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MINANGKABAU TRADITIONAL DIET AND CARDIOVASCULAR DISEASE RISK IN WEST SUMATRA, INDONESIA

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To my husband, *Deddi Prima Putra*

our children, *Indah Fauzia & Ilham Firdausi*

my mother, *Nurhuda Jamil*

and

in memory of my beloved father, *Benyamin Lipoeto*

SUMMARY

Minangkabau Traditional Diet and Coronary Heart Disease in West Sumatra, Indonesia

Diet is known to be involved in the pathogenesis of the major health problems of newer economies, namely cardiovascular disease, diabetes mellitus, osteoporosis and also in the development of obesity and certain cancers. Experimental and metabolic studies suggest that coconut in diets can cause hyperlipidaemia and atherosclerosis. This thesis focuses on food culture and its relationship with coronary heart disease amongst the Minangkabau people in West Sumatra, who are high consumers of coconut. Results presented in this thesis involved three major studies, the Focus Group Discussions, the Case-Control Study and the Intervention Study.

The present thesis has found through the Focus Group Discussions that rice, fish, coconut, green vegetables and chilli are the basic daily foods for the Minangkabau. Meals have little variation from breakfast through to the evening meal. The methods of food preparation and the taste preferences have remained remarkably consistent in recent generations although the amounts of food eaten have changed and novel foods from other parts of Indonesia have become integral to Minangkabau food culture. So far there have been little Western influences on Minangkabau people. West Sumatra, as is the case in many developing communities, is experiencing a nutrition transition reflected in rapid changes in diet structure and dramatic shifts in the causes of death.

The Case-Control study aimed to investigate the pattern of coconut consumption and the risk factors for Coronary Heart Disease, and compare that between patients with Coronary Heart Disease and their gender- and age-matched Controls. This study showed that some lifestyle factors were important in Coronary Heart Disease events in the Minangkabau. Being physically active was protective against Coronary Heart Disease, while high perceived stress and cigarette smoking increased the risk for Coronary Heart Disease events.

The intakes of dietary fat, especially saturated fat, was not significantly different between the cases and the controls and was unlikely to be predictors for Coronary Heart Disease. However, the intakes of more animal-derived foods, protein and dietary cholesterol and less plant-derived food, were predictors of increased Coronary Heart Disease.

There were associations of increased Coronary Heart Disease risk with higher blood pressure and lower high-density lipoprotein cholesterol. However, the present study failed to demonstrate an increase in Coronary Heart Disease risk for those with higher total cholesterol, low-density lipoprotein cholesterol, triglyceride concentrations and obesity.

A Food Intervention Study was conducted to examine how coconut food protect against coronary heart disease risks. In this study, the Coconut group not only had a higher intake of coconut products, but also of fish. The increased intake of coconut products was positively correlated with intakes of fish, vegetables, fruit and meat. Increased coconut intake prevented the small sixth week increase in total cholesterol concentration seen in the Non-coconut group (although this was not the case in the third week). There were no differences in HDL- and LDL-cholesterol, lipoprotein (a) and triglyceride concentrations at the 6^{th} week of the study between the Coconut and the Non-coconut groups. Also no significant differences were found in the concentrations of glucose or insulin. For plasma phospholipid fatty acid composition, at the end of the study, the Coconut group had significantly higher proportions of palmitic acid (C16:0), n-6 docosapentaenoic acid (C22:5n-6), α-linolenic acid (C18:3n-3 docosahexaenoic acid (C20:5n-3), n-3 docosapentaenoic acid (C22:5n-3), docosahexaenoic acid (C22:6n-3), docosahexaenoic acid/ docosahexaenoic acid ratio and a lower n-6/n-3 fatty acid ratio. A comparison between the Minangkabau in West Sumatra and the Chinese and the Anglo-Celtic populations in Melbourne showed that the arachidonic acid status in Melbourne was much higher than for the Minangkabau.

Significant differences were not found in any anthropometric or body composition indices between the Coronary Heart Disease cases and their controls. Amongst these measurements, only height was able to account for Coronary Heart Disease events. In the coconut intervention study, mean weight, body mass index, skinfold thickness, abdominal circumference or waist-to-hip ratio did not change. These findings are reassuring for a coconut based food culture.

In conclusion, a traditional diet based on mainly coconut milk and coconut meat might actually be protective for the Minangkabau. Health authorities could emphasise their traditional culture in dietary recommendations. The recommendation in Indonesia to reduce saturated fatty acids, in particular from coconut, requires re-evaluation.

CANDIDATE'S STATEMENT

This thesis contains no material which has been accepted for the award of any degree or diploma in any university or institution.

To the best of my knowledge, the thesis contains no material previously published or written by any person, except where due reference is made in the text of the thesis.

The author consents to the thesis being made available for photocopying and loan accepted for the award degree

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Nur Indrawaty Lipoeto

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LIST OF ABBREVIATIONS

% CI	% confidence interval
AHR	abdominal-to-hip ratio
BMI	body mass index
BMR	basal metabolic rate
CHD	coronary heart disease
CVD	cardiovascular disease
DHA	docosahexaenoic acid
EE	energy expenditure
EI	energy intake
EPA	eicosapentaenoic acid
EPA/AA	eicosapentaenoic acid to arachidonic acid ratio
EPA/DHA	eicosapentaenoic acid to docosahexaenoic acid ratio
FFQ	Food Frequency Questionnaire
FGD	focus group discussion
g/d	gram/day
GNP	gross national product
HDL	high density lipoprotein
ICD	international statistic classification of diseases
IUNS	the International Union of Nutritional Sciences
LCFAs	long chain fatty acids
LDL	low density lipoprotein
MCFAs	medium chain fatty acid
MCTs	medium chain triglyceride
MUFAs	monounsaturated fatty acids
OR	odds ratio
P:S ratio	polyunsaturated to saturated fatty acid ratio
PAL	physical activity level
PUFAs	polyunsaturated fatty acids
RCT	Reserve Cholesterol Transport
r_s	Spearman correlation coefficient
SCFAs	short chain fatty acids
SD	standard deviation
SKRT	Household Health Survey
SUSENAS	National Socio-economic Survey
WHO	World Health Organization
WHR	waist-to-hip ratio

PUBLICATIONS ARISING FROM THIS THESIS

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CHAPTER 1

Introduction and Statement of Hypotheses

1.1 BACKGROUND

Diet is known to be involved in the pathogenesis of the major health problems of newer economies, namely cardiovascular disease (CVD), diabetes mellitus, and osteoporosis, and also in the development of obesity and certain cancers. The prevalence of CVD is increasing in Indonesia. The Ministry of Health in Indonesia (Mihardja et al., 1997) revealed that CVD was 13% of all causes of death for all of Indonesia except Java and Bali, in 1995, and increased from 8% to 11% in 1986 and 1992, respectively. According to the reports, the mortality rate per 100,000 of 67.8 was due to CVD in 1986. The rate is comparatively lower to other countries, but obviously there has been an increase in the CVD prevalence in Indonesia, as well as in West Sumatra.

Several nutrition and non-nutritional pathways have been suggested in the development and occurrence of CVD. The increasing intake of saturated fat in different populations has been confirmed by the elevation of serum cholesterol concentrations and the mortality in coronary heart disease (CHD) (Barr et al., 1992; Keys, 1986b; Kromhout et al., 2000; Shekelle et al., 1981). It has been suggested that a high intake of coconut oil may significantly contribute to this relationship (Anderson et al., 1976; Pronczuk et al., 1991; Remla et al., 1991; Soma et al., 1985; Van and Zilversmit, 1988). But other studies (Hajri et al., 1998; Mensink et al., 1994) also found that the medium chain fatty acids (lauric and myristic acids) increased both low-density lipoprotein (LDL) as well as high-density lipoprotein (HDL) cholesterol.

However, several population studies reported that hyperlipidaemia and vascular diseases were uncommon among populations who consumed coconut. Studies in Papua New Guinea and Polynesia (Lindeberg et al., 1994; 1996 & 1997a; Lindeberg and Lundh, 1993; Prior et al., 1981) found that despite high consumption of fat, which came mostly from coconut, these populations were lean, and rarely had vascular diseases. The investigators concluded that there was no evidence of coconut consumption having a harmful effect in these populations. Studies in India

and Sri Lanka (Atukorala and Jayawardene, 1991; Kumar, 1997) found that coconut consumption had no relationship with CHD and CHD risks.

Traditionally, coconut is consumed with other foods such as fish, fruits, vegetables and it is prepared with herbs, which are mostly anti-atherogenic. Anti-atherogenic foods and several nutritional pathways are important in the development of CVD (Wahlqist, 1986).

The Minangkabau are an ethnic group in Indonesia who are high consumers of coconut. A previous study among the Minangkabau reported that fat consumption in West Sumatra was about 16% of total energy in rural areas and 20% in urban areas (Oenzil, 1997; Ratna Wilis, 1995).

Minangkabau food culture provides a unique opportunity to look at how newer economies have impacted on a traditional economy. Experimental and metabolic studies suggest that coconut intake can cause hyperlipidaemia and atherosclerosis, but whether it does so within the Minangkabau food culture is another issue.

1.2 HYPOTHESES

The hypotheses considered in this thesis are:

1. Coconut is an important part of the Minangkabau food culture.

2. The consumption of coconut and/or coconut products is not the major determinant of CHD events in the Minangkabau food culture.

3. High coconut consumption is not necessarily associated with CHD.

4. Traditional Minangkabau food culture has a role in the protection against cardiovascular risk.

1.3 OBJECTIVES

1. To use a*nthropological methods* to document food culture in relation to the manner and style of preparation of basic coconut foods, to determine changes in food habits amongst Minangkabau people.

2. To use a *Case-Control Study* to investigate the pattern of coconut and non-coconut consumption and the risk factors for CVD. Patients with CHD (Cases) were recruited from the Cardiovascular Units of five hospitals, and the gender- and age- matched patients without CHD (Controls) were recruited from the same hospitals and from the same population from which the Cases were obtained.

3. To conduct a *Food Intervention Study* to examine how coconut food protects against CVD risks. Participants were recruited from two villages located in the semi urban area of West Sumatra.

CHAPTER 2

Literature Review

2.1 THE MINANGKABAU

The Minangkabau are one of some 140 ethnics groups scattered over 3000 islands in Indonesia. Most of the Minangkabau inhabit the province of West Sumatra. West Sumatra is one of the twenty-six provinces of Indonesia. It is bordered by the Indian Ocean in the west, the Jambi and Bengkulu provinces in the south, the Riau province in the east and the province of North Sumatra in the north. According to the 1999 census, the province of West Sumatra was inhabited by about 4.542 million people. West Sumatra has six municipalities, eight districts and one administrative city. It covers an area of approximately 42,297 square kilometres or 2.2% of the whole land mass of Indonesia. Although the area contains a substantial number of Chinese, Javanese, and Batak people, around 91% of this population are Minangkabau (Central Bureau Statistics of West Sumatra, 2000). The land of Minangkabau is generally fertile with a varied agriculture. Wet rice is the major agricultural crop. Palm oil, rubber, copra, coffee, gambier, cinnamon and cloves are some of the important cash crops. Annual crops such as corn, chilies, peanuts and cabbage are also planted and are usually traded within West Sumatra. In addition to agriculture, artisanship such as weaving, is widely practiced in some parts of West Sumatra, particularly around Bukittinggi and Pariaman (Central Bureau Statistics of West Sumatra, 2000).

According to Kato (1982) there are three characteristics most often associated with the Minangkabau: a strong Islamic faith, migration (merantau) and matrilineal customs. Merantau means to leave one's home village in search of wealth, knowledge and fame. According to Lekkerkerker (1916) a Minangkabau man, consciously or not is always looking for a place where he can find his 'freedom' and 'personality', from the bondage of "matriarchy" (Kato, 1982). As a result of migration, the Minangkabau can be found in all parts of Indonesia. Despite their inclination to migrate, the Minangkabau maintain a strong sense of their homeland identity. The Minangkabau are a unique combination of patrilineally-oriented Islam and Minangkabau matriliny, aside from the relatively rarity of the matrilinear system itself. The Minangkabau are probably one of the largest matrilineal societies in the

world (Kato, 1982). A wide range of studies have investigated the matrilinear system of the Minangkabau (Kahn, 1993; Kato, 1982). Quite surprisingly to Western scholars, the matrilinear system is still much a part of the Minangkabau life. In the matrilinear society, the family name is not passed down from the father to his children as it is in most cultures. Instead, the family name is inherited from the mother. Furthermore, inheritance continues through the generations from mother to daughter. This is especially true of the ancestral home, which remains the property of the women. In the matrilineal culture, Minangkabau women play an important role in the leadership and decision-making process of the traditional village government.

In addition to the three characteristics mentioned previously, another distinguishing characteristic is the Minangkabau food culture. Minangkabau food is popular among Indonesians and Minang-Restaurants can be easily found in almost every corner of Indonesia (Owen, 1980). However, little has been written about the Minangkabau food culture.

2.2 CHD AND THE RISK FACTORS

Cardiovascular disease (CVD) comprises all diseases of the heart and blood vessels (Australian Institute of Health and Welfare, 1994). Ischemic heart disease more commonly known as coronary heart disease (CHD) is the most common form of CVD. CHD, characterised by a limited supply of oxygen to the heart muscle, has clinical manifestations ranging from angina pectoris to myocardial infarction and sudden death. The World Health Organization (WHO) has set criteria for the diagnosis of CHD (Rose et al., 1982). The WHO diagnosis is based on symptoms such as typical chest pain, changes in electrocardiogram (ECG) or the elevation of creatinine phosphokinase and other enzymes.

2.2.1 PATHOGENESIS OF ATHEROSCLEROSIS

Atherosclerosis is the major pathological component involved in CVD (Getz et al., 1969). The characteristic lesion of atherosclerosis is a fibrous plaque. The plaque consists of a cap of smooth muscle cells and fibrous tissue covered by a layer of endothelium, and a core containing lipid (Pearson et al., 1977; Thompson, 1994). Several hypotheses have been put forward to explain the process involved in plaque formation. Ross and Harker (1976) proposed that endothelial damage is the crucial initiating event in atherogenesis and subsequent changes were in response to the

injury sustained by the endothelium. The injury, resulting in the entry of platelets into arterial wall, could stem from various risk factors, including toxic damage mediated by cigarette smoke (carbon monoxide, CO), hypercholesterolaemia and hypertension (Ross and Harker, 1976).

Ross (1986a) hypothesised that hypercholesterolaemia leads to a clustering of monocytes that attach to the endothelial surfaces of arteries. These monocytes penetrate the endothelium, accumulate lipid and turn into foam cells. Foam cells became visible to the naked eye as fatty streaks. When fatty streaks retract, they expose the underlying macrophages to the circulating blood, resulting in the adherence of platelets into a mural thrombus. This leads to the hyperplasia of smooth muscle cells and the eventual conversion of a fatty streak into proliferative lesion. In the early 1990s, Witzum (1993) and Parthasaraty et al (1992) proposed that lipid peroxides were the principle mediators of endothelial injury (Parthasarathy and Rankin, 1992; Witztum, 1993). They proposed that small low density lipoprotein (LDL) particles would pass from the plasma through the endothelium into the intimal space. Here the LDL would undergo oxidation before being taken up by macrophages to form foam cells.

2.2.2 AGE, GENDER AND LIFESTYLE FACTORS

2.2.2.1 Age and Gender

Epidemiological data has shown a positive relationship between increasing age and the incidence and severity of atherosclerosis (Strong et al., 1978). The absolute risk is greater at age of 65 years than at age of 35 years but, with increasing serum cholesterol concentrations, the relative risk increases more steeply in 35 year olds than in 65 year olds.

The risk of CVD and the incidence of CHD are significantly higher in men than women up to the age of 75 years (National Heart Foundation of Australia, 1991). The risk is three times greater in men than women, at least until menopause (Pugeat et al., 1995; Ridker et al., 1993). In a study describing the changes in CVD risk factors during the perimenopausal and early postmenopausal years of women from Allegheny County, Pennsylvania it was suggested that the absolute risk for CVD increases substantially in midlife, with lipid metabolism being adversely affected at menopause (Matthews et al., 2001). Premenopausal women should be targeted for intervention if identified with risk factors for CVD.

In some non-Westernised populations such the Kitavan in Papua New Guinea, risk factor variables for CVD such as weight, body mass index (BMI) and triceps skinfold decrease linearly with age from 30 to 40 years onward. (Lindeberg et al., 997; Mancilha-Carvalho and Crews, 1990; Strickland and Ulijaszek, 1993; Trowell and Burkitt, 1979). In contrast, in Western populations, weight and BMI tend to increase from 20 to 65 years of age.

2.2.2.2 Education and Occupation

In developing countries, a strong association between high socio-economic class (indicated by higher education and job) and CHD has been found repeatedly (Singh et al., 1999a; INCEN, 1999). People in the higher socio-economic class of developing countries are from the urban areas where obesity, central obesity, hypertension, diabetes mellitus and a high total cholesterol are more common (Singh et al., 1999b). Urbanisation has been suggested as a cause of the increased prevalence of CHD among the Malays in Malaysia (Khor, 1994). The lack of association between low socio-economic class and CHD is possibly due to the fact that people from this class tend to have lower serum cholesterol concentrations, eat more fruits and vegetables, less animal foods (because they are too poor) and usually have physically demanding occupations (Oenzil, 1997).

In contrast, in more developed countries, low socioeconomic status, (indicated by low education attainment) has been associated with an increased risk for CHD (Chandola 1998; Wamala et al., 1999; Wamala et al., 2001). The Stockholm Female Coronary Risk Study, a population-based case-control study, examined 292 women aged 65 years and under with CHD and 292 age-matched controls using a wide range of socioeconomic, behavioural, psychosocial and physiological risk factors. The investigators concluded that being socio-economically disadvantaged in either early or later life was associated with an increased CHD risk (in women). Exposure to socioeconomic disadvantage in later life seemed to be more harmful for a woman's cardiovascular health than early life exposure to socioeconomic disadvantage (Wamala et al., 2001).

2.2.2.3 Smoking

Cigarette smoking is one of the most important risk factors for CHD and other macrovascular diseases. Several reports consistently indicate that the risk of CHD is two to four times higher among men and women who are heavy smokers (usually defined as those who smoke 20 or more cigarettes per day), compared to men and

women who do not smoke. (Kromhout et al., 2000; Willett et al., 1987). There is a clear dose-response relationship between smoking and CHD. Light smokers (those who smoke one to four cigarettes per day) have more than twice the risk of CHD compared with non-smokers (Willett et al., 1987). Case- control studies in Italy have found that the relative risk among smokers did not always show a clear dose-relationship. The relative risk continues to increase up to 10 cigarettes per day, but beyond that the relative risk alters little (Negri et al., 1993 & 1994). Furthermore, in populations with low average plasma cholesterol concentrations, the association was much weaker (Pooling Project Research Group, 1978; Robertson et al., 1977). This suggests that smoking became an important risk factor only in the presence of elevated plasma cholesterol levels.

Women who smoke are more susceptible to CHD and they more likely to be somewhat oestrogen-deficient (Baron et al., 1990). Several case-control and cohort studies have found that the odds ratio for CHD risk is higher in women than that found in men (Bosetti et al., 1999; Prescott and Hippe, 1998; Tverdal et al., 1993). In the Nurses' Health Study, (Hu et al., 2000), evaluated the effects of changes in diet and lifestyle on trends in coronary disease. They found that a reduction in smoking, an improvement in diet and an increase in postmenopausal hormone use, accounted for much of the decline in the incidence of coronary disease in this group of women. However, an increasing prevalence of obesity among these women appears to have slowed the decline in the incidence of coronary disease.

The mechanisms by which smoking predisposes to CHD are still unclear. Smokers tend to have in lower high density lipoprotein (HDL) cholesterol concentrations (Criqui et al., 1980; Raftopoulos et al., 1999, 1985). One mechanism may relate to the response to injury hypothesis (Ross, 1986b) where chemicals, especially free radicals, present in cigarette smoke may cause endothelial damage. Then in the presence of high concentrations of cholesterol, lesions may develop. Benditt and Benditt (1973) hypothesise that mutagenic chemicals present in cigarette smoke may be required for lesion initiation and progression. Smoking probably also increases the risk of thrombosis independently of atherosclerosis (Benditt and Benditt, 1973; Friedman et al., 1979).

2.2.2.4 Physical Activity

Results from the Harvard Alumni Health Study clearly indicate that physical activity (even short sessions) is associated with a decreased risk in CHD (Lee et al., 2000). Physical activity increases HDL-cholesrerol concentrations (Spate-Douglas and

Keyser, 1999) and decreases CHD risk (Eaton et al., 1995). Physical inactivity appears to be more common among some populations susceptible to CHD (Crespo et al., 2000). A case-control study by Eaton et al (1995) reported that the risk of CHD doubled in sedentary women. This study was done among 50 cases and 150 age-matched controls. The Odds Ratio (OR) of CHD in sedentary women was 2.1 (95% CI 1.0–4.3, *P=0.046*) and this altered little after adjusting for potential confounding factors. Among the persons with type 2 diabetes, increased physical activity, including regular walking has been associated with a substantially reduced risk for cardiovascular events (Hu et al., 2001).

2.2.2.5 Stress

The mechanism through which psycho-social stress relates to CHD is unknown. One hypothesis is that psychosocial stress may have indirect influence on lipid and haemostatic profiles through smoking and lack of exercise (Brunner, 1997). Another hypothesis is that psycho-social stress may result in pathogenic physiological changes. Stress hormones, including cathecholamines, have a pronounced effect on hemodynamics, lipid metabolism, hemostasis and other aspects of metabolism (Bjontorp, 1976; Brindley et al., 1999). In developed countries, low socioeconomic status appears to be linked to psychosocial stress and lifestyle factors (Wamala et al., 1999).

