat fish and marine mammals contain larger amounts of long chain n-3 fatty acid, EPA, and DHA. The longer-chain n-6 fatty acids, such as arachidonic acid, are found in foods of animal origin, including organ meats [44]. Fish are classified broadly into "lean" fish that store their reserve fats as triacylglycerols in the liver (e.g cod),or "fatty" or "oily"fish" (e.g mackerel, herring) where the fat is located in the flesh. The oils have a high content of fatty acids with 20 or more carbon atoms which are either predominantly MUFA or PUFA [65].

Fiber intake in this study population was lower than the recommendation. While most consumed vegetables daily, this was not the case for fruits. Researchers classify dietary fibers by their ability to dissolve in water as soluble fiber and insoluble fiber. Oat bran, legumes, soybean fiber, and some fruits and vegetables are rich in soluble fiber. Soluble fibe benefits both glycaemic and lipid metabolism and appears to reduce fasting or basal glucose and lowering blood cholesterol levels. Improved insulin sensitivity is postulated to be the mechanism by which soluble fiber improves hyperglycaemia [41].

5.2 Lifestyle and Health-Related Practices (Physical Activity, Smoking Habit, Drinking Alcohol)

Physical activity among subjects in this study was mostly at moderate stages (MET value per week). Few subjects had daily walking for 15 minutes and medium intensity of sports 3-7 days/week. Multiple studies have demonstrated that routine physical activity was critical to long-term maintenance of weight loss. As a complement to energy restriction, increased physical activity provides the advantages of improving cardiovascular conditioning and insulin sensitivity. Kelley and Goodpaster, (1999) also concluded that physical activity can reduce insulin resistance and improve glucose intolerance among obese individuals (Kelly, 2000, p.114). Exercise improves insulin sensitivity in skeletal muscles and fat tissue, reducing both fasting blood sugar and insulin levels. Horber et al, (1996) demonstrate that consistent exercise training, even without accompanying improvements in body composition, improve peripheral insulin activity in subjects with impaired glucose tolerance (Kelly, 2000, p.114). Increased physical activity can be achieved with a combination of regular exercise several times weekly plus daily "step-losing" activities, such as walking or climbing stairs instead of driving or using the elevator. For obese persons, aerobic exercises such as walking, swimming, bicycling, and low-impact dance and exercise classes were recommended to minimize damage to weight-bearing joints [12,66].

Results in this study showed that majority of subjects did not smoke every day. Many research showed that chronic cigarette smokers were likely to be insulin resistant, hyperinsulinemic, and dyslipidemic when compared with matched groups of non-smokers. Facchini et al, 1992 demonstrated that chronic cigarette smoking markedly aggravated insulin resistance in patients with type-2 diabetes melitus (Kelly, 2000, p.114). This situation can be caused by nicotine. Eliasson et al, 1996 in their findings suggested that long-term use of nicotine-containing chewing gum was associated with insulin resistance, and the degree of insulin resistance was correlated to the extent of nicotine used (Kelly, 2000,

p.114). Other finding (Assali,1999) also reported that nicotine replacement therapy resulted in a decrease in insulin sensitivity (Kelly, 2000, p.114) [66].

In this study, most of subjects did not consume alcohol in the last one year. Individuals with diabetes should have restricted on or abstinence from alcohol to prevent from hypoglycemic unawareness, neuropathy, poor control of blood glucose or blood lipids. Alcohol adds calorie without nutritional benefit and has been shown to supplement rather than displace other calories. Suter et al, 1992 and Flatt, 1992 demonstrated that, whereas carbohydrate and protein oxidation was mostly unaffected by the ingestion and metabolism of alcohol, fat oxidation was decreased by about 5% (Chalmers, 2005, p. 624). Ingestion of alcohol increases the synthesis of lipoprotein, especially very low density lipoprotein hypertriglyceridemia, and because alcohol has the potential to raise hypertriglyceride levels, alcohols ingestion should be discouraged [40].

Other lifestyle that now has been thought to influence insulin level was coffee. Nowadays, coffee was a widely consumed beverage around the world, including Indonesia. One constituent of coffee was caffeine, but caffeine in the coffee was different with caffeine alone as alkaloid. Battram and colleagues (2006) study showed that caffeine elevated blood glucose about 160%, compared to caffeinated coffee, and about 320% compared to decaffeinated coffee (Tunnicliffe and Shearer, 2008). Insulin was also significantly higher with alkaloid caffeine than either placebo or decaffeinated coffee, but not caffeinated coffe. Epidemiological data showed that chronic coffee consumption in dose-responsive and at the highest level of consumption reduces incidence of type-2 diabetes melitus by 50%. It is recommended that individual at risk for metabolic syndrome, glucose intolerance, and T2DM consumed decaffeinated coffee [67]. Coffe drinkers among subjects was 31.75%, which is caffeinated coffee.

5.3 Insulin Profiles and Insulin Resistance

Insulin levels among abdominal obese adult men in this study showed the desirable value. There is evidence suggesting that an early abnormality in type-2 diabetes melitus was hyperinsulinemia, which is associated with insulin resistance. Initially, these patients were similar to obese non-diabetic adults, in that they were hyperinsulinemic but usually have normal or near normal glucose tolerance [27]. Based on clinical experiences since 1997, Tjokroprawiro (2001) has hypothesized the link between insulin resistance-obesity and T2DM, and termed as clinical classification of insulin resistance-linked obesity and T2DM. There were four grades of insulin resistance classification. Patients in grade I still have normal insulin blood levels whereas insulin resistance may be detected by HOMA-IR. Grade I usually happens in patients with BMI>30 kg/m^2 or visceral obesity [10]. Grade II can be subdivided into A, B and C, while grade III, and IV can be subdivided into A and B. Grade II can be found in patient with hyperinsulinemia with various glucose profile (normal, intolerance, and T2DM) with or without visceral obesity. Grade III usually happens in patient with normo/low insulinemia plus T2DM with visceral obesity. Grade IV was found in patient with hypoinsulinemia and this patient become totally insulin dependent [10]. In this study, most of subjects (57.9%) can be classified into grade I characterized by desirable insulin level (normoinsulinemia) and visceral obesity.

5.4 Correlation between Fatty Acid Intake and Others Factors with Insulin Level

Result of this study showed that there is no correlation between intake of different fatty acids and insulin levels. This finding was not in line with finding by Lovejoy et al (2001)[68], which found significant correlations between plasma insulin/glucose and total dietary fat in grams, dietary fat, MUFA and SFA as a percentage of total calories and dietary cholesterol. Previous study (Lovejoy et al, 2001) was conducted to a group of 38 men and women with wide range of glucose tolerance. Other study done by Harding et al (2001) [69] showed that there

was association between total fat and fasting insulin after adjusted with the energy among 815 non-diabetic men and women aged 30-71 year. This association was not significantly after adjustement with BMI, WHR, age, sex, family history of DM, smoking and alcohol use. Besides fat intake, other factors play a role on fasting plasma insulin. This study found that waist circumference and BMI were significantly correlated with fasting plasma insulin. In Cavallo-Perin et al study (1985), weight loses and a decrease in the waist-hip ratio amongst obese men were closely related with improved insulin sensitivity (Kelly, 2000, p.112). In this study, waist circumference was negatively correlated with odds of having insulin resistance (Table in appendix 8) which might be explained by the negative correlation between waist circumference and percentage of total fat and percentage of SFA intake (due to higher energy intake in those with higher waist circumference). This finding was different with study by Krachler et al (2006, p.9) [70] showing an association between fat intake and abdominal obesity whereby increased consumption on hamburger and french fried potatoes, which is generally considered as markers of a diet high in fatty acids, was associated with an increase in waist circumference. Our study found that peanut was the main source of total PUFA and MUFA intake among the subjects. Meanwhile coconut milk in vegetable dish *(lodeh)*, was the main source of SFA intake. Omega-3 and omega-6 PUFA intake were found in peanut and in several marine fish. With this intake profile, peanut and marine fish were foods which can acts as protective factor among the subjects.

The previous two studies (Lovejoy et al, 2001 and Harding et al, 2001) had wide range of glucose tolerance. In Harding et al (2001) study, subjects consisted of non diabetic group having normal glucose, impaired glucose tolerance and non diagnostic diabetes melitus. Meanwhile in our study, majority of subjects (81.7%) were in normal glucose. Nevertheless, positive correlation between fasting plasma glucose and fasting plasma insulin was found in our study. Insulin was hormonal products of glucose production. Moderately increased fasting glycemia in some nondiabetics and in mildly hyperglycemic

noninsulin-dependent diabetics appeared to be primarily a result of decreased insulin action [15].

Other factors that contributed to insulin level was FFA plasma. In our study, FFA plasma was positively correlated with fasting plasma glucose. In the study of Bogardus et al (1984, p.1238) [71], FFA was correlated with fasting plasma glucose in non diabetic subjects. Circulating FFAs were elevated in many insulin-resistant states and have been suggested to play a central role in the pathogenesis of the insulin resistance. FFAs inhibit insulin-stimulated glucose uptake at the level of glucose transport and/or phosphorylation, inhibit insulin-stimulated glycogen synthesis and inhibit insulin-stimulated glucose oxidation. FFAs might have a special role in the insulin resistance associated with central obesity. A common link between increased levels of FFAs and the insulin resistance in type-2 diabetes, obesity and syndrome X could be accumulation of triglycerides in muscle. Factors leading to the accumulation of triglycerides is derived from elevated levels of both circulating FFAs and triglycerides and is also the result of reduced muscle fat oxidation [71].

There was positive correlation between PUFA intake (percentage of total calories) and fasting plasma FFA in our findings. Kiyonuri et al (2003, p.3643) study found positive correlations between plasma of EPA, DHA and (n-3) high unsaturated fatty acid (HUFA) with dietary intakes of marine oil. The limitation of fasting plasma FFA is that it is not specific biomarker of individual fatty acid intake. FFA released into the circulation were only free in the sense that they were non-esterified. FFA in circulations act as lipid transport system [42]. Nevertheless, biomarkers of fatty acid intake offer an alternative to dietary assessment because they reflect actual rather than reported [22].

In this study, intake of SFA is higher (21% of total calories) and omega-3 PUFA (alpha-linolenic acid) and omega-6 PUFA (linoleic acid) were lower than recommended (IOM) (0.9 g/day vs 1.6 g/day and 15.37 g/day vs 17 g/day). Nutritional factors may play a critical role in controlling insulin secretion. Long-

chain fatty acids increase insulin secretion of islet cells in vivo and in vitro in the short term. This situation is due to a stimulation of insulin release by long-chain fatty acid CoA. In contrast, incubation of islet cells in the presence of elevated levels of fatty acid over several days impairs insulin release. This effect is concomitant with acumulation of triglyceride in the islet cells. It is therefore possible that energy and fat overfeeding decrease insulin secretion through a lipotoxicity on islet beta cells [72]. Our study found that there is positive correlation between fasting plasma insulin and triglyceride, contrastly with omega-3 and omega-6 PUFA intake (as gram or percentage of total calories) (Table in appendix 8).

In multiple logistic regression, waist circumference acts as protective factor of having insulin resistance. This mechanism can be explained by the negative correlation found between SFA intake (percentage of total calories) and waist circumference; i.e. since people with more central obesity consumed more energy. Small range of waist circumference however is limitation in this study. Fasting plasma glucose acts as protective factor of having insulin resistance. In our findings, there was positive correlation between fasting plasma insulin and fasting plasma glucose (r_p=0.243, $p<0.01$), but negatively correlated with fasting plasma FFA (although not significantly). Meanwhile, fasting plasma FFA was positively correlated with fasting plasma glucose (r_p=0.215, $p<0.05$). This findings indicated that fasting FFA plasma had two roles among abdominal obese such as increasing gluconeogenesis and suppresion secretion of insulin, although the latter was not significant in our study. Other study, preferably with wider data distribution, is needed to confirm this findings.

There were several mechanisms for fatty acid intake induced insulin resistance among abdominal obese men. Our study showed that in abdominal-obese subjects there was no relationship between intakes of different fatty acids and plasma insulin; however the effect may be mediated through its indirect effect with FPG. Further study is needed to have clear understanding of the relationship

between intakes of different fatty acids and plasma insulin preferably in subjects with more diverse obesity and insulin profiles i.e. including obese and non-obese subjects as well as subjects with and without insulin resistance.

PART 6
CONCLUSIONS AND RECOMMENDATIONS

6.1 CONCLUSIONS

1. Fatty acid intake (% of total calories) among abdominal obese adult men were unfavorable when compared to existing recommendations (PERKENI, NCEP, AHA and ADA), except for PUFA. Mean intake (as percentage of total calories) of fat was 41.23%, SFA 21.51%, MUFA 9.32% and PUFA 6.87% (within recommended range of PERKENI). Meanwhile omega-3 and omega-6 were did not meet the standard based on IOM, 2002 (1.0 g vs 1.6 g and 16.77 g vs 17 g).

   Lipid profiles of study population identified hypercholesterolemia and hypertriglyceridemia amongst the subject, and HDL-cholesterol did not meet the criteria for desirable level (high).

2. Among these abdominal obese adult men with no diabetic and other metabolic abnormalities, the fasting plasma insulin was in normal range (mean: 7.63 u/L). Mean fasting plasma glucose as one of the exclusion criteria was 92.63 mg/dl.

3. Most of respondents (59.3%) had moderate physical activity, but only a few of them (11.9% and 13.5%) had daily walking of 15 minutes each and moderate intensity of sport of 3-7 days/week.

   Fibre intake as one influencing factors was also lower value than the NCEP recommendations (10 g vs 20-30 g).

4. There was no association between intakes of fatty acids (SFA, MUFA, PUFA, omega 3 and omega 6) and plasma insulin in this study. However, PUFA intake was positively correlated with plasma FFA (as percentage of total calories) and plasma glucose (as gram intake); and the latter was positively correlated with insulin level

6.2 RECOMMENDATIONS

1. Further study needs to be conducted to have clear understanding of the relationship between intake of different fatty acids and insulin level preferably in subjects with more diverse obesity and insulin profile i.e including obese and non-obese subjects as well as subjects with and without insulin resistance.

2. Intake of different fatty acids among abdominal obese adult men in Indonesia should be modified in particular intake of MUFA. Lipid profile indicated hypercholesterolemia and hypertriglyceridemia and the risk of having coronary heart disease. Recommendations for dietary fat should not focus only in quantity but also in quality/composition of fatty acids. This recommendation has to be translated into food-based messages. This may include, based on this study, messages to reduce fat intake from animal sources, coconut milk and frying (palm) oil, to minimise obvious fat from meat as much as possible (e.g. skin-less poultry), to increase intake of unsaturated fatty acid and omega-3/omega-6 sources (e.g. peanut, soybean, marine fish) and to increase fiber intake (i.e. by increasing fruit intake and ensuring daily consumption of vegetables).

3. In addition to message on fat intake, physical activity should be encouraged especially among abdominal obese person, as this factor was important in the weight loss management among abdominal obese person.

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Appendix 1

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MANUSCRIPT FOR PUBLICATION

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RELATIONSHIP BETWEEN INTAKES OF DIFFERENT FATTY ACIDS AND INSULIN LEVEL IN ABDOMINAL OBESE ADULT MEN

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Manuscript

ABSTRACT Obesity is known as the major global health problems, including in Indonesia.This situation is associated with nutritional transitional leading to the prevailing degenerative diseases. Waist circumference is strong predictor of insulin resistance, an initial phase for development of type 2 diabetes melitus. High intake of SFA is contributed to insulin resistance. Data on the relations between intake of fatty acids and insulin resistance in Indonesia are very limited. A cross-sectional study was undertaken to examine the association between intake of different fatty acids and insulin level in abdominal obese adult men in Jakarta. Dietary fatty acids was obtained through validated fat SQ-FFQ to 126 men with abdominal obesity aged 30-50, who pass the screening procedure through clinical and blood asssessment. Structured interview, anthropomethric assessments and biochemichal assessments were used in collecting the data. Intake of total fat, SFA, MUFA and PUFA (% of total calories) were found higher than that of the PERKENI/NCEP/AHA/ADA recommendations (41.23% , 21.51% and 9.32%), except PUFA intake based on PERKENI (6.87%). Intake of omega-3 and omega-6 PUFA did not meet the requirement suggested by IOM. Hypercholesterolemia and hypertriglyceridemia were found among subjects. Mean fasting plasma insulin was found within desirable range (7.63 u/L). There is no correlation between intakes of different fatty acids and insulin level in abdominal obese adult men, but FFA plasma were positively correlated with PUFA intake (% of total calories) (r_p=0.190, $p<0.05$) and fasting plasma glucose (r_p=0.193, $p<0.05$). Further study need to be conducted to have clearly understanding of the relationship between intake of different fatty acids and insulin level between abdominal obese and non-abdominal obese or insulin resistance and non insulin resistance.

KEYWORD : fatty acids intake, insulin, abdominal obese

BACKGROUND Increasing prevalence of diabetes melitus type 2 worldwide and national (the second cause of death for > 15 year), emphazise the importance to identify pre-diabetic condition. This may subclinical appear in a person. Insulin resistance believed as the initial phase of type 2 diabetes melitus and other metabolic abnormalities such as hypertension, dyslipidemia and hyperuricemia [1-3]. The cause of insulin resistance remains unclear, but it believed that overweight,

physically inactive and intake of excessive amount of fat are modifiable (nutritional) determinants of insulin resistance and hyperinsulinemia. Other determinant that is believed as good predictor of insulin sensitivity is waist circumference. Women and men are different in their distribution of fat, which is men were likely to have abdominal fat. In Indonesia, prevalence of central/abdominal obesity starts to increase in the 35-44 year age group (24.4%) and was the highest in 45-54 year age group (26.1%) as the data from Ministry of Health [2,4,5].

Changing lifestyle including food pattern can be contributable factors of the increasing prevalence, include modern Western diets which is high in fat especially saturated fatty acids (SFA) and can promote excessive energy intake. Previous study of nutrient intake and their relations to lipid profiles in four ethnics in Indonesia showed that the mean of the contribution of total fat to energy intake ranged from 29.5% to 35.8%. The percentage of SFA to total calories was also high around 20%. Meanwhile, the percentage of PUFA ranged from 2.6% to 4.6%. The percentage of MUFA among four ethnic groups were still inadequate based on the recommended value (2.6%-6.1%). The P/S ratio also showed lower value (0.15-0.25) [6-7].