2.2.2.6 Family History

Familial aggregation of CHD has been reported in several studies. (Allayee et al., 1998; Caicoya et al., 1999; Canani et al., 1998; Hotopf et al., 1999; Lagstrom et al., 1999; Zureik et al., 1999). The specific underlying mechanisms and the relative contribution of atherosclerosis to subsequent CHD events in subjects with a family history are not well established. Zureik et al (1999) examined the association of parental history of premature death from CHD with ultrasound carotid measurements of atherosclerosis in a population of 1040 subjects aged 59 to 71 years. Subjects who reported that one or both parents had died suddenly or died of a myocardial infarction before the age of 65 years were considered to have a parental history of premature death from CHD. The prevalence of atheromatous plaques was higher in subjects with a history of premature death from CHD compared with those without such a history. The age- and sex-adjusted odds ratio of atheromatous plaques associated with a parental history of premature death from CHD was 2.85 (95% CI 1.60–5.08; *P<0.001*). Furthermore, multivariate adjustment for the major cardiovascular risk factors did not markedly alter the results (OR 2.70;

P<0.002). The findings demonstrated that a parental history of premature death from CHD was strongly associated with CHD.

The risk associated with a family history of CHD may be partly due to a genetic predisposition towards high blood pressure, hypercholesterolaemia, diabetes and obesity. Genetic factors may be involved, but it cannot be ruled out that the observed relationship may be confounded by other risk factors, especially the aggregation of family life-styles and the environment (Ciruzzi et al., 1997; Friedlander et al., 1998; Leander et al., 2001).

2.2.3 NUTRIENT INTAKES

2.2.3.1 Dietary Fat

The effect of saturated fatty acids (SFAs), polyunsaturated fatty acids (PUFAs), and cholesterol on serum cholesterol concentrations has been well established through a number of experimental studies (Hegsted et al., 1965; Keys et al., 1965a), as well as epidemiological studies (Schaefer, 1997; Shekelle et al., 1981). A diet high in saturated fat and cholesterol predisposes one to the development of risk factors associated with premature CVD. The National Cholesterol Education Program of the United States have recommended that the intake of total fat and saturated fat should not be more than 30% and 10% of energy intake, respectively and, that the intake of cholesterol should not exceed 300 mg/d (Schaefer, 1997).

2.2.3.2 Dietary Fat and Cholesterol

Studies examining the relationship between dietary fat and cholesterol with CHD began in the 19th century. In 1847, Vogel described cholesterol in atheromatous tissue. In 1908 Ignatowski demonstrated that high protein diets fed to rabbits led to atherosclerosis (Quiney and Watts, 1989). Anitschkow and Chalatow (1913) found that feeding cholesterol to rabbits led to atherosclerosis, and the severity of this disease was associated with the amount of cholesterol ingested by the animals. In 1913, Bacmeister and Henes found an association between elevated serum cholesterol and certain diseases including atherosclerosis (Bacmeister and Henes, 1913).

By postulating that a raised serum cholesterol caused atheroma, it was Anitschkow (1913) who put forward the "lipid hypothesis". The work of Mueller in 1938 supported this hypothesis. He described tendon xanthomata, angina pectoris, and

hypercholesterolaemia in patients with an elevated serum cholesterol. Since then an enormous number of observations and experimental studies have supported this hypothesis (Anitschkow and Chalatow, 1913).

For about 40 years after the work of Anitschkow, very few studies examined the relationship between cholesterol and atherosclerosis. Then in the 1950's Keys and others began publishing a series of classic papers which culminated in the Seven Countries Study (Keys et al., 1957a & 1957b; 1958a & 1958b; 1959; 1963; 1965a; 1965b & 1965c) . In the Seven Countries Study, Keys was able to show that both dietary saturated fat and cholesterol were associated with CHD incidence rates in seven countries that had different rates of CHD (Keys, 1967). Despite this, Keys acknowledged that there was very little direct evidence to show the effect of the diet on human arteriosclerosis. Another study around the time of Keys' early work found that the type of fat in the diet was an important determinant of serum cholesterol levels. Saturated fat was associated with a rise in serum cholesterol, and polyunsaturated fat resulted in falls in serum cholesterol (Kinsell et al., 1952).

In a study preceding the Seven Countries Study, Keys and others examined the relationship between the consumption of different fatty acids and serum cholesterol levels and derived equations for predicting the expected changes in serum cholesterol as a result of changes in the type of fat in the diet (Keys et al., 1957b; 1965a; 1965b & 1965c). Similar studies were also conducted by (Hegsted et al., 1965). Equations derived from these studies, predict the increase in serum cholesterol with increases in cholesterol and saturated fat in the diet, and the fall in serum cholesterol with more polyunsaturated fat in the diet (Hegsted et al., 1965; Hegsted, 1986; Keys et al., 1965a; 1965b & 1965c).

In 1958 Keys and colleagues studied Japanese living in Japan, Hawaii and Los Angeles to determine whether diet or racial factors were responsible for the observed difference in CHD rates between countries. In this study the relationship between dietary fat and cholesterol, and the rates of CHD were found (Keys et al., 1958b). Although not conclusive, these results are consistent with the hypothesis that dietary saturated fat, exerting its effects via serum cholesterol, is a determinant of CHD. This is known as the "diet-heart" hypothesis.

The "diet-heart" hypothesis was proposed to explain the relationship between diet and heart disease. According to this hypothesis, a high intake of saturated fat and cholesterol, and a low intake of polyunsaturated fat, increases the level of serum cholesterol, which leads to the development of atheromatous plaques in the arteries.

Accumulation of these plaques leads to the narrowing of the coronary arteries, reduced blood flow to the heart muscle, and finally myocardial infarction or "heart attack" (Willett, 1998b).

This hypothesis was derived from the work on cholesterol and atherosclerosis during the early 1900's, as well as the following studies, which relate diet to CHD rates. The most influential of these was the Seven Countries Study (Keys, 1970). These studies have related dietary factors to serum cholesterol and, in separate studies, serum cholesterol to the risk of CHD. The relationships between dietary factors and blood lipid levels are best studied in humans through controlled, preferably double blind, feeding trials. These studies have shown that a higher intake of cholesterol and saturated fat and a low intake of unsaturated fat, increases blood total cholesterol levels (Ahrens et al., 1957 & 1959; Hegsted et al., 1965; Keys et al., 1965a). As a result of these studies, SFAs were seen as the major dietary factor in the development of atherosclerosis.

Subsequent studies confirm the association between saturated fatty acids (SFAs) and atherosclerosis. In animal and human feeding studies, SFAs consistently raise serum cholesterol levels compared with unsaturated fatty acids (Grundy and Denke, 1990). Also, populations consuming high intakes of SFAs have higher rates of CHD than populations who consume lesser amounts (Keys, 1970). Recent reviews by Lichtenstein & Schwab (2000) and other studies (Heilbron et al., 1999; Vogel et al., 1997) also suggest that those with high intakes of fat are more prone to disturbances in glucose metabolism and impaired endothelial function and therefore CHD (Heilbronn et al., 1999; Lichtenstein and Schwab, 2000; Vogel et al., 1997). However, one study failed to find an association between high intakes of fat and disturbances in glucose metabolism and impaired endothelial function (Nelson et al., 1995). These investigators argued that the inconsistencies in data might be attributable to the clustering of high intakes of dietary fat (especially animal fat) with obesity and inactivity.

2.2.3.3 Saturated Fatty Acids (SFAs)

Experimental studies have found different classes of SFAs have different effects on plasma lipid and lipoprotein concentrations (Kelly et al., 2001; Nicolosi, 1997). SFAs with less than 10 carbons and stearic acid (C18:0) are considered neutral fats, suggesting the cholesterol-raising effects of SFAs can be attributed to lauric acid (C12:0), myristic acid (C14:0), palmitic acid (C16:0) (Katan et al., 1994; Mensink et al., 1994). Among these medium chain fatty acids, it seems that lauric acid, palmitic

and myristic acid differ in their cholesterol raising effects (Khosla et al., 1997; Mensink, 1993; Mensink et al., 1994; Ng et al., 1992; Pronczuk et al., 1995; Sundram et al., 1994). Lauric acid raises total serum and LDL-cholesterol concentrations slightly less than palmitic acid while myristic acids raises it more than palmitic acid. Myristic acid raises HDL-cholesterol (Hajri et al., 1998). Palmitic acid increases total serum and LDL-cholesterol, as well as HDL-cholesterol. Further studies have shown that dietary palmitic acid has similar effects to oleic acid on serum cholesterol and lipoprotein profiles in normocholesterolemic men and women (Hayes and Khosla, 1992; Ng et al., 1992). Stearic acid has only a slight effect on serum LDL- and HDL- cholesterol levels. Scwab US (Schwab et al., 1995) found lauric and palmitic acid had a minimal impact on serum lipids, lipoprotein concentrations and glucose metabolism, and compared to trans-fatty acids, lauric acid proved to be more favourable (de Roos et al., 2001).

The differential effects of specific saturated fats on plasma lipids and lipoproteins imply that these fatty acids may have different effects on the risk of CHD. As the intake of saturated fat increases in different populations so does serum cholesterol concentrations and CHD mortality (Barr et al., 1992; Keys, 1986; Kromhout et al., 2000; Shekelle et al., 1981). The clear relationship between the intake of SFAs and serum cholesterol and Apo-B concentrations have been seen in a number of controlled intervention studies, especially in serum cholesterol concentration over 5.78 mmol/L (Hayes, 1993; Katan et al., 1994).

Up to date, epidemiological studies have assessed only the association of CHD risk with total SFAs, and only a few studies have examined the association between individual SFAs and CHD risk within populations. A prospective cohort study by Hu et al. (1999) revealed that the intakes of short- to medium-chain SFAs (C4:0–C10:0) were not significantly associated with CHD risk (Hu et al., 1999a). Whereas the intake of each longer-chain SFAs (C12:0 – C18:0) was associated with a small increase in risk. In contrast to previous metabolic studies, the relative risk for every 1% increase in energy from stearic acid was 1.19 (95% CI 1.02–1.37). the authors concluded that stearic acid should not be separated from other saturated fats when giving dietary advice to reduce the risk of CHD (Hu et al., 1999a). However, it should be noted that this prospective study was conducted among populations whose intake of SFAs were dominated by palmitic and stearic acids. One could expect different results with diets of containing different fatty acid profiles.

2.2.3.4 Monounsaturated Fatty Acids (MUFAs)

The major MUFA in the diet is oleic acid (C18:1 n-9). Until recently it was thought that MUFAs were neutral in relation to serum cholesterol lowering. The term neutral, however, applies only under iso-caloric conditions MUFAs have similar effects on serum total cholesterol as carbohydrate (Mensink, 1994).

The effects of MUFAs on serum cholesterol have been re-examined in a number of studies (Grundy, 1986; Grundy et al., 1988; Mattson and Grundy, 1985; Mensink and Katan, 1987). It was found in all of these studies that MUFA did not elevate serum cholesterol concentrations and that diets high in MUFAs did not lower HDL-cholesterol concentrations, as does the substitution of SFAs with carbohydrates. A meta-analysis of various studies comparing low-saturated-fat, high carbohydrate diets and high-monounsaturated-fat diets on patients with type 2 diabetes, revealed that high-monounsaturated-fat diets reduce fasting plasma triglycerides, very low density lipoprotein (VLDL) cholesterol concentrations and cause a modest increase in HDL-cholesterol concentrations (Garg, 1998). MUFAs are either neutral, or may even lower, LDL-cholesterol concentrations.

Results from several studies suggest that diets high in MUFAs have a more favourable effect on total cholesterol (Mensink, 1994), and appear to be cardioprotective (de Lorgeril et al., 1998).

2.2.3.5 Polyunsaturated Fatty Acids (PUFAs) and Essential Fatty acids (EFAs)

Most fatty acids can be synthesised in vivo. However there are two series of fatty acids, which can not be synthesised in vivo, and must be obtained from food. These are known as the essential fatty acids (EFAs). The essential fatty acids, linoleic acid (C18:2 n-6) and alpha-linolenic acid (C18:3 n-3), are the major precursors of the omega-6 or n-6, and the omega-3 or n-3 series. The parent n-3 PUFA is α-linolenic acid (C18:3 n-3). α-Linolenic is the precursor of the longer chain fatty acids such as eicosapentanoic (EPA, C20:5 n-3) and docosahexaenoic acids (DHA, C22:6 n-3). The parent n-6 PUFA is linoleic acid (C18:2 n-6), which can produce γ linolenic acid (C18:3 n-6) and arachidonic acid (C20:4 n-6) (Cook, 1991).

Competition occurs between fatty acids of the n-3 and n-6 series. An abundance of α-linolenic (C18:3 n-3) can effectively decrease the formation of arachidonic acid (C20:4 n-6) from linoleic acid (C18:2 n-6). Simultaneous feeding of linoleic acid (C18:2 n-6) and α-linolenic acid (C18:3 n-3) indicates that the conversion of α-

linoleic acid (C18:3 n-3) to EPA and DHA is much greater than the conversion of linoleic acid (C18:2 n-6) to arachidonic acid (C20:4 n-6) (Cook, 1991). Furthermore, diets high in α-linolenic acid (up to 15 grams/day for 4 weeks) increase α-linolenic, EPA and C22:5 n-3 concentrations in plasma triglyceride and phospholipids, but there is very little, if any, detectable increases in DHA (Sinclair and Li, 2000). In a population where the PUFA intake is dominated by linoleic acid, the consumption of a low fat diet alters the fatty acid pattern in a manner similar to that observed with the feeding of (n-3) long-chain fatty acids. Raatz et al. (2001) evaluated the effect of a low fat diet (20% of total energy) versus a high fat diet (45% of total energy). They found that the low fat diet resulted in a significantly greater total of (n-3) fatty acids, EPA (C20:5 n-3) and DHA (C22:6 n-3) levels in plasma phospholipid fatty acids (Raatz et al., 2001).

The protective effects of foods rich in n-3 PUFAs derived from fish, shellfish and marine mammals on cardiovascular risk has been of interest for the past 20 years. Low rates of coronary heart disease reported in the Eskimo population exposed to a diet rich in fish oil, has led to several studies exploring the antiatherogenic, antithrombotic and antiarrhytmic effects of n-3 (Simopoulos et al., 1991).

In a clinical trial where patients were randomly assigned to receive an n-3 oil supplement, the investigators found that the treatment with n-3 significantly lowered the risk of death, non-fatal myocardial infarction and stroke (Anonymous, 1999). Furthermore, in a prospective cohort study of 76,283 women, Hu et al concluded that a higher intake of α-linolenic acid was protective against fatal CHD (Hu et al., 1999b).

Linoleic acid lowers total serum and LDL-cholesterol levels when it was used as a substitute for saturated fat. It also lowers the HDL-cholesterol level when consumed in high quantities (Almario et al., 2001). The n-3 PUFAs (fish oils) apart from being the most potent dietary inhibitors of thrombosis, also reduce serum LDL-cholesterol levels, but these fats do not influence HDL-cholesterol when it is used as a substitute for SFAs (Grundy, 1986). Early clinical investigations focused on linoleic acid because it appeared to induce a greater lowering of serum cholesterol levels than alternative nutrients, e.g. oleic acid and carbohydrate (Keys et al., 1965b&c). Another recent clinical trial reported that the plasma cholesterol ester of linoleic acid was inversely associated with CHD risk. (Bemelmans et al., 2000).

However, other studies do not support the findings. Hodgson et al (1993) reported a positive correlation between adipose tissue and platelet linoleic acid

concentrations with the degree of coronary artery disease (Hodgson et al., 1993). More recent case-control studies have shown that plasma linoleic acid is higher in CHD patients compared to their healthy controls. (Leng et al., 1999; Pedersen et al., 2000).

2.2.4 THE EQUATIONS OF KEYS AND HEGSTED

In a prospective study by Shekelle et al. (1981), it was demonstrated that two scores, one developed by Keys et al. (1965a) and another developed by Hegsted et al. (1965b), which took into account dietary cholesterol, saturated fat and polyunaturated fat intake were related to both serum cholesterol levels and the risk of coronary death.

The equations to predict the expected change in serum cholesterol with changes in SFAs, PUFAs and cholesterol were developed separately by Keys et al (1965a) and Hegsted et al (1965). Two of these equations are given below:

(i) serum cholesterol change = 1.35 (2S – P) + 1.52Z (Keys et al., 1965a)

(ii) serum cholesterol change = 2.16S – 1.65P + 0.097C (Hegsted et al. 1965)

S = Change in the percentage of calories from saturated fat.

P = Change in the percentage of calories from polyunsaturated fat

Z = The difference between the square root of the initial intake of cholesterol and the square root of the subsequent intake of cholesterol.

C = The difference between the cholesterol intake of two diets in mg/1000 kcal.

The pioneering studies of Keys in the Seven-Country study demonstrated a significant association between the dietary intake of SFAs and cholesterol and mortality from CHD (Keys, 1986). In another study, a significant relationship was found between the percentage of calories derived from fat and the prevalence of raised atherosclerostic lesions in autopsy cases (Scrimshaw & Guzman, 1968). It has been concluded that, relative to carbohydrates, SFAs elevate serum cholesterol while PUFA (linoleic acid) lowers serum cholesterol, and MUFA (oleic acid) has no statistically significant effect (Hegsted et al., 1965; Keys et al., 1957a).

What is more important, is that Keys and Hegsted equations are used to predict CHD events and mortality rather than simply blood lipids. An important study by

Kushi et al., (1985) known as the Ireland-Boston Diet-Heart Study found a J-shaped relationship between the cholesterol scores and mortality and, an inverse linear relationship for plant food and CHD mortality (Kushi et al., 1985).

2.2.5 THE QUESTIONABLE ROLE OF DIETARY FAT IN CARDIOVASCULAR DISEASE

A growing number of recent reviews and studies do not support a linear relationship between fat and cholesterol intakes with CHD and CHD mortality. Hooper et al (2001) reviewed 27 studies comprising 40 distinct intervention arms and over 30,902 person years of observation (Hooper et al., 2001). They found that alteration of dietary fat intake reduced the pooled rate ratio of mortality to 0.98 (95% CI 0.86–1.12), while the reduction in cardiovascular mortality and cardiovascular events were 9% and 16% respectively. This result indicates little, if any effect on modifying the intake of dietary fat on CHD and CHD mortality. Previous studies in USA by Taylor et al. (1987) and Browner et al. (1991) have led to predictions for life expectancy when the consumption of saturated fat is reduced to 10% of total energy and total fat intake is reduced to 30% (Browner et al., 1991; Taylor et al., 1987). These life expectancies are estimated by combining USA National Vital Statistic data with the cholesterol risk factor data from the Framingham Heart Study. Taylor et al found that unhealthy individuals with a high risk of CVD could expect to gain, on average, one extra year by reducing saturated fat, while healthy individuals with one CVD risk factor, might live 3 to 90 days more. Furthermore, Browner and colleague concluded that reducing fat consumption would delay 42,000 deaths each year, but the net increase in life expectancy would average out to only 3 to 4 months. To be more precise, a woman who might otherwise die at 65 could expect to live two extra weeks after a lifetime of avoiding saturated fat.

A recent cross sectional study in Sweden described the intake of dietary fatty acids among 94 healthy adolescents and related this dietary intake of fat with serum lipid measurements. This study found that dietary fat intake adversely affected serum cholesterol concentrations and each fatty acid (C4:0–C10:0, C12:0, C14:0) was inversely related to serum cholesterol concentrations (Samuelson et al., 2001).

Ravnskov reviewed a wide range of studies based on the diet-heart hypothesis (Ravnskov, 1995 & 1998). The review included ecological, dynamic population, cross sectional, cohort and case-control studies, as well as controlled, randomised

trials that looked at the effect of fat reduction. He found that the positive ecological correlations between national intakes of total fat and saturated fat and cardiovascular mortality found by previous author were absent or negative in the larger, more recent studies by Yerushalmy and Hilleboe (1957) (Yerushalmy and Hilleboe, 1957). Secular trends of national fat consumption and mortality due to CHD in 18-35 countries (four studies) during different time periods diverged from each other as often as they coincided. In cross sectional studies of CHD and atherosclerosis only one group of studies was supportive, six gave partly supportive, partly contradictive results, and in seven studies the findings were contradictory. No significant difference in fat intake was noted in six case-control studies of CVD patients and CVD-free controls. Neither total or CHD mortality were lowered in a meta-analysis of nine randomised, controlled dietary trials with substantial reductions in dietary fat, and in six trials combining the addition of PUFAs. It was concluded that the harmful effect of dietary saturated fat and the protective effect of dietary PUFAs on atherosclerosis and CVD were questionable.

A cohort study by the Harvard Group, which followed 43,757 health professionals from 1986 concluded that their findings did not support the strong association between the intake of saturated fat and the risk of coronary heart disease suggested by other studies (Ascherio et al., 1996). Another study by Grundy and colleague concluded that there is only little support for the claim that a high proportion of dietary fat predisposes to CHD (Grundy, 1999).

Most of these studies were done in populations with high intakes of fat. In populations with low fat intakes, the risk of CHD tends to be low (Dreon et al., 1999; Grundy et al., 1982) as are plasma lipid concentrations (Raeini-Sarjaz et al., 2000). A recent study by Raatz et al (2001) found that consumption of a low fat diet alters plasma fatty acid patterns in a manner similar to that observed with the feeding of n-3 long-chain fatty acids (Raatz et al., 2001).

2.2.6 Total Energy, Carbohydrates, Protein and Dietary Cholesterol Intake

2.2.6.1 Total energy intake

Excess energy intake in relation to energy expenditure results in weight gain and eventually obesity. Some studies have found a positive association between total energy intake and the risk of CHD (Kromhout et al., 1988; Tzonou et al., 1993). However, this association is not supported by other studies (Kushi et al., 1985) .

Gordon et al. (1981) reviewed the consumption of total energy in 16,349 men aged 45 – 64 years from three prospective studies (the Framingham Study, the Honolulu Heart Study and the Puerto Rico Heart Health Program), based on their 24-hour dietary recalls. It was concluded that men who had a greater caloric intake or greater caloric intake per kilogram of body weight were less likely to develop CHD, suffer a myocardial infarction or die from CHD, even though men of greater weight are more likely to develop CHD (Gordon et al., 1981). Presumably physically active individual need to eat more and so are less likely to develop.

2.2.6.2 Carbohydrates

High-carbohydrate, low-fat diets have been widely recommended as a way to reduce the risk of CHD since populations with low intakes of fat tend to be at lower risk (Grundy, 1999; Vogel et al., 1997). A study among the Pima Indians found that a high carbohydrate diet decreased the conversion of VLDL to LDL, which in turn contributed to a decrease in LDL. This study also found evidence to show an increase in the activity of the LDL apoB clearance mechanism, and a decrease in the LDL-cholesterol to apoB ratio (Abbott et al., 1990; Gordon et al., 1981). Moreover, other studies have confirmed that high carbohydrate diets not only lower total cholesterol but also LDL and HDL-cholesterol concentrations (without modifying the ratio of LDL to HDL). Furthermore, high carbohydrate diets have little or no effect on triglyceride concentrations (Vidon et al., 2001; Vogel et al., 1997). High carbohydrate diets eaten in association with a weight reduction diet have similar effects to a high monounsaturated diet (Walker et al., 1996). Some studies have shown that a high-carbohydrate diet can reduce high-density lipoprotein (HDL) cholesterol levels, raise fasting levels of triglycerides (Dreon et al., 1999; Mensink and Katan, 1992; Nelson et al., 1995) and reduce LDL particle size (Dreon et al., 1994 & 2000; Dreon and Krauss, 1997).

2.2.6.3 Protein

There were only a few case-control studies examining the association of CHD with protein intake. A study by Smit et al (1999) found a significant positive association between CHD and protein intake (Smit et al., 1999). They found a lower percentage of animal protein intake among persons in the lowest quartile of total serum cholesterol and apolipoprotein B concentrations compared with persons in highest quartile. Although most analyses show an association between protein intake and CHD risk these analyses are difficult to interpret because they are not adjusted for the intake of specific types of fat(Smit et al., 1999).

2.2.7 FOOD INTAKE

A high fish intake, which is associated with a higher intake of n-3 fatty acids, has been shown to reduce very-low density lipoprotein, inhibit thromboxane production, increase prostacyclin synthesis, reduce blood viscosity, and reduce the likelihood of thrombosis and cardiac arrythmias (Agren et al., 1997; Krauss et al., 2000; Leaf and Weber, 1988; Smit et al., 2000). Populations with a high intake of fish such as the Eskimos and the Japanese have long been known to have low rates of coronary heart disease (Bang et al., 1980; Hirai et al., 1980; Kromann and Green, 1980). However, some studies done in North America and European countries, have found no correlation between fish intake and CHD incidence (Ascherio et al., 1995; Kromhout et al., 1985; Norell et al., 1986) and CHD mortality (Kromhout et al., 1996). A possible explanation for the apparently discordant results between fish consumption and CHD may be due to the fact that the incidence of CHD relates to other factors and the reduction in CHD mortality may be due to the effect of fish (n-3 fatty acids) in reducing the risk of fatal arrhythmia

In ecologic and epidemiologic studies, animal protein consumption has been shown to be directly correlated with CHD risks, (Smit et al., 1999) and CHD events (Hu et al., 1999a). The consumption of vegetable protein on the other hand, is inversely correlated with CHD risks (Smit et al., 1999).

Vegetables and fruit are associated with a low risk of CVD (Ness and Powles, 1997). Vegetables and fruit are sources of a variety of nutrients, including vitamins, trace minerals, dietary fibre and many other kinds of biologically active compounds. These compounds known as phytochemicals can have complimentary and overlapping mechanisms of action, including antioxidant activity, decreasing platelet aggregation, altering cholesterol metabolism, and reducing blood pressure (Lampe, 1999).

2.2.8 COCONUT

The coconut, *Cocos nucifera,* is found throughout wet tropical lowlands. It grows almost anywhere if there is sufficient rain and warmth. It has been grown for thousands of years in tropical countries, and it seems most likely that the coconut originated from Indonesia or Malaysia (Child, 1964; Piggott, 1964). Apart from coconut oil, there are many kinds of coconut-products used by the Minangkabau, namely coconut meat, coconut water, copra, and desiccated coconut (Figure 2.1). The part of the coconut that is most used for food is the meat. Coconut milk is the

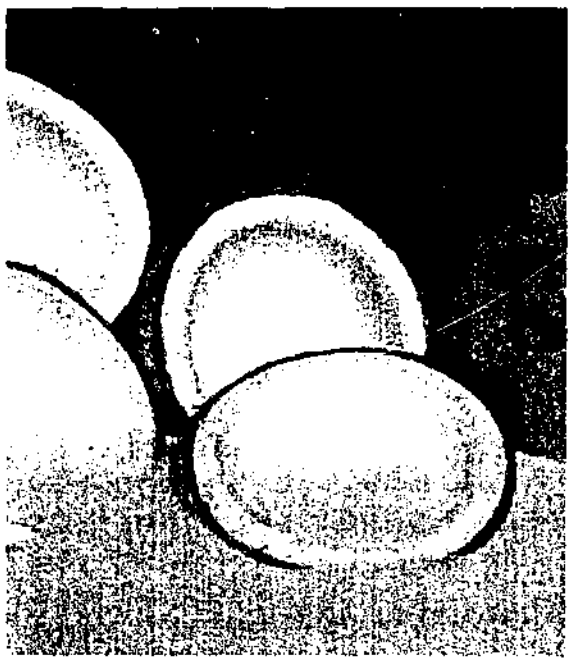
liquid that is expressed from fresh grated coconut meat. To produce the milk, the freshly grated coconut is mixed with water and then pressed. The flesh of one coconut will give about half a litre of coconut milk. Coconut milk is used to thicken sauces and to add flavour. Freshly grated coconut is also used as part of the ingredients in vegetable dishes. The coconut shell is a thin hollow sphere with a rather flat base and a slightly conical shaped top. The enclosed space is filled with coconut water, which is used as a beverage. A fully mature nut contains about 250 g of water. Copra is prepared by drying coconut meat over a slow fire. Copra is a source of coconut milk. Desiccated coconut is the shredded dehydrated meat prepared from the fresh kernel of a coconut (Banzon, 1990).

Up until 1996 the first major source of vegetable oil in Indonesia and West Sumatra was coconut oil. The consumption of coconut oil in 1993 was 12 mL/day (Central Bureau Statistics of West Sumatra, 2000; Simatupang and Purwoto, 1996). Since 1997, the price of coconut oil has risen, the palm oil industry has flourished and coconut oil has been gradually replaced by palm oil.(Piggott, 1964; Prior et al., 1981).

2.2.8.1 Fatty acid composition of Coconut Products

Investigations by Pehowich et al (2000) reported that fatty acid composition of coconut water, milk, cream and the meat of the coconut contained similar relative compositions of individual fatty acids (Central Bureau Statistics of West Sumatra, 2000; Pehowich et al., 2000; Simatupang and Purwoto, 1996). Coconut water contains very few lipids of any type, while coconut oil is about 96% fat. Coconut milk contains about 24% of the fatty acid content of coconut oil, while coconut cream and meat each contain about 34% of the fatty acid content of coconut oil (Table 2.1).

Figure 2.1 Coconut food products



Coconut flesh/meat
white solid endosperm

Coconut water
liquid endosperm

Coconut milk/ coconut cream
liquid obtained by manual or mechanical extraction of grated coconut meat with or without added water

Copra
dried meat of mature coconut from which oil is extracted

Desiccated coconut
edible, shredded dehydrated flesh prepared from fresh kernel

Young coconuts produce sweet coconut meat and coconut water.

Harvesting coconuts

Table 2.1: The fatty acid content of coconut oil and coconut milk (per 100 gram of edible portion)

Fatty acid (g/100 g edible portion)	Coconut oil	Coconut milk	Coconut water	Coconut cream	Coconut meat
Saturated fatty acids	**86.50**	**21.14**	**0.19**	**30.7**	**29.7**
C4:0	0.00	0.00	0.00	0.00	0.0
C6:0	0.60	0.14	0.01	0.2	0.2
C8:0	7.50	1.67	0.01	2.4	2.3
C10:0	6.00	1.33	0.01	1.9	1.9
C12:0	44.60	10.58	0.10	15.4	14.9
C14:0	16.80	4.18	0.04	6.1	5.9
C16:0	8.20	2.02	0.02	2.9	2.8
C18:0	2.80	1.23	0.01	1.8	1.7
Monounsaturated fatty acids	**5.80**	**1.01**	**0.02**	**1.5**	**1.4**
C16:1	0.00	0.00	0.00	0.0	0.0
C18:1	5.80	1.01	0.02	1.5	1.4
C20:1	0.00	0.00	0.00	0.0	0.0
C22:1	0.00	0.00	0.00	0.0	0.0
Polyunsaturated fatty acids	**1.80**	**0.26**	**0.00**	**0.4**	**0.4**
C18:2	1.8	0.26	0.00	0.4	0.4
C18:3	0.00	0.00	0.00	0.0	0.0
C18:4	0.00	0.00	0.00	0.0	0.0
C20:4	0.00	0.00	0.00	0.0	0.0
C20:5	0.00	0.00	0.00	0.0	0.0
C22:5	0.00	0.00	0.00	0.0	0.0
C22:6	0.00	0.00	0.00	0.0	0.0
Cholesterol	**0.00**	**0.00**	**0.00**	**0.0**	**0.0**

Sources:- USDA Nutrient Database for Standard reference (www.nal.usda.gov/fnic/cgi-bin/list_nut.pl)
- Pehowich et al., 2000.

NA: data not available

2.2.8.2 Medium Chain Triglycerides in Coconut Oil and its Metabolism

Unlike other oils such butter, lard, soybean, corn oils, which are dominated by long chain saturated triglycerides, coconut oil contains mostly medium chain triglycerides. Only coconut and palm kernel oils (among the commercially available oils) contain medium chain triglycerides (Banzon, 1990). Medium chain triglycerides (MCTs) yield medium chain fatty acids (MCFAs) upon hydrolysis, and consist of molecules comprising 6 to 10 carbon atoms (Bach et al., 1996). Sometimes lauric acid (C12:0) is classified as MCTs (Papamandjaris et al., 2000). A number of studies indicate that MCTs increase feelings of satiety, reduce energy density, undergo rapid entero-portal transportation and intrahepatic oxidation. The latter leads to less MCTs being incorporated into complex lipids, thus making more energy available (Bach et al., 1996).

MCTs are metabolised differently to long chain triglycerides as shown in Figure 2.2. A growing number of studies have confirmed that the medium-chain fatty acids are more prone to oxidation; therefore less likely to lead to obesity (Baba et al., 1982; Bray et al., 1980; Cotter et al., 1989; DeLany et al., 2000; Geliebter et al., 1983; Hill et al., 1989; Papamandjaris et al., 2000; Seaton et al., 1986). Among the fatty acids, MCFAs are highly oxidised, whereas the polyunsaturated and monounsaturated fatty acids are oxidised less. The oxidation of SFAs decreases with increasing carbon length (lauric acid > myristic acid > palmitic acid > stearic acid). MCFA are rapidly cleared from the circulation and absorbed into blood from the intestinal lumen (Bach and Babayan, 1982). Moreover, medium-chain fatty acids are not re-esterified and are more easily transported into the mitochondria for subsequent oxidation compared with the fatty acids from long chain triglycerides (LCTs) (Bach and Babayan, 1982). LCTs are not completely oxidised and some long-chain fatty acids continue to be re-esterified to triglycerides in the liver (Goodenough and Wolfe, 1984). On the other hand, MCTs are more rapidly cleared from circulation (Dawes et al., 1986) and metabolised faster (Deckelbaum et al., 1990). MCTs are thought to enter the cell mitochondria for metabolism relatively independently of the carnitine acyltransferase transportation system required for long-chain fatty acids (Bach and Babayan, 1982). Due to the higher degree of oxidation and minimal re-esterification, MCTs are cleared more efficiently leading to a lower concentration of triglyceride (Cotter et al., 1989; Seaton et al., 1986).

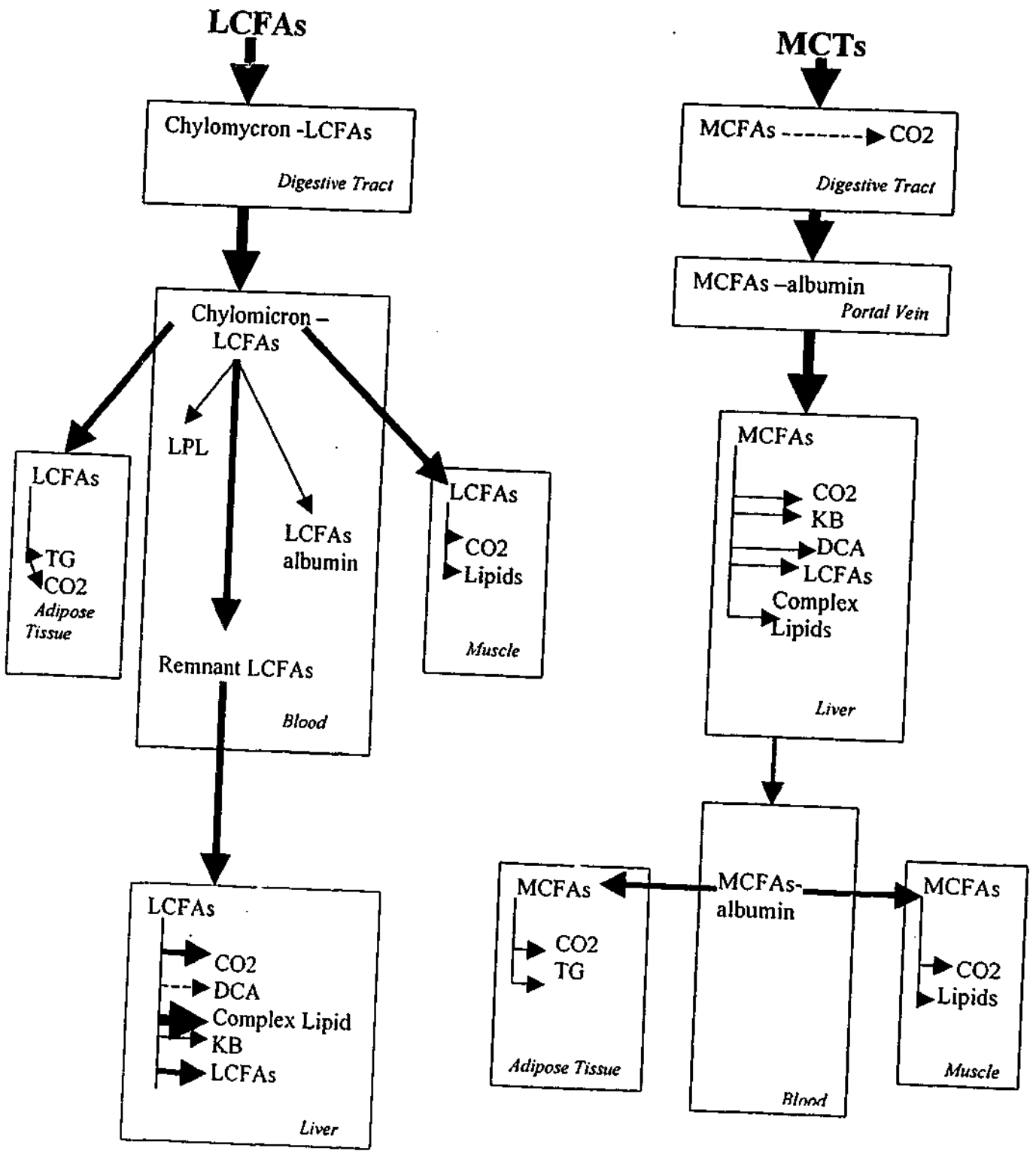


Figure 2.1: Transport, distribution, and metabolic fate of exogenous fatty acids according to their chain length (modified from Bach et al., 1996)

DCA: dicarboxylic acids; KB: ketone bodies; LCFAs: long-chain fatty acids; LCFAs': de novo synthesized, elongated and/or unsaturated LCFAs; LCTs: long-chain triglycerides; LPL: lipoprotein lipase; MCFAs: medium chain fatty acids; MCTs: medium chain triglycerides; TG: triglycerides.

The width of the arrows reflects the relative importance of the pathway.

Furthermore, when excessive MCTs are ingested, fat deposition is diminished (Baba et al., 1982; Geliebter et al., 1983). The reduction in the deposition of fat may be explained by the obligatory oxidation of MCT–derived fatty acids in the liver after being transported there via the portal vein, leaving almost no MCTs for incorporation into body fat. The decrease in the deposition of fat on MCT diets is related to an increase in metabolic rate and thermogenesis (Baba et al., 1982; Hill et al., 1989; Seaton et al., 1986).

2.2.8.3 Coconut and CHD Risk

High intakes of coconut oil have been correlated with elevated cholesterol levels in metabolic studies. SFAs from coconut oil may have been shown to inhibit the release of prostanoids in rats and to increase total plasma and HDL-cholesterol (Cox et al., 1995; Rodriguez-Vico et al., 1993). These effects are often more exaggerated when excessive cholesterol is present in the diet (Elson, 1992; Van and Zilversmit, 1988). In another study examining the effect of dietary coconut oil on lipoprotein composition of young chicks, Rodriguez-Vico et al., (1993) found after two weeks of coconut oil feeding that the composition of HDL changed drastically, total and VLDL-cholesterol increased significantly while triglyceride remained. Coconut oil has also been implicated in the development of hypertension (Hassall and Kirtland,). However, in a study comparing the effects of hydrogenated coconut oil with safflower and evening primrose oil, Soma et al (1985) demonstrated that the development of hypertension on a coconut oil diet was not significantly different from other oil diets (Soma et al., 1985).

In contrast to previous studies, recent studies have shown that populations consuming high amounts of coconut are less likely to have a stroke and ischaemic heart disease and, are more likely to have significantly lower serum cholesterol concentrations, lower diastolic blood pressure and be lean (Lindeberg et al., 1994; 1996; 1997a&b; Lindeberg and Lundh, 1993; Prior et al., 1981). A series of reports by Lindeberg et al have been published comparing the cardiovascular risk factor levels of traditional populations on the islands of Kitava and Trobriand in Papua New Guinea with healthy Swedish populations living in Sweden. The Kitavan diet consists of tubers, fruit, coconut, fish and vegetables. The intake of Western food and alcohol is negligible. Total fat intake is 21% of total energy, while saturated fat intake from coconuts is high (17% of total energy, mainly in the form of lauric and myristic acid). Compared with Sweden, the Kitavan population had a substantially lower diastolic blood pressure, body mass index and triceps skinfold thickness. Fasting serum cholesterol and apolipoprotein B were also 10-30% lower and even

though triglycerides were higher, HDL-cholesterol was similar and apolipoprotein (a) tended to be lower. A previous study of high consumers of coconut was done by Prior et al (1981) among two populations of Polynesians, the Pukapuka and the Tokelau. The habitual-diet of the two communities was high in saturated fat but low in dietary cholesterol and sucrose. Coconut was the chief source of fat. An analysis of a variety of food samples and human fat biopsies showed a high concentration of lauric and myristic acids. Vascular disease was uncommon in these populations and it was concluded that there was no evidence of a high coconut intake having a harmful effect. In India and Sri Lanka, where coconut is also used, two studies suggest there was no relationship between coconut consumption and the risk of CHD or CHD events (Atukorala and Jayawardene, 1991; Kumar, 1997).

2.3 LIPIDS AND GLUCOSE MEASUREMENTS

2.3.1 LIPOPROTEINS: THEIR PROPERTIES AND MEASUREMENTS

The major lipids found in human plasma include triglycerides, phospholipid and cholesterol esters. These molecules are all esters of medium and long chain fatty acids and together they comprise the lipid moiety of lipoproteins. The major function of lipoproteins is make lipids soluble in the blood. Lipoproteins also contain proteins, known as apolipoproteins. The main functions of apolipoproteins are to solubilise cholesterol esters and triglycerides, assist in the regulation of enzymes reactions important in the metabolism of lipoproteins, and bind to cell surface receptors. Fatty acids may also exist in plasma as the free, non-esterified form, mostly bound to albumin.

The blood lipid measurements that are usually performed in order to make a prediction of CHD risk are serum or plasma total cholesterol, triglycerides and HDL-cholesterol. There is a systematic difference between cholesterol measured in plasma and serum. However this difference is small. Total cholesterol from here on will therefore relate to either serum or plasma total cholesterol. LDL-cholesterol is most often calculated from the above measurements using the Friedewald formula (Friedewald et al., 1972). Direct methods for the measurement of LDL-cholesterol are available. These involve either ultra-centrifugation (Havel et al., 1955) or precipitation (Burstein and Scholnick, 1973). These methods are relatively time-consuming. Because most of the cholesterol is carried in LDL, there is a good correlation between total cholesterol and LDL-cholesterol, and an observed correlation with LDL-cholesterol. LDL-cholesterol is a better predictor of CHD

because it does not take into account HDL-cholesterol. However because of the difficulty of obtaining a direct measurement of LDL-cholesterol, most of the larger studies have used total cholesterol rather than a direct LDL measurement.

2.3.2 CHYLOMICRONS, VLDL AND FASTING TRIGLYCERIDES

Dietary fat containing triglyceride and cholesterol is digested and absorbed in the small intestine. Once taken up as monoglyceride and free fatty acids into the intestinal epithelial cells, absorbed lipids are re-esterified and packaged inside a monolayer of phospholipids, free cholesterol and lipoproteins to form large triglyceride-rich particles called chylomicrons. Nascent chylomicrons are secreted into the circulation via the lymph system, where they undergo rapid modification, with some components leaving the particle and others joining it (Calvert and Barter, 1981). One important modification is the progressive removal of triglyceride from the chylomicron remnant particles. Ingestion of a cholesterol rich meal may result in an increase in circulating chylomicron remnants and poor clearance in certain individuals. These chylomicron remnant particles are relatively cholesterol rich and may be atherogenic. Cholesterol from chylomicron remnants may be taken up by macrophages in the arterial wall, a process which may be important in the development of atherosclerosis (Van Lenten et al., 1985). A delayed clearance, which may occur in particular individuals, may be a risk factor for atherosclerotic vascular disease due to the atherogenic nature of the chylomicron remnants. In these individuals this might be an important pathway to atherosclerosis.