The increasing prevalence of diabetes mellitus and obesity in Indonesia also followed by unfavorable intake of different fatty acids can lead to insulin resistance. In order to obtain data about associaton between various intake of different fatty acids with the high prevalence of T2DM and obesity in Indonesia, a cross sectional study was conducted to examine the relationship between intakes of different fatty acids and insulin level in abdominal obese adult men in Jakarta as capital city of Indonesia.

SUBJECT AND METHODS

Subject. Subjects were recruited from government instution, private office in Central Jakarta and East Jakarta. The Committee of The Medical Research Ethics of the Faculty of Medicine, University of Indonesia approved this study and all subject gave informed consent before taking the data. Subject were excluded if they did not meet the inclusion criterias such as man aged 30- 50 years old, had

waist circumference ≥ 90 cm and meet the exclusion criterias such as FPG ≥ 126 mg/dl, SGPT ≥ 110 U/L, creatinin serum ≥ 1.6 mg/dl, blood pressure systolic ≥ 140 mmHg and diastolic ≥ 90 mmHg, consume hypolipidemia medicine within one month. For the reliable result of biochemichal assessment, subject were not fever at the time of blood collection (body temperature ≤ 37.5 °C). A hundred and sixty eight subjects participated in this study, but only 126 meet the inclusion and exclusion criteria.

Methods. This study consisted of several data collection procedures. Before blood assessment, subject had to follow screening procedure to minimize the drop-out of the subject. The screening procedure consisted of taking medical history, anthropomethric assessment for body weight using calibrated electric SECA model 872, height using microtoise, waist circumference using waist-watcher tape measure, blood pressure using Riester mercury sphygmomanometer and Litmann sthetoscope and body temperature using mercury termometer. All the screening procedures were done by train enumerators. Weighing was done to subject with minimal clothing and no heavy things carried in body as well as when do height assessment with no head accesories and stand erect in correct position (Frankfurt plane position). Waist circumference was measured in the fasting condition or one hour post prandial and wear minimized clothing to allow the tape to be correctly positioned. To perform the assessment at the natural waist, the lowest rib margin (the tenth rib) was first located then palpated the iliac crest in the midaxillary line. A waist-watcher tape was applied horizontally midway between the lowest rib margin and the lateral iliac crest made into a complete loop and fitted firmly around the waist by a spring mechanism, which is controlled by a pushbutton. Blood pressure assessment were done by trained person (medical doctor and health workers). Thermometer was putted in axilla at ipsilateral of the immobilized elbow for 5-10 minutes [8-10].

Analysis of fasting plasma insulin, fasting plasma glucose, fasting plasma FFA. Blood samples were taken from fasting subject. After an overnight fast of at least 10 hours, venous blood was collected into vacutainers containing disodium EDTA. Fasting plasma glucose was quantitatively analyzed by using standard procedure utilizing The ADVIA Chemistry Systems Glucose Hexokinase II (GLUH)

while fasting plasma glucose was quantitatively analyzed by using standard procedure utilizing IMMULITE/IMMULITE 1000 Insulin. Fasting plasma FFA was a biomarker of dietary fat. This was quantitatively analyzed by using standard procedure based on calorimetri. All blood assessment were done at Prodia Laboratory.

Assessment of dietary intake. Dietary fat assessment was conducted using validated SQ-FFQ and other nutrients (energy, protein, carbohydrate, fiber) was assessed by using 3-d non-consecutive recall. The development and validation of the food list rich in fat and the fat SQ-FFQ were done before this fat SQ-FFQ was used. Analysis for relatively validation of SQ-FFQ used three statistical tests, such as mean difference Paired t-test, Spearman's correlation test and cross-classification. Fat SQ-FFQ was valid, indicated by no significance difference of mean, relatively moderate coefficient correlation and relatively good cross-classification. Dietary fat assessment was done on March - May 2009 using validated SQ-FFQ by three trained enumerators with food picture which came from 2-3 times sampling of food and food models. Subjects were asked to recall their frequency and portion size of consumption of food items over preceding 1 month. Intake of carbohydrate and fiber, other macronutrient was assesed by using 3-day non-consecutive 24 hr recall.

Analysis of other determinants of insulin levels. STEPS and GPAQ questionnaire (interview-administered) were adopted to use as tools for obtaining other determinants of insulin levels such as smoking habit, alcohol consumption, fruit and vegetables consumption and physical activity [11].

Statistical analysis. Data entry and statistical analysis was done using SPSS program version 15.Total physical activity was analyzed as MET value/week and categorized as high, moderate and low based on physical activity cut-off value. Descriptive statistics was done to describe frequency, mean, median, standard deviation and 25th-75th percentile value. Kolmogorov-Smirnov test was used to test the normality of sample distribution. Pearson's correlation test was administered to find out the correlation between intake of different fatty acids, fasting plasma insulin, fasting plasma glucose, FFA plasma and partial correlation for controlling confounder with age (year), WC (cm), physical activity (MET value/week), smoking habit (Y/N), alcohol use (Y/N), fiber intake (g). (SPSS version 15).

Multiple logistic regression was used to examine the trend of having insulin resistance with other confounding factors such as age (year), waist circumference (cm), total physical activity (high, moderate, low of MET value/week), FPG (mg/dl), FFA (mM/l), smoking habit (Y/N), alcohol use (Y/N), fiber intake (favor of g/unfavor of g),.

RESULTS

Table. 1 presents socio-economic-demographic characteristics of the study population. This study involved 126 subject, with proportion of group aged 30-39 and 40-50 (51.6% vs 48.4%). Most of subject were Javanese (63.5%) . Almost half of them were diploma/bachelor degree (46.8%) and majority worked as government employee (68.3%).

Most of subjects (51.2%) had income between IDR 2.000.000- IDR 5.000.000 per month. Expenditures between food and non food items per month were almost equal as individual (IDR 400.000 vs IDR 500.000 per month) but as family expenditure, food expenditure was almost twice than the non food (IDR 1.000.000 vs IDR 550.000 per month).

Table. 1 Socio-economic-demographic characteristics of the study population

Characteristics	Total	
	n	%
Subject	126	100
Age group (year):		
• 30-39	65	51.6
• 40-50	61	48.4
Ethinicity		
• Javanese	80	63.5
• Betawinese	7	5.6
• Sundanese	13	10.3
• Sumatran	20	15.9
• Celebesian	4	3.2
• Balinese	2	1.6
Education		
• 9-year schooling	3	2.4
• Senior high school	30	23.8
• Diploma/bachelor degree	59	46.8
• Master degree	34	27
Occupation		
• Civil workers	86	68.3
• Private employee	18	14.3
• Policeman	18	14.3
• Others	4	3.2

Table. 1 (continued)

Characteristics	Total	
	n	%
Subject	126	100
Income (IDR/month)	n=125	
▪ >10.000.000	19	15.2
▪ 5.000.000- 10.000.000	24	19.2
▪ 2.000.000-< 5.000.000	64	51.2
▪ 1.000.000-< 2.000.000	16	12.8
▪ <1.000.000	2	1.6

Table. 2 lists anthropomethric and lifestyle-health related characteristics of the study population. WHO classification of BMI placed most (61.1%) of subject as pre-obese while the Asian adult population classification put most respondent (72.2%) as obese I. Most of subject (60.3%) did not smoke every day and majority of subject (79.4%) did not drink alcohol in past one year. Consumption on fruit was relatively low which is showed by fifty three of subject (42.1%) consume fruit every day. Contrastly, seventy two of subject (57.1%) eat vegetable every day. Most of subject (59.3%) were in moderate physical activity.

Table. 2 Anthropomethric-health related and lifestyle-healt related characteristics of the study population

Characteristics	Total	
	n	%
Subject	126	100
Anthropomethric-health related		
Waist circumference (cm)	97 (92.75-101)	
BMI		
WHO classification		
▪ Overweight (kg/m^2) :		
o 25.00-29.99 (pre-obese)	77	61.1
o 30.00-34.99 (obese class I)	40	31.7
o 35.00-39.99 (obese class II)	7	5.6
o ≥ 40 (obese class III)	2	1.
Adult Asian population classification		
▪ 23.00-27.49 (overweight)	35	27.8
▪ ≥27.5 (obese I)	91	72.2
Lifestyle-health related		
Smoking every day	50	39.7
Drink alcohol past one year	26	20.6
Fruit consumption (days/week)		
▪ 7 (every day)	53	42.1
▪ 0-6 (not every day)	73	57.9

Table. 2 (continued)

Characteristics	Total	
	n	%
Subject	126	100
Vegetable consumption (days/week)		
▪ 7 (every day)	72	57.1
▪ 0-6 (not every day)	54	42.9
Physical activity minutes per week (MET value) :		
▪ High	8	6.3
▪ Moderate	75	59.3
▪ Low	43	34.1

Dietary intake profile of the study population was present in table 3. Differently with intake of total fat, SFA, MUFA and PUFA as percentage of total calories, this study had higher figure than the previous (Djuwita-Hatma, 2001) (41.23% vs 29.5%-35.8%, 21.51% vs 20%, 9.32% vs 2.6%-6.1%; 6.87% vs 2.6%-4.6%). Ratio of P(M)S also showed higher figure than previous study (0.75 vs 0.15-0.25).Intake omega-3 and omega-6 PUFA were not meet the standard based on IOM, 2002 (1.0 g vs 1.6 g and 16.77 g vs 17 g) (Table 3). Inversely with fiber intake, this study showed lower figure than the PERKENI and NCEP recommendation (10 g vs 25 g and 30 g).

Table. 3 Dietary intake profile of the study population

Characteristics	Total	
	n	% E
Subject	126	
Fat intake (SQ-FFQ)		
▪ Total fat (g)	87.10 ± 39.77	41.23 ± 5.66
▪ SFA (g)	45.64 ± 22.77	21.51 ± 4.50
▪ MUFA (g)	19.54 ± 8.95	9.32 ± 1.71
▪ PUFA (g)	14.51 ± 7.52	6.87 ± 2.02
• n-6 (g)	16.77 ± 9.30	8.00± 3.01
• n-3 (g)	1.00 (0.60-1.70)	0.54 (0.35 - 0.76)
Ratio		
▪ P(M)S (%)	0.75 (0.64 - 0.88)	
▪ n-6 :n-3 (%)	14.27 (10.90 - 21.42)	
Others intake (3d-recall)		
▪ Energy intake (Kcal)	1879, 68 ± 411.25	
▪ Protein intake (g)	66.5 ± 16.55	14.21 ± 2.1
▪ Fat intake (g)	71.02 ±24.41	33.57 ± 6.65
▪ Carbohydrate intake (g)	241.65 ± 53.73	51.75 ± 6.46
▪ Fiber intake (g)	10 (8-12)	

Laboratory profile (Table 4) showed that fasting plasma insulin were in desirable range (<27 u/L), but the fatty acid profile indicated hypercholesterolemia and hypertrigliseridemia.This conditions refer to the mean cholesterol total was 210.33 mg/dl, cholesterol LDL was 142.46 mg/dl, triglyceride was 152.27 mg/dl (normal value, cholesterol total < 200 mg/dl, cholesterol LDL < 100 mg/dl, triglyceride <150 mg/dl). Cholesterol HDL in this study was not meet the criteria of NCEP, 2001 for high (desirable) 41.53 mg/dl vs ≥ 60 mg/dl. Total cholesterol to HDL cholesterol was 5.19, indicated there was high risk of coronary heart disease. Fasting plasma glucose were positively correlated with fasting plasma insulin and fasting plasma FFA (r=0.243, p<0.01, Pearson correlation test; r=0.215, p<0.05, Pearson correlation test).

Table. 4 Laboratory profile of the study population

Characteristics	Total
	n
Subject	126
Fasting plasma insulin (u/L)	7.63 ± 4.57
Fasting plasma glucose (mg/dl)	92.63 ± 9.15
Fasting plasma FFA (mM/l)	0.55 ± 0.17
Cholesterol total (mg/dl)	210.33 ± 33.22
Cholesterol LDL direk (mg/dl)	142.46 ± 31.6
Cholesterol HDL (mg/dl)	41.53 ± 7.29
Triglyceride (mg/dl)	152.27 ± 60.18
Total chol/HDLC	5.19 ± 1.08
LDLC/HDLC	3.51 ± 0.90
TG/HDLC	3.31 (2.54-4.81)

Results in this study showed that there were no correlations between intake of individual fatty acids and fasting plasma insulin (Table. 5). PUFA intake was positively correlated with fasting plasma glucose (unadjusted) as gram and with fasting plasma FFA (unadjusted) as percentage of total calories (r=0.175, p<0.05, Pearson correlation test and r=0.180, p<0.05, Pearson correlation test).

Table 5. Correlation coefficient between fatty acid intake with fasting plasma insulin, fasting plasma glucose and fasting plasma FFA in abdominal obese adult men

	Correlation coefficient (r)					
	Fasting plasma insulin		Fasting plasma glucose		Fasting plasma FFA	
	Unadjusted	Adjusted^	Unadjusted	Adjusted^	Unadjusted	Adjusted^
Total fat intake (g)	0.117	0.129	0.153	0.167	-0.063	-0.076
SFA intake (g)	0.104	0.131	0.119	0.141	-0.084	-0.082
MUFA intake (g)	0.130	0.136	0.136	0.145	-0.062	-0.081
PUFA intake (g)	0.075	0.053	0.175*	0.164	0.035	0.009
Total fat intake (% of total calories)	-0.064	0.009	0.051	0.037	-0.022	-0.020
SFA intake (% of total calories)	-0.025	0.052	0.000	0.005	-0.080	-0.063
MUFA intake (% of total calories)	-0.049	-0.004	-0.026	-0.024	-0.010	0.006
PUFA intake (% of total calories)	-0.076	-0.094	0.101	0.046	0.180*	0.161
Ratio P(M)S (%)	-0.021	-0.051	0.048	0.026	0.120	0.108
Ratio n6 : n3 (g)	0.092	0.155	0.008	-0.030	0.015	0.014

*significant correlation at p<0.05, Pearson correlation test

^adjusted with age,WC, physical activity, smoking habit, alcohol use,fibre intake

A multiple logistic regression analyses were performed to examine the odds ratio having insulin resistance. This odds ratio was not conclusive in this study, but seen as the trend of having insulin resistance. Prevalence of insulin resistance (IR) among subject was 32.5%. Results of multiple logistic regression showed that in predicting insulin resistance, there three determinants were significantly associated with insulin resistance , i.e waist circumference, fasting plasma glucose and MUFA intake (percentage of total calories).

DISCUSSION

Result of this study showed that there is no correlation between intake of different fatty acids and insulin levels. This finding was not in line with finding by Lovejoy et al (2001)[12], which found significant correlations between plasma insulin/glucose and total dietary fat in grams, dietary fat, MUFA and SFA as a percentage of total calories and dietary cholesterol. Previous study (Lovejoy et al, 2001) was conducted to a group of 38 men and women with wide range of glucose tolerance. Other study done by Harding et al (2001) [13] showed that there was association between total fat and fasting insulin after adjusted with the energy among 815 non-diabetic men and women aged 30-71 year. This association was not significantly after adjustement with BMI, WHR, age, sex, family history of DM,

smoking and alcohol use. Besides fat intake, other factors play a role on fasting plasma insulin. This study found that waist circumference and BMI were significantly correlated with fasting plasma insulin.. In Cavallo-Perin et al study (1985), weight loses and a decrease in the waist-hip ratio amongst obese men were closely related with improved insulin sensitivity [13]. In this study waist circumference was negatively correlated with odds of having insulin resistance *(data not shown)* which might be explained by the negative correlation between waist circumference and percentage of total fat and percentage of SFA intake (due to higher energy intake in those with higher waist circumference). This finding was different with study by Krachler et al (2006) [14] showing an association between fat intake and abdominal obesity whereby increased consumption on hamburger and french fried potatoes, which is generally considered as markers of a diet high in fatty acids, was associated with an increase in waist circumference. Our study found that peanut was the main source of total PUFA and MUFA intake among the subjects. Meanwhile coconut milk in vegetable dish *(lodeh)*, was the main source of SFA intake. Omega-3 and omega-6 PUFA intake were found in peanut and in several marine fish. With this intake profile, peanut and marine fish were foods which can acts as protective factor among the subjects.

The previous two studies (Lovejoy et al, 2001 and Harding et al, 2001) had wide range of glucose tolerance. In Harding et al (2001) study, subjects consisted of non diabetic group having normal glucose, impaired glucose tolerance and non diagnostic diabetes melitus. Meanwhile in our study, majority of subjects (81.7%) were in normal glucose. Nevertheless, positive correlation between fasting plasma glucose and fasting plasma insulin was found in our study. Insulin was hormonal products of glucose production. Moderately increased fasting glycemia in some nondiabetics and in mildly hyperglycemic noninsulin-dependent diabetics appeared to be primarily a result of decreased insulin action [15].

Other factors that contributed to insulin level was FFA plasma. In our study, FFA plasma was positively correlated with fasting plasma glucose. In the study of Bogardus et al (1984), FFA was correlated with fasting plasma glucose in non diabetic subjects. Circulating FFAs were elevated in

many insulin-resistant states and have been suggested to play a central role in the pathogenesis of the insulin resistance. FFAs inhibit insulin-stimulated glucose uptake at the level of glucose transport and/or phosphorylation, inhibit insulin-stimulated glycogen synthesis and inhibit insulin-stimulated glucose oxidation. FFAs might have a special role in the insulin resistance associated with central obesity. A common link between increased levels of FFAs and the insulin resistance in type-2 diabetes, obesity and syndrome X could be accumulation of triglycerides in muscle. Factors leading to the accumulation of triglycerides is derived from elevated levels of both circulating FFAs and triglycerides and is also the result of reduced muscle fat oxidation [15].

There was positive correlation between PUFA intake (percentage of total calories) and fasting plasma FFA in our findings. Kiyonuri et al (2003) [16] study found positive correlations between plasma of EPA, DHA and (n-3) high unsaturated fatty acid (HUFA) with dietary intakes of marine oil. The limitation of fasting plasma FFA is that it is not specific biomarker of individual fatty acid intake. FFA released into the circulation were only free in the sense that they were non-esterified. FFA in circulations act as lipid transport system [17]. Nevertheless, biomarkers of fatty acid intake offer an alternative to dietary assessment because they reflect actual rather than reported [18].