Cholesterol once delivered to the liver may be packaged into very low density lipoproteins (VLDL). Cholesterol is also synthesised in vivo. The major site of cholesterol biosynthesis is the liver. The role of VLDL is to transport endogenously synthesised triglyceride (including some cholesterol) to the peripheral tissues. Very low density lipoprotein synthesis is promoted by an increase in the flux of free fatty acids (FFAs) to the liver, or when the rate of hepatic synthesis of fatty acids increases (Thompson, 1994). VLDL particles show a considerable size variation. Because chylomicrons are normally cleared quickly from the circulation, a fasting plasma triglyceride measurement gives a measure of VLDL triglyceride, as well as triglyceride associated with chylomicron remnants and VLDL remnants. As with chylomicrons, lypolisis of VLDL fatty acids results in a smaller lipoprotein known as a VLDL remnant, beta-VLDL, or intermediate density lipoprotein (IDL). An intermediate density lipoprotein is also an atherogenic particle (Thompson, 1994). Intermediate density lipoproteins may then be converted to LDL.

2.3.3 LIPIDS AND LIPOPROTEINS, AND CHD

2.3.3.1 Cholesterol

Since the 1940s and 1950s, studies within populations as well as cross-cultural comparisons produced evidence that higher serum cholesterol levels were associated with an increased risk of CHD (Anderson et al., 1976; Levy et al., 1996). Total cholesterol (TC) comprises low-density lipoprotein (LDL) cholesterol, high density lipoprotein (HDL) cholesterol and triglyceride (TG). LDL-cholesterol is highly atherogenic while HDL-cholesterol is anti-atherogenic and thus protective. In terms of CHD risk, total cholesterol concentration may be misleading if the HDL level is unknown. TC/HDL-C ratio is used to predict risk of CHD (Brochu et al., 2000). An average or low total cholesterol level may be associated with a high risk of CHD if the HDL fraction is low (Forge, 1999).

Findings from many epidemiological studies have shown that the risk of CHD increases sharply when TC levels exceed 6.5 mmol/L (Gouldbourt and Medalie, 1979; Kannel et al., 1971; Nobili et al., 1994; Verschuren et al., 1995). For TC levels between 5.2 and 6.5 mmol/L, there is a moderate but not always clear relationship with CHD events (Deslypere, 2000; Kannel et al., 1971; Pooling Project Research Group, 1978).

A report from the 25-year follow-up of the Seven Countries Study revealed that cholesterol was linearly related to CHD mortality, and the relative increase in CHD mortality rates with a given cholesterol increase was the same. The baseline data of the Seven Countries Study was completed from 1958 to 1964 from five European countries, the United States and Japan (Verschuren et al., 1995). The Framingham study began in 1948. It was a prospective study of 5,127 subjects who did not have demonstrable CHD at entry. The relationship between plasma lipids and the risk of CHD was then examined. Total cholesterol concentrations were found to be directly associated with CHD risk (Kannel et al., 1971).

In 1978, the Pooling Project Research Group analysed and reported on pooled data from eight individual studies. The report found that elevated total cholesterol levels were associated with an increase in the incidence of major coronary events. The relationship between total cholesterol and CHD has now been demonstrated within many populations (Ducimetie're et al., 1980; Goldbourt et al., 1985; Holme et al., 1981; Jonhnson et al., 1968; Rose and Shipley, 1980). Another prospective study of a population from Shanghai in China (a population where total cholesterol concentrations were low compared to Western populations), has shown that the

relationship between total cholesterol and CHD also operates at lower concentrations of total cholesterol (Chen et al., 1991).

However, the concept of an adverse cholesterol profile with CHD does not remain constant with age. Abbott et al. (1997) described the levels of total cholesterol and HDL-cholesterol in a group of elderly men and then compared these levels to those that were observed 20 years later (Abbott et al., 1997). They found that men with prevalent CHD at the end of the 20-year follow-up period experienced significantly greater reductions in total cholesterol, followed by men who developed CHD later and men who remained disease free. It suggests that large reductions in total cholesterol in the elderly may signal occult disease or declines in overall health, although the changes may be part of natural aging.

2.3.4 LOW DENSITY LIPOPROTEIN (LDL)

Elevated blood levels of LDL are considered to be a major risk factor for the pathogenesis of atherosclerosis. There is accumulating evidence that oxidative damage to LDL creates a modified form of LDL, which is more rapidly taken up by "scavenger" receptors on monocytic macrophages in the arterial wall, leading to foam cells, an early step in the genesis of atherosclerosis (Ross, 1986).

Several epidemiological studies have shown that the risk of CHD increases sharply when LDL-cholesterol level exceeds 4.9 mmol/L, a level that corresponds to a cholesterol level of about 6.5 mmol/L (Deslypere, 2000; Kannel et al., 1971; Pooling Project Research Group, 1978). The US National Cholesterol Education Program has released guidelines that focused on LDL as the primary target for intervention. In the guidelines, patients without CHD and who have an LDL – cholesterol level of more than 4.1 mmol/L are considered at high risk. A 20% to 30% reduction in serum LDL-cholesterol levels may enable sufficient extraction of LDL-cholesterol from the atherosclerotic lesion to stabilise it, thereby preventing plaque disruption and coronary events (Deslypere, 2000).

2.3.5 HIGH DENSITY LIPOPROTEIN (HDL)

An increased level of HDL is a particularly strong predictor of a decreased risk of CHD in men and women (Jacobs et al., 1990). Several studies have shown that low HDL-cholesterol had been associated with CHD, even without elevated LDL- or total cholesterol (Assmann et al., 1998; Castelli et al., 1986; Gordon et al., 1977;

Wilt et al., 1997). The risk of CHD in relation to HDL is also influenced by the prevailing LDL concentration. A study found CHD mortality was not increased in subjects who had isolated low HDL-cholesterol compared with those with both low HDL-cholesterol and high total cholesterol (Goldbourt et al., 1997).

Reverse cholesterol transport (RCT) describes the metabolism and important antiatherogenic function of HDL (von Eckardstein, 2001). The HDL-mediated efflux of cholesterol from non-hepatic cells is delivered to the liver and steroidogenic organs, where it is used for the synthesis of lipoproteins, bile acids, vitamin D, and steroid hormones (Genest, 1999; Stein and Stein, 1999). Distortion of RCT can favour the deposition of cholesterol within the arterial wall and therefore promote the development of arteriosclerosis (Glomset, 1968).

2.3.6 TRIGLYCERIDES

Several epiemiological studies have indicated that hypertriglyceridaemia is an independent risk factor for CHD. High triglycerides were found to be an independent risk factor for CHD in 2,300 Swedish males who were followed for 7-10 years (Aberg et al., 1985). In 1,500 females followed for 12 years, hypertriglyceridaemia was found to be a risk factor for fatal myocardial infarction, stroke and total mortality (Lapidus et al., 1985). A high relative risk for CHD has also been associated with elevated serum triglycerides, as reported in the PROCAM study, (Assmann and Schulte, 1992), the Helsinki studies (Manninen et al., 1992), and the Framingham Study (Castelli, 1988). It has been postulated that subjects with increased serum triglycerides and reduced HDL often have a different form of LDL whether or not the LDL concentrations are elevated. This is called pattern B LDL, often characterised by LDL particles, which are smaller and more dense. Pattern B LDL is atherogenic and present in 80 % of subjects with triglycerides of 2.3 mmol/L or higher (Austin et al., 1990).

2.3.7 LIPOPROTEIN (a)

A number of studies have found that an increased Lp(a) concentration is associated with CHD (Berg et al., 1974; Dahlen et al., 1986; Hearn et al., 1990; Kostner, 1989; Murai et al., 1986; Rhoads et al., 1986; Rosengren et al., 1990; Sandkamp et al., 1990). Lp(a) has been shown to be a genetically determined independent risk factor for coronary artery disease (Dahlen et al., 1986).

The role of Lp(a) in atherosclerosis and thrombosis has been controversial, however the evidence for an association between Lp(a) levels and CHD is highly suggestive. It has been found that Lp(a) prolongs the time required for fibronolysis. The mechanism for this appears to be the inhibition of the conversion of plasminogen to plasmin (Edelberg et al., 1990). These functions may relate to an increased risk of myocardial infarction through the thrombogenic arm of such an event.

2.3.8 PREDICTION OF CHD WITH LIPIDS AND LIPOPROTEIN MEASUREMENTS

Total cholesterol, LDL-cholesterol, triglycerides, HDL-cholesterol can all be used as indicators of CHD risk. Various combinations of the lipoprotein measurements have also been used in attempt to improve the accuracy of predicting CHD. The ratio of total cholesterol or LDL-cholesterol to HDL-cholesterol is often used.

2.3.9 INSULIN AND GLUCOSE

Epidemiological data supported by three large prospective surveys have suggested a role for hyperinsulinaemia as an independent risk factor for CHD (Depres et al., 1996). In a 17-year prospective study, Moller and Jespersern (1995), found that fasting serum insulin levels in both men and women were a good predictor of the development of CHD and CVD, even after controlling for relevant confounding factors (Moller and Jespersen, 1995). The Paris prospective study, a long term, large scale study investigating the factors predicting CHD, suggested that plasma insulin levels following a two hour post glucose load, predicted coronary heart mortality (Fontbonne, 1991). Hyperinsulinaemia was also reported to be a predictors of future diabetes in subjects with normal glucose tolerance (Haffner et al., 1990; Saad et al., 1988; Sicree et al., 1987). Furthermore, insulin resistance contributes to hypertriglycidaemia and low HDL-cholesterol concentrations (Wahlqvist, 2001).

The extent to which hyperglycaemia and insulin-resistance contribute independently to CVD is debatable and depends on the coexistence of other risk factors. The site of adverse action of these factors may be either at the arterial wall or in the large body organs. Arterial damage reduces the capacity for blood flow and also the use of glucose as a fuel which is critical in anaerobic metabolism due to the ischaemia that reduces the oxygen supply (Wahlqvist, 2001).

2.4 BODY COMPOSITION

The degree of obesity is assessed using the body mass index, which is calculated by dividing weight by height squared. Apart from obesity, the distribution of body weight is also important. Fat distribution differs according to gender, as well as showing individual variation. In general, gluteal-femoral fat distribution is more common to women, whereas in men, an abdominal or upper body distribution is more common. The waist-to-hip circumference ratio (WHR) is most often used as a measure of fat distribution.

2.4.1 BODY MASS INDEX (BMI)

Historically, ideal or desirable weights have been defined as those associated with the lowest morbidity. Sjostrom (1992) reported that in a comprehensive study of 40 subjects, a curvilinear relationship was observed between BMI and mortality (Sjostorm, 1992a). There was therefore an increase in cardiovascular disease at both a low and high BMI. A BMI greater than 35 kg/m^2 was associated with an approximately 2-fold increase in total mortality, and several-fold increase in mortality due to diabetes, CVD, and certain forms of cancer (Sjostorm, 1992b). Further prospective study have also shown a positive association between BMI and CHD incidence (Waaler, 1984). Another prospective study by Willett et al (1995), reported a strong positive association between BMI and risk of CHD during 14 years of follow-up among women in the Nurses' Health Study (Willett et al., 1995). Case-control studies have also found a positive correlation between body weight, BMI, height and CHD events (D'Avanzo et al., 1994; Tavani et al., 1997).

Weight gain after 18 years of age has been found to be a strong predictor of CHD risk. Because increases in weight largely reflect fat mass, which may anticipate risk within ranges of attained weights that are plausibly due to differences in muscle or bone mass (Willett et al., 1995). Middle age women who gained weight more than 5 kg from 18 years of age appeared to have higher risk for CHD (Willett et al., 1995). Excess weight change has also been shown to promote atherosclerosis (Stevens et al., 1998).

However some studies found no statistically significant association between BMI measurements with CHD (Paffenbarger et al., 1986; Rosengren et al., 1999). The inconsistency of data in some studies can be explained by the lack of numbers and/or the uncommon prevalence of overweight and obesity in the study subjects. In the WHO MONICA Project, the sensitivity of measurement to identify subjects

with overweight or obesity was generally lower in populations in which overweight was relatively uncommon (Molarius et al., 1999a).

2.4.2 ABDOMINAL FAT DISTRIBUTION AND CHD

A central distribution of body fat, indicated by a high waist to hip ratio, has been shown to be associated with CHD risk (Lapidus et al., 1984). Studies carried out in Goternburg, Sweden, showed that even with a low body mass index, abdominal fat (body fat with the highest risk) was associated with cigarette smoking, insulin resistance, hyperinsulinaemia, diabetes, high blood pressure; and an abnormality in plasma lipoprotein lipids (DeFronzo and Ferrannini, 1991). Furthermore, obesity (particularly the android pattern of fat), has been associated with an increased risk for developing CVD (Bjontorp, 1992; Peiris et al., 1991).

The distribution of adipose tissue which indicates possible obesity-associated complications clearly depends on the visceral component of excess body fat, and does not necessarily need to be combined with general/total obesity (Bjontorp, 1992). The abdominal distribution of fat is often measured as WHR. There are better anthropometric measurements for the estimation of abdominal fat distribution, however the WHR, has a unique power as a predictor of various diseases, including CVD (Pihl and Jurimae, 2001). Waist circumference alone has also been recommended as a useful measure of abdominal fat and risk of CVD (Molarius et al., 1999a & 1999b; Molarius et al., 1999). The WHO Monitoring Trends and Determinants in Cardiovascular Disease (MONICA) Project and other studies have suggested a waist circumference cut off point of 94 cm for men and 80 cm for women. Men with a waist circumference ≥ 94 cm and women with a waist circumference ≥ 80 cm should gain no further weight (waist action level 1) and, men with a waist circumference ≥ 102 cm and women with a waist circumference ≥ 88 cm should reduce their weight (waist action level 2) (Lean et al., 1995; Molarius et al., 1999a & 1999b).

Ethnic differences in fat distribution have been reported in the literature. A greater tendency toward upper body obesity has been documented in black women compared to Caucasian women (Kumanyika et al., 1993). The tendency has been observed in Mexican Americans, compared with Caucasians regardless of gender. Anthropometric comparisons have shown that although Asians usually have a lower BMI than whites, they have more upper body subcutaneous fat than Caucasians of both genders (Wang et al., 1996).

2.5 BLOOD PRESSURE

Prospective studies have demonstrated a strong association exists between elevated blood pressure and CHD (Levy et al., 1996; Njolstad et al., 1996). In a follow up study of the original Framingham Study subjects and their offsprings, it was observed that hypertension antedated the development of congestive heart failure, and was associated with a three-fold risk of developing CHD (Levy et al., 1996). The relationship between hypertension and obesity has been fully documented in cross sectional studies of differing socio-economic, racial and ethnic groups. In the multi-centre Community Hypertension Evaluation Clinic study, the prevalence of hypertension was 50% to 300% higher in those who were overweight compared with those who were of normal weight (Stamler, 1978; Stamler et al., 1993). According to reports from the Framingham Study, hypertension increases with adiposity (Kannel et al., 1967 & 1990). Weight gain has been shown to increase both systolic and diastolic blood pressure (Sinaiko et al., 1999).

The mechanisms by which high blood pressure causes CHD relates to the response to injury hypothesis (Ross, 1985). An increased blood pressure places higher stress on the arterial wall, which may result in endothelial damage, initiating the atherosclerosis. It is also possible that the atherosclerotic changes that occur in the arterial wall result in an elevation of blood pressure. There may therefore be synergy between atherosclerosis and hypertension.

2.6 CONCLUSION

On the basis of the uncertainty about how food, and it food components such as fat, predict not only serum lipoprotein status, but also CHD outcome, the present study on the coconut-based Minangkabau culture has been devised.

CHAPTER 3

Subjects and Methods

3.1 INTRODUCTION

The methods used in all the studies presented in this thesis are described in this chapter.

3.2 SUBJECT POPULATION

Results presented in this thesis involve three major studies:

a) the Focus Group Discussion study, which is presented in Chapter 4

b) the Case-Control Study, which is presented in Chapters 5-7

c) the Intervention Study, which is presented in Chapters 8-9.

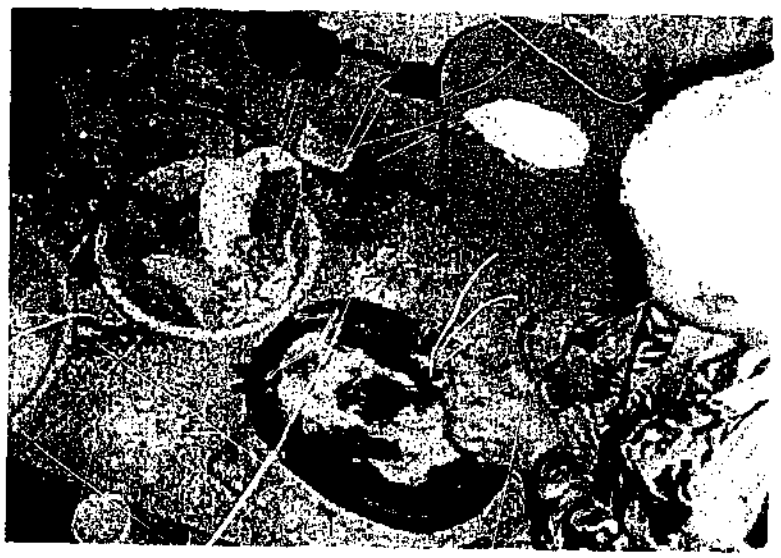
This research project was approved by the Monash University Standing Committee on Ethics in Research Involving Humans in November 1998 (Project Number 98/458) and acquired written permission from the local government in West Sumatra, Indonesia. All subjects gave written informed consent prior to their participation in the study.

3.2.1 THE FOCUS GROUP DISCUSSIONS

This study obtained in-depth opinions from two different generations of the Minangkabau people about their food culture in regard to the manner and style of food preparation, to gain a better understanding of dietary patterns, using a questionnaire (Appendix 1). The Focus Group Discussions took place in four villages in West Sumatra from January to March 1999. Two of the villages, Naras and Kalumbuk, are located in the coastal regions of Pariaman and Padang municipalities, respectively. The other two villages, Pincuran Panjang and Kubang, are in the mountainous regions of Tanah Datar and 50 Koto districts, respectively. Thirteen women aged 46 to 67 years, participated in the discussion group in Naras.

Focus Group Discussion in Nareh, Pariaman

Prof. Wahlqvist visited one of the Focus Group Discussion groups



The Minangkabau used a wide range of herbs and spices along with the coconut milk.

In Kalumbuk, nine women aged 30 to 67 years joined in the discussion. In Pincuran Panjang, seven women aged 46 to 81 years old and thirteen women in Kubang, aged 33 to 77 years old were also included in the discussion. Each session was tape-recorded and lasted up to one and half-hours.

3.2.2 THE CASE-CONTROL STUDY

This study examined the difference in food patterns and CHD risk factors between the CHD cases and their gender- and age-matched healthy subjects serving as the controls. The study was conducted from February to August 1999. All patients diagnosed with CHD in the last 6 months were eligible for this study. All patients enrolled in the study had to have received the diagnosis from cardiologists. The diagnosis followed the WHO criteria which was based on typical symptoms, typical changes in ECG or enzymes (Rose et al., 1982).

Eligible patients were identified through the co-operation of five participating hospitals located in two cities in West Sumatra. The two cities were Padang, which is the capital of the West Sumatra province and Bukittinggi, a more rural county situated in a mountainous area 88 km north of Padang. Subjects in the Case group were recruited from the outpatient clinic of the Cardiovascular Unit in the five hospitals in Padang and Bukittinggi. As the largest hospital in West Sumatra and located in the capital city of the province, majority of the Case group (81 cases) were recruited from M Jamil General Hospital. Other hospitals in Padang where the recruitment took place were Heart Foundation Hospital (7 cases) and Yos Sudarso Hospital (5 cases) and the two hospitals in Bukittinggi were Ahmad Mukhtar General Hospital (12 cases) and YARSI Hospital (3 cases). Subjects in the Control group were recruited from the outpatient Ear, Nose and Throat and the Eye clinics from the same hospitals and came from the same areas as the cases. The controls were randomly selected from people matched to the Case subjects on the basis of age and gender. Subjects in the Control group who had health problems related to cardiovascular diseases, such as hypertension and diabetes mellitus, were not included. Pregnant women were also excluded.

A total of 108 eligible cases were interviewed, but 15 of them did not turn up on the examination day. Among the controls, 220 were interviewed but only 189 had anthropometric measurements and blood samples taken. One subject in the Control group did not complete the Food Frequency Questionnaire. Eligible subjects in this study, came not only from Padang and Bukittinggi but also from almost all parts of West Sumatra: Pariaman, Painan, Agam, Padang Panjang, Batusangkar, Payakumbuh and Solok.

3.2.3 THE INTERVENTION STUDY

This study examined the effect of increased coconut consumption on food patterns, serum lipid, glucose, phospholipid fatty acid concentrations and body composition. The study was conducted from May to July 2000. The participants were recruited from two villages located in the semi urban area of West Sumatra. Subjects in the Coconut group were chosen from the villagers who lived in Nareh, in the southern part of Padang Pariaman Municipality, while those in the Non-coconut group were from Kapalo Koto village, District of Pauh in Padang Municipality. The majority of Kapalo Koto villagers were farmers while Nareh was predominantly a fishing village. This Intervention study was conducted simultaneously in these two villages in order to minimise the communications between the two groups.

A screening interview was conducted with individuals from the two villages who responded to the announcements made by the head of the villages, nurses and midwives of the two community health centres and also by word of mouth. Selection was based on the following:

1) being willing to comply with the requirements of the programme over the period of the intervention
2) having no health problems
3) not being pregnant

A total of 46 subjects (12 men and 34 women), aged 28-81 years, from Nareh were assigned as the Intervention group, while a total of 42 subjects (3 men and 39 women), aged 29-75 years, from Kapalo Koto were assigned as the Control group.

3.3 DATA COLLECTION

3.3.1 CONDUCT OF THE STUDY

After the subjects agreed to participate in the study and before an interview took place, they were given an introductory information package written in Indonesian. They were then invited to the Department of Nutrition, Medical Faculty of Andalas University in Padang, where the interviews were conducted by three dietitians who had experience in nutrition surveys. Anthropometric, bio-impedance, blood pressure and electrocardiogram measurements were performed by a single examiner (the candidate) in order to achieve standardised measures. Blood taken was done by trained health professionals. For subjects who live in Bukittinggi and surrounds, the interviews and examinations were done in the local hospitals or in the villages. Personal examination results were reported to the subjects within 6 weeks after the

interview. Access to the research data was limited to research team members to ensure privacy and confidentiality.

Data was obtained by:

1. Interviews using the following structured questionnaires
   - Demography, health status and life style questionnaire
   - Food habits: Eating practices questionnaire, and
   - Dietary intake: food frequency intake questionnaire.

   All interviews and questionnaires were in Indonesian.
2. Anthropometric, bio-impedance analysis and blood pressure measurements.
3. Biochemical measurements of blood samples

3.4 DEMOGRAPHY, HEALTH STATUS AND LIFESTYLE INFORMATION

A questionnaire was used to obtain information on demographic and socio-economic characteristics (Appendix 2, questions 1-7), self-reported health conditions, medication use (Appendix 2, questions 8-26) and information on life style factors such as physical activity and stress (Appendix 2, questions 27-30).

3.4.1 STRESS AND PHYSICAL ACTIVITY

The questions on stress referred to their self-assessment of stress as to whether it adversely affected their health or not and the stress levels the subjects usually experienced every day.

The level of physical activity was assessed by combining leisure activity scores with occupational activity scores. Leisure and occupational activity levels were categorised on a four level scale: sedentary (score=1), light (score=2), moderate (score=3) and heavy (score=4) levels. Physical activity indices were then classified as low (0-2), moderate (3-5), or high (6-8).

Leisure activities were scored according to the intensity and duration of the activity engaged in. For example, activities of low intensity that could be done sitting down

or lying down, were generally home bound and, involved no outside activity were considered sedentary and included activities such as reading and watching television (score=1). Activities such as warming up or toning exercises, light gardening, house cleaning and cooking were categorised as light activities if they lasted longer than 30 minutes (score=2). Activities such as strolling or walking were given a moderate activity score if they lasted longer than 30 minutes (score=3). Activities of high intensity such as weight lifting, power walking or jogging, or other competitive sports with a duration of 30 minutes or longer were given a high activity score (score=4).

Occupational activities were scored in a similar way. The unemployed or office workers such as administrators and clericals who were required to sit all day, received a sedentary activity level score (score=1), while those who worked in trading, carpentry and farming received a moderate activity score (score=3). Mining or quarry workers received a high activity level score (score=4).

3.4.2 Cigarette Smoking and Alcohol Consumption

Information on smoking status and alcohol consumption was obtained from a questionnaire on demography, health status and lifestyle (Appendix 2, questions 31-32 and 53-55). Smoking status was identified as non-smokers, ex-smokers (those who had given up smoking for more than one year) or current smokers. Information on the number of cigarettes smoked in a day and the number of years of smoking was also obtained to quantify their smoking habit. Details on the number of glasses drunk, the types of drinks, and the frequency of drinking were asked to obtain information on alcohol consumption.

3.4.3 Eating Practice Questionnaire

Information on food eating patterns, food choices and food preparation methods (Appendix 2, Questions 35-52) was very important and was used to identify food culture. Questions were asked about coconut milk consumption patterns, use of dietary fat and oil, the amount of salt and sauce, the frequency of some spices used, preference of cooking methods and dietary change.

3.5 FOOD INTAKE INFORMATION

For both Case-Control and Intervention studies, food intake information was obtained using a Food Frequency Questionnaire (FFQ). In the Case-Control study, recollection of the food habit before diagnosis was probably best measured by the questions about past diet, although there was a strong influence of current diet (Willett, 1998). In this study, eligible cases were diagnosed within 6 months prior to the participation in the study. Within this timeframe, the subjects would have been able to recall their food habits and lifestyles before being diagnosed as having CHD.

An FFQ is a semi-quantitative 12-month recall method. The questionnaire contains a total of 215 food items of popular Indonesian foods and dishes, and traditional Minangkabau foods and dishes (Appendix 3). The questionnaire was modified using the Nutrition and Metabolic Study of Indonesian Elderly (Purba, 2000). Food items listed in the questionnaire were cooked using various methods, such as deep frying, stir-frying, grilling, stewing or boiling. Information on the amount and frequency of each food item consumed was obtained, provided that the food was eaten at least once a month. The frequency of food consumption was expressed in terms of 'per day', 'per week' or 'per month'. To obtain information on portion sizes, the food frequency questionnaires were completed with the aid of food models developed by the Nutrition Research Centre in Bogor, Indonesia. These food models were commonly used for dietary assessment. Where appropriate, household measures such as cups, glasses, teaspoons, tablespoons were also used.

3.5.1 VALIDATION OF FOOD FREQUENCY QUESTIONNAIRE

It is difficult to assess the validity of measurement of dietary intake in free-living subjects, because all methods rely on information given by the subjects themselves (Bingham,1994). A ratio of reported energy intake (EI) to basal metabolic rate can be used to validate dietary questionnaires (Bingham, 1994; Black et al., 1991). This method uses the principle of energy physiology to obtain cut-off values for physical activity levels (PAL), expressed as multiples BMR for evaluating reported energy intake against the expected level of energy expenditure (EE) in a sedentary population. It is based on the notion that non-obese individuals are in energy balance as shown in the equation:

$$\text{Energy intake } (EI) = \text{Energy expenditure } (EE)$$
$$\text{or } EI/EE = 1$$

If EE = PAL*BMR; then PAL = EE/BMR (James et at., 1988). BMR was calculated from standard equations based on body weight (Schofield, 1985).

Table 3.1: Basal metabolic rate

Subject group			Basal metabolic rate
Adults	30 – 60 years	Male	(0.048 x weight) + 3.653
		Female	(0.034 x weight) + 3.538
	> 60 years	Male	(0.049 x weight) + 2.459
		Female	(0.038 x weight) + 2.755

The PAL value of 1.27 when substituted in the equation EE = 1.27*BMR is regarded as the "minimum energy requirement" needed for survival, but it is not compatible for long-term health (FAO/WHO/UNU, 1985). The average value of 1.55, also used in the same report, is associated with a sedentary life. Another report by Goldberg et al. (1991) suggested a cut-off value of 1.35 as the lowest value for the habitual energy intake level of an individual. This is compatible with a normal (not bed bound) lifestyle; and they also suggested that the PAL value of 1.55 was the lowest value that could reflect actual energy intake over a given measured period. Bingham (1994) suggested using a PAL cut-off of 1.35 if BMR had been measured using doubly labelled water because the limits of precision would be smaller.

The method described by Goldberg et al. (1991) and Black et al. (1991) was used to validate the FFQ in this study. BMR was first calculated for all subjects using the Schofield equation. Energy intake in megajoules (MJ) was calculated from the FFQ. The ratio of energy intake to BMR (EI:BMR) was then calculated for each subject to assess the PAL value. EI:BMR ratios were compared to the cut-off values 1.2 and 1.55. Energy intake values less than 1.2*BMR indicated erroneous estimates of habitual food intake resulting from under-reporting. Then the population average EI:BMR ratio was compared to 1.55.

Results of the determination of EI:BMR ratio in the Minangkabau population are shown in Tables 3.2 and 3.3

Table 3.2: Distribution of EI:BMR ratio in the Minangkabau population

	N	Mean ±	SD	Minimum	Maximum
Case group	93	1.35 ±	0.45	0.57	3.14
Men	61	1.33 ±	0.42	0.59	2.95
Women	32	1.39 ±	0.47	0.57	3.14
Control group	188	1.27 ±	0.39	0.56	2.85
Men	112	1.22 ±	0.36	0.57	2.08
Women	76	1.33 ±	0.42	0.56	2.85

Table 3.3: Percentage of subjects with EI:BMR ratio above and below 1.27

Energy intake	Case		Control	
	Men	Women	Men	Women
Less than or equal to (1.27*BMR)	31 (50.8	16 (50.0	66 (57.9	39 (50.7
Greater than (1.27*BMR)	30 (49.2	16 (50.0	48 (42.1	38 (49.4

A dietary method can be regarded as invalid if the population habitual mean intake is less than 1.55 times the PAL calculated from the ratio of EI:BMR, as suggested by Goldberg et al. (1991) and Black et al. (1991). Table 3.2 shows that the mean EI:BMR ratio of both the Case and Control groups was less than 1.5, suggesting under-reporting of this study population. Table 3.3 shows that approximately more than half of the population under-reported their habitual food intake. However, it was observed that among the under-reporters, only 3% of the cases and 5% of the controls had BMI above 28 kg/m^2. Several studies of Asian populations have documented under-reporting, and it is very pronounced in obese subjects (Bingham, 1994; Hulshof et al., 1995; Wolmarans et al., 1999).

It is also important to note that BMR used in this study is derived from equations based on Caucasian subjects. Several studies show that these equations tend to

overestimate the BMR of Asians, as well as of some Caucasians (Lapidus et al., 1985; Leung et al., 2000; Liu et al., 1995).

3.5.2 FOOD GROUPS

The total 215 food items in the FFQ were grouped into 13 food groups, according to their biological classification (Table 3.4). The consumption of each food group (grams per day) was the sum of various food items in the same food group.

Table 3.4: List of food groups

Major food groups	Food items included in food groups	Source
Fish, seafood and their products	All types and forms of fish, crustacea and mollusca, canned fish and fish products	Animal
Meat	Chicken, beef, lamb, organ meat	Animal
Eggs	Hen eggs (whole, white and yolk), duck eggs and quail eggs	Animal
Dairy products	Milk, cheese, yoghurt	Animal
Soy products	Tempeh, tofu and soy milk	Plant
Legumes and nuts	Mungbean, peanut, kidney bean, coconut milk	Plant
Coconut products	Coconut milk, desiccated coconut	Plant
Rice and other cereals	Bread, noodles, corn, cassava,	Plant
Vegetables	Green leafy vegetables, root vegetables, flowers, onions, peppers, tomatoes	Plant
Fruits	Tropical fruits, banana, orange, melon, apple, pear, passion fruit, stone fruits	Plant
Sugar and sugar products	Soft drinks, cakes, chocolate bar and syrups	Not classified
Oils and spreads	Coconut oil, palm oil, margarine, butter.	Not classified
Beverages	Tea, coffee	Not classified

Of the 13 food groups, 10 were further collapsed into two major food groups: animal food groups (meat + egg + fish + dairy products) and plant food groups (legumes and nuts + soy products + rice and other cereals + vegetables + fruit + coconut milk).

3.5.3 NUTRIENTS

Information on the nutrient content of food items was obtained from the Nutrient Composition Tables of Indonesian foods (Mukrie et al., 1995), and the Nutrient Composition Tables of Malaysian foods (Tee et al., 1997), and was used to convert food items to total energy, dietary fat, protein, carbohydrate and micronutrients. Data on the fatty acid composition of various foods was taken from a USDA Nutrient Database for Standard Reference, www.nal.usda.gov/fnic/cgi-bin/list_nut.pl, retrieved on the 27th February 2001, and used to calculate individual fatty acid intakes (United State Department of Agriculture, 2001).

Subjects whose daily energy intake was implausibly low or high, for example, less than 2094 kJ (500 kcal) or more than 14,650 kJ (3500 kcal), were not included in the further data analyses of food or nutrient intakes (Willett, 1998c). Nutrient intake was presented in grams per day, as percentage of total energy intake, and as nutrient density, which was calculated as follows.

Nutrient density (g/kcal) = (nutrient quality in grams/total energy in kcal) * 1000

3.5.4 FOOD VARIETY

Previous studies of food variety have determined a food variety score representing the total number of foods consumed in a given time period. Food variety indices for specific food groups have also been used (Savige et al., 1997). In this study, food variety scores were constructed in order to describe the variety of food consumed within a food group.

Food group variety scores include:

Meat variety score (0-44/week)
Fish variety score (0-23/week)
Dairy variety score (0-8/week)
Legume variety score (0-27/week)
Cereal variety score (0-33/week)
Vegetable variety score (0-42/week)
Fruit variety score (0-23/week)
Sugar variety score (0-7/week)
Other variety score (0-22/week)

Broader food groups and total variety scores are listed below.

Animal variety score (0-75/week):

meat + fish + dairy varieties

Plant variety score (0-125/week):

legumes + cereals + vegetables + fruit varieties

Total food variety score (0-220/week):

animal + plant + other food varieties

Sugar products and other food varieties were not included in the plant and animal variety scores. A medium serving of a food or mixed dish within a food group had to be consumed at least once a week or more to obtain a score.

3.6 CLINICAL EXAMINATION

3.6.1 ANTHROPOMETRIC MEASUREMENTS

Standard anthropometric measurements used in this study were based on Lohman et al. (1988). All measurements were performed in duplicate on the dominant side of the body determined by whether or not a person was right-handed or left-handed.

3.6.1.1 Stature and Body Weight

Stature was measured to the nearest milimetre using a microtoir fixed to the wall. Subjects stood on a flat floor in barefeet and wore minimal clothes. Body weight was measured using a single-beam balance scale.

3.6.1.2 Skinfold Thickness

Skinfold thickness measurements for body fat estimation were performed at four sites: biceps, triceps, subscapular and suprailiac using standard Harpenden skinfold calipers. The thumb and index finger of the left hand were used to elevate a double fold of skin and subcutaneous adipose tissue about 1 cm proximal to the site at which the skinfold was to be measured. The jaws of the caliper were placed so that the thickness of skinfold was measured perpendicular to its long axis. Two readings were recorded, each to the nearest 0.2 mm.

Biceps skinfold was measured as the thickness of a vertical fold on the anterior surface of the biceps, midway between the anterior auxiliary fold and the antecubital fossa.

Triceps skinfold was measured as the thickness of a vertical fold on the posterior midline of the upper arm, over the triceps muscle, halfway between the acromion process (bony process on top of the shoulder) the olecranon process (bony process on the elbow).

Subscapular skinfold was picked up on a diagonal, inferior-lateral incline approximately 45° to the horizontal plane in the natural cleavage lines of the skin. The site was just inferior to the inferior angle of the scapula. The jaws of the callipers were applied 1 cm infero-lateral to the thumb and the index finger raising the fold.

Suprailiac skinfold was measured in the midaxilllary line immediately superior to the iliac crest. An oblique skinfold was grasped just posterior to the midaxillary line following the natural cleavage lines of the skin. It was aligned inferomedially at 45° to the horizontal plane.

3.6.1.3 Waist and Abdominal Circumferences

Waist circumference was measured by positioning a non-stretchable fibreglass tape either at:

a) the level of natural waist which was the narrowest part of the abdominal circumference, or if this was not apparent for the very obese

b) 12 cm from the xiphisternal notch.

Abdominal circumference is measured mid-way between two bony landmarks: the inferior point of the rib cage and the superior iliac crest.

The maximal abdominal circumference was taken at the level of the umbilicus. The maximal abdominal circumference was the greatest circumference around the abdomen. Three readings were taken at each position to the nearest 0.1 cm, and the average of the three readings was recorded.

The hip circumference is a measure of external pelvis size and it reflects the amount of adipose tissue in the region. The maximum (gluteal) hip circumference was

measured by placing a non-stretchable fibreglass tape around the buttocks at the area of maximum gluteal protrusion in a horizontal plane without compressing the skin. Three readings were taken to the nearest 0.1 cm and the average recorded.

Abdominal-to-hip ratio (AHR), a measure of abdominal fat distribution, was calculated by dividing the maximal abdominal circumference by hip circumference.

3.6.2 Blood Pressure

Blood pressure was measured in the morning on the subject in a sitting position using the auscultatory method with a standard sphygmomanometer and cuff. The occurrence of sounds (phase I) was recorded as the systolic blood pressure, and the disappearance of sounds (phase V) was recorded as diastolic blood pressure (WHO, 1996). All measurements were performed in duplicate.

3.6.3 Electrocardiography (ECG)

An electrocardiographic (ECG) method has been used in population studies for years to assess a subject's heart condition (Pipberger et al., 1982). An ECG machine model HP4745A was used in this study. The ECG was read and the heart condition diagnosed by a medical intern. ECG was used only for checking entry criteria.

3.7 BIOCHEMICAL MEASUREMENTS

Prior to blood collection, the subject was asked to fast overnight. All blood samples were collected by trained health professionals. About 30 mL venous samples were collected in evacuated glass tubes with or without citrate (Vacutainer®, Becton Dickinson Ltd, Cowley, UK). Blood samples were then stored in ice and transported to the Laboratory Unit of Yos Sudarso Hospital where blood samples were centrifuged. Plasma samples were collected in small portions in plastic tubes and stored at –70°C until further analyses in Melbourne, Australia.

3.7.1 Total Cholesterol, HDL-Cholesterol, LDL-Cholesterol and Triglycerides

Total cholesterol was measured enzymatically using cholesterol oxidase/peroxidase system of Abbott Spectrum cholesterol reagent (Abbott Laboratories, USA,

Diagnostics Division, Abbott Park, IL 60064, USA, list number 1375-04). Total cholesterol measurements were calibrated against a serum based Abbott Spectrum multi calibrator set (list number 1357-01). HDL-cholesterol was measured as for total cholesterol, and the measurements were calibrated against Abbott Spectrum HDL calibrators or equivalent (list number 13711-01). Triglycerides were measured enzymatically using glycerol kinase Abbott Spectrum cholesterol reagent (list number 1351-03). Total and HDL-cholesterol and triglyceride measurements were performed on a CCX Abbott Spectrum Autoanalyser. LDL-cholesterol was then calculated using Friedewald formula (Friedewald et al., 1972).

LDL cholesterol = Total cholesterol – HDL cholesterol - (Triglycerides/5)

3.7.2 LIPOPROTEIN (a)

Lipoprotein(a) [Lp(a)] was analysed by the Department of Clinical Biochemistry, Monash Medical Centre, Clayton, Victoria, Australia using an immuno-turbidimetric method, using an in-house ITA on a Kone Progress Selective Chemistry Analyser (KONE, Finland). Lp(a) anti-human antisera was from Behring (Behringerwerkr AG, Marburg, Germany) and Lp(a) standards and controls were from Incstar (Stillwater, MN, USA).

3.7.3 PLASMA PHOSPHOLIPID FATTY ACIDS

Phospholipid fatty acid profile was measured by first extracting plasma lipids with chloroform:methanol 1:1, containing 10 mg/L butylated hydroxytoluene (Labco, Victoria, Australia), and 10 mg/L C17:0 phospholipid (L-α-phosphatidylcholine diheptadecanoyl, Sigma-Aldrich, Pty. Limited, NSW, Australia) as internal standard (Sinclair and Mann, 1996). The plasma phospholipid fractions were then separated using Thin Layer Chromatography. The methyl esters of fatty acids were prepared by saponifacation using potassium hydroxide (KOH 0.68 mol/L in methanol) followed by *trans*-esterification with 20% boron trifluoride (BF3) in methanol. The fatty acid concentrations were determined using Gas Liquid Chromatography (Sinclair et at., 1987). The percentage of coefficient of variation (%CV) for fatty acid analysis was less than 5%. The analysis of fatty acids was performed at the Department of Food Science, Royal Melbourne Institute of Technology, in Melbourne, Australia.

3.7.4 GLUCOSE AND INSULIN

Fasting serum glucose was analysed using an adaptation of the hexokinase and glucose-6-phosphate dehydrogenase method provided by the Abbott Spectrum glucose reagent from Abbott Laboratories, USA, Diagnostic Division (Abbott Park, IL 60064).

Plasma insulin was assayed using the AIA-PARK IRI (Cat. No. 020260) and the TOSOH 1200/600 system (TOSOH Corporation, Tokyo, Japan). The AIA-PACK IRI is a two-site immuno-enzymometric assay which is performed entirely in the AIA-PACK. This measurement was performed by the Department of Clinical Biochemistry, Monash Medical Centre, Clayton, Victoria, Australia.

3.8 DATA MANAGEMENT AND STATISTICAL ANALYSIS

3.8.1 DATA MANAGEMENT

All data relating to demography, food intake, body composition, biochemistry, were entered into separate files using Microsoft Excel 97 software programme. These files were then converted into dataset files using the Statistical Analysis System (SAS) software version 6.12 (SAS Institute, Cary, NC, USA).

3.8.2 STATISTICAL METHODS

The Statistical Analysis System (SAS software version 6.12 for Windows, SAS Institute Inc., NC, USA) was used for all data analyses. All data analysis procedures were performed using the SAS/ASSIST function, which is an integrated component of SAS system that enables the user to complete different types of statistical analyses without a knowledge of programming language.

3.8.3 DESCRIPTIVE ANALYSES

Descriptive statistics were used to report sample distributions and attributes for confounders and antecedent factors. Mean, standard deviation (SD) and percentiles were used for continuous variables, whereas for discrete variables, frequency and percentage were derived.

were used for continuous variables, whereas for discrete variables, frequency and percentage were derived.

3.8.4 MULTIPLE COMPARISONS

Analysis of variance (ANOVA) was applied for group comparisons. Differences between the continuous variables of two groups (two levels of class variable) were tested by the Student's *t*-test. A paired *t*-test was used to examine cohort changes in comparable variables between the follow-up and baseline studies. General linear modelling (GLM) (Tukey's test) was used for the multiple group comparison and allowed for the adjustment of confounding factors. For the discrete variables, a Chi-square (χ^2) test (2x2 or NxN table) was used to examine the differences between frequency distributions.

3.8.5 CORRELATION ANALYSES

Pearson and Spearman correlation coefficients were used to describe the relationships between antecedent factors and outcome variables, such as correlation between age and CHD risk factors, and between intakes of saturated fatty acids, in the Case-Control Study. In the Intervention Study, the relationships were examined between the consumption of coconut and that of selected food groups.

3.8.6 LOGISTIC REGRESSION ANALYSES

In the Case-Control Study, a logistic regression analysis was used to determine the association between CHD and a variable of interest (or a risk factor). Subjects were divided into a number of groups according to their lifestyle levels such as smoking status, stress level and physical activity. Each group was assigned with a number ranged from 1 to 3 or 4, depending on the number of groups. For each lifestyle level, an OR was computed as the rate in the group assigned with a highest value divided by that in the group assigned with a lowest value. For the intakes of various foods and nutrients, subjects were divided into four equal groups according to the quartile values, and an OR was computed as the rate in the highest quartile divided by that in the group with the lowest quartile. In multivariate models, other significant variables in lifestyle, biochemistry, anthropometry assessments and, food and nutrient intakes were simultaneously included.