In this study, intake of SFA is higher (21% of total calories) and omega-3 PUFA (alpha-linolenic acid) and omega-6 PUFA (linoleic acid) were lower than recommended (IOM) (0.9 g/day vs 1.6 g/day and 15.37 g/day vs 17 g/day). Nutritional factors may play a critical role in controlling insulin secretion. Long- chain fatty acids increase insulin secretion of islet cells in vivo and in vitro in the short term. This situation is due to a stimulation of insulin release by long-chain fatty acid CoA. In contrast, incubation of islet cells in the presence of elevated levels of fatty acid over several days impairs insulin release. This effect is concomitant with acumulation of triglyceride in the islet cells. It is therefore possible that energy and fat overfeeding decrease insulin secretion through a lipotoxicity on islet beta cells [19]. Our study found that there is positive correlation between fasting plasma insulin and triglyceride, contrastly with omega-3 and omega-6 PUFA intake (as gram or percentage of total calories) (*data not shown*).

In multiple logistic regression , waist circumference acts as protective factor of having insulin resistance. This mechanism can be explained by the negative correlation found between SFA intake (percentage of total calories) and waist circumference; i.e. since people with more central obesity consumed more energy. Small range of waist circumference however is limitation in this study. Fasting plasma glucose acts as protective factor of having insulin resistance. In our findings, there was positive correlation between fasting plasma insulin and fasting plasma glucose (r_p=0.243, $p<0.01$), but negatively correlated with fasting plasma FFA (although not significantly). Meanwhile, fasting plasma FFA was positively correlated with fasting plasma glucose (r_p=0.215, $p<0.05$). This findings indicated that fasting FFA plasma had two roles among abdominal obese such as increasing gluconeogenesis and suppresion secretion of insulin, although the latter was not significant in our study. Other study, preferably with wider data distribution, is needed to confirm this findings.

There were several mechanisms for fatty acid intake induced insulin resistance among abdominal obese men. Our study showed that in abdominal-obese subjects there was no relationship between intakes of different fatty acids and plasma insulin; however the effect may be mediated through its indirect effect with FPG. Further study is needed to have clear understanding of the relationship between intakes of different fatty acids and plasma insulin preferably in subjects with more diverse obesity and insulin profiles i.e. including obese and non-obese subjects as well as subjects with and without insulin resistance.

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MANUSCRIPT FOR PUBLICATION

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VALIDATION OF A SQ-FFQ ON MEASURING FAT INTAKES AMONG MEN AGED 30-50 YEARS IN JAKARTA

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ABSTRACT. Nowadays coronary heart disease is increasing in modern society. This is related to changing of food habit and lifestyle. Jakarta as the capital city of Indonesia has higher prevalence of cardiovascular disease and obesity among men than the national figure. Dietary fatty acids may take an important role on chronic degenerative disease including coronary heart disease. Food frequency questionnaire is the obvious choice for assessing food and nutrient intake in epidemiological studies and thus we developed and validated a SQ-FFQ for dietary fatty acids among men aged 30-50 years old in Jakarta. Forty four respondents are eligible for validation analysis of fat SQ-FFQ with 6 day record. The result of relative validity analysis showed that this fat SQ-FFQ was valid through there is no significance difference between energy, total fat, SFA, MUFA and PUFA intake of 6-day records and SQ-FFQ, coefficient correlation were in moderate correlation (rs: 0.183-0.304) with SFA intake statistically significant (rs= 0.304, $p<0.05$) , thus 63.5%-74.9% of subjects were correctly classified or classified with one adjacent quartile and only MUFA intake has classification > 10% in extremely quartile. Further improvement is required to assist respondent in recalling fat intakes consumed with or without meal dan to take into account under or over reporting , using adjustment of number of reported meal vs actual meal based on daily pattern.

KEYWORD : SQ-FFQ, fatty acid intake, relative validity

BACKGROUND. Cardiovascular disease (CVD) is responsible for up to 80 per cent of the deathness 3.2 million people around the world from complications associated with diabetes. The prediction that diabetes incidence will double by 2025 and parallel rise in cardiovascular-related illness[1]. In Indonesia, based on RISKESDAS 2007, prevalence of cardiovascular disease is 7.2% and Jakarta has higher prevalence than nasional (8.1%). Ischemic heart disease is the fourth cause of death of men aged 45-55 years old [2]. Coronary heart disease is increasing in modern society with changing of food habit and liestyle [3]. Several risk factors of coronary heart disease are related to diet [4].

Saturated fatty acids intake can increase LDL cholesterol, while essential fatty acids can reduce the risk of having fatal coronary heart disease cases [5,6]. Previous study on lipid profiles among

four ethnics in Indonesia found unfavorable of intakes of fatty acids [7]. Prevalence of obesity in Jakarta is higher than national (15% vs 10.3%). By gender, in Jakarta prevalence of obesity among men was higher than national figure (22.7 % vs 13.9%) and the second highest in Indonesia [2]. Unfortunately, investigations of the relationship between dietary fatty acids and disease in general population has been limited due lack of the methods suitable for population studies measuring dietary fatty acids intake in Indonesia.

The great potential of dietary fatty acids for epidemiological and clinical applications seems strengthened by the fact that dietary fatty acids may have role on chronic degenerative disease such as diabetes melitus and coronary heart disease [6]. For dietary fatty acids to be used insuch studies, simple and reliable instruments have to developed for its assessments. The FFQ is the obvious choice for assessing food and nutrient intake in epidemiological studies [8], and thus we developed and validated a SQ-FFQ for dietary fatty acids.

SUBJECT AND METHODS

Subjects. Fifty one adult men aged 30-50 years were recruited from private office and government instution. Four respondents dropped-out due to unfinished 7d-records, non-Indonesian man and surrogate interviewer. The cut-off for underreporting energy intake with 99.7% confidence limits was 0.92 for individual with 6 days. One subjects also excluded, who has higher ratio EI and BMR (over reporting). Thus, 44 respondents were eligible to have validation analysis.

Methods. SQ-FFQ design and validation. The development and validation of fat SQ-FFQ were done on December 2008 – February 2009. Firstly, a long list of foods (single and composite dishes), which are potentially important fat sources, was selected using Indonesian food composition tables, existing fat questionnaires (from other studies) and results of existing surveys on adult men consumption. Then, three series of focus group discussion (FGD) among 8 adult men were conducted to elicit foods, focusing on fat rich foods that are usually (typically) consumed by adult men in the study area. Thirdly, the selected food list (step 1) was compared to the results of the FGD (step 2), the food items

that were never consumed (or not commonly eaten) by adult men under study were excluded. On the other hand, the food items obtained exclusively from the FGD, which may generally be consumed by adult men was added to the list. Subsequently, a rough questionnaire was designed using the food list derived from step 3 and asked for the last FGD to see the response, which is identified as potentially major sources of dietary fat, incorporating the frequency response and the portion size questions. The response of specific food sources of fat ranging from monthly, weekly, daily and never for the frequency of consumption of specified serving sizes based on household measures (e.g cups, spoons) and natural unit (e.g. 1 slice bread).

The inclusion criteria of fat rich food in fat SQ-FFQ were source of fat, high g total fat/portion (≥ 0.2 g tot fat/portion), consume ≥ 10 % of the subjects. Grouping of food rich fat by composite foods and single foods, which consist of dairy products, meat, egg, fish and other seafood, cake, baked food, fried food and salty snacks.

Additional questions were added to collect information on the trimming of visible fat from meat, frequency of fried at home and fried takeaway food and specific types of fats and oils used on bread, vegetables and in cooking. Questions on the use of self-prescribed nutritional supplements are also included. Respondent were asked to recall their frequency of consumption of food items over the preceding 1 month.

After run and analyze the last FGD then the definitive form of fat SQ-FFQ were ready, the data collection for measuring its relative validity with the 7-day dietary records was conducted to cover "true intake" of different type of fatty acids especially SFA, MUFA, PUFA. Ultimately, a validation of fat SQ-FFQ was done on 51 respondents include obese and non obese adult men with food picture which came from 2-3 times sampling of food and food models..

7D-RC. The 7d-records was chosen as the reference method because it is reliable in measuring fatty acids intake (7). After brief instructions, each respondent was supplied with dietary record form for two days. A 24-hr recall was examined to respondents to check the ability of respondents on recording

the food and give another two record form. Three days form record are given in the last visit with 24 hr-recall. The forms consist of identity, guidelines for recording the food, example of food record, blank food record with column of time of eating, place of eating, food, food items (composite food), method of cooking, portion size, and others information (price, etc).

*Anthropomethric measurements.*Body weight, height, and waist circumference were assessed among respondents. Anthropomethric measurement for body weight using calibrated electric SECA model 872, height using microtoise, waist circumference using waist-watcher tape measure. All the assessment either dietary and anthropomethric were done by two trained enumerators. Weighing was done to respondents with minimal clothing and no heavy things carried in body as well as when do height measurement with no head accesories and stand erect in correct position (Frankfurt plane position). Waist circumference was measured in the fasting condition or one hour post prandial and wear minimized clothing to allow the tape to be correctly positioned. To perform the measurement at the natural waist, the lowest rib margin (the tenth rib) was first located then palpated the iliac crest in the midaxillary line. A waist-watcher tape was applied horizontally midway between the lowest rib margin and the lateral iliac crest made into a complete loop and fitted firmly around the waist by a spring mechanism, which is controlled by a pushbutton. [9-11].

Statistical analysis. Data record for analyzing was used for 6-day because there is one day that the record was poor. This 6-day records was the number required to estimate true average fat intake for groups of individuals. The cut-off for underreporting energy intake with 99.7% confidence limit was 0.92 for individual (n=1) with 6-day records. According to this criteria, data from 45 subjects can be used for validation analysis. One subject was excluded due to the highest ratio of EI/BMR (overreporting). With all the criteria, 44 subjects were eligible to have validation analysis. Data entry and statistical analysis was done using SPSS program version 15. Descriptive statistics was done to describe frequency, mean, median, standard deviation and range value. Kolmogorov-Smirnov test was used to test the normality of sample distribution. Mean difference with Paired t-test was used to assess mean difference of SQ-FFQ and 6d-records. Spearman's correlation test was administered to find out

the correlation between SQ-FFQ and 6d-records. Cross-classification done to examine correctly classified and extremely classified between SQ-FFQ and 6d-records. (SPSS version 15).

RESULTS

In this study, (Table. 1) the majority of respondents (77.3 %) were in younger age group (30-39 year). Most of the respondents were bachelor degree (45.5%). More respondents were private employee (40.9%) than government employee (22.7%).

Table. 1 Socio-demographic characteristics of subjects (n=44)

Characteristics	n (%)
Age :	
• 30-39 year	34 (77.3)
• 40-50 year	10 (22.7)
Education :	
• 9-year schooling	8 (18.2)
• senior high school	11 (25)
• bachelor degree	20 (45.5)
• master degree	5 (11.4)
Occupation :	
• private employee	18 (40.9)
• government employee	10 (22.7)
• medical doctors	5 (11.4)
• others	11 (25)

Table. 2 showed that most of respondents (61.4%) had normal waist circumference. According to WHO classification on BMI, most (50%) of subjects were in normal range, but fourteen (31.8%) of the subjects were in pre-obese stage. Meanwhile, based on Asian Adult Population classification on BMI, more comparable on subjects with normal range and obese I (27.3%).

Table. 2 Antropomethric characteristics of subjects (n=44)

Characteristics	n (%)
Waist circumference :	
• < 90 cm (normal)	27 (61.4)
• ≥ 90 cm (abdominal obese)	17 (38.6)
BMI :	
Based on WHO classification :	
• < 18.50 kg/m^2 (mild underweight)	3 (6.8)
• 18.50-24.99 kg/m^2 (normal)	22 (50)
• ≥ 25.00 kg/m^2 (overweight)	
o 25.00 -29.99 kg/m^2 (pre-obese)	14 (31.8)
o 30.00-34.99 kg/m^2 (obese class I)	4 (9.1)
o 35.00-39.99 kg/m^2 (obese class II)	0 (0)
o ≥ 40 kg/m^2 (obese class III)	1 (2.3)
Based on Asian Adult Population classification :	
• < 18.50 kg/m^2 (underweight)	3 (6.8)
• 18.50-22.99 kg/m^2 (normal range)	12 (27.3)
• 23.00-27.49 kg/m^2 (overweight)	17 (38.6)
• ≥ 27.50 kg/m^2 (obese I)	12 (27.3)

Analysis of relative validation used three types of statistical analysis such as mean differences with Paired t-test, Spearman's correlation test and cross-classification test. The result of relative validity analysis showed that this fat SQ-FFQ was valid through there is no significance difference between energy, total fat, SFA, MUFA and PUFA intake of 6-day records and SQ-FFQ (Table. 3), coefficient correlation were in moderate correlation (r: 0.183-0.304) with one significant coefficient ($p<0.05$) (Table. 4), there were 27.3%-36.3% correctly classified to individual fatty acids intake and just one fatty acid intake was in the extreme classification with >10% (MUFA intake).

Table. 3 Mean intake of total fat, SFA, MUFA, PUFA in 6d-records and in fat SQ-FFQ (n=44)

	Fat SQ-FFQ	6 day- records
Energy (Kcal)	1878.16 ± 811.31	2047.55 ± 357.88
Total fat (g)	85.64 ± 43.44	79.77 ± 19.76
SFA (g)	39.36 ±19.45	33,41 ± 10.15
MUFA (g)	21.59 ± 12.06	20.82 ± 5.50
PUFA (g)	16.11 ± 12.06	17,57 ± 4.54

Table. 4 Correlation coefficient between 6d-records and fat SQ-FFQ (n=44)

	Coefficien correlation (r) between 6d-records and fat SQ-FFQ
Total fat	0,185
SFA	0,304*
MUFA	0,183
PUFA	0,222

*significant correlation at the level p<0.05, Spearman correlation test

Table. 5 Cross-classification of fat SQ-FFQ and 6d-records (n=44)

	Correctly classified into the same quartile(%)	Classified into one adjacent quartile(%)	Classified into extreme quartiles (%)
Total fat	27.2	40.8	4,6
SFA	36.3	38.6	6.8
MUFA	27.2	43.1	11.3
PUFA	27.3	36.2	9

DISCUSSION

The present study shows that mean fatty acid intake in SQ-FFQ were higher than those obtain by 6d-records but not statistically significant. This was similiar to finding by Broadfield et al, (2003)[12] in which, mean SQ-FFQ was higher than 7d-records. This study was more diverse on respondents characteristics than study by Broadfield et al, (2003). Ideally, the subjects in a validation study should be a random sample of the study population in which the questionnaire is being used, since food sources in developing countries are correlated with socio-economic condition.[8]. Representation of subjects with different socio-economic results in more representative food items rather than if subjects come from similiar background (e.g student or university staff). Based on this diversity, our SQ-FFQ was consist of 7composite food and 75 single food items. Six- day records were used, as the require minimum day to estimate true average fat intake for groups of individuals [13]. Food item, portion sizes and number of day in this SQ-FFQ are believed to represent usual fatty acid intake in this population.

The weakness of SQ-FFQ are that it relies on memory and estimated portion sizes (average portion sizes). Portion sizes in this study were developed by using 2-3 times sampling of food items in the area of respondents and options were made to allow for small, medium and large portion

sizes.Therefore, subject's memory to recall the usual frequency of intake may account more to the result. Also, men are usually less aware of their foods compared to women. It is known that, weighing would not rely on memory and would give detailed data on portion size consumed [13] Unfortunately, weighed record can not be done because limitation of subject condition (working).

Total fat in this fat SQ-FFQ was higher than that found in other study on fatty acid intake among four ethnicity in Indonesia (Djuwita-Hatma, 2001) 40.37% vs 29-35%,.most likely because almost half of respondents in thisstudy were obese and abdominal obese. MUFA and PUFA intake as percentage of total calories were comparable with that found by Masson et al, (2003)[14] (MUFA ≈ 10.04% vs 11.9%, PUFA ≈ 7.55% vs 5.50% for this study and Masson et al, respectively). Coefficient correlation of MUFA intake was also comparable with previous study (Broadfield et al, 2003) 0.183 vs 0.15. Cross-classification test showed that correct classification was 27.2%-36.3% and classification within one-adjacent quartile was 36.2%-43.1%, thus 63.5%-74.9% of subjects were correctly classified or classified within one adjacent quartile. Based on that, we consider this fat SQ-FFQ has the sufficient ability to obtain useful and reliable information about dietary fatty acids intake in adult men. Further work needs to be done if this SQ-FFQ is to be used for subjects with other characteristics (sex, age, body composition profile). Future improvement of this SQ-FFQ by using weighing (diverse subjects) and addressed as meal base including carbohydrate intake to allow for energy adjustment. The latter has been done in our study, but further improvement is required to assist respondent in recalling fat intakes consumed with or without meal dan to take into account under or over reporting , using adjustment of number of reported meals vs actual meals based on daily food pattern.

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Appendix 2

GUIDELINE FOR AUTHORS

Appendix 2 Guideline for Authors (1)

MANUSCRIPT PREPARATION

JANUARY 2009 - NEW SAMPLE FILES ADDED TO ASSIST AUTHORS WITH MANUSCRIPT PREPARATION

See links below under "Manuscript Preparation" to view sample files for a submitted manuscript, tables, figures, and an editorial checklist.

GUIDELINES, INCLUDING MANUSCRIPT WORD LIMIT - PLEASE READ CAREFULLY

Manuscripts longer than 7500 words will be returned without review. Word count includes the abstract, all text and the references (but excludes tables.) *The Journal* is limited in the number of pages that can be published each year and article length is a consideration in the editorial process.

Manuscript Preparation:

- Prepare your manuscript in Word 6.0 or later, saving the file in the .doc format (please note: the Word 2007 .docx file format is not accepted). Please consult the "Help" feature in Word for assistance with fonts, line numbering, etc.
- Times, Times Roman, Courier, Helvetica and Arial are the recommended text fonts. Please see section on Tables and Figures for information on figure fonts. For best quality conversions of special characters and symbols, use the Symbol font.
- Papers must be completely double-spaced.
- Papers must have consecutively numbered lines from the first line, first manuscript page throughout the last line, last manuscript page. Do not number the Literature Cited section. If you are uncertain about how to do this, please consult the 'Help' feature in Word.
- Figures and tables should be clearly labeled (Fig. 1, Fig 2, etc. or Table 1, Table 2,etc).
- Please refer to "Manuscript Digital Files" for information on electronic file requirements.
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- Use only standard abbreviations.
- Use standard chemical and biochemical terms and follow ASN nomenclature policy.
- Include Conflict of Interest and Funding Disclosure footnotes.
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  (1) The editorial checklist that is completed for manuscripts being returned to authors for revision or resubmission.
  (2) A JN manuscript with comments, and
  (3) Several JN tables and figures with comments.

Your Manuscript should include:

A) Title Page

B) Abstract Page

C) Introduction

D) Materials and Methods

E) Results and Discussion

F) Literature Cited

G) Acknowledgement (if applicable)

A) TITLE PAGE: **The title page must include:**

1. The title should be composed as a single declarative statement. The title should be focused on the results presented in the manuscript. Please do not use a colon or semicolon in the title. Please keep the title as generally applicable as possible. It usually is not necessary to include the exact study location or a specific study name in the title, as this information can be included in the abstract.
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Appendix 2 (continued) (1)

Conflict of Interest and Funding Disclosure: Any existing financial arrangements between an author and a company whose product figures prominently in the submitted manuscript or between the author and any company or organization sponsoring the research reported in the submitted manuscript should be brought to the attention of the Editor in the cover letter that accompanies the manuscript submission. In addition, all authors must declare all sources of funding for research reported in their manuscript and report all potential conflicts of interest in separate footnotes on the manuscript title page. If an author has no conflicts of interest, the footnote should list the author's name, followed by "no conflicts of interest". A conflict of interest includes, but is not limited to:

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Individuals who are asked to review a manuscript should decline the solicitation if they have:
(1) served as an adviser or advisee to an author on the manuscript
(2) collaborated or served as a coauthor with an author of the manuscript during the past 3 years;
(3) are currently affiliated with, were previously employed within the past 12 months by, or are being considered for employment at the institution of an author;
(4) participated in a consulting/financial arrangement with an author in the past 3 years; or
(5) are the spouse, child, sibling, parent, partner, or close friend, or otherwise have a relationship that might affect judgment, or could be seen as doing so by a reasonable person familiar with the relationship.

B) ABSTRACT PAGE: The abstract must be a single paragraph of no more than 250 words summarizing the relevant problem addressed by the study and the theory or hypothesis that guided the research. The abstract should include the study design/methodology and clear statements of the results, conclusions and importance of the findings.

C) INTRODUCTION: Background to the research conducted and specific objectives should be clearly indicated. This should not be a comprehensive review of the literature, however.

D) MATERIALS AND METHODS: Documentation of methods and materials used should be sufficient to permit replication of the research. State the source of specialized materials, diets, chemicals, and instruments and other equipment, with model or catalog numbers, where appropriate. Specify kits, analyzers, and commercial laboratories used. Cite references for methods whenever possible and briefly explain any modifications made.

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DIETS. Composition of control and experimental diets must be presented. When a diet composition is published for the first time in *The Journal of Nutrition,* utilize a table or a footnote to provide complete information on all components. If previously described in *The Journal of Nutrition* or *The American Journal of Clinical Nutrition,* a literature citation may be used. State specifically any modifications made to the published diet compositions. The proximate composition of closed formula diets should be given as amounts of protein, energy, fat, and fiber. Components should be expressed as g/kg diet. Vitamin and mineral mixture compositions should be included using *Journal of Nutrition* units and nomenclature. For a discussion of the formulation of purified animal diets, refer to Baker (4) and to a series of ASN publications (5-8).

STATISTICAL METHODS. Describe all statistical tests utilized and indicate the probability level (P) at which differences were considered significant. If data are presented in the text, state what they represent (e.g. means ± SEM). Indicate whether data were transformed before analysis. Specify any statistical computer programs used.

Present the results of the statistical analysis of data in the body of each and on figures per se. Use letters or symbols to indicate significant differences; define these in a table footnote or the figure legend. Provide the appropriate statistics of variability. An estimate of the error variance (SD or SEM) of group means should be displayed in figures. Standard ANOVA methodology assumes a homogeneous variance. If error variance is tested and found to be heterogeneous, data should be transformed before ANOVA, or nonparametric tests should be used. For a discussion of variability calculations and curve-fitting procedures, see Baker (4).

E) RESULTS AND DISCUSSION Report the results of the study. Discuss the significance of the findings, interpret the results and conclusions.

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Literature Cited
References and further reading
Bibliography
Literature

G) ACKNOWLEDGMENTS. Technical assistance and advice may be acknowledged in a section at the end of the text. Only named individuals should be included in this section. Authors are responsible for obtaining written permission from everyone acknowledged by name and for providing copies of signed permission statements to *The Journal of Nutrition*. These statements should be submitted to *The Journal* along with the manuscript Authors' Statement/Copyright Release Form.

UNITS OF MEASURE. Most measurements must conform to *le Systeme Internationale d'Unites* (SI) (9). The metric system and the Celsius scale (° C) must be used. Concentrations should be expressed on a molar basis. Except for diet composition, convert to substance concentration, e.g., mol/L. The denominator should be L. Do not use *M, mM, N,* etc. Use one of three acceptable options to express measurements. (a) Use SI units exclusively. (b) Use SI units and, if appropriate, provide conventional units parenthetically in the text and give conversion factors in table footnotes and figure legends. (c) Use conventional units, if appropriate, and provide SI units parenthetically in the text and give conversion factors in table footnotes and figure legends. Units should not be pluralized. Useful websites are:

- SI conversion website: http://www.ex.ac.uk/cimt/dictunit/dictunit.htm
- Clinical SI conversions: http://www.unc.edu/~rowlett/units/scales/clinical_data.html
- Clinical SI conversions: http://dwjay.tripod.com/conversion.html

ABBREVIATIONS. Use only standard abbreviations. Table 2 is an abridged list of abbreviations that may be used without definition in Journal of Nutrition articles. Other standard abbreviations are listed in Scientific Style and Format (1).

If there are three or more abbreviations defined in the text, define each the first time it is used in the text and prepare an abbreviation footnote. The footnote should be associated with the first abbreviated term in the text and should be an alphabetized listing of all author-defined abbreviations and their definitions. Abbreviations should not be followed by a period and should not be pluralized (e.g. AA should represent both "amino acid" and "amino acids"). Use the verb (e.g."is" or "are") that is consistent with the context in which the abbreviation is used in the sentence. Units and statistical terms also should not be followed by a period or pluralized. Use the standard abbreviations for SI prefixes found in Young (9) and in Table 3 and those for units of measure in Table 4. Abbreviations used only in tables and figures must be separately defined in the footnotes or legend for each table or figure. Abbreviations that are in the abbreviation footnote should not be redefined in table footnotes or figure legends.

NOMENCLATURE. Chemical and biochemical terms and abbreviations and identification of enzymes must conform to the recommended usage of the International Union of Biochemistry and Molecular Biology (10). Names for vitamins, related compounds, and abbreviations for amino acids should follow the ASN nomenclature policy (11,12).

Previous Page
Top

Last updated 07/07/2009 19:25:16

Appendix 2 (continued) (2)

Guide to Authors

Welcome to the electronic manuscript submission website for *European Journal of Clinical Nutrition*. The instructions below are structured so you can quickly and easily answer the following questions:

1. Is my manuscript suitable for *European Journal of Clinical Nutrition*? (Scope + Editorial Note)
2. How do I format my manuscript for *European Journal of Clinical Nutrition*? (Format of Papers)
3. How do I submit my manuscript to *European Journal of Clinical Nutrition*? (Submission of Papers)

OTHER LINKS

- About the journal
- Format of papers
  - Preparation of Original Articles
  - House style
- Submission of papers
- Editorial policy
  - Ethics and bioethics
  - Peer review policy
  - Corrections
  - About npg online services

ABOUT THE JOURNAL

Scope

European Journal of Clinical Nutrition is an international, peer-reviewed journal publishing articles related to human and clinical nutrition. We aim to cover all aspects of human and clinical nutrition:

- Clinical and whole body metabolic investigations
- Epidemiological, social and behavioural studies in nutrition
- Nutritional determinants of growth, development and health
- Assessment and measurement of nutritional status and indicators, and their relation to function and health
- Nutritional causes and effects of disease
- Community nutrition and education
- Nutrition, population health and health promotion

Topics Covered
The scope of the journal includes publishing original articles, short communications and case reports based on clinical, metabolic and epidemiological studies that describe methodologies, mechanisms, relationships and benefits of nutritional interventions for disease and health promotion. Editorials, commentaries, reviews, book reviews and letters to the editor will also feature in EJCN

Editor	Professor Prakash S Shetty, Institute of Human Nutrition, School of Medicine, University of Southampton, Southampton, UK
Frequency	12 issues a year
Abstracted in	Current Contents Current Contents Clinical Medicine Current Contents Life Sciences EMBASE/Excerpta Medica Elsevier BIOBASE/Current Awareness in Biological Sciences Index medicus

Appendix 2 (continued) (2)

Science Citation Index
BIOSIS
CAB Abstracts
CAB Health and Nutrition Newsletter

Editorial Note

Manuscripts based on animal nutrition and in vitro studies will not be considered. Papers reporting validation of generally accepted methodologies in specific population groups and prevalence or incidence data on nutritional problems from countries have very low priority. When validation studies and prevalence or incidence data specific to countries are submitted for publication to EJCN, they will be processed only if they are submitted as a short communication with the clear understanding that supplementary data will be made available by the authors to anyone interested in compiling regional or global comparisons. EJCN also does not encourage submissions based on testing clinical or commercial food, and nutritional or clinical products.

If a manuscript, previously considered for publication in another journal, is submitted to EJCN, we would appreciate receiving copies of the comments from reviewers and Editors. This will enable an early decision by the Editorial team and the possibility to fast track the same with fewer peer reviews.

Finally, please make sure covering letters highlight the unique features of the submitted manuscript and make the case why this manuscript deserves publication in EJCN.

FORMAT OF PAPERS

Article Types Table

Article Type	Description	Approximate Word Count
Original Article	These are reports of current basic or clinical research and should follow the structure outlined below this table.Reports of Randomised controlled trials (RCTs) submitted to EJCN must adhere to the CONSORT statement, (CONsolidated Standards Of Reporting Trials). This guideline provides an evidence-based, minimum set of recommendations for reporting RCTs, it comprises a list of items to report and a patient flow diagram. For other study designs, EJCN strongly encourages authors to consult reporting guidelines relevant to their specific research design: studies of diagnostic accuracy (STARD); observational studies in epidemiology (STROBE); systematic reviews and meta-analyses (QUOROM); and meta-analyses of observational studies in epidemiology (MOOSE). All these reporting guidelines can be found at http://mts-ejcn.nature.com/"http://www.equator-network.org"	Word limit: 3,000 words excluding abstract, references, figures and tables. The abstract should be no longer than 250 words and structures, as outlined in the Abstract and Keywords section below. References: 50 references maximum (as far as possible only recent references). Display items: No more than six display items (e.g. figures and/or tables) should accompany the manuscript. Multiple figures (1a, 1b, 1c etc.) will count as individual figures.
Short Communications	Short Communications are studies that fall short of the criteria for full Original Articles (e.g. preliminary experiments limited by sample size or duration, or novel hypotheses). Apart from the abstract, there is no obligation to divide the text into sections.	Word limit: 1,000 words. Abstract: unstructured paragraph of 150 words maximum. References: 10 references maximum. Display items: No more than 2 display items (e.g.

Appendix 2 (continued) (2)

		figures and/or tables) should accompany the manuscript.
Invited Reviews	Reviews are comprehensive analyses of specific topics that are solicited by the Editor by invitation. Proposals for reviews may be submitted; however, authors should only send an outline of the proposed paper for initial consideration. All invited reviews will undergo peer review prior to acceptance. A maximum of three authors is preferred for Review articles.	Word limit: 5,000 words excluding the abstract, references, figures and tables. Abstract: Structured paragraph of 250 words maximum with headings: 'Background and Aims', 'Methods' (to include search strategy), and 'Results and Conclusions'. References: 100 references maximum.
Letters to the Editor	Letters to the Editor will be considered for publication, subject to editing. Letters must contain information critical to a certain area or must be referencing data recently published in the *European Journal of Clinical Nutrition*. A Letter must reference the original source, and a Response to a letter must reference the Letter in the first few paragraphs. Letters can use an arbitrary title, but a Response must cite the title of the Letter: e.g. Response to [title of the Letter].	Word limit: 500 words. Abstract: No abstract required for this manuscript type. References: Three references maximum in addition to the reference to the original article if correspondence relates to a publication in EJCN.
Editorials (only by invitation of the Editors)	Proposals for Editorials may be submitted; however, authors should only send an outline of the proposed paper for initial consideration.	Word limit: 1,000 words. Abstract: No abstract required for this manuscript type. References: Five references maximum.
Book Reviews (only by invitation of the Editors)	Frank opinion about the scope, contents, quality, and usefulness of the book.	Word limit: 250 words maximum. Abstract: No abstract required for this manuscript type. References: Five references maximum.
Case Reports	Interesting case studies relevant to nutrition in clinical practice.	Word limit: 500 words maximum. Abstract: No abstract required for this manuscript type. References: Five references maximum. Display items: One clinical photo or other display item (table/figure).
Commentaries, Points-of-View	Commentaries discuss a paper published in a specific issue and should set the problems addressed by the paper in the wider context of the field. Invited commentaries may also provide a 'point-of-view' related to a	Word limit: 1,000 words. References: 10 references

Appendix 2 (continued) (2)

and Debates (only by invitation of the Editors)	controversy or debate in nutrition. Debates will address an area of research that is of major present interest and for which there are substantially different views. The subject to be debated and the authors producing the opposing views will be selected by the Editors.	maximum. Display items: No more than one display item.
Continuing Educational/Guidelines/Nutrition in Clinical Practice (only by invitation of the Editors)	Highlights in boxes are encouraged	Word limit: 3,000 words maximum excluding the abstract, references, figures and tables. Abstract: Unstructured abstract of 250 words maximum. References: 50 references maximum (as far as possible only recent references). Display items: No more than 8 display items (e.g. figures and/or tables) should accompany the manuscript.

Preparation of Original Articles

Abstract and Keywords
Articles must be prepared with a structured abstract designed to summarize the essential features of the paper in a logical and concise sequence under the following headings, which are mandatory.

- **Background/Objectives:** What was the main question or hypothesis tested?
- **Subjects/Methods:** How many subjects were recruited, how many dropped out? Was the study randomized, case-controlled etc? Interventions/methods used, and duration of administration.
- **Results:** Indicate 95% confidence intervals and exact P value for effects.
- **Conclusions:** Answer (significant or not) to main question.
- **Keywords:** preferably use up to 6 words chosen from MEDLINE MeSH, which best describe your paper. These will be used for indexing your paper in EJCN and also help retrieval from computer databases.

House Style

Statistical methods: For normally distributed data, mean (SD) is the preferred summary statistic. Relative risks should be expressed as odds ratios with 95% confidence interval. To compare two methods for measuring a variable the method of Bland & Altman (1986, Lancet 1, 307–310) should be used; for this, calculation of P only is not appropriate.

Units and Abbreviations: Use metric units (SI units) as fully as possible. Preferably give measurements of energy in kiloJoules or MegaJoules with kilocalories in parentheses (1 kcal = 4.186kJ). Use % throughout. Very common abbreviations such as **FFA, RNA**, need not be defined; on first using an abbreviation place it in parentheses after the full item. Note these abbreviations: gram **g**; litre **l**; milligram **mg**; kilogram **kg**; kilojoule **kJ**; megajoule **MJ**; weight **wt**; seconds **s**; minutes **min**; hours **h**. Do not add s for plural units. insertText3();

References
References in text are indicated in the text by name and date e.g. (Pampiglione & Ricciardi, 1986) and (Kusin *et al.*, 1994), and listed at the end of the paper in alphabetical order of first author. References should be listed and journal titles abbreviated according to the style used by Index Medicus, examples are given below. All authors should be quoted for papers with up to six authors; for papers with more than six authors, the first six only should be quoted, followed by *et al.* Please use recent references wherever possible.

Appendix 2 (continued) (2)

Examples of article references:

- Kusin KA, Kardjati S and Renqvist UH (1994): Maternal body mass index: the functional significance during reproduction. *Eur. J. Clin. Nutr.* **48**, Suppl, 3 S56-S67.
- Martin JC, Bourgnoux P, Fignon A, Theret V, Antoine JM, Lamisse F *et al.* (1993): Dependence of human milk essential fatty acids on adipose stores during lactation. *Am. J. Clin. Nutr.* **58**, 653-659.
- Friedman MI, Gil KM, Rothhopf MM and Askanazi J (1986): Post-absorptive control of food intake in humans. *Appetite* **7**, 258 (abstract).

Articles in books:

- Pampiglione S and Ricciardi ML (1986): Parasitological survey of Pygmy groups. In *African Pygmies*, ed. LL Cavalli-Sforza, pp 153-165. New York: Academic Press.

Personal communications must be allocated a number and included in the list of references in the usual way or simply referred to in the text; the authors may choose which method to use. In either case authors must obtain permission from the individual concerned to quote his/her unpublished work.

SUBMISSION OF PAPERS

First, if you have not done so already, register for an account. HOME http://mts-ejcn.nature.com/letters/ejcn_copyright.pdf

European Journal of Clinical Nutrition open

Upon submission of an original research paper, authors can indicate within the manuscript tracking system whether they wish to pay a one off fee to allow their article to become freely available immediately upon publication. The fee is £2,000/$3,000/€2,400 (plus VAT where applicable) and can be paid via credit card or by requesting an invoice be raised.

Upon acceptance, authors must fill out and send back a payment form. By paying this fee authors are permitted to post the final, published, PDF of their article on a website, institutional repository or other free public server immediately on publication.

Authors must still sign and return the licence to publish form, which has been amended to offer authors the choice of which licence to use on their paper. The first licence is the *Creative Commons Attribution-Noncommercial-No Derivative Works 3.0 Unported Licence* which allows readers to download the article and share it with others as long as they mention the author and link back to the original article. The article cannot be changed in any way or used commercially. The second option is the *Creative Commons Attribution Noncommercial Share Alike 3.0 Unported Licence* which allows readers to alter, transform, or build upon the article and then distribute the resulting work under the same or similar license to this one. The work must be attributed back to the original author and commercial use is not permitted.

For further information please see the *European Journal of Clinical Nutrition* Open FAQs and payment form.

Colour charges

Number of colour illustrations		1	2	3	4	5	6	7+	
Cost	Rest of world USA	£494 $987	£735 $1,470	£977 $1,953	£1,124 $2,247	£1,271 $2,541	£1,397 $2,793	£126 $252	per additional colour figure

Further information

To find out who to contact for advertising, subscriptions, permissions, papers in production or publishing a supplement, please visit our publisher's contacts page. Alternatively, you can write to: European Journal of Clinical Nutrition, Nature Publishing Group, The Macmillan Building, 4 Crinan Street, London, N1 9XW, UK.