An OR was calculated as following:

$$\text{Odds ratio (OR)} = \frac{\text{Odds of the exposed (to the risk factor) subjects}}{\text{Odds of the unexposed (to the risk factors) subjects}}$$

$$= \frac{\text{Ratio of Cases and Controls exposed to the risk factor}}{\text{Ratio of Cases and Controls unexposed to the risk factor}}$$

In general, if an OR is greater than 1.0, it is suggested that the variable of interest is a risk factor for CAD. If an OR is less than 1.0, the variable of interest could be considered to be a protective factor for CAD, provided the 95% confidence interval (CI) of the OR does not include 1.0 in both cases.

Stepwise selection was used with significance set at 0.15.

3.8.7 SIGNIFICANT LEVELS

Statistical significance was set to a probability level of 5% (*P=0.05*), unless otherwise specified. The following symbols are used throughout this thesis to represent significance levels: *, *P<0.05*; **, *P<0.01*; ***, *P<0.001*; ****, *P<0.0001*.

CHAPTER 4

Food Culture and Nutrition Transition in the Minangkabau

4.1 INTRODUCTION

Food behaviour and cuisine constitute the food culture of people, and its exploration through socio-anthropology can allow new insight into food-health relationship. Minangkabau food culture provides a unique opportunity to look at the impact of newer economies on a traditional economy and food supply centred on women. In transitional economy, like those seen in the Indonesian archipelago, lifestyle diseases, like heart disease, are superimposed on a background of food insecurity and deficiency disorders. However, Minangkabau culture, where views and decisions of women are influential, has provided and organised the economy with regard to food distribution in family and society.

Indonesia, like many other developing countries, is experiencing rapid urbanisation characterised by a double burden of disease in which chronic diseases become more prevalent while infectious diseases remain undefeated. A nutrition transition, where societies suffering the problems of both the undernutrition and the overnourished and also changes in diet and health, is a universal trend which is dominating the health profile of increasingly large numbers of people in developing countries (Lohman et al., 1988; Romieu et al., 1997; Vorster et al., 1999; Worsley, 1998).

The epidemiologic transition and concurrent shift in diet, activity and body composition in many developing countries has been rapid, unlike the gradual transition in the United States and most European countries (Popkin, 1998). Reports from Asian countries such as Korea, India, Japan, and from countries in South America indicated that a rapid change in the dietary habits and body composition occurred after their countries achieved dietary sufficiency at the national level (Kato et al., 1987; Kim et al., 2000; Lohman et al., 1988; Monteiro et al., 1995; Romieu et al., 1997; Vio and Albala, 2000; Worsley, 1998). In India, rapid socio-economic transition has resulted in rapid changes in dietary patterns (Singh et al., 1999). Obesity has become a public health problem, especially amongst children (Drewnowski and Popkin, 1997; Popkin, 1998; Schneider, 2000). In Korea,

however, after an acceleration in economic growth for three decades, the nutrition transition has been reflected in dramatic shifts in causes of death, although there are movements to keep to traditional dietary patterns (Kim et al., 2000). Since 1966, a remarkable transformation in the Indonesian economy has changed the social demographic structure and contributed to large shifts in overall dietary patterns in Indonesia. Only a few investigators have discussed aspects of nutrition transition in Indonesia (Boedhi-Darmojo, 1993; Gopalan, 1996; Oenzil, 1993).

This chapter describes the Minangkabau food culture, which was obtained from a Focus Group Discussion (FGD). For a thorough understanding of nutrition transition over the period of 1983 to 1999, trend changes in food and nutrient intakes amongst people in West Sumatra were also explored. Included in this chapter are also the comparisons made between data obtained from healthy subjects who participated in the Case-Control study in 1999, and information collected from published reports and articles.

4.2 THE FOCUS GROUP DISCUSSION (FGD)

The FGD was carried out on the Minangkabau to obtain and in-depth understanding of their food culture. Information on food habits and food preparation and changes to these from 1983 to 1999 was gathered and explored in the focus groups. Also, the participants were asked for their opinion about the role of coconut in their food culture.

FGD has been widely used as one of the basic anthropological methods, especially in research to determine health-seeking behaviour (Scrimshawand Hurtado, 1987). The focus group approach is a qualitative research methodology used to provide insight and understanding of a target group's perceptions and beliefs regarding a particular topic or program (Ramirez and Shepperd, 1988). In this study, focus group discussion was not used to ascertain perceptions about a new topic or program. The FGD was intended to document Minangkabau food culture and to determine any changes in dietary pattern among the people.

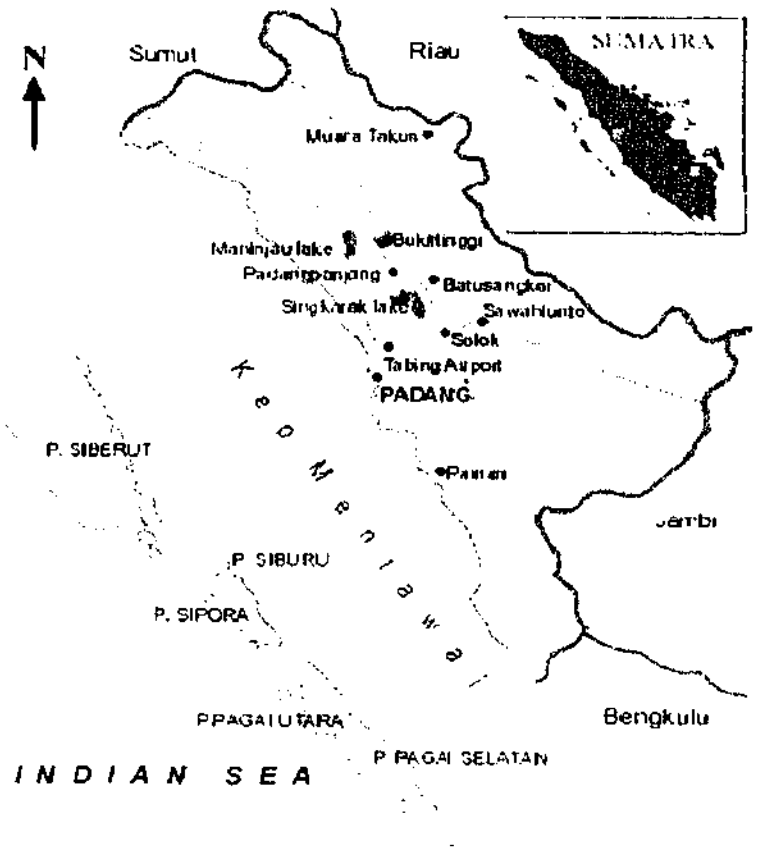
4.2.1 SUBJECTS AND METHODS

The FGDs took place in four villages in West Sumatra from four different municipalities. Padang is the capital city of West Sumatra where Kalumbuk was situated (Figure 4.1). Kalumbuk is about 10 km from Padang. From the abundance

of natural resources, one would not expect Kalumbuk to be a poor village. Irrigated rice fields produce crops two to three times per year and mixed cropping is also common. Kuranji River crosses along the village and is used to provide fish for the villagers. The second village was Nareh, situated in the coastal region south of Padang Pariaman Municipalities; it is about 45 km from Padang. Nareh is a fertile village where coconut trees are available in abundant quantities. While most of the men were fishermen, the women have developed the village as embroidery centre in West Sumatra. These textiles have been imported to other parts of Indonesia and also exported to Malaysia and Brunei. The other two villages, Pincuran Panjang and Kubang are in the mountainous region, in the municipalities of Tanah Datar and Limapuluh Koto respectively. These two villages are in the heartland of West Sumatra, where Minangkabau culture actually originated. Pincuran Panjang is about 60 km from Padang, while Kubang is about 110 km from Padang. Both of the villages are fertile - irrigated rice fields dominate the scenery. In Kubang almost all of the houses had fish ponds. By contrast, only one fish pond was found in Pincuran Panjang, although in the past ponds were found in every home.

The FGD took place in each village and was conducted from January to March 1999 (Figure 4.2). Invitations to participate in the FGD were disseminated through the village mayor, and also through doctors and midwives. Invitations were intended for adult women in the villages. The characteristics of the participants involved in the FGD are presented in Table 4.1. Each session was tape-recorded and lasted up to one and half-hours.

Figure 4.1: Indonesia and West Sumatra



Three generations of Minangkabau women and children.

The candidate and some of the participants in Kapalo Koto, Padang.

Thirteen women participated in the FGD in Naras, aged from 46 to 67 years. In Kalumbuk, nine women joined in the discussion, aged 30 to 67 years. While in Pincuran Panjang, 7 women aged 46 to 81 years old and thirteen women in Kubang, aged 33 to 77 years old participated in the FGD.

Some other complementary methods were used to obtain data about the food culture including informal interviews, observations, and the knowledge of the Minangkabau author.

Table 4.1: Characteristics of participants involved in the Focus Group Discussions

	Kalumbuk (n = 9)	**Nareh (n = 20)**	**Pincuran Tinggi (n =7)**	**Kubang (n = 13)**
Age (years)	36 – 67	35 – 71	40 – 81	33 – 77
Highest education level				
No formal education	3	4	2	1
Elementary school	5	6	3	3
High school	1	10	2	9
College or university	0	0	0	0

4.2.2 THE TRADITIONAL MINANGKABAU DIET AND MEAL PATTERN

Basic daily dishes of the Minangkabau consist of steamed rice, a hot fried dish and a coconut milk dish, with minimal variation from breakfast to evening meals. Like other parts of Indonesia and Southeast Asia, rice is the most important food, along with fish, coconuts, vegetables and chilli. Throughout Southeast Asia, although a variety of special dishes play an important role, the prestige food is rice(Wilson, 1986). In Indonesia and also for the Minangkabau, the word 'rice', *nasi,* is synonymous with food, 'to eat' is to 'to eat rice'.

A "three meals a day" pattern is common amongst the Minangkabau, in contrast to the "two meals a day" pattern found in rural West Java (Howden et al., 1993). The meal patterns of the Minangkabau were described in Tables 4.2-4.4. Each meal pattern consists of three basic components: 'the core' or staple items of a particular meal, 'the secondary core' or those items which are either added to or substituted

for the basic core, and 'the peripheral' diet or those items which are used infrequently by most people (adapted from Jerome, 1980).

4.2.2.1 Breakfast

Almost all participants in the FGD always had breakfast, especially those in Kubang and Pincuran Panjang, who were mostly farmers. A survey conducted in other part of Indonesia, West Java, showed that the incidence of missing breakfast regularly was about 1% (Howden et al., 1993). Breakfast could be either sweet food or hot food that contains chillies (Table 4.2).

Table 4.2: Meal components and preparation methods: determinants of the breakfast pattern

Ccmponent			Preparation method	Meal pattern
Core item	**Secondary core item**	**Peripheral diet**		
Carbohydrate				**Pattern I**
Rice	Banana	Noodle	Boiling with or without coconut milk	Rice
	Cassava			Fish
	Glutinous rice			Samba-lado *
	Mung bean	White bread		Vegetable
	Pumpkin			Water
Protein				
Fish		Meat	Frying	**Pattern II**
Egg				Lontong
Vegetable				Vegetable
Cucumber			Raw	Kerupuk **
Green-leafy vegetable			Boiling with coconut milk	Sweet tea
Beverage				**Pattern III**
Water	Tea + raw egg	Chocolate + milk		Banana
Tea	Coffee + milk			Glutinous rice
Coffee				Grated coconut
				Sweet coffee

** Samba-lado: grated chilli with onions, tomatoes and salt;*

*** Kerupuk: crackers*

Food is usually prepared mid-morning, and is consumed as lunch and dinner on that day, and breakfast the following morning. Hot food eaten as breakfast is usually dinner left over from the previous evening, such as fried rice or porridge

(*lontong*) and vegetables in coconut milk. Sweet food is made from fried banana with boiled glutinous rice, spread with desiccated young coconut or mung bean boiled with coconut milk, sweetened with brown sugar. Traditional cakes are also made from cassava, rice or wheat flour. Drinks are often plain water, tea or coffee. A drink called *teh-telur*, a mixture of raw egg and tea is very popular amongst the Minangkabau men, and is usually one component of breakfast. Milk is rarely consumed. Breakfast time is 7.00 to 8.00 am. A more Westernised breakfast, such as bread and butter, is not common.

4.2.2.2 Lunch and Evening Meal

Lunch is usually similar to dinner (Table 4.3). Lunch is considered to be the most important meal of the day for the Minangkabau. The core component of lunch and dinner is usually rice. Other carbohydrate-rich foods include cassava, corn, sago or noodles, although they are sometimes considered as snacks. In other parts of Indonesia, such as in Mentawai islands, the people consume sago. Corn is consumed in the eastern part of Indonesia (some parts of Sulawesi and Malucca). In some parts of Java, rice is substituted with cassava during the harsh times. Noodles, especially instant noodles, are recognised as a novel food. Noodles were introduced into the diet about 10 to 15 years ago and have now become very popular and consumed at least 3 times a week.

The protein source consumed daily by most Minangkabau is fish. In the present study, participants in the three of the four villages reported consuming fish 4 to 6 times a week. In the mountainous region of Kubang, there was a fishpond in most of the houses in the villages; in the coastal region of Naras, fresh fish was readily available. In a village far from the seashore, Kalumbuk, fish used to be conveniently caught from a river that ran along the village, but currently there is a shortage of fish in the river and fresh fish has become more expensive. Therefore, Kalumbuk people use alternative protein sources in their diet, including tofu (made of soy) or tempeh (fermented soy) and egg. In the village of Pincuran, which was also far from the coast and where people did not have fish, fresh fish bought from the market, was consumed about 2 to 3 times a week and salty fish was eaten more frequently.

Table 4.3: Meal components and preparation methods: determinants of the lunch and dinner pattern

Component			Preparation method	Meal pattern
Core item	**Secondary core item**	**Peripheral diet**		
Carbohydrate				
Rice	Noodle		Boiling	**Pattern I**
Protein				Rice
Fish	Tofu/tempeh	Chicken	Frying	Fish
Egg		Beef	Boiling with coconut milk	Vegetable
Vegetable				Crackers
Green-leafy vegetable			Boiling with coconut milk	Water
Seasoned vegetable			Stir frying	
Beverage				**Pattern II**
Water				Rice
Fruit				Chicken
Banana	Seasoned fruit			Vegetable
Papaya				Banana
Dessert				Jelly
		Jelly	Boiling with coconut milk	Water
		Glutinous rice		

Beef and chicken are mainly prepared for special occasions. *Rendang*, a popular meat dish from Minangkabau, is a beef-dish cooked with a large quantity of spices, herbs and a liberal amount of coconut milk. It is cooked mostly four to five times in a year, namely in the beginning and end of *ramadhan*, the birthday of the Prophet *Mohammad*, and on the *Haj* day. Rendang is one of the ritual foods (*makanan-adat*) for the Minangkabau that are served on the *adat*-occasions and it has been identified as one of the Minangkabau culture characteristics. In general, there are two kinds of *rending* - dried and wet. Dried *rendang* can be kept for three or four months and still palatable throughout the period. It is to be served only on the *adat*-occasions or to honour guests. Wet *rendang* can be found in *Minang* restaurants and it should be consumed within a month.

Other protein sources such as tempeh and tofu are novel to the Minangkabau. Originated from Java, tempeh and tofu became popular when transmigration

programs began in West Sumatra in the late 1970s. As tofu and tempeh are cheaper than fish, they have become common on menus since the recent economic crisis of 1997-1999.

Vegetables are usually consumed two to three times a day. Green leafy vegetables, such as *kangkung (Ipomoea reptans;* swamp cabbage), spinach and cassava leaves (*Manihot utilissima*), are principally consumed. Other vegetables commonly found in households include cucumber, long beans, cabbage, young jackfruit, gourd, cauliflower, carrots and bean sprout. Most fruits are seasonally available, except for banana, papaya and citrus (orange and lime) which can be easily found all year round. However, fruit consumption is not a traditional food habit for most Minangkabau. Fruit is consumed as a snack only when available. In the high season of some fruits, such as mango, rambutan (*Nephelium lappaceum*), duku (*Lansium domesticum*), and durian (*Durio zibethinus*), they are consumed everyday in ample amounts.

Food for the Minangkabau has many social roles. This is shown by the prominence of food in ceremonies honouring religious and life-cycle rites. In *ramadhan*, the meal pattern is different from that in other months. Foods and drinks are prohibited from dawn to dusk. The fasting day is broken by one obligatory feast such as the features of seldom-eaten beef and chicken (Table 4.4). Like in Malaysia where Minangkabau culture is also found, the fasting period of each day throughout the month is ended by eating sweet foods exchanged between households before the evening meal is served (Wilson, 1986). The evening meal in *ramadhan* is often more special than is dinner in other months.

4.2.2.3 Variation in Snacking Behaviour

For people in the villages, snacks are not often consumed, except during the harvest times which are two to three times a year, and in *ramadhan*. In the harvest times, snacks are served around 3 p.m. between lunch and dinner, for those who work in the rice fields. In *ramadhan*, snacks are consumed before the main menu at dinnertime. For people in the urban areas, snacks are more varied and consumed more often. Sometimes snacks contribute a significant amount of nutrients; amongst school age children snacks contribute 38.7% of total daily energy (unpublished data). Snacks can be divided into hot and cold snacks. Hot snacks are usually more filling, spicy, and usually have a larger serving size. Snacks can be consumed as desserts or consumed between two main meal times in the morning or afternoon. Coconut milk is often used in a dessert with glutinous rice, cassava, rice-

flour, wheat flour, and mung beans and is sweetened with white sugar or brown sugar. Table 4.5 shows a list of traditional snacks that are commonly found in West Sumatra.

Table 4.4: Meal components and preparation methods: determinants of the dinner pattern in *ramadhan*

Component			Preparation method	Meal pattern
Primary core item	**Secondary core item**	**Peripheral diet**		
Carbohydrate				**Pattern I**
Rice	Banana		Boiling with or without coconut milk	Kolak *
	Cassava			Rice
	Glutinous rice			Fish
	Pumpkin			Vegetable
Protein				Crackers
Fish	Chicken		Frying	Diluted syrup
Egg	Beef		Boiling with coconut milk	
Vegetable				**Pattern II**
Green-leafy vegetable			Boiling with coconut milk	Onde-onde **
Seasoned vegetable			Stir frying	Rice
Beverage				Chicken
Water				Vegetable
Tea/coffee				Banana
Diluted syrup				Ice-tea
Fruit				
Banana	Seasoned fruit			
Papaya				
Dessert				
Banana			Boiling with coconut milk	
Cassava				
Glutinous rice				
Pumpkin				

* *Kolak: Manioc cooked coconut milk, sweetened with brown sugar could be mixed with banana, mung bean;*

** *Onde-onde: Round of glutinous rice flour and water; centre filled with brown sugar, boiled, coated with grated coconut.*

Table 4.5: Selected traditional cakes and snacks made or sold in West Sumatra, Indonesia

Traditional cakes and snacks	Description
Made of rice	
Lontong	Rice porridge, hardened and then cut into compact square. Eat with young jack fruit or other vegetables cooked with coconut milk
Tapai pulut	Fermented red glutinous rice
Nasi Kuning	Glutinous rice cooked or steamed with coconut milk coloured with turmeric
Ketupat	Glutinous rice covered in coconut leaves cooked with coconut milk
Lemang	Glutinous rice put in bamboo, mixed with coconut milk and baked
Bika	Rice flour mixed with grated coconut and sugar, then baked
Lepat Bugis	Rice flour mixed with grated coconut and sugar, covered with banana leaves, then steamed
Onde-onde	Round of glutinous rice flour and water; centre filled with brown sugar, boiled, coated with grated coconut
Kue talam	Rice flour, coconut milk, brown sugar put into a square plate, steamed.
Made of manioc	
Kolak	Manioc cooked coconut milk, sweetened with brown sugar could be mixed with banana, mung bean.
Kerupuk balado	Manioc fried in deep oil, rolled in grated chilli and sugar.
Tapai Ubi	Fermented manioc
Tumbang	Round boiled manioc, centre filled with brown sugar, coated with grated coconut
Made of banana	
Pisang goreng	Fried banana, coated with flour, eat with boiled glutinous rice spread with grated coconut
Nagasari	Small slice of banana mixed with rice flour, sugar, covered with banana leaves, steamed
Other	
Sate	Beef boiled with spices, cut into small slices and grilled, eat with lontong and rice flour sauce
Soto	Beef boiled with spices and herbs, eat with rice
Martabak Kubang	Beef mixed with wheat flour and spices, herbs and stirred with a little cooking-oil.

4.2.2.4 Changes in Meal Patterns

Changes in meal patterns in West Sumatra appear to have occurred, partly due to the ease with which food can now be obtained, and the acculturation process. According to the older participants in the FGD, when they were young (about 50 to 60 years ago), rice was sometimes eaten only once a day; they consumed more cassava, and more green vegetables which were cooked with coconut milk; they also consumed more salty fish mixed with desiccated young coconut. They seldom bought cooked food. In present days, the young generation can easily purchase cooked food, such as noodles and meatball soup. According to a report from the Bureau of Statistics of West Sumatra, 10% of total expenditure in 1996 was for cooked food, increasing from 6.7% in 1990 and 8.6 % in 1993 (Central Bureau Statistics of West Sumatra, 2000).

The participants in the FGD indicated that the elderly today were healthier and stronger than their ancestors and were sick less often than their parents. They suggested that this could have been because their parents were "over-worked" with excessive physical activity. On the other hand, they also recognised that food was more available at present time than before. And this may play a role.

4.2.3 Food Preparation

Although the Minangkabau is a matrilineal society, the preparation of food provides further elaboration of gender difference in food culture. Similar to Indonesians in Bali, Minangkabau women cook everyday meals, while men would prepare beef dishes only for special occasions (Manderson, 1986). The head chefs in every Minang restaurant are always men. Meals for breakfast, lunch and dinner are generally cooked once in a day, normally in the morning between 9 am to 12 noon, and for dinner, only rice is cooked in the afternoon.