EDITORIAL POLICY

Statement of Ethics

Appendix 2 (continued) (2)

Peer-review policy

Correction and retraction process

Non-native speakers of English
Non-native English-speaking authors often receive negative comments from referees or editors about the English usage in their manuscripts, and this can contribute to a negative decision. To help reduce the possibility of such problems, we strongly encourage such authors to take at least one of the following steps:

- Ask a colleague whose native language is English to review your paper for clarity.
- Use a service such as one of those listed below. An editor will improve the English to ensure that your meaning is clear and identify problems that require your review. Note that the use of such a service is at the author's own expense and risk and does not guarantee that the article will be accepted. NPG accepts no responsibility for the interaction between the author and the service provider or for the quality of the work performed.

American Journal Experts
Inter-Biotec (Inter-Biotec also provides a free online writing course to help biomedical scientists whose first language is not English to write and publish their papers in English-language journals)
SPI Professional Editing Services
Write Science Right .

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Appendix 3

ETHICAL APPROVAL

Appendix 3 Ethical Approval

UNIVERSITAS INDONESIA
FAKULTAS KEDOKTERAN

Jalan Salemba Raya No. 6 Jakarta Pusat
Pos Box 1358 Jakarta 10430
Kampus Salemba Telp. 31930371, 31930373, 3922977, 3927360, 3912477, 3153236, Fax. : 31930372, 3157288, e-mail : office@fk.ui.ac.id

NOMOR : 21 /PT02.FK/ETIK/2009

KETERANGAN LOLOS KAJI ETIK

ETHICAL --- CLEARANCE

Panitia Tetap Penilai Etik Penelitian, Fakultas Kedokteran Universitas Indonesia dalam upaya melindungi hak asasi dan kesejahteraan subyek penelitian kedokteran, telah mengkaji dengan teliti protokol berjudul:
The Committee of The Medical research Ethics of the Faculty of Medicine, University of Indonesia, with regards of the Protection of human rights and welfare in medical research, has carefully reviewed the proposal entitled:

"RELATIONSHIP BETWEEN INTAKES OF DIFFERENT FATTY ACIDS AND HYPERINSULINEMIA IN ABDOMINAL OBESE ADULT MEN IN JAKARTA".

Peneliti Utama : **dr. ANDI YASMIN SYAUKI**
Name of the principal investigator

Nama Institusi : **SEAMEO-TROPMED UI**

dan telah menyetujui protocol tersebut di atas.
and approved the above mentioned proposal.

Jakarta, 19 Januari 2009

Chairman
Ketua

Prof. Dr. dr. Agus Firmansyah, SpA(K)

-Peneliti wajib menjaga kerahasiaan identitas subyek penelitian.

Appendix 4

INFORMED CONSENT

LEMBAR INFORMASI UNTUK SUBJEK

Hubungan antara asupan berbagai jenis lemak dengan hiperinsulinemia pada laki-laki dewasa dengan obesitas sentral di Jakarta

Peneliti :
dr. Andi Yasmin Syauki, Dr. Ir. Umi Fahmida, M.Sc, dr. Widjaja Lukito, Sp.GK, Ph.D,
Andi Mariyasari Septiari, M.Sc, Dra. Indriyanti R.S, M.Si

Anda diundang untuk turut serta dalam suatu penelitian dengan judul **Hubungan antara asupan berbagai jenis lemak dengan hiperinsulinemia pada laki-laki dewasa dengan obesitas sentral (lingkar pinggang ≥ 90cm) di Jakarta** yang dilaksanakan oleh mahasiswi SEAMEO-TROPMED Regional Center For Community Nutrition (Pusat Gizi Regional) Fakultas Kedokteran Universitas Indonesia bekerja sama dengan mahasiswi Fakultas Kedokteran program Doktoral Universitas Hasanuddin. Informasi dalam lembar informasi ini akan menjelaskan apa yang akan terjadi selama penelitian. Setelah membaca dengan teliti anda dapat mengajukan pertanyaan dan dapat membicarakannya dengan petugas.

Prosedur Penelitian
Pada penelitian ini, berikut adalah hal-hal yang akan di jalani oleh anda:

1. Anda akan diminta untuk menandatangani suatu persetujuan kesediaan mengikuti penelitian ini.
2. Penelitian ini terdiri atas 2 tahap :
   a. tahap pertama/tahap skrining subjek dimana anda akan menjalani wawancara mengenai identitas pribadi dan riwayat kesehatan, pengukuran anthropometrik, pemeriksaan klinis dan pemeriksaan biokimia darah.
   b. tahap kedua/ tahap pengambilan data bagi responden dimana bila hasil dari pemeriksaan darah anda memenuhi kriteria dari penelitian kami, maka anda akan diwawancara mengenai identitas lengkap, gaya hidup, aktivitas fisik, dan asupan makanan terdiri atas asupan lemak, energi, karbohidrat terutama serat, dll.
3. Pada tahap pertama anda akan di wawancara mengenai identitas singkat.
4. Untuk pengukuran antropometrik, anda akan di ukur lingkar pinggang, tinggi badan, dan berat badan.
5. Untuk pemeriksaan klinis, anda akan diukur tekanan darah dan suhu tubuh.
6. Untuk pemeriksaan biokimia darah, anda akan diperiksa kadar glukosa, fungsi hati, fungsi ginjal, profil lemak, dan insulin dalam keadaan puasa untuk mengetahui apakah anda memenuhi kriteria penelitian kami. Pengambilan darah vena sebanyak 5 ml dikerjakan oleh staf Laboratorium Klinik Prodia yang sudah terampil pada pembuluh darah di lipatan siku dengan alat pengambil darah sekali pakai. Pengambilan darah mungkin menyebabkan memar, infeksi, perdarahan tidak berhenti, dan shok. Untuk menghindari terjadinya perdarahan yang tidak berhenti ditanyakan apakah subyek kalau berdarah cepat membeku atau tidak. Memar tidak akan terjadi bila pengambilan darah dilakukan oleh orang yang sudah terlatih, dan infeksi dihindari dengan melakukan pengambilan darah secara steril. Untuk menghindari shok, maka sebelum pengambilan darah dilakukan pemeriksaan kelopak mata untuk melihat ada tidaknya anemi, pengambilan darah sebaiknya diambil pada subyek dalam posisi berbaring. Setelah pengambilan dilakukan pengamatan sekurang-kurangnya 10 menit. Darah yang diambil tadi akan digunakan untuk pemeriksaan di Laboratorium Klinik Prodia.
7. Pada tahap kedua, subjek yang memenuhi kriteria kami akan menjadi responden dalam penelitian kami.

8. Anda akan diwawancara mengenai identitas lengkap, karakteristik sosio-ekonomi, gaya hidup (kebiasaan merokok, konsumsi alkohol, konsumsi sayur dan buah, dan aktivitas fisik).
9. Untuk pengambilan data asupan makanan lemak, anda akan diwawancara dengan menggunakan kuesioner yang telah disediakan, sedangkan untuk asupan energi, karbohidrat terutama serat, dll akan dilakukan selama 3 hari yang telah ditentukan.
10. Partisipasi anda dalam penelitian ini bersifat sukarela. Anda dapat sewaktu-waktu mengundurkan diri dalam penelitian ini. Bila anda mengundurkan diri dalam penelitian ini maka hasil pemeriksaan darah tidak dapat anda terima.

Manfaat Penelitian :
Partisipasi anda akan memberikan informasi yang berharga mengenai keadaan profil asupan lemak, status insulin, glukosa, lipid dan hubungannya. Hasil penelitian akan memberikan informasi mengenai profil lemak maupun jenis makanan apa saja yang mempengaruhi peningkatan kadar insulin pada laki-laki dewasa dengan obesitas sentral.
Perlu anda ketahui bahwa semua pemeriksaan pada penelitian ini tidak dipungut biaya, informasi yang anda berikan kepada kami akan tetap dirahasiakan dan identitas anda tidak akan dipublikasikan.

Demikian penjelasan kami tentang penelitian ini dan kami memohon kesediaan anda untuk turut berpartisipasi dalam penelitian ini. Bila anda menyetujui untuk ikut serta dalam penelitian ini, kami harapkan anda dapat memberikan tanda tangan pada lembar persetujuan dan mengembalikannya kepada kami. Bila ada pertanyaan mengenai penelitian ini, silahkan menghubungi dr. Andi Yasmin Syauki and Dra. Indriyanti R.S, MSi.

Hormat kami,

dr. Andi Yasmin Syauki
Gedung SEAMEO TROPMED RCCN-UI, Kampus UI Salemba
Jl. Salemba Raya no. 6 Jakarta Pusat
Telepon: (021) 3909205; Fax: (021) 3913933,HP: 08124232132

LEMBAR PERSETUJUAN

Hubungan antara asupan berbagai jenis lemak dengan hiperinsulinemia pada laki-laki dewasa dengan obesitas sentral

Peneliti :
dr. Andi Yasmin Syauki, Dr. Ir. Umi Fahmida, M.Sc, dr. Widjaja Lukito, Sp.GK, Ph.D, Andi Mariyasari Septiari, M.Sc, Dra. Indriyanti R.S, M.Si

SEAMEO-TROPMED Regional Center for Community Nutrition
University of Indonesia
Jl. Salemba Raya 6, Jakarta. Telepon: (021) 3909205; Fax: (021) 3913933

Setelah membaca dan mendengar penjelasan mengenai tujuan dan manfaat dari penelitian ini, maka dengan ini saya menyatakan bahwa saya:

1. Bersedia untuk mengikuti penelitian ini.
2. Bersedia mengikuti tahapan dalam penelitian ini.
3. Bersedia di wawancara mengenai kondisi identitas pribadi, riwayat kesehatan, karakteristik sosio-ekonomi, gaya hidup (kebiasaan merokok, konsumsi alkohol kebiasaan makan sayur dan buah, dan aktivitas fisik).
4. Bersedia diwawancarai mengenai asupan makanan lemak selama sehari serta asupan energi, karbohidrat terutama serat, dll selama 3 (tiga) hari yang telah ditentukan.
5. Bersedia diukur lingkar pinggang, tinggi badan, berat badan, tekanan darah, dan suhu tubuh.
6. Bersedia untuk diperiksa kadar glukosa, fungsi hati, fungsi ginjal, insulin, dan profil lemak dengan cara pengambilan darah yang dilakukan oleh tenaga terampil dari Laboratorium Klinik Prodia.

Dengan membubuhkan tanda tangan saya dibawah ini saya setuju untuk ikut berpartisipasi secara sukarela dan bersedia melaksanakan semua prosedur yang telah ditentukan dalam penelitian ini.

Jakarta, 2009

Pewawancara

(Nama lengkap)

Peserta Penelitian

(Nama lengkap)

Appendix 5

OFFICIAL PERMIT LETTER

Appendix 5 Official Permit Letter

PEMERINTAH PROVINSI DAERAH KHUSUS IBUKOTA JAKARTA

BADAN KESATUAN BANGSA

Jl. Medan Merdeka Selatan 8 - 9 Telp.3800590, 3822670 lokal 2070
Jakarta

Kode Pos 10110

Nomor : 346/1.502 18 Desember 2008
Sifat : Penting
Lampiran :
Hal : Izin penelitian

Kepada

Yth. Kepala Biro Administrasi Wilayah Setda
Provinsi DKI Jakarta
di
Jakarta

REKOMENDASI

Sehubungan dengan surat Deputi direktur Bidang Penelitian SEAMEO TROPMED Nomor : 100/SEAMEO-RES/XII/2008 Tanggal : 09 Desember 2008, hal surat ijin penelitian, dengan ini diberikan rekomendasi kepada:

Nama	: dr. Andi Yasmin
Alamat	: Jl. Sunu Komp.UNHAS Blok.AX No.18 Makasar
Pekerjaan	: Peneliti
No. Mahasiswa/KTP	: 21.5008.511180.0004
Tingkat	: -
Univ/fakultas/jurusan	: SEAMEO-TROPMED
Tujuan	: Penelitian berjudul " Hubungan antara asupan berbagai jenis lemak dengan hiperinsulinemia pada laki-laki dewasa dengan obesitas sentral di Jakarta "
Waktu	: Desember 2008 s.d. Februari 2009
Peserta	: 1 (satu) orang
Lokasi	: DKI Jakarta
Penanggung jawab	: Drg. Rosnani V.Pangaribuan,Dr.rer.nat

Untuk melakukan Penelitian dimaksud, dengan ketentuan :

1. Sebelum melakukan penelitian, terlebih dahulu melapor kepada pimpinan daerah/wilayah setempat.
2. Mematuhi peraturan-peraturan yang berlaku di daerah/wilayah setempat.
3. Tidak dibenarkan melakukan penelitian yang materinya bertentangan dengan topik/judul penelitian dimaksud.
4. Setelah selesai melakukan penelitian, segera melaporkan hasilnya kepada Gubernur Provinsi DKI Jakarta melalui Badan Kesatuan bangsa.

Demikian disampaikan untuk menjadi bahan lebih lanjut.

A.n.KEPALA BADAN KESATUAN BANGSA
PROVINSI DKI JAKARTA
KEPALA BIDANG PEMANTAPAN
KESATUAN BANGSA

Drs. TAFDIL, M.Si
NIP 470054155

Tembusan :
1. Gubernur Provinsi DKI Jakarta
2. Sekda Provinsi DKI Jakarta

Form – A12

Appendix 6

FOCUS GROUP DISCUSSION

GUIDELINE QUESTIONS FOR FOCUS GROUP DISCUSSION TO ELICIT FOODS USUALLY CONSUMED BY ADULT MEN (30-50 YEARS) IN JAKARTA (FOCUSING ON FAT RICH FOODS)

Date : _____
Name of sub-district : _____
Moderator : _____
Recorder : _____
Duration : _____
Number of participant : _____
Place of FGD : _____

CHARACTERISTIC OF RESPONDENTS

No	Name	Age	Last education	Occupation
1				
2				
3				
4				
5				
6				
7				
8				
9				
10				

General guidelines:

1. Introduce yourself and the purpose of meeting
2. Ask for their consent about using tape recorder to record the conversation
3. Let them know that they can speak freely and there is no wrong or right answer
4. Ask their name and let they introduce themselves
5. Use the guideline and probe the answer
6. Note the time (± 60 minutes)
7. Do not forget to ask them whether they have any additional comments and thank them afterwards
8. Do not forget to summarize and confirm the given answer to the participants

Guidelines questions :

1. What are you usually have for breakfast?
2. What are you usually have for lunch?
3. What are you usually have for dinner?
4. What are you usually have for snacks (morning, afternoon, and evening snacks)?

For questions no 1 to 4, probe :

a. When do you usually use margarine (brand)? How often do you use margarine? What kind of margarine do you usually use?

b. When do you usually use butter (brand)? How often do you use butter? What kind of butter do you usually use?

c. When do you usually eat fried food at home? How often do you eat fried food at home? What kind of food do you usually fried?

d. When do you usually eat fried food outside home? How often do you eat fried food outside home? What kind of fried food do you eat (fried chicken, fried fish, fried potato)?

e. When do you usually eat meat? How often do you usually consume meat? What kind of meat do you usually consume (beef, chicken, duck, lamb, bacon)? What part of meat do usually consume (leg, head, wing, liver)? How do the food prepare? Did you eat the skin too?

f. When do you usually eat egg? What kind of egg do you usually consume (chicken, duck, fish, bird)? What part do you usually consume? How often do you usually consume it?

g. When do you usually eat fish? How often do you usually consume fish? What kind of fish do you usually consume (kakap, kembung, lele, mas, mujair)?

h. When do you usually eat sea food (crabs, shellfish)? How often do you usually eat it?

i. When do you usually use oil? What kind of oils do you usually use (ask the brand)? How often do you use it?

j. When do you usually eat canned food (sausage, corned beef)? How often do you usually eat it?

k. When do you usually eat nuts? How often do you usually eat it? What kind of nuts do you usually eat ?

l. When do you usually eat baked cookies and cakes, salty snack foods such as chips? How often do you usually consume baked cookies and cakes, salty snack foods such as chips? What kind of baked cookies and cakes, salty snack (brand)?

QUESTIONNAIRE INTAKE LAST MONTH
ADULT MEN 30-50 YEARS

Name : _____

Age : _____

Last education : _____

Occupation : _____

Time	Name of meal	Food item	Portion size
Breakfast			
Morning snack			
Lunch			
Afternoon snack			
Dinner			
Supper			

QUESTIONNAIRE OF FAT INTAKE LAST MONTH
ADULT MEN 30- 50 YEARS

Name : _____
Age : _____
Last education : _____
Occupation : _____

In last month, what kind of food do you usually consume.. Give number in daily, weekly and monthly column and give ✓ if "never"					Usual portion size	Brand/other information
Food item	Daily	Weekly	Monthly	Nevr		
Margarine						
Butter						
Fried food at home :						
•						
•						
•						
•						
•						
•						
Fried food outside home :						
•						
•						
•						
•						
•						
•						
Chicken						
• Breast and wing						
• BreastDada						
• WingSayap						
• Thigh						
• Leg						
• Brain						
• Liver						
•						
•						
•						
Meat						
• Liver						
• Kidney						
•						
•						
•						

Appendix 7

QUESTIONNAIRES

Appendix 7 Questionnaires (1)

Screening's ID number [ ] A [ ][ ] B [ ][ ][ ] C

Repondent's ID number [ ] A [ ][ ] B [ ][ ][ ] C

SCREENING OF THE SUBJECT FOR STUDY OF RELATIONSHIP BETWEEN INTAKE OF FATTY ACIDS AND INSULIN RESISTANCE AMONG ABDOMINALOBESE ADULT MEN IN JAKARTA

South East Asian Ministers of Education Organization (SEAMEO)
Tropical Medicine and Public Health (TROPMED)
Regional Center for Community Nutrition (RCCN)
University of Indonesia (UI)
Jln. Salemba Raya 6 Jakarta Pusat
Phone : (021) 3913932 / 330205, Fax : (021) 3913933

INTERVIEWER AND DATE

Interviewer : 1. 2. 3. [ ]

Field supervisors : 1. dr. Andi Yasmin Syauki 2. Dra. Indriyanti Rafi Sukmawati, M.Si [ ]

Date of screening: [ ][ ] - [ ][ ] - [ ][ ][ ][ ] (dd - mm - year)

Date of completion: [ ][ ] - [ ][ ] - [ ][ ][ ][ ] (dd - mm - year)

Screening result : 1. Qualify 2. Not qualify [ ]

If the result is qualify, the subject will be the respondent of the study (fill the respondent's code and questionnaires)
If the result is not qualify, the subject will not be the respondent of the study (do not fill the respondent's code and questionnaires)

LOCATION AND SCREENING'S CODE

I. District Code : 1. Central Jakarta 2. South Jakarta 3. North Jakarta 4. West Jakarta 5. East Jakarta 6. Others ..... [ ]

II. Institution/company's code : [ ][ ]

III. Screening's code : [ ][ ][ ]

LOCATION AND RESPONDENT'S CODE

IV. District Code : 1. Central Jakarta 2. South Jakarta 3. North Jakarta 4. West Jakarta 5. East Jakarta 6. Others ..... [ ]

V. Institution/company's code : [ ][ ]

VI. Respondent's code : [ ][ ][ ]

Screening's ID number [ ] A [ ][ ] B [ ][ ][ ] C

Repondent's ID number [ ] A [ ][ ] B [ ][ ][ ] C

SUBJECTS IDENTITIES

A. Name : .....

B. Address : Jl.....

Municipality.....Post Code.....

C. Telephone : .....(Home)..... (seluler)

D. Birth date : [ ][ ] - [ ][ ] - [ ][ ][ ][ ]
dd mm years

E. Age : [ ][ ] years

MEDICAL HISTORY

1. Do you have disease recently? 1. Yes 2. No [ ]

2. Do you have history of disease? 1. Yes 2. No [ ]

3. If there are two answers of no, go to anthropomethric measurements
If there are one or two answers of yes, please fill the table below

Do you have one or more of diseases/disorders/behaviors below this?	(1) Yes (2) No	Information from : (1) medical record (2) respondent (3) others:.....	Time of disease : (1) Recently (< 1 month) (2) Pastly (> 1 month)
Diabetes mellitus			
Hypertension			
Kidney disease			
Liver disease			
Consume hypolipidemia drugs			

If there are all "NO" in the table, go to "ANTHROPOMETHRIC" measurements
If there is ONE OR MORE "YES" in the table, stop the screening (go to the CONSULTATION ROOM)

ANTHROPOMETHRIC ASSESSMENTS

WAIST CIRCUMFERENCE

4. Device ID for waist : [ ]

5. Waist circumference : Reading 1 [ ][ ][ ],[ ] in centimetres (cm)

Reading 2 [ ][ ][ ],[ ] in centimetres (cm)

Average [ ][ ][ ],[ ] in centimetres (cm)

If waist circumference is < 90 cm, stop the screening (go to the CONSULTATION ROOM)

Screening's ID number [ ] A [ ][ ] B [ ][ ][ ] C

Repondent's ID number [ ] A [ ][ ] B [ ][ ][ ] C

If waist circumference is ≥ 90 cm, continue to **HEIGHT, WEIGHT MEASUREMENTS AND CLINICAL ASSESSMENTS**

HEIGHT AND WEIGHT

6. Device IDs for height and weight: height [ ]
   weight [ ]

7. Height : Reading 1 [ ][ ][ ],[ ] in centimetres (cm)
   Reading 2 [ ][ ][ ],[ ] in centimetres (cm)
   Average [ ][ ][ ],[ ] in centimetres (cm)

8. Weight : Reading 1 [ ][ ][ ],[ ] in kilograms (kg)
   Reading 2 [ ][ ][ ],[ ] in kilograms (kg)
   Average [ ][ ][ ][ ] in kilograms (kg)

9. Body mass index (BMI) : [ ][ ][ ],[ ]

CLINICAL ASSESSMENTS

BLOOD PRESSURE

10. Device ID for blood pressure : [ ]

11. Blood pressure : [ ][ ][ ],[ ] systolic (mmHg)
    [ ][ ][ ],[ ] diastolic (mmHg)

12. During the past two weeks, have you been treated for raised blood pressure with drugs (medication) prescribed by a doctor or other health worker? [ ]
    1. Yes
    2. No

If **systolic pressure ≥ 140 mmHg** and **diastolic pressure ≥ 90 mmHg**, stop the screening (go to **CONSULTATION ROOM**)
If **systolic pressure < 140 mmHg** or **diastolic pressure < 90 mmHg**, go to **BODY TEMPERATURE** measurement

BODY TEMPERATURE

13. Device ID for temperature : [ ][ ]

14. Temperature : [ ][ ],[ ] in Celsius degree (°C)

Screening's ID number [ ] A [ ][ ] B [ ][ ][ ] C

Repondent's ID number [ ] A [ ][ ] B [ ][ ][ ] C

If body temperature is 36,5 - 37,1°C go to **BIOCHEMICHAL** measurement (blood taken by phlebotomist)
If body temperature is ≥ 37,2°C, **BIOCHEMICHAL** measurement **DELAY** for 2 days until body temperature is normal (go to **CONSULTATION ROOM**)

BIOCHEMICHAL ASSESSMENTS

15. During the last 10 hours have you had anything to eat or to drink, other than water? [ ]
    1. Yes
    2. No

16. Technician ID [ ]

17. Device ID : [ ]

18. Time of day blood specimen taken (24 hour clock) : [ ][ ] : [ ][ ]
    hrs mins

19. Fasting blood glucose : [ ][ ][ ] , [ ] in mmol/L

20. SGPT : [ ][ ][ ] , [ ] in mm/Ul

21. Creatinin : [ ][ ][ ] , [ ] in mg/dl

Finish the screening test

Screening 's identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's identity [ ] A [ ][ ] B [ ][ ][ ] C

SEMI- QUANTITAVE FOOD FREQUENCY QUESTIONNAIRE IN STUDY OF "RELATIONSHIP BETWEEN INTAKES OF DIFFERENT FATTY ACIDS AND INSULIN LEVEL IN ABDOMINAL OBESE ADULT MEN IN JAKARTA"

South East Asian Ministers of Education Organization (SEAMEO)
Tropical Medicine and Public Health (TROPMED)
Regional Center for Community Nutrition (RCCN)
University of Indonesia (UI)
Jln. Salemba Raya 6 Central Jakarta
Telephone : (021) 3913932 / 330205, Fax : (021) 3913933

INTERVIEWER AND DATE

Interviewer : 1. Risha Hakimah [ ]
2. Sriani
3. A. Yasmin Syauki
4. Desi

Supervisors :1. dr. Andi Yasmin Syauki [ ]
2. Dra. Indriyanti Rafi Sukmawati, M.Si

Date of interview : [ ][ ] - [ ][ ] - [ ][ ][ ][ ]
date month year

Interview result : 1. Complete [ ]
2. Uncomplete

LOCATION AND SCREENING'S CODE

A. District's code : 1. Central Jakarta 4. West Jakarta [ ]
2. South Jakarta 5. East Jakarta
3. North Jakarta 6. Other : .....

B. Institution's/company's code: 1. DIKTI 3. DKP [ ][ ]
2. BAPEPAM 4. Other :.....

C. Screening's code : [ ][ ][ ]

LOCATION AND SCREENING'S CODE

A. District's code : 1. Central Jakarta 4. West Jakarta [ ]
2. South Jakarta 5. East Jakarta
3. North Jakarta 6. Other .....

B. Institution's/company's code: 1. DIKTI 3. DKP [ ][ ]
2. BAPEPAM 4. Other :.....

C. Respondent's code : [ ][ ][ ]

Screening 's identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's identity [ ] A [ ][ ] B [ ][ ][ ] C

RESPONDENT'S IDENTITY

1. Name : .....

2. Age : [ ][ ] year

1. 30-39 year
2. 40-50 year

[ ]

3. Last education :
1. Non formal education
2. Ungraduated elementary school
3. Graduated elementary school
4. Graduated junior high school
5. Graduated senior high school
6. Bachelor degree
7. Master degree
8. Other : .....
88. Do not know

[ ]

4. Last occupation :
1. Civil servant
2. Private employee
3. Enterpreneur
4. Non- paid
5. Student
6. Home industry
7. Retired
8. Not working (able)
9. Not working (unable)
10. Other : .....
88. Do not know

[ ]

MEAL FREQUENCY

A. How many time do you eat in a day ?

Type of food	Frequency in a day
a. Main meal	
b.Snack	

B. How many time in a week do you eat ?

Food items	Frequency in a week
a. Dairy product	
b. Meat (beef, goat, chicken, other and the product)	
c. Egg (chicken, duck, quail and other)	
d. Fish and seafood	
e. Fried food as a snack	
f. Food contain coconut milk/peanut	

Screening 's identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's identity [ ] A [ ][ ] B [ ][ ][ ] C

GUIDELINE TO FILL :

a.Food which asked were food consumed in LAST ONE MONTH.

b.In FREQUENCY part, ask how often those food were consume, fill number in appropriate column (in a month, in a week and a day). Fill √ in colum never if the answer was never.

c. In the PORTION SIZE part, ask what was the average those food was usually consume. Use MEDIUM PORTION as REFERENCE OF PORTION SIZE, give √ in the colum of small (S), medium (M), large (L).

SMALL PORTION : ≤ ½ of MEDIUM PORTION

LARGE PORTION : ≥ 1½ of MEDIUM PORTION

COMPOSITE FOOD

	FREQUENCY How often did you consume in the last one month?				PORTION SIZE What was the average of those food do you usually consume ?					NOTE (Price, place and other)
	NEVER	A MONTH (1x-3x)	A WEEK (1x-6x)	A DAY (1x-6x)	MEDIUM PORTION	S	M	L	Other portion (mention)	
UDUK RICE (NASI UDUK)										If buy : - Price :IDR. - Place :
Coconut rice (nasi santan)					1 ½ rice ladle (150 g)					
Fried noodle rice (bihun goreng)					5 tbsp (50 g)					
Tofu cooked with soybean sauce/ fried tofu* (tahu semur/goreng)*					1 slice (31 g)					
Tempe cooked with soybean sauce/fried tempe* (tempe semur/goreng) *					1 slice (27 g)					
Tempe cooked with peanut and soybean sauce (tempe orek)					21/2 tbsp (43 g)					
Boiled egg/omelette/scrambled egg (telur rebus/dadar/orak arik)*					½ egg (25 g) -> egg with boiled					
Flour cooked with frying oil and egg (bakwan)					1 slice (36 g)					
Peanut sauce (saus kacang)					3 tbsp (30 g)					
Soybean sauce soup (kuah semur)					1 bowl (60 g)					
FRIED RICE (NASI GORENG)										If buy : - Price :IDR. - Place :
Fried rice and scrambled egg (nasi goreng dan telur orak arik					2 rice ladle (200 g)					
Fried sunny side egg /omelette* (telur ceplok/dadar)*					1 egg (60 g)					
Fried chicken with crispy flour (ayam goreng tepung)					1 slice, chicken breast meat(153 g)					
Chicken meat (daging ayam suir)					1 tbsp (12 g)					
Beef meat ball (bakso sapi)					1 tbsp (10 g)					
Tapioca starch, crisp (kerupuk aci)					1 package (24 g)					
MIXED VEGETABLES (GADO-GADO)										If buy : - Price :IDR. - Place :
Rice cake boiled in rhombus packedo of plaited young coconut leaves/rice stemed (ketupat/lontong)					1 piece (100 g)					
Fried tofu (tahu goreng)					1 slice (18,5 g)					
Fried tempe (tempe goreng)					1 slice (23 g)					
Fried peanut crisp (rempeyek)					1 piece (14 g)					
Boiled egg (irisan telur rebus)					½ egg (26 g)					
Fried crisp (kerupuk goreng)					1 package (20 g)					
Peanut sauce (saus kacang)					1 portion (149 g)					

Screening 's identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's identity [ ] A [ ][ ] B [ ][ ][ ] C

	FREQUENCY How often did you consume in the last one month ?				PORTION SIZE What was the average of those food do you usually consume ?					NOTE (Price, place and other)
	NEVER	A MONTH (1x-3x)	A WEEK (1x-6x)	A DAY (1x-6x)	MEDIUM PORTION	S	M	L	Other portion (mention)	
CHICKEN PORRIDGE (BUBUR AYAM)										If buy : - Price :IDR. - Place :
Porridge (bubur nasi)					1 bowl (280 g)					
Chicken meat (ayam suir)					3 tbsp (15 g)					
Flour cooked with frying oil (cakwe goreng)					3 tbsp(15 g)					
Yellow soybean (kacang kedelai)					1 tbsp (5 g)					
Peanut sauce (saus kacang)					2 tbsp (22 g)					
Chicken broth (kuah kaldu ayam)					7 tbsp (42 g)					
Chicken liver (hati ayam)					1 stick (22 g)					
Chicken intestinal (usus ayam)					1 coil (12 g)					
Tapioca starch crisp (kerupuk aci)					2 handful (20 g)					
FRIED NOOODLE (MIE GORENG)										If buy : - Price :IDR. - Place :
Fried noodle (mie goreng)					1 portion (200 g)					
Chicken meat (daging ayam suir)					31/2 tbsp (34 g)					
Scrambled egg (telur orak-arik)					21/2 tbsp (30 g)					
Beef meat ball (bakso sapi)					1 tbsp (12 g)					
Tapioca starch crisp (kerupuk aci)					11/2 handful (15 g)					
Fried shrimp (udang goreng)					1 portion (9 g)					
GUDEG RICE (NASI GUDEG)										If buy : - Price :IDR. - Place :
Rice (nasi putih)					1 plate (150 g)					
Coconut milk, jackfruit vegetable (sayur nangka santan)					1 tbsp (23 g)					
Chicken meat, breast and wing (daging ayam, dada dan sayap)					1 slice chicken thigh (75 g)					
Boiled egg (telur rebus)					1 egg(46,5 g)					
Coconut milk (kuah santan)					1 spoon ladle vegetable (30 g)					
Kulit crisp (kerupuk kulit)					1 portion (54 g)					
Krecek soup (kuah krecek)					1 spoon ladle vegetable (25 g)					
CHICKEN SOTO (SOTO AYAM)										If buy : - Price :IDR. - Place : - Coconut milk/soup*
Rice (nasi putih)					1 plate (150 g)					
Chicken meat (daging ayam suir)					31/2 tbsp (34 g)					
Fried potato (kentang goreng)					21/2 tbsp (25 g)					
Boiled egg (irisan telur rebus)					½ egg (32 g)					
Coconut milk (kuah santan)					1 bowl (249 g)					
Soup (kuah bening)					1 bowl (367 g)					
Jointfir spinach crisp (kerupuk emping)					1 package (23 g)					
BETAWIAN SOTO (SOTO BETAWI)										If buy : - Price :IDR. - Place : - Coconut milk/soup*
Rice (nasi putih)					1 plate (150 g)					
Beef meat (daging sapi)					2 tbsp (30 g)					
Beef tripe (babat sapi)					1 tbsp (14 g)					
Beef lung (paru sapi)					1 tbsp (13 g)					
Beef feet (kaki sapi)					2 tbsp (36 g)					
Beef intestinal (usus sapi)					1 tbsp (13 g)					
Fried potato (kentang goreng)					3 tbsp (30 g)					
Coconut milk (kuah santan)					1½ glasses (300 g)					
Jointfir spinach crisp (krupuk emping)					1 package (18 g)					

Screening 's identity [ ] [ ][ ] [ ][ ][ ]
A B C

Responden's identity [ ] [ ][ ] [ ][ ][ ]
A B C

	FREQUENCY How often did you consume in the last one month ?				PORTION SIZE What was the average of those food do you usually consume ?					NOTE (Price, place and other)
	NEVER	A MONTH (1x-3x)	A WEEK (1x-6x)	A DAY (1x-6x)	MEDIUM PORTION	S	M	L	Other portion (mention)	
FRIED FOOD AS MAIN MEAL										
Fried tempe (tempe goreng)					1 slice (31 g)					
Fried tofu (tahu goreng)					1 slice (47 g)					
Tempe cooked with peanut and soybean sauce (tempe orek)					1 portion (83,5 g)					
Flour cooked with frying oil, maize and egg (bakwan jagung)					1 slice (29 g)					
Flour cooked with frying oil, shrimp and egg (bakwan udang)					1 slice (27 g)					

Screening 's identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's identity [ ] A [ ][ ] B [ ][ ][ ] C

DAIRY PRODUCTS

	FREQUENCY How often did you consume in the last one month ?				PORTION SIZE What was the average of those food do you usually consume ?					NOTE (Brand, flavor and other)
	NEVER	A MONTH (1x-3x)	A WEEK (1x-6x)	A DAY (1x-6x)	MEDIUM PORTION	S	M	L	Other portion (mention)	
Butter (add in food or in a bread), not used for cooking (mentega (tambahan pada makanan atau pada roti), diluar pemakaian untuk memasak)					½ tbsp (5 g)					Brand:
Margarine(add in food or in a bread), not used for cooking (margarin (tambahan pada makanan atau pada roti), diluar pemakaian untuk memasak)					½ tbsp(5 g)					Brand :
Milk cow powder (susu bubuk (sapi))					2 ½ tbsp (45 g)					Brand : Flavor:
Energy drink (chocolate flavour) i.e Milo or ovaltine (minuman berenergi (rasa coklat) seperti milo atau ovaltine)					powder: 2 tbsp (20 g)					Brand :
					box : 1 box 250 ml					
					sachet : 1 sachet (14 g) (regular Milo)					
					sachet : 1 sachet (35 g) (Milo 3 in 1)					
Milk, cow,sweetened, condensed (susu kental manis)					can : 3 tbsp (30 g)					Brand : Flavor :
					sachet : 1 sachet (42 g) (plain frisian flag)					
Food or drink contain milk or dairy product (creamer in coffee, mayoinese in a burger, other) consume minimally ≥ once a week										
	[redacted]									

Screening 's identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's identity [ ] A [ ][ ] B [ ][ ][ ] C