Fish is usually fried or boiled with coconut milk. Some herbs and spices, including turmeric, ginger, galanga (*Languas galangal*), chilli, onions, turmeric leaves, lemon grass, small green chilli and lime juice, are mixed with the fish before it is cooked with coconut milk. The most important herbs and spices used in almost every coconut milk dish are chilli, turmeric, ginger and galanga. In meat preparation, herbs, spices and coconut contribute flavours and are useful for preservation. Meat could be deep-fried, boiled with or without coconut milk, or grilled (as satay). *Rendang* is an example of meat preparation, in which meat is cooked with a full range of herbs and spices, and a large quantity of coconut milk. Rendang is minced

meat cooked with ginger, galanga, chilli, onions, garlic, turmeric leaves, lemongrass and lime leaves. In other parts of West Sumatra, rendang is also mixed with cloves, cinnamon, nutmeg, coriander and white pepper. Usually four coconuts are used for one kilogram of meat, but only one piece of coconut is used with one kilogram of fish. The meat is simmered slowly with light heat until the coconut milk has dried; the entire process takes about three to four hours, but it may take longer to cook dried rendang which can be kept for months. Rendang can be made from either beef, chicken, or some vegetables such as young jackfruit or cassava. Due to limited availability and unfavourable taste, lamb, goat and wild game are rarely eaten. Pork is not halal and therefore not eaten by Muslim Minangkabau people.

Tempe and tofu sometimes are combined with fish or some kinds of beans, such as red beans, green beans, or young banana, and then cooked with coconut milk or fried with a large quantity of hot chillies.

Almost all vegetables in West Sumatra can be cooked with coconut milk. In Naras and Kubang where coconut is used more frequently, vegetables are cooked with coconut milk and flavoured with fish or a small quantity of meat. In Padang Panjang and Kalumbuk, vegetables are usually stir-fried with a small amount of coconut oil, while fresh salads or boiled vegetables are rarely consumed. Another vegetable dish that is common is *anyang*, in which the vegetables are boiled and then mixed with desiccated coconut, chillies, onions and salt.

4.2.4 Coconut

Coconut plays an important role in Minangkabau food culture. For those not familiar with West Sumatra, Minang food may be identified as food that is cooked with coconut milk (*gulai*). All participants in the FGD agreed that coconut was an important ingredient. Characteristic expressions including (a) coconut improves food flavour, (b) coconut is a trademark of Minangkabau dishes, (c) coconut is used in most dishes of Minangkabau food, were made. In Naras and Kubang where the lands are fertile, coconut trees can be found easily in villages where most households would use at least one coconut a day. To make dishes, the coconut is pressed for the milk, and the flesh is cooked with fish, vegetables, meat, or chicken.

In Padang Panjang and Kalumbuk, coconut milk was used less frequently, compared to the other two villages. Coconut trees in these villages were cut down and replaced with houses. The villagers had to purchase coconuts from the market. In these villages, almost all dishes are fried with cooking oil and then mixed with a

moderate amount of hot chillies. Since 1995, coconut oil was replaced by palm oil due to the increasing price of coconut oil. Some participants made some comments that fried dishes were easily prepared and also more economical than coconut milk dishes. In addition, some younger participants mentioned that their children preferred fried dishes to coconut milk dishes. During times of hardship, such as during the economic crisis in 1997, coconut milk dishes were not used at all. According to the participants, an average of 250 g of cooking palm oil was used in a day in a household with three or four children.

4.2.5 CHANGES IN FOOD PREPARATIONS

All participants agreed that there was no difference in food preparation between the two generations interviewed. The younger generation continued to use the same ingredients and same processes in food preparation. Any possible changes were in cooking utensils. Presently, electrical appliances such as rice cookers, food blenders and gas stove, can be found in many households in the villages.

All participants agreed there has been little or almost no difference in food tastes between the two generations interviewed. Minangkabau traditional food was still the first choice. Taste preference among Minangkabau people was a combination of the hot and spicy taste and the flavour of herbs and spices, with a little bit of salt. Western food such as McDonald™ was hardly known to the villagers. However, the older generation mentioned that, compared to 40 to 50 years ago, more food was now available. Restaurants and food outlets can now be found everywhere. Novel foods have also been introduced, such as instant noodles, tofu and tempeh, which were now commonly used in households.

4.3 NUTRITION TRANSITION IN WEST SUMATRA

The major source of information for nutrition transition data was from the PhD thesis of Malik (1986) and the present study done in 1999. Cause of death data was from the National Socio-Economic Survey (SUSENAS).

Malik (1986) collected dietary data from a simple random sampling of populations in four villages in Kuranji District of Padang Municipality. The actual data was collected in the year of 1983. Malik collected his dietary data by using 24 hour food recalls and FFQ on 98 subjects from 15 to 45 years old, comprised of 48 men and 50 women (Malik, 1986). The FFQ was comprised of 21 food items.

The present study was part of the Case-Control study. The data was collected from 220 healthy subjects, aged from 35 to 82 years. The population in this study was from Padang and Bukittinggi, and also from almost all parts of West Sumatra: Pariaman, Painan, Agam, Padang Panjang, Batusangkar, Payakumbuh and Solok. The FFQ, which was comprised of 215 food items, were used in this study.

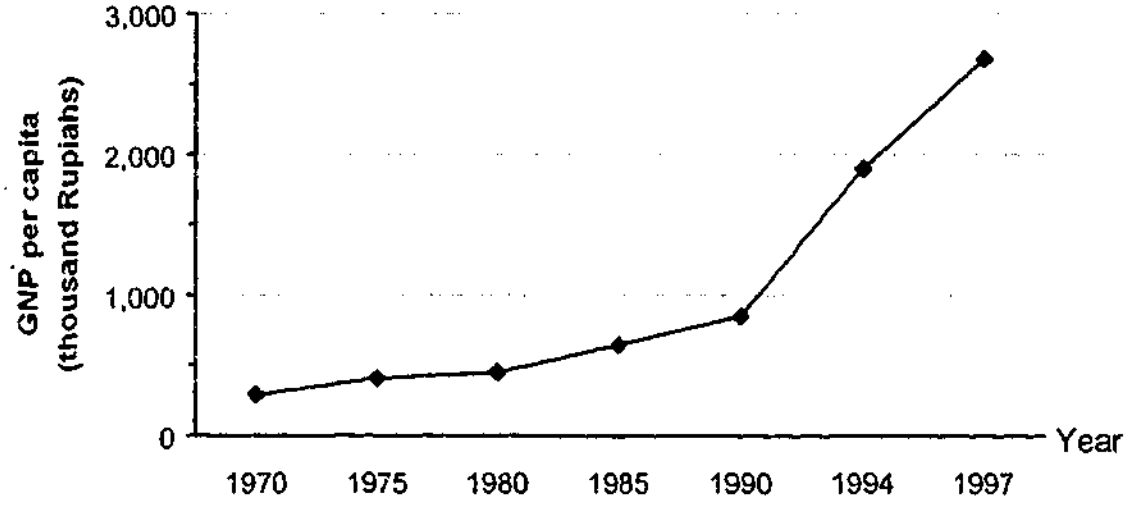
Data on work force structure were obtained from Central Bureau of Statistics of Indonesia and West Sumatra. For economic trends, the national income data were used from the official estimates of gross national product (GNP) per capita from 1966 to 1997 as established by the World Bank. GNP per capita was expressed in thousand Rupiahs.

No data were available for the causes of death for West Sumatra, so national data were used. A regular House-hold health survey (SKRT) has been done in Indonesia since 1972 by Ministry of Health of Indonesia. This survey was conducted to collect health information on Indonesians and the cause of death (Soemantri et al., 1997). In this chapter, cause of death trends were presented as percentages of total deaths for 4 disease categories in which the death rates changed noticeably in Indonesia during the two decades. Diseases were classified according to the World Health Organization's International Statistical Classification of Diseases (ICD-10). The 4 categories chosen to illustrate major shifts in disease patterns from infectious disease were: 1) infectious and parasitic diseases (eg tuberculosis, malaria), 2) diseases of respiratory system (eg, pneumonia and bronkhitis), 3) diseases of the circulatory system (e.g. rheumatic heart disease, hypertension, ischemic heart disease, cerebrovascular disease, and diseases of the pulmonary circulation), and 4) malignant neoplasma (e.g. cancers of breast, lung, liver, stomach)

4.3.1 INDONESIAN ECONOMY

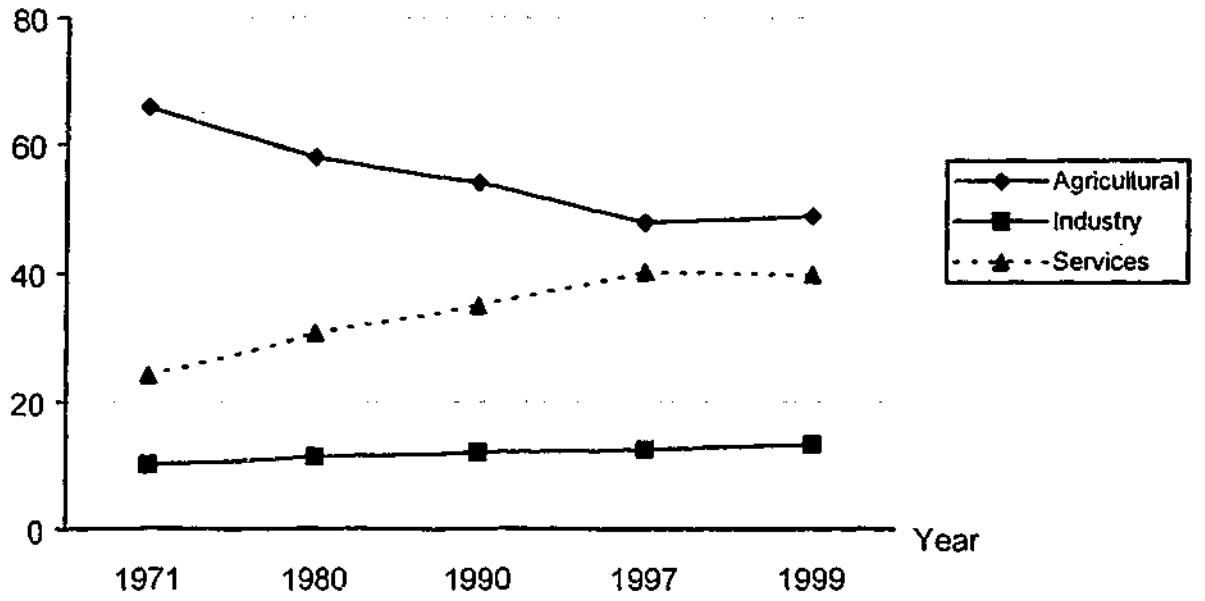
There have been remarkable changes in the Indonesian economy over the past 35 years, including the economic crisis in mid 1997. GNP per capita increased dramatically from 1970 to the mid 1990s (Figure 4.1). In 1970, the nominal value of per capita national income was 295 thousand rupiahs, then it increased to 2,680 thousand rupiahs before the crisis in 1997 (WHO, 2000). This was in line with the high average economic growth of 7.8% in 1970s, 6.5% in 1980s and 7.2% in 1990s. Such high economic growth increased food availability and enhanced purchasing power of the people, which accelerated nutrition transition (WHO, 2000).

Figure 4.1: Trends in gross national product (GNP)



The rapid shift in income was associated with changes in occupation distribution. The West Sumatra labour fource data shown in Figure 4.2 indicated a clear trend over the four population surveys (SUSENAS 1971–1997). The share of the agricultural labour force fell continually, from almost three-quarters of the total in 1961 to less than one-half in 1997. During the same period, that of industry doubled, while that of services rose by about 75% (Hill, 1996). A shift alteration from labour-intensive occupations in the rural primary product sectors of agriculture, forestry and fisheries, to occupations in the services and manufacturing was in agreement with the marked increase in the GNP of Indonesia. The current occupational structure of Indonesia is similar to that of most Western countries. This transition is linked to a major reduction in energy expenditure at work.

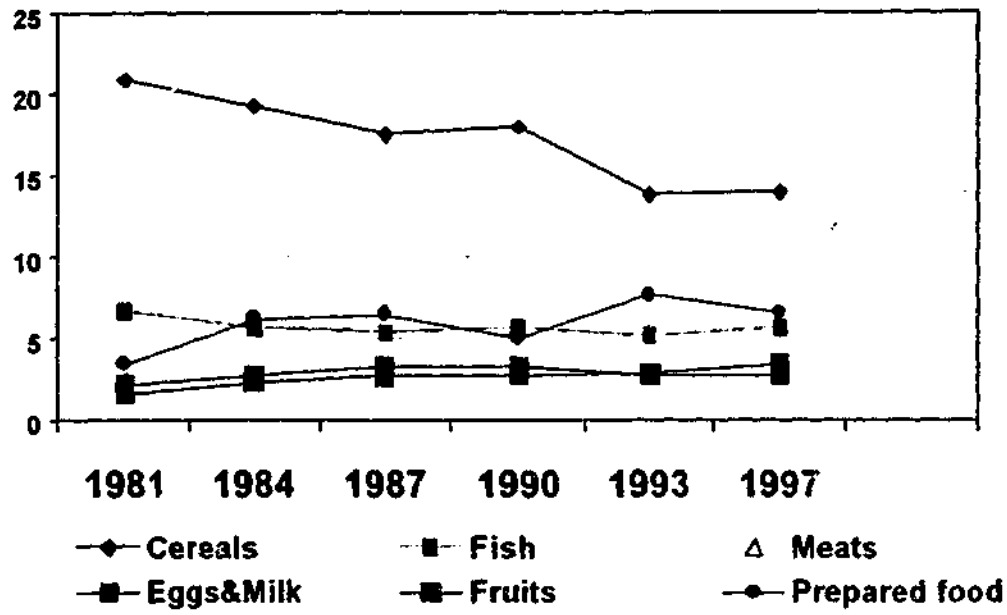
Figure 4.2: Trends in the distribution of occupation in West Sumatra, Indonesia, 1971-1999



4.3.2 FOOD INTAKE

Trends in the patterns of household expenditure and food consumption confirm the picture on nutrition transition. Consistent with significant improvements in living standards, the proportion of household expenditure on food fell steadily since 1969-1970, with most of the decline accounted for by the cereal and tuberous food groups. Correspondingly, the share of non-food items rose and there was a sharp increase in housing and utilities. Expenditure for meats, eggs and milk increased significantly (Figure 4.3). Expenditure for prepared food increased by 100% over the period of 17 years, more than any other food items. This was due to more women entering the labour force, only 32.6% in 1980, 39.6% in 1985 and 49.93% in 1997, and this could have resulted in the reduction in their available time to prepare food at home (Central Bureau Statistics of West Sumatra, 2000; Soemantri et al., 1997).

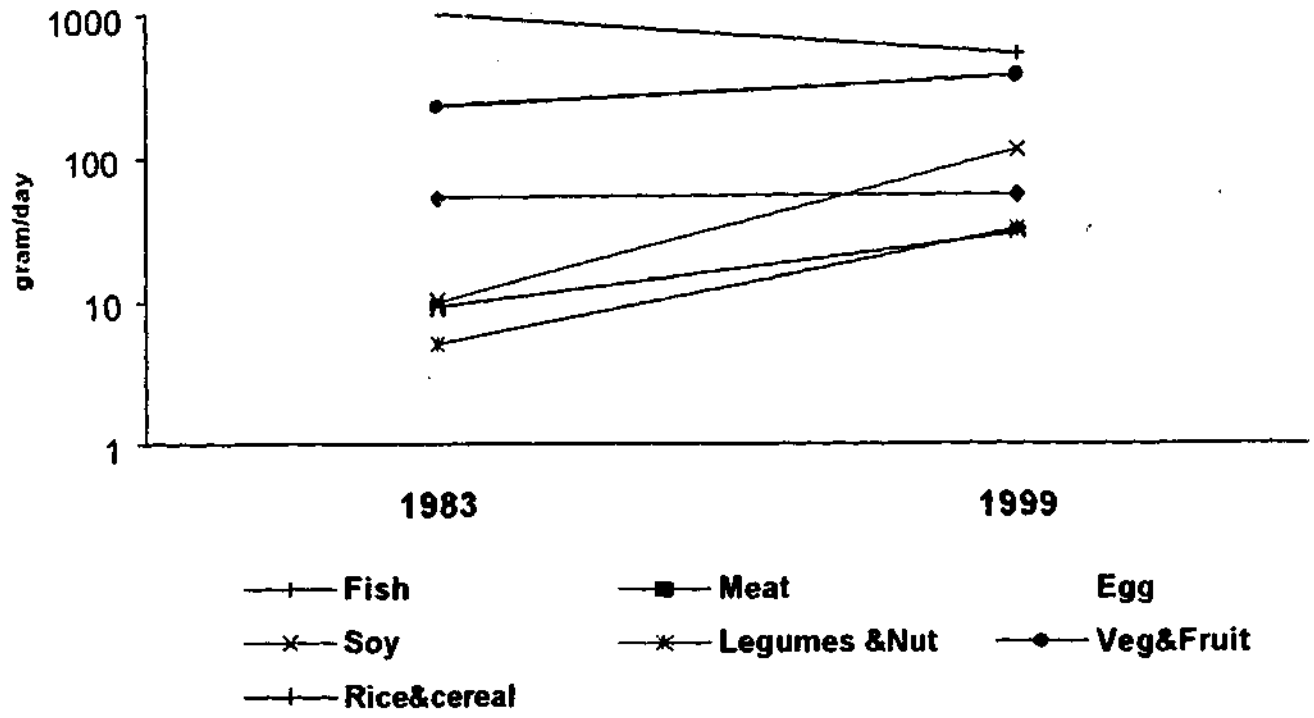
Figure 4.3: Changes in expenditure by food groups, 1981 to 1997



Source: Central Bureau of statistics of West Sumatra, 1993, 1996 & 1999

Figure 4.4 shows the change in food intake during the period of 1983 and 1999. Fish consumption remained similar during the period from 52 g/day in 1983 to 54 g/day in 1999. The largest increases were found in soy and to a lesser extent in meat, egg and dairy products. In 1983, soy was not known by the largest part of Minangkabau people. Consumption of soy in 1983 was 10 g/day. But in 1999, soy became more popular, the consumption increased to 110 g/day. In contrast, rice and cereal consumption decreased significantly from 1007 g/day to 512 g/day in 1999.

Figure 4.4 Changes in food consumption in West Sumatra during 1983 to 1999



4.3.3 NUTRIENT INTAKES

Nutrient intake data can be used to capture the nutrition transition in West Sumatra. To demonstrate the trends in proportion of dietary nutrient intakes, results from two case studies from West Sumatra are reported. There was a dramatic change in macronutrient intake (computed as % contribution of total energy intake), as summarised in Table 4.7. The average total energy intake was significantly different between the two periods; the average total energy intake was 2,722 kcal and 1,740 kcal for men and women in 1983 (Malik, 1986), while it was 1,511 kcal and 1,353 kcal for men and women in 1999. The energy intake for men remained significantly higher than that of women.

Table 4.7: Daily intakes of energy and macronutrients for men and women, (mean ± SD) obtained from 24-hour recall data, 1983 and 1999

	Year 1983		Year 1999	
	Men	**Women**	**Men**	**Women**
	n = 48	*n = 50*	*n = 116*	*n = 77*
Energy (kcal)	2722 ± 137	1740 ± 72	1666 ± 475	1643 ± 507
Protein (g)	54.3 ± 3.1	39.7 ± 2.4	80.4 ± 28	77.7 ± 27
Fat (g)	23.2 ± 1.5	19.2 ± 1.5	43.9 ± 18.1	44.2 ± 19.3
Carbohydrate (g)	562 ± 30	346 ± 1.6	205.7 ± 49.9	203.1 ± 57.4
% Energy from protein	7.8	8.8	19.1	18.9
% Energy from fat	9.1	12.1	23.2	23.6
% Energy from carbohydrate	81.5	77.9	57.7	57.4

The change in % energy contribution from carbohydrate, protein and fat may give a broad picture of the nutrition transition of Indonesia. The ratio of % energy from carbohydrate:protein:fat was 82:8:10 in 1983, indicating that energy intake was mainly from carbohydrate and that fat or protein did not contribute much. After 13 years, the ratio shifted to 57:19:23, which showed that carbohydrate still contributed a great proportion of energy but to a much lesser extent, and an increase in fat and protein consumption.

The increases in protein intake parallelled the substantial increases in meat and dairy products consumption, as described in the Section 4.3.2. Fat-derived energy intake increased throughout the period, from 10% to 23%. However, it was still lower than that that of many other Asian countries, and even much less than that of most the Western countries.