MEAT

	FREQUENCY How often did you consume in the last one month ?				PORTION SIZE What was the average of those food do you usually consume ?					NOTE (Part, price, place, fried/boiled, other)
	NEVER	A MONTH (1x-3x)	A WEEK (1x-6x)	A DAY (1x-6x)	MEDIUM PORTION	S	M	L	Other portion (mention)	
CHICKEN MEAT (DAGING AYAM)										
Fried chicken (ayam goreng pecel)					1 slice, breast (113 g)					Part :
Fried chicken (beside fried chicken in pecel) (ayam goreng tanpa tepung (selain pecel ayam))					1 slice , breast (113 g)					Part :
Fried chicken with crispy flour (ayam goreng tepung)					1 slice, breast (122 g)					Part :
Chicken roasted (ayam bakar)					1 slice breast and wing (120 g)					Part:
					1slice thigh (85 g)					
Chicken soup (sup ayam) : - chicken meat (ayam suir)					1 portion (56g)					If buy : - Price :IDR. - Place :
					1 portion (5 g) Mc Donald					
- Chicken feet (ceker)					1 slice (5 g)					
- Soup (kuah sup ayam)					1 glasss (142 g)					
Chicken satay (sate ayam) : -chicken meat (daging ayam)					10 sticks (80 g)					with/without skin*
- peanut sauce (saus kacang)					1 tbsp (17 g)					
Chicken curry (gulai ayam): - chicken meat (ayam)					1 slice thigh (65 g)					If buy : - Price :IDR.
- Coconut milk (kuah santan)					1 bowl (64 g)					- Place :
Chicken gizzard (rempelo ayam)					1 slice (16 g)					Fried/other^ Other :
Chicken brains (otak ayam)					1 slice (60 g)					Fried/other^ Other :
Other food contain chicken and other part of it (intestine, liver, other) consume minimally ≥ once in a week										
BEEF MEAT (DAGING SAPI)										
Sausages (sosis)					1 little slice (23,5 g)					fried/roasted/boiled*
Meat simerred in spice and coconut milk (rendang)					1 slice (27 g)					fried/roasted/boiled*
Rendang sauce (saus rendang)					1 tsp (17 g)					
Beef meat ball (bakso sapi)					5 meat balls (75 g)					fried/roasted/boiled*
					1 big meat ball (32 g)					
					1 meat ball + egg (81 g)					
Corned beef (komet sapi)					1 tsp (15g)					fried/roasted/boiled*
Comet in a burger (komet di burger)					1 slice (33 g)					fried/roasted/boiled*
Tripe soto (soto babat): - tripe (babat					2 slices (80 g)					If buy : - Price :IDR. - Place :
- Coconut milk (kuah santan)										

Screening 's identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's identity [ ] A [ ][ ] B [ ][ ][ ] C

	FREQUENCY How often did you consume in the last one month ?				PORTION SIZE What was the average of those food do you usually consume ?					NOTE
	NEVER	A MONTH (1x-3x)	A WEEK (1x-6x)	A DAY (1x-6x)	MEDIUM PORTION	S	M	L	Other portion (mention)	
Tail soup (sop buntut) : - tail meat (buntut)					1 portion (196 g)					If buy : - Price :IDR. - Place :
-soup (sop)					11/2 glasses (300 g)					
Other food contain beef meat and other part (abon, semur, burger, steak, other) consume minimally ≥ once in a week										
GOAT MEAT AND OTHERS (DAGING KAMBING DAN DAGING LAINNYA)										
Goat tongseng (tongseng kambing) : - goat meat (daging kambing)					1 portion (50 g)					If buy : - Price :IDR. - Place :
- coconut milk (kuah santant)					11/2 glasses (182 g)					
Goat satay (sate kambing): -goat meat (daging kambing)					10 sticks (90 g)					If buy : - Price :IDR. - Place :
-peanut sauce (saus kacangO					1 portion (17 g)					
Other food contain goat meat or other meat (beacon, other) consume minimally ≥ once in a week										

Screening 's Identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's Identity [ ] A [ ][ ] B [ ][ ][ ] C

EGG (BESIDE IN THE MAIN MEAL)

	FREQUENCY How often did you consume in the last one month ?				PORTION SIZE What was the average of those food do you usually consume ?					NOTE (with or without n-3, n-6 PUFA, other)
	NEVER	A MONTH (1x-3x)	A WEEK (1x-6x)	A DAY (1x-6x)	MEDIUM PORTION	S	M	L	Other portion (mention)	
CHICKEN EGG (TELUR AYAM)										
Chicken egg in instant noodle (telur ayam di mie instant)					1 egg (60 g)					broiler/local* regular/low cholesterol*
Sauce chicken egg (telur ayam balado)					1 egg (60 g)					broiler/local* regular/low cholesterol*
Boiled chicken egg (telur ayam rebus)					1 egg (60 g)					broiler/local* regular/low cholesterol*
Fried sunny side egg (telur ayam mata sapi)					1 egg (50 g)					broiler/local* regular/low cholesterol*
Omelette chicken egg (telur ayam dadar)					1 egg (64 g)					broiler/local* regular/low cholesterol*
Chicken egg yolk (kuning telur ayam mentah)					1 egg (15 g)					broiler/local* regular/low cholesterol*
Other food contain chicken egg consume minimally ≥ once in a week (egg in siomay, egg in pempek)										
										broiler/local* regular/low cholesterol*
										broiler/local* regular/low cholesterol*
										broiler/local* regular/low cholesterol*
DUCK EGG (TELUR BEBEK)										
Saltened duck egg (telur bebek asin)					1 egg (66,4 g)					Cooking method :
Duck egg (telur bebek)					1 egg (66,4 g)					Cooking method :
Other food contain duck egg consume minimally ≥ once in a week										
										Cooking method :
										Cooking method :
QUAIL EGG					1 egg (10 g)					Cooking method :
Other food contain quail egg consume minimally ≥ once in a week										
										Cooking method :
										Cooking method :
Other egg (se turtle egg,fish egg, other) consume minimally ≥ once in a week										

Screening 's identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's identity [ ] A [ ][ ] B [ ][ ][ ] C

FISH, SHRIMP AND OTHER ANIMAL SEA

	FREQUENCY How often did you consume in the last one month ?				PORTION SIZE What was the average of those food do you usually consume ?					NOTE (method of cooking, other)
	NEVER	A MONTH (1x-3x)	A WEEK (1x-6x)	A DAY (1x-6x)	MEDIUM PORTION	S	M	L	Other portion (mention)	
MARINE FISH (IKAN AIR LAUT)										
Sardines fish (ikan sarden)					1 slice (26,4 g)					fried/roasted/ boiled/other* Other :
Mackerel fish (ikan kembung)					1 slice (52,3 g)					fried/roasted /boiled/other* Other :
King mackerel fish (ikan tengiri)					1 slice, body (108 g)					fried/roasted/ boiled/other* Other :
Anchovy dried fish (ikan teri nasi)					1 tbsp(4 g)					fried/roasted/ boiled/other* Other :
Anchovy fresh fish (ikan teri)					1 portion (33 g)					fried/roasted/ boiled/other* Other :
Tuna fish (ikan tongkol)					1 slice (61,5 g)					fried/roasted/ boiled/other* Other :
Sea perch fish (ikan kakap merah)					1 slice (358 g) 1slice, tail (104 g) 1 slice, head (164 g)					fried/roasted/ boiled/other* Other :
Gray mullet fish (ikan belanak)					1 slice (97 g)					fried/roasted/ boiled/other* Other :
Marine fish or their product that contain marine fish consume minimally ≥ once in a week										
										fried/roasted/ boiled/other* Other :
										fried/roasted/ boiled/other* Other :
										fried/roasted/ boiled/other* Other :
										fried/roasted/ boiled/other* Other :
										fried/roasted/ boiled/other* Other :

Screening 's identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's Identity [ ] A [ ][ ] B [ ][ ][ ] C

	FREQUENCY How often did you consume in the last one month ?				PORTION SIZE What was the average of those food do you usually consume ?					NOTE (method of cooking, other)
	NEVER	A MONTH (1x-3x)	A WEEK (1x-6x)	A DAY (1x-6x)	MEDIUM PORTION	S	M	L	Other portion (mention)	
FRESH WATER FISH (IKAN AIR TAWAR)										
Catfish (ikan lele)					1 slice (71 g)					fried/roasted/ boiled/other* Other :
Carp fish (ikan mas)					1 slice (79 g)					fried/roasted/ boiled/other* Other :
Milk-fish (ikan bandeng)					1 slice head and body (63 g)					fried/roasted/ boiled/other* Other :
					1 slice body and tail (51 g)					
Snakehead fish (ikan gabus)					1 slice (20 g)					fried/roasted/ boiled/other* Other :
Fresh water fish or their product consume minimally ≥ once in a week (tilapia fish, pompret black fish, gouramy fish, other)										
										fried/roasted/ boiled/other* Other :
										fried/roasted/ boiled/other* Other :
										fried/roasted/ boiled/other* Other :
Shrimp (udang)					1 tbsp (medium) 4 pieces (20 g)					fried/roasted/ boiled/other* Other :
Clam (kerang)					1 tbsp (7 r)					fried/roasted/ boiled/other* Other :
Crab (fresh water) (kepiting)					1 portion (50 g)					fried/roasted/ boiled/other* Other :
Squid (cumi-cumi)					1 small piece (15 g)					fried/roasted/ boiled/other* Other :
Other animal sea that consume minimally ≥ once in a week										
										fried/roasted/ boiled/other* Other :
										fried/roasted/ boiled/other* Other :
										fried/roasted/ boiled/other* Other :

Screening 's identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's identity [ ] A [ ][ ] B [ ][ ][ ] C

BAKED FOOD, SALTED SNACK, FRIED FOOD SNACK AND OTHER

	FREQUENCY How often did you consume in the last one month				PORTION SIZE What was the average of those food do you usually consume ?					NOTE : (flavor, price, material, brand, other)
	NEVER	A MONTH (1x-3x)	A WEEK (1x-6x)	A DAY (1x-6x)	MEDIUM PORTION	S	M	L	Other portion (mention)	
Pastry (kue kering)					4 pieces (60 g)					Flavor :
Sponge cake (kue bolu)					(B) 2 slices (50 g)					Price : IDR Flavor :
					(M) 1 slice (25 g)					Flavor :
Fried chips (krupuk goreng)					1 package (white) (15 g) 1 piece of shrimp crisp (17 g)					Material : Price : IDR
Fried tempe with crispy flour (tempe goreng tepung)					(B) 2 slices (52 g)					Price : IDR
					(M)) 3 slices (78 g)					
Fried tofu with crispy flour (tahu goreng tepung)					(B) 2 slices (52 g)					Price : IDR
					(M) 2 slices (52 g)					
Fried banana (pisang goreng)					(B) 3 slices (156 g)					Price : IDR
					(M) 3 slices (156 g)					
Fried bakwan (bakwan goreng)					(B) 2 slices (50 g)					
					(M) 3 slices (75 g)					
Cassava raw crisp (rengginang)					1 piece (23 g)					Price : IDR
Frying flour with milk, egg, margarine (risol)					2 slices (62 g)					
Pasty (pastel)					1 slice (65 g)					Price : IDR
Glutionous rice flour cover with grated coconut (onde-onde)					1 slice (63 g)					
Cassava crisp (keripik singkong)					1 package (60 g)					
Peanut (kacang tanah)					2 packages (54 g)					Brand:
Crackers, i.e Malkist, Jacob's, Gabin,other (kraker seperti Malkist, Jacob's dan lainnya)					1 small package (80 g) -> Jacobs					Brand :
Other baked food,salted snack or other fried food (bread with substance, cucur, molen banana, pancong cake, sukro peanut, martabak fried rice glutinous, chitato, batagor, fried sweet potato, fried emplang, combro, pangsit, misro, muffin, peanut) minimally consume ≥ once a week										

Note :

* : circle the answer

B : buy in street vendor

M : made

Screening 's identity [ ] A [ ][ ] B [ ][ ][ ] C

Responden's identity [ ] A [ ][ ] B [ ][ ][ ] C

OTHER FOOD CONTAIN FAT (COCONUT MILK AND PEANUT)

	FREQUENCY How often did you consume in the last one month				PORTION SIZE What was the average of those food do you usually consume ?					NOTE
	NEVER	A MONTH (1x-3x)	A WEEK (1x-6x)	A DAY (1x-6x)	MEDIUM PORTION	S	M	L	Other portion (mention)	
VEGETABLE										
Coconut milk , vegetable dish (lodeh)					1 spoon landle vegetable (175 g)					liquid/thick
Peanut , vegetable dish (asem)					1 tbsp (10 g)					
Other vegetable contain coconut milk consume minimally ≥ once in a week (coconut milk in cassava leaves vegetable, other)										
OTHER CAKE/PORRIDGE										
Coconut milk in bean mung porridge					1 portion (65 g)					encer/kental
Other cake contain coconut milk (pudding, other) consume minimally ≥ once a week										

a.What do you do with the visible fat on your meat ?
1. eat most of it
2. eat some of it
3. eat as little as possible
4. not consume at all

[ ]

b. If you see meat with the skin, what do you do?
1. take ou the skin before eat
2. do not take out the skin before eat

[ ]

c. What kind of fat do you usually use for :
Choice : 1. palm oil
2. soybean oil
3. corn oil
4. canola oil
5. butter
6. margarine
7. Other : _____

Type of cooking	Type of oil
- baked	
- frying	

d. What kind of oil do you usually use for frying? _____(brand)

e Do you use a microvawe oven?
1. Yes, if yes for how many years....., what do you use for?.....
2. No

[ ]

f Are you currently on a special diet?
1. Yes, if yes for how many years/month.....and type of diet.....
2. No

[ ]

g. Did you consume multivitamin?

	Yes (1), If yes, what is the brand	No(2)
Multiple vitamin,		
Vitamin A		
Vitamin C		
Vitamin E		

[ ]
[ ]
[ ]
[ ]

---------- finish SQ-FFQ----------

Screening's code ☐ (A) ☐☐ (B) ☐☐☐ (C)

Repondent's code ☐ (A) ☐☐ (B) ☐☐☐ (C)

QUESTIONNAIRE STUDY OF RELATIONSHIP BETWEEN INTAKE OF FATTY ACIDS AND INSULIN RESISTANCE AMONG ABDOMINAL OBESE ADULT MEN IN JAKARTA

South East Asian Ministers of Education Organization (SEAMEO)
Tropical Medicine and Public Health (TROPMED)
Regional Center for Community Nutrition (RCCN)
University of Indonesia (UI)
Jln. Salemba Raya 6 Jakarta Pusat
Phone : (021) 3913932 / 330205, Fax : (021) 3913933

INTERVIEWER AND DATE

Interviewer : 1. 2. 3. ☐

Field supervisors : 1. dr. Andi Yasmin Syauki 2. Dra. Indriyanti Rafi Sukmawati, M.Si ☐

Date of interview : ☐☐-☐☐-☐☐☐☐ (dd mm year)

Date of completion: ☐☐-☐☐-☐☐☐☐ (dd mm year)

LOCATION AND SCREENING'S CODE

A. District Code : 1. Central Jakarta 2. South Jakarta 3. North Jakarta 4. West Jakarta 5. East Jakarta 6. Others : ..... ☐

B. Subdistrict code : ☐☐

C. Screening's code : ☐☐☐

LOCATION AND RESPONDENT'S CODE

A. District Code : 1. Central Jakarta 2. South Jakarta 3. North Jakarta 4. West Jakarta 5. East Jakarta 6. Others : ..... ☐

B. Subdistrict code : ☐☐

C. Respondent's code ☐☐☐

Screening's code [ ] [ ][ ] [ ][ ][ ]
A B C

Repondent's code [ ] [ ][ ] [ ][ ][ ]
A B C

CONSENT, INTERVIEW LANGUANGE, AND TIME OF INTERVIEW

D. Consent has been read out to the respondent : 1. Yes
2. No, If NO read out the informed consent [ ]

E. Consent has been obtained writtenly : 1. Yes
2. No, If NO, END [ ]

F. Interview languange : 1. Bahasa
2. Local languange :..... [ ]

G. Time of interview : .....hrs [ ]

.....mins [ ][ ]

RESPONDENT CHARACTERISTICS AND DEMOGRAPHIC INFORMATION

1. Family name : .....
2. First name : .....
3. Address : Jl.....
Municipality.....Post Code.....
4. Telephone : .....(Home)..... (seluler)
5. What is your birth date? : [ ][ ] - [ ][ ] - [ ][ ][ ][ ]
dd mm years
6. How old are you? : [ ][ ] years
7. What is your ethinicity? : 1. Betawian 5. Sumateranese [ ]
2. Javanese 6. Others :.....
3. Sundanese 88. Refused
4. Buginese
8. What is highest level of education you have completed ? [ ]
1. No formal schooling 5. High school completed
2. Less than primary school 6. College/University completed
3. Primary school completed 7. Post-graduate degree
4. Secondary school completed 88. Refused
9. Which of the following best describes your main work status over the last 12 months? [ ]
1. Government employee 7. Retired
2. Non-government employee 8. Unemployed (able to work)
3. Self-employed 9. Unemployed (unable to work)
4. Non- paid 88. Refused
5. Student
6. Homemaker

Screening's code	A	B	C
Repondent's code	A	B	C

10. What is your marital status? : 1. Married 3. Single
2. Divorced

11. Taking the past year, can you tell me an estimate of the annual household income? (read the options)
1. > IDR 10.000.000
2. IDR 5.000.000 – IDR 10.000.000
3. IDR 2.000.000 – < IDR 5.000.000
4. IDR 1.000.000 – <IDR 2.000.000
5. < IDR 1.000.000

10. Taking the past year, can you tell me an estimate of the annual household/single expediture?
1. Food : .....
2. Non food : .....

TOBACCO USE

Now I am going to ask you some questions about various health behaviours. This includes things like smoking, drinking alcohol, eating fruits and vegetables and physical activity. Let's start with tobacco.

11. Do you currently smoke any tobacco products, such as cigarettes,cigars, or pipes?
1. Yes
2. No, if no go to # 16

12. If yes, Do you currently smoke tobacco products daily?
1. Yes
2. No, if no go to 16

13. How old were you when you first started smoking daily?
1. .....years, if know go to # 15
77. Do not remember

14. Do you remember how long ago it was? (record only one not all three)
1. .....(in years) if know go to # 15
or 2. .....(in months) if know go to # 15
or 3. .....(in weeks)
77. Do not remember

15. On average, how many of the following do you smoke each day? (record for each type)
1. manufacutered cigarettes
2. hand-rolled cigarettes
3. pipes full or tobacco
4. cigars, cheroots, cigarillos
5. others .....

16. In the past, did you ever smoke daily?
1. Yes
2. No, if no go to # 19

17. If Yes, How old were you when you stopped smoking daily?
1. .....(age) years, if know go to # 19
77. Do not know

Screening's code [ ] [ ][ ] [ ][ ][ ]
A B C

Repondent's code [ ] [ ][ ] [ ][ ][ ]
A B C

18. How long ago did you stop smoking daily?
1. ..... years ago, if know go to # 19
or 2. .....months ago, if know go to # 19
or 3. .....weeks ago
77. Do not know

19. Do you currently use any smokeless tobacco such as [snuff, chewing tobacco, betel]?
1. Yes
2. No, if no go to # 22

20. If Yes,Do you currently use smokeless tobacco products daily?
1. Yes
2. No, if no go to # 22

21.On average, how many times a day do you use ? (record for each type)
1. snuff, by mouth
2. snuff, by nose ....
3. chewing tobacco
4. betel, quid
5. other.....
77. Do not know

22. In the past, did you ever use smokeless tobacco such as [snuff, chewing tobacco, or betel] daily?
1. Yes
2. No, if no go to # 22

ALCOHOL CONSUMPTION

The next questions ask about the consumption of alcohol.

23. Have you consumed alcohol (such as beer, wine, spirits,fermented cider or [add other local examples] within the past 12 months?
1. Yes
2. No, if no go to # 30

24. In the past 12 months, how frequently have you had at least one drink?
1. Daily
2. 5-6 days per week
3. 1-4 days per week
4. 1-3 days per month
5. Less than once a month
66. Not applicable

25. When you drink alcohol, on average, how many drinks do you have during one day?
1. .....numbers
66. Not applicable
77. Do not know

26. Have you consumed alcohol (such as beer, wine, spirits,fermented cider or [add other local examples] within the past 30 days?
1. Yes
66. Not applicable
2. No, if no go to # 28

27. During each of the past 7 days, how many standard drinks of any alcoholic drink did you have each day?
1. Monday
2. Tuesday
3. Wednesday
4. Thursday

Screening's code ☐ ☐☐ ☐☐☐ (A B C)

Repondent's code ☐ ☐☐ ☐☐☐ (A B C)

5. Friday ☐☐

6. Saturday ☐☐

7. Sunday ☐☐

66. Not applicable ☐☐

77. Do not know

28. In the past 12 months, what was the largest number ofdrinks you had on a single occasion, counting all types of standard drinks together? ☐☐

1. .....largest numbers 66. Not applicable

77. Do not know

29. In the past 12 months, on how many days did you have five or more standard drinks in a single day? ☐☐

1. .....numbers of days 66. Not applicable

77. Do not know

DIET

The next questions ask about the fruits and vegetables that you usually eat. I have a nutrition card here that shows you some examples of local fruits and vegetables. Each picture represents the size of a serving. As you answer these questions please think of a typical week in the last year.

30. In a typical week, on how many days do you eat fruit? (☐☐

1. .....numbers of days (If zero days, go to # 32)

77. Do not know

31. How many servings of fruit do you eat on one of those days? ☐☐

1. .....numbers of servings

77. Do not know

32. In a typical week, on how many days do youeat vegetables? ☐☐

1. .....numbers of days (If zero days, go to # 34)

77. Do not know

33. How many servings of vegetables do you eat on those days? ☐☐

1. .....numbers of servings

77. Do not know

PHYSICAL ACTIVITY

Next I am going to ask you about the time you spend doing different types of physical activity in a typical week. Please answer these questions even if you do not consider yourself to be a physically active person. Think first about the time you spend doing work. Think of work as the things that you have to do such as paid or unpaid work, study/training, household chores, harvesting food/crops, fishing or hunting for food, seeking employment. [Insert other examples if needed]. In answering the following questions 'vigorous-intensity activities' are activities that require hard physical effort and cause large increases in breathing or heart rate, 'moderate-intensity activities' are activities that require moderate physical effort and cause small increases in breathing or heart rate.

Screening's code [ ] A [ ][ ] B [ ][ ][ ] C

Repondent's code [ ] A [ ][ ] B [ ][ ][ ] C

ACTIVITY WORK

34. Does your work involve vigorous-intensity activity that causes large increases in breathing or heart rate like [carrying or lifting heavy loads, digging or construction work] for at least 10 minutes continuously?
    1. Yes
    2. No, if no go to # 37 [ ]
35. In a typical week, on how many days do you do vigorous intensity activities as part of your work?
    1. .....number of days [ ][ ]
    77. Do not know
36. How much time do you spend doing vigorous-intensity activities at work on a typical day?

    Hours : minutes [ ][ ] : [ ][ ]
    hrs mins
37. Does your work involve moderate-intensity activity, that causes small increases in breathing or heart rate such as brisk walking [or carrying light loads] for at least 10 minutes continuously?
    1. Yes
    2. No, if no go to # 40
38. In a typical week, on how many days do you do moderateintensity activities as part of your work?
    1. .....number of days [ ][ ]
    77. Do not know
39. How much time do you spend doing moderate-intensity activities at work on a typical day?

    Hours : minutes [ ][ ] : [ ][ ]
    hrs mins

TRAVEL TO AND FROM PLACES

The next questions exclude the physical activities at work that you have already mentioned.
Now I would like to ask you about the usual way you travel to and from places. For example to work, for shopping, to market, to place of worship. [insert other examples if needed]

40. Do you walk or use a bicycle (pedal cycle) for at least 10 minutes continuously to get to and from places?
    1. Yes
    2. No, if no go to # 43 [ ]
41. In a typical week, on how many days do you walk or bicycle for at least 10 minutes continuously to get to and from places? Number of days #$& P8 [ ]
    1. .....number of days
    77. Do not know
42. How much time do you spend walking or bicycling for travel on a typical day?

    Hours : minutes [ ][ ] : [ ][ ]
    hrs mins

RECREATIONAL ACTIVITIES

The next questions exclude the work and transport activities that you have already mentioned.
Now I would like to ask you about sports, fitness and recreational activities (leisure), [insert relevant terms].

43. Do you do any vigorous-intensity sports, fitness or recreational (leisure) activities that cause large increases in breathing or heart rate like [running or football,] for at least 10 minutes continuously?
    1. Yes
    2. No, if no go to # 46 [ ]

Screening's code ☐ (A) ☐☐ (B) ☐☐☐ (C)

Repondent's code ☐ (A) ☐☐ (B) ☐☐☐ (C)

44. In a typical week, on how many days do you do vigorousintensity sports, fitness or recreational (leisure) activities? ☐☐
1. .....number of days
77. Do not know

RECREATIONAL ACTIVITIES

45. How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?
Hours : minutes ☐☐ (hrs) : ☐☐ (mins)

46. Do you do any moderate-intensity sports, fitness or recreational (leisure) activities that causes a small increase in breathing or heart rate such as brisk walking,(cycling, swimming, volleyball) for at least 10 minutes continuously? ☐
1. Yes
2. No, if no go to #

47. In a typical week, on how many days do you do moderate-intensity sports, fitness or recreational (leisure) activities? ☐☐
1. .....number of days
77. Do not know

48. How much time do you spend doing moderate-intensity sports, fitness or recreational (leisure) activities on a typical day?
Hours : minutes ☐☐ (hrs) : ☐☐ (mins)

SEDENTARY BEHAVIOUR

The following question is about sitting or reclining at work, at home, getting to and from places, or with friends including time spent [sitting at a desk, sitting with friends, travelling in car, bus, train, reading, playing cards or watching television], but do not include time spent sleeping.

49. How much time do you usually spend sitting or reclining on a typical day?
Hours : minutes ☐☐ (hrs) : ☐☐ (mins)

Finish the questionnaire

Appendix 8

OTHER RESULTS

Table Correlation Coefficient between Total Fat, SFA, MUFA, PUFA Intake as (g) and (percentage of total calories) in Abdominal Obese Adult Men in Jakarta

	Coefficient correlation (r)							
	Total fat intake (g)	SFA intake (g)	MUFA intake (g)	PUFA intake (g)	Total fat intake (% of total calories)	SFA intake (% of total calories)	MUFA intake (% of total calories)	PUFA intake (% of total calories)
Total fat intake (g)	1.000							
SFA intake (g)	0.961**	1.000						
MUFA intake (g)	0.921**	0.801**	1.000					
PUFA intake (g)	0.851**	0.718**	0.833**	1.000				
Total fat intake (% of total calories)	0.391**	0.418**	0.285**	0.295**	1.000			
SFA intake (% of total calories)	0.348**	0.518**	0.105	0.052	0.797**	1.000		
MUFA intake (% of total calories)	0.127	-0.018	0.374**	0.194*	0.481**	-0.019	1.000	
PUFA intake (% of total calories)	0.152	0.004	0.187*	0.566**	0.394**	-0.088	0.418**	1.000

* significant correlation at p<0.05, Pearson correlation test
** significant correlation at p<0.01,Pearson correlation test

Food Sources of Different Fatty Acid Intake among Abdominal Obese Adult Men in Jakarta

Table 1. Food Sources of Total Fat Intake

No	Food	Total fat (g)
1	*Ayam goreng* (fried chicken)	9,55
2	*Kacang sukro* (fried peanut)	7,93
3	*Saus kacang* (peanut sauce)	7,75
4	*Daun singkong* (coconut milk and vegetable)	7,72
5	*Ikan bawal goreng* (fried pompret black fish)	7,28
6	*Lodeh* (coconut milk and vegetable)	6,94
7	*Donat* (doughnut)	6,50
8	*Semur daging ayam* (chicken cooked in sweet-soybean sauce)	6,20
9	*Nasi goreng* (fried rice)	6,00
10	*Tempe goreng* (fried tempe)	4,96

Table 2. Food Sources of SFA Intake

No	Food	SFA intake (g)
1	*Lodeh* (coconut milk and vegetable)	6,16
2	*Ikan bawal goreng* (fried pompret black fish)	5,88
3	*Ayam goreng* (fried chicken)	4,86
4	*Semur daging ayam* (chicken cooked in sweet-soybean sauce)	4,80
5	*Ikan gurami goreng* (fried gouramy fish)	3,37
6	*Kuah semur* (soybean sauce)	2,97
7	*Ikan tuna goreng* (fried tuna fish)	2,92
8	*Bakwan goreng* (frying oil and flour)	2,86
9	*Tahu goreng* (fried tofu)	2,85
10	*Kuah santan soto ayam* (coconut milk and chicken soup)	2,56

Table 3. Food Sources of MUFA Intake

No	Food	MUFA intake (g)
1	*Saus kacang* (peanut sauce)	3,87
2	*Kacang sukro* (fried peanut)	3,50
3	*Ayam goreng* (fried chicken)	2,41
4	*Daging bebek* (duck meat)	1,80
5	*Santan sayur daun singkong* (coconut milk and s vegetable)	1,72
6	*Donat* (doughnut)	1,60
7	*Nasi goreng* (fried rice)	1,55
8	*Pepes ikan mas* (steamed carp fish)	1,20
9	*Batagor* (fried meat ball and tofu)	1,03
10	*Kacang tanah dengan kulit* (peanut with shell)	0,93

Table 4. Food Sources of PUFA Intake

No	Food	PUFA intake (g)
1	*Donat* (doughnut)	3,60
2	*Nasi goreng* (fried rice)	3,15
3	*Kacang sukro* (fried peanut)	2,93
4	*Saus kacang* (peanut sauce)	2,45
5	*Kentang goreng* (potato crisps)	2,40
6	*Martabak* (frying oil, egg and vegetable)	1,82
7	*Tempe goreng* (fried tempe)	1,81
8	*Uli goreng* (frying oil, flour rice)	1,60
9	*Kue pati singkong goreng* (frying oil and cassava)	1,58
10	*Martabak manis* (frying oil, flour, egg, milk)	1,50

Table 5. Food Sources of omega-3 PUFA Intake

No	Food	ω-3 PUFA intake (g)
1	*Rempeyek* (fried peanut crisp)	0,60
2	*Kerang* (clam)	0,24
3	*Telur dadar* (omelette)	0,20
4	*Kacang kedelai* (yellow soybean)	0,16
5	*Nasi goreng + telur orak arik* (fried rice and scrambled egg)	0,16
6	*Ikan tuna goreng* (fried tuna fish)	0,16
7	*Ikan tenggiri* (king mackerel fish)	0,15
8	*Telur ceplok* (fried sunny-side egg)	0,14
9	*Mie goreng* (fried noodle)	0,11
10	*Telur balado* (egg and chili)	0,11

Table 6. Food Sources of omega-6 PUFA Intake

No	Food	ω-6 PUFA intake (g)
1	*Saus kacang* (peanut sauce)	5,76
2	*Rempeyek* (fried peanut crisp)	4,65
3	*Ayam goreng* (fried chicken)	2,07
4	*Telur dadar* (omelette)	1,29
5	*Kacang kedelai* (yellow soybean)	1,23
6	*Ayam bakar* (roasted chicken)	1,15
7	*Ayam goreng tepung* (fried chicken with crispy flour)	1,15
8	*Nasi goreng + telur orak arik* (fried rice with scrambled egg)	1,04
9	*Telur ceplok* (fried sunny-side egg)	0,99
10	*Telur ayam* (chicken egg)	0,75

Others Factors Related to Fasting Plasma Insulin

Table 1. Correlation Coefficient between Fasting Plasma Insulin and Other Factors Related in Abdominal Obese Adult Men

	Coefficient correlation (r)
	Fasting plasma insulin
Age	0.033
Waist circumference	0.401**
Body mass index (BMI)	0.415**
Omega-3 PUFA	0.124
Omega-6 PUFA	0.080
Triglyceride	0.237**
Smoking habit	0.108
Alcohol use	0.110
Physical activity	0.005

**significant correlation at p<0.01, Pearson correlation test

Table 2. Correlation Coefficient between Different Fatty Acids intake and Waist Circumference in Abdominal Obese Adult Men

	Coefficient correlation (r)
	Waist circumference
Total fat intake (g)	-0.041
Total fat intake (% of total calories)	-0.215*
SFA intake (g)	-0.071
SFA intake (% of total calories)	-0.196*
MUFA intake (g)	-0.022
MUFA intake (% of total calories)	-0.133
PUFA intake (g)	0.015
PUFA intake (% of total calories)	-0.020
n-3 PUFA (g)	0.063
n-3 PUFA (% of total calories)	-0.010
n-6 PUFA (g)	-0.058
n-6 PUFA (% of total calories)	-0.139
Ratio PMS	-0.061
Ratio n-6 : n-3 PUFA	-0.130

*significant correlation at p<0.05, Pearson correlation test

Multiple Logistic Regression of Insulin Resistance

Table 1. Logistic Regression of Likelihood of having Insulin Resistance {total fat intake as % of total calories (categorical)}

Variable	Odds ratio	p
Total fat intake (% of total calories)	1.305 (0.317-5.373)	0.713
WC	0.834 (0.766-0.908)	< 0.001
Age	0.999 (0.928-1.076)	0.976
FPG	0.939 (0.890-0.992)	0.024
FFA plasma	6.794 (0.450-102.590)	0.167
Smoking	1.197 (0.470-3.049)	0.706
Physical activity	0.934 (0.440-1.981)	0.934
Alcohol use	1.375 (0.409-4.618)	0.606
Fibre intake	0.856 (0.064-11.527)	0.907

$R^2 = 0.316$

Table 2. Logistic Regression of Likelihood of Having Insulin Resistance {MUFA intake as % of total calories (categorical)}

Variable	Odds ratio	p
MUFA intake (% of total calories)	1.209 (0.462-3.165)	0.699
WC	0.834 (0.766-0.909)	< 0.001
Age	0.998 (0.927-1.075)	0.962
FPG	0.942 (0.892-0.994)	0.030
FFA plasma	5.724 (0.397-82.505)	0.200
Smoking	1.209 (0.473-3.090)	0.692
Physical activity	0.936 (0.441-1.985)	0.863
Alcohol use	1.318 (0.389-4.462)	0.657
Fibre intake	0.926 (0.067-12.691)	0.954

$R^2 = 0.316$

Table 3. Logistic Regression of Likelihood of Having Insulin Resistance {total fat and MUFA intake as % of total calories (categorical)}

Variable	Odds ratio	p
Total fat intake (% of total calories)	1.360 (0.325-5.684)	0.674
MUFA intake (% of total calories)	1.242(0.470-3.280)	0.662
WC	0.835 (0.767-0.909)	< 0.001
Age	0.999 (0.927-1.075)	0.969
FPG	0.941 (0.891-0.994)	0.028
FFA plasma	6.407 (0.418-98.299)	0.182
Smoking	1.224 (0.477-3.139)	0.674
Physical activity	0.953 (0.447-2.033)	0.901
Alcohol use	1.330 (0.392-4.515)	0.648
Fibre intake	0.884 (0.063-12.308)	0.927

$R^2 = 0.317$

Table 4. Logistic Regression of Likelihood of Having Insulin Resistance {PUFA intake as % of total calories (categorical)}

Variable	Odds ratio	p
PUFA intake (% of total calories)	1.009 (0.237-4.304)	0.990
WC	0.834 (0.765-0.908)	< 0.001
Age	0.999 (0.927-1.076)	0.969
FPG	0.940 (0.891-0.993)	0.026
FFA plasma	6.138 (0.403-93.399)	0.191
Smoking	1.188 (0.466-3.024)	0.718
Physical activity	0.921 (0.436-1.947)	0.830
Alcohol use	1.360 (0.406-4.553)	0.618
Fibre intake	0.897 (0.066-12.241)	0.935

$R^2 = 0.315$

Table 5. Logistic Regression of Likelihood of Having Insulin Resistance {Total fat and PUFA intake as % of total calories (categorical)}

Variable	Odds ratio	p
Total fat intake (% of total calories)	1.316 (0.313-5.530)	0.708
PUFA intake (% of total calories)	1.056 (0.243-4.586)	0.942
WC	0.834 (0.766-0.908)	< 0.001
Age	0.999 (0.927-1.076)	0.971
FPG	0.940 (0.890-0.992)	0.025
FFA plasma	6.985 (0.418-116.787)	0.176
Smoking	1.200 (0.470-3.064)	0.703
Physical activity	0.933 (0.440-1.980)	0.857
Alcohol use	1.377 (0.410-4.627)	0.605
Fibre intake	0.867 (0.063-11.909)	0.915

$R^2 = 0.316$

Table 6. Logistic Regression of Likelihood of Having Insulin Resistance {MUFA and PUFA intake as % of total calories (categorical)}

Variable	Odds ratio	p
MUFA intake (% of total calories)	1.209 (0.462-4.305)	0.998
PUFA intake (% of total calories)	0.998 (0.232-4.305)	0.998
WC	0.834 (0.766-0.909)	< 0.001
Age	0.998 (0.927-1.075)	0.962
FPG	0.942 (0.892-0.995)	0.031
FFA plasma	5.720 (0.368-88.954)	0.213
Smoking	1.209 (0.472-3.095)	0.693
Physical activity	0.936 (0.441-1.985)	0.863
Alcohol use	1.318 (0.389-4.464)	0.657
Fibre intake	0.925 (0.066-13.025)	0.954

$R^2 = 0.316$