4.3.4 Causes of Death

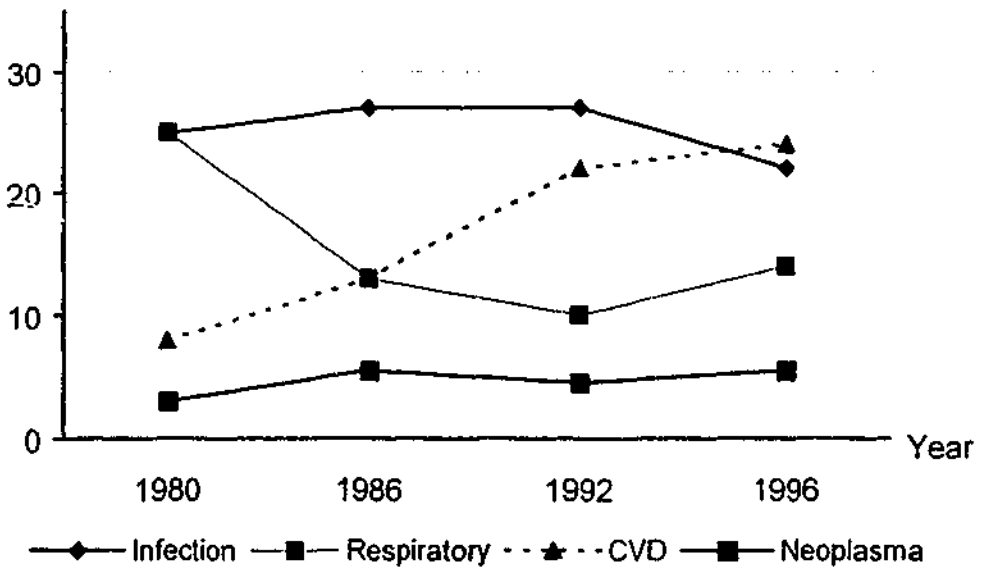
The trend in nutrition intake in Indonesia have the shifted from dietary deficit and food insecurity to overconsumption. One therefore could expect concurrent and possibly related shifts in the causes of death (Figure 4.5). It was only since 1992 that diseases of the circulatory system have been the leading causes of death, accounting for 24.2% of all mortality, especially in Java and Bali, where rapid economic growth was more dominant. In other provinces, the recent data in 1996 indicated that the

diseases were in the third position as the leading cause of death, accounting for 13.8% of all mortality. Death from cardiovascular disease began to accelerate, especially from mid 1980s. In 1986, cardiovascular disease as the leading cause of death accounted for 13% of all mortality and became 24% in 1996 (Djaja et al., 1999; Soemantri et al., 1997). Other non-communicable diseases such as cancer, was still the fifth major cause of death in Indonesia.

The disease classification system used by Central Bureau Satistics does not distinguish individual diseases. For example, diseases of the circulatory system included rheumatic heart disease, hypertensive disease, ischemic heart disease, cerebrovascular disease, and other diseases of the pulmonary system. Data presented in the report did not classify the diseases individually.

Changes in demography are also worth considering. Life span has changed dramatically in Indonesia from 42 years and 47 years for men and women in 1967, to 62 years and 66 years in 1996 (Central Bureau Statistics of West Sumatra, 2000). The subsequent increase in the relative size of the elderly population may have made the increasing trend in chronic disease apparent. The elderly population (aged 65 years and over) rose from 2.5% in 1971 to 4.2 % in 1995 (Central Bureau Statistics of West Sumatra, 2000).

Figure 4.5: Trends in causes of death in Indonesia, 1980-1996



4.4 DISCUSSION

Indonesia has been far away in its nutrition transition. It has been reflected in changes in the proportion of the macronutrients and food intake, and in dramatic shifts in causes of death from infectious to chronic non-communicable diseases.

4.4.1 FOOD AND NUTRIENT INTAKE

A dramatic difference in food usage is reflected in the changing percentage of energy consumed over the period of 1983-1999. Carbohydrate is still the main source of energy for the Minangkabau. In 1983 carbohydrate contributed 80% of total energy intake, where as in 1999 it had dropped to 62%. This is in line with the decrease in rice and cereal consumption. Protein consumption increased from 8.3% to 10.4% from 1983 to 1996. The increase in protein intake paralleled the substantial increase in meat and dairy product consumption during the period as described in the previous section. Fat consumption has doubled from 11% to 22% energy intake for the same period. Compared to other countries in Asia, fat consumption in West Sumatra is relatively lower. In China, dietary fat intake ranges from 22% to 33% of total energy intake in rural and urban areas (Chen et al., 1998).

There has been only a slight change in food preferences in West Sumatra. Mostly the Minangkabau have not changed their dietary pattern, as shown from the Focus Group Discussions. The study found that there was little or almost no change in food taste between younger and older generations. Many studies have indicated that peoples who have deviated from their traditional ways of eating and living, begin to suffer severe consequences in the form of chronic diseases (Bell et al., 1999; Boyce and Swinburn, 1993; Shintani and Hughes, 1994). A study in Japan suggested an association between the increase of Western style fat-rich foods such as butter and margarine, cheese, bread and ham & sausage with an increase in mortality from cancer and breast cancer (Kato et al., 1987).

4.4.2 CAUSES OF DEATH

Changes in the leading causes of death from infectious diseases to chronic diseases in Indonesia occurred significantly since the early 1990s. In other Asian countries such as Korea, cancer and cardiovascular-related deaths became predominant since the early 1970s (Kim et al., 2000), whilst the changes have happened over the last 50 years in Jamaica (Wilks et al., 1998).

The potential measurement errors of the household health data might lead to the under-reporting of the causes of death. Some respondents with limited education may not have known the exact age of their late relatives. Birth or death certificates may have been misplaced or they may not have been issued. Although the interviews were done by doctors in the villages, the report of clinical signs and symptoms of the illness before death were very dependent on the recollection of the respondents (Djaja et al., 1999).

4.5 CONCLUSIONS

Rice, fish, coconut, green vegetables and chilli are the basic daily foods for the Minangkabau with little variation from breakfast to evening meal. Breakfast can be hot which means it consists of the basic foods, or it can be a sweet breakfast, which consists of sweet snacks. Food for lunch is usually similar to dinner (evening meal). In religious and life-cycle rites, food has an important role in Minangkabau society. Ramadhan is an example of where meal pattern differs for one month in the year.

The methods of food preparation and the taste preferences are remarkably consistent across recent generations, although the amount of food eaten has changed and more novel foods have been imported from other parts of Indonesia. So far there has been little Western influence on the Minangkabau people. Until 1997, Western fast food has not appeared in West Sumatra and it would be valuable to monitor changes in the immediate future.

Indonesia, like many other developing countries, is experiencing nutrition transition which is being reflected in rapid changes to diet structure, and dramatic shifts in causes of death.

CHAPTER 5

A Case-Control Study of Nutrition and Coronary Heart Disease:

Lifestyle Patterns

5.1 INTRODUCTION

There are only a few studies from developing countries in which CHD and CHD risk factors have been studied in relation to socio-economic and lifestyle factors. Studies amongst various populations in China, Thailand and India have consistently shown that BMI and CHD risk factors were significantly related to higher socio-economic classes (Singh et al., 1999; INCEN, 1999). In contrast, studies in Europe and North America reported that low educational attainment and low income were associated with an increased risk of cardiovascular morbidity (Kaplan and Keil, 1993; Winkleby et al., 1992) and mortality (Lenfant, 1996; Wamala et al., 1999).

Rapid nutrition transition in developing countries which changed diet and lifestyle in the higher social class have been suggested to be related to the increase in coronary heart disease incidence (Singh et al., 1999). There is definite evidence that certain lifestyle patterns, such as smoking, less physical activity, stress, and family history, increase CVD risk. (Bosetti et al., 1999; Crespo et al., 2000; Kromhout et al., 2000; Negri et al., 1993; Negri et al., 1994; Wamala et al., 1999). World Development Report indicated that tobacco consumption in developing countries increased rapidly, in conjunction with sedentary lifestyle (World Bank, 1993). From the point of view of prevention, a better understanding in the developing countries of the influence of socio-economic status and lifestyle on CHD is important. It also provides new ideas for aetiological research.

This chapter describes socio-demographic and lifestyle characteristic of individuals who had CHD (Case group) and their age- and gender-matched healthy subjects (Control group). Their relationships with conventional CVD risk factors, such as serum lipids (cholesterol, low and high cholesterol and triglyceride), serum glucose, and body composition, were also examined.

5.2 SUBJECTS AND METHODS

A total of 108 eligible cases (71 men and 37 women) and 220 controls (128 men and 92 women) were recruited for this Case-Control study. Information on demography, health status, smoking, physical activity, stress and health status was obtained from an interview questionnaire (Appendix 2).

5.3 SOCIO-DEMOGRAPHIC CHARACTERISTICS

5.3.1 AGE AND GENDER

The age distribution of the population by gender is shown in Table 5.1. In men, the age ranged from 37 to 86 years for the cases, and from 42 to 79 years for the controls. While in women, the age ranged from 37 to 78 years for the cases, and from 34 to 82 years for the controls. No differences were found in mean age between the cases and the controls. The highest proportion of participants in this study was the 60 to 69 years old for both cases and controls.

Table 5.1: Age distribution by gender

	n	Mean ± SD	Min	25th	50th	75th	Max
Cases							
Men	71	59.8 ± 8.7	37	53	61	65	86
Women	37	55.8 ± 11	37	46	57	63	78
Controls							
Men	128	61.2 ± 8.9	42	56	62	68	79
Women	92	55.9 ± 9.7	34	48	56	62	82

5.3.2 LEVEL OF EDUCATION

There were no differences in education level between the Case and Control groups. About 3% of subjects in both groups had never attended formal education and over 60% had finished senior high school or had some form of tertiary education in college or university, as shown in Table 5.2.

The available information on educational attainment of the West Sumatran general population was reported by the Central Bureau of Statistic of West Sumatra (2000) and shown in Table 5.2. However, it was found that the distribution of educational attainment of subjects in the present study was markedly different from that of the general population in West Sumatra (Central Bureau Statistics of West Sumatra, 2000). This could be due to the difference in age range of the two populations. The subjects in the present study aged from 34 to 86 years, while those in the report from the Statistic Bureau of West Sumatra were 10 years old or over.

Table 5.2 Highest education level of study population and that of West Sumatran general population aged over 10 years

Highest level of education	Cases		Controls		West Sumatran general population*
	n	%	n	%	%
No formal education	3	2.8	7	3.2	3.8
Elementary school (Year 1-6)	19	17.6	44	20.0	58.4
Junior high school (Year 7-9)	18	16.7	32	14.6	16.8
Senior high school (Year 10-12)	27	25.0	81	36.8	17.4
College or university	41	38.0	56	25.5	3.6

**, Source: Central Bureau of Statistics of West Sumatra, 2000.*

When subjects were grouped according to their education level, it was found that those who had a higher education level were younger, both in the Case and Control groups as shown in Table 5.3

Table 5.3: Mean age of subjects with different educational levels of the Case and Control groups

Education level	Cases (n = 108)		Controls (n = 220)	
	n	Mean ± SD	n	Mean ± SD
None/primary/junior	37	63.3 ± 9,8	76	61.5 ± 9.3
High school/University	71	55.8 ± 8.7	144	57.8 ± 9.5

5.3.3 Occupational Category

Table 5.5 presents the distribution of subjects according to their occupation. No difference in the distribution of occupation was observed between the cases and the controls. Also, nearly 50% of subjects in both groups were retired.

Table 5.4: Distribution of subjects according to their occupation

Occupational category	Cases		Controls		Total	
	n	%	n	%	N	%
Public officer	31	28.7	44	20.0	75	22.9
Trades	11	10.2	16	7.3	27	8.2
Farmer	2	1.8	6	2.7	8	2.4
Domestic duties	14	13.0	47	21.4	61	18.6
Retired (55+ years)	50	46.3	107	48.6	157	47.9

5.3.4 Marital Status

Table 5.5 shows the distribution of the subjects by marital status. The majority of the subjects in this study were living with their spouse (83% in the cases and 80% in controls). The divorce rate in both groups was very low. No significant difference was observed in marital status between the Case and Control groups.

Table 5.5: Marital status of study population

Marital status	Cases		Controls		Total	
	n	%	n	%	N	%
Single/never married	0	0.0	1	0.4	1	0.3
Married	90	83.3	176	80.0	266	81.1
Divorced	1	0.9	3	1.4	4	1.2
Widowed	17	15.7	40	18.2	57	17.4

5.4 LIFESTYLE FACTORS

5.4.1 STRESS

When asked about stress, more subjects in the Case group (65%) reported that they were easily stressed, compared with the Control (46%) (*P=0.002*).

Table 5.6 shows distribution of reported stress level in Cases and Controls, again more subjects in the Case group suffered from stress, either little or a lot, compared with those in the Control group (*P=0.019*). The odds ratio for those under a little and a lot of stress compared with those who were rarely under stress between the Cases and the Control were 2.13 (95% CI 1.25–3.63).

Table 5.6: Stress level experienced by the subjects

Stress level	Cases		Controls		Total	
	n	%	n	%	N	%
Rarely or never under stress	74	68.5	181	82.3	255	77.7
Under a little stress	33	30.6	38	17.3	71	21.6
Under a lot of stress	1	0.9	1	0.4	2	0.6

5.4.2 PHYSICAL ACTIVITY

Assessment of activity levels showed that more subjects in the Control group had a high level of physical activity, compared with the Case group (*P=0.022*), as shown in Table 5.7. The Control group also spent a longer time in leisure and occupational activities than the Case group (4 hours/week vs 2.7 hours/week, *P=0.0008*). Compared to those having a low level of physical activity, the odds ratio was 0.46 (95% CI 0.25–0.84) for those who had a high level of activity and 0.36 (95% CI 0.15–0.85) for those who had a moderate level of activity.

Table 5.7: Odd ratios, with 95% confidence interval, of physical activity level

Level of physical activity	Cases	Controls	Odd ratio	95% CI
	n (%)	n (%)		
Low level of activity	13 (12.2)	10 (4.6)	1	
Moderate level of activity	88 (88.2)	188 (85.5)	0.36	0.15 – 0.85
High level of activity	6 (5.5)	22 (10)	0.46	0.25 – 0.84

5.4.3 Smoking Habit of Study Subjects

Table 5.9 shows that, amongst men, there were less current smokers, and more ex-smokers in the Case group, compared to the Control group. In addition, cigarette smoking was not prevalent amongst women. Only one woman in the Control group currently smoked, and 9 women (7 in the Case group and 2 in the Control group) quitted smoking for at least one year. Most of the smokers began smoking in their late teens (19 years). For current smokers, the average number of cigarettes smoked per day was 10 for the Case group and 14 for the Control group. Amongst women, compared to smokers, the odds ratio for the non-smokers was 0.2 (95% CI 0.04–0.7).

Table 5.9: Smoking status in the Case and Control groups

Smoking status	Cases (n = 108)			Controls (n = 220)		
	Total N (%)	Men n (%)	Women n (%)	Total N (%)	Men n (%)	Women n (%)
Non cigarette smokers	54 (50)	23 (32.4)	31 (83.8)*	118 (53.6)	30 (23.4)	88 (95.7)
Ex-cigarette smokers	43 (39.8)***	37 (52.1)**	6 (16.2)**	39 (17.7)	37 (28.9)	2 (2.2)
Current cigarette smokers	11 (10.2)***	11 (15.5)***	0 (0.0)	63 (28.6)	62 (48.4)	1 (1.1)

*Significantly different from the Control group: *, P<0.05; **, P<0.01, ***,P<0.001*

5.4.4 WEIGHT HISTORY

The only difference observed in weight history between the Case and Control group was the maximum weight in men, 64.1 kg for the Case and 61.1 kg for the Control (*P=0.02*). It was also found that, although there was no difference in weight at the age of 21 years, women in the Case group had a higher BMI, compared to women in the Control group (Table 5.10).

Table 5.10: Weight history of the Case and Control groups

Weight history	Total	Men	Women
	Mean ± SD	Mean ± SD	Mean ± SD
Cases			
Weight at 21 y of age	49.5 ± 5.5	50.3 ± 5.4	47.5 ± 5.4
BMI at 21 y of age	20.1 ± 2.3	19.5 ± 0.2	21.5 ± 0.2*
Maximum weight	63.0 ± 10.1 **	64.1 ± 9.6 *	60.8 ± 10.7
Minimum weight	48.1 ± 6.5	49.8 ± 5.9	44.2 ± 6.3
Controls			
Weight at 21 y of age	48.3 ± 6.8	50.5 ± 6.9	45.1 ± 5.4
BMI at 21 y of age	19.8 ± 2.6	19.6 ± 2.7	20.0 ± 0.3
Maximum weight	59.8 ± 8.3	61.1 ± 7.8	58.1 ± 8.8
Minimum weight	46.5 ± 6.5	47.1 ± 14.8	43.5 ± 5.2

*Significantly different from the Control group: *, P<0.05; **,P<0.01.*

In the Case group, the average weight changes (from the weight at 21 years old to maximum weight) were 8 kg and 6 kg for men and women (ranged from –11 to 25 kg), while in the Control group they were 7 kg and 9 kg for men and women (ranged from –16 to 32 kg). However, it was also found that those who gained more weight from the age of 21 years had a lower BMI (r_s = –0.4, *P<0.001)* for the Case and Control groups. Among the subjects in the Case group, 21% of them were advised to lose weight by their doctors, compared to 8% in the Control (*P=0.001*). The average weight recommended to lose was 7 kg, for the Case and Control groups.

5.4.4.1 Measured Practised for Weight Maintenance

About 46% of the Case and 33% of the Control reported practising measures to maintain a satisfactory weight. Table 5.11 lists various measures used by subjects to

maintain their weight. The major measures were avoiding particular food, exercising and consuming a special diet.

Table 5.11 Measures practised to maintain body weight

	Cases		Controls		Total	
	n	%	n	%	N	%
Special diet	5	10.0	1	32.6	6	5.2
Avoiding any particular food	34	68.0	29	43.9	63	54.3
Herbs	0	0.0	5	7.6	5	4.3
Exercise	11	22.0	27	40.9	38	32.8
Stress management	0	0.0	2	3.0	2	1.7
Medication	0	0.0	1	1.5	1	0.9
Smoking	0	0.0	1	1.5	1	0.9

5.4.4.2 Major Events Resulting in Weight Increase

In the response to the question regarding events that could have led to a drastic increase in body weight, about 70% and 82% of the Case and the Control group reported that they never experienced a drastic increase in body weight. Among those who gained weight, getting married was a major event that contributed to their weight gain. Other factors for the men were quitting smoking and major stressful events. Females reported gaining weight after their first pregnancy and the after commencement of use of oral contraception.

5.4.5 Family History

Table 5.12 shows that having a family history of CHD was more common among the Case group, compared to the Control. About 25% of the Case group had at least one parent or sibling with angina or myocardial infarction, compared to 7% in the Control group ($P<0.001$). It was also found that, amongst subjects in the Case group, those with family history of CHD were heavier, had a higher BMI, and higher LDL concentration, compared to those without family history (Table 5.13).

Table 5.12: Prevalence of family history of CHD in the Case and Control groups

	Cases		Controls		Total	
	n	%	n	%	N	%
CHD affected parents or sibling	27	25.0	16	7.3	43	13.1
No CHD affected parents or sibling	81	75.0	204	92.7	285	86.9

Table 5.13: Comparison of selected conventional CVD risk factors between those with and without family history in the Case group

Parameter	With family history (n = 27)	Without family history (n = 81)
	Mean ± SD	Mean ± SD
Weight (kg)	57.5 ± 7.9 ***	56.0 ± 10.1
Height (cm)	156.3 ± 8.0	156.8 ± 8.1
Body mass index (kg/m^2)	23.5 ± 2.9 ****	22.8 ± 3.4
Systolic blood pressure (mmHg)	141.9 ± 20.3	137.3 ± 22.9
Diastolic blood pressure (mmHg)	90.0 ± 11.0	82.6 ± 12.5
Total cholesterol (mmol/L)	5.9 ± 1.2	5.4 ± 1.5
HDL-cholesterol (mmol/L)	1.3 ± 0.3	1.2 ± 0.3
LDL-cholesterol (mmol/L)	3.9 ± 1.0 *	3.3 ± 0.8
Triglyceride (mmol/L)	1.6 ± 0.7	1.6 ± 0.8
Glucose (mmol/L)	5.6 ± 2.1	5.5 ± 1.9

*Significantly different from those without a family history: *, P<0.05; ***, P<0.001; ****, P<0.0001.*

Apart from a family history of CHD, it was also observed that about 69% of the cases had a family history of CHD risks or conditions relevant to CHD, compared to 36% for the controls.

Table 5.14: Family history with diseases or conditions related to CVD of the Case and Control groups

Family history	Cases (n=108)		Controls (n=220)		Total (n=328)	
	n	%	n	%	N	%
Angina/infarction	27	25.0	16	7.3	43	13.1
Hypertension	22	20.4	30	13.6	52	15.9
Diabetes	10	9.3	19	8.6	29	8.8
Stroke	4	3.7	3	1.4	7	2.1
Obesity	7	6.5	8	3.6	15	4.6

5.5 ASSOCIATIONS OF AGE WITH SELECTED CHD RISK FACTORS

The results of correlation analyses between age and selected CHD risk factors are presented in table 5.15. It was found that weight and BMI increased in the age group 40 to 50 and decreased thereafter. While triceps, biceps, subscapular and suprailiac skinfold thicknesses decreased after 40 y. Furthermore, no association was found between age and diastolic blood pressure but systolic pressure increased linearly after 40 y. Inverse relations were found with age for cholesterol, LDL-cholesterol, and triglycerides in males.

Table 5.15: Spearman correlation coefficients (r_s) describing relationships between age and selected CHD risk factors

CHD risk factors	Cases			Controls		
	Total	Men	Women	Total	Men	Women
Weight (kg)	–0.38**	–0.46**	–0.39*	–0.24**	–0.33**	–0.24*
Height (cm)	–0.22	–0.28*	–0.11	0.11	0.02	–0.25*
BMI (kg/m^2)	–0.44**	–0.41**	–0.42*	–0.31**	–0.36**	–0.14
Triceps skinfold (mm)	–0.35**	–0.17	–0.50**	–0.34**	–0.26**	–0.30**
Biceps skinfold (mm)	–0.30**	–0.15	–0.43*	–0.30**	–0.19*	–0.26*
Subscapular skinfold (mm)	–0.46**	–0.37**	–0.53**	–0.41**	–0.35**	–0.26*
Supra-iliac skinfold (mm)	–0.41**	–0.49**	–0.23	–0.41**	–0.37**	0.01
Σ skinfold thickness (mm)	-0.46**	-0.40**	-0.47**	-0.42**	-0.36**	-0.31**
Systolic blood pressure (mmHg)	0.18	0.02	0.40	0.38**	0.360'	0.40**
Diastolic blood pressure (mmHg)	–0.15	–0.22	–0.08	–0.03	–0.11	–0.04
Total cholesterol (mmol/L)	–0.14	–0.26*	0.02	–0.09	–0.12*	0.12
LDL-cholesterol (mmol/L)	–0.19	–0.24	–0.09	–0.10	–0.20*	0.13
HDL-cholesterol (mmol/L)	0.07	0.16	0.09	0.05	0.15	0.16
Triglyceride (mmol/L)	–0.13	–0.25*	0.02	–0.13	–0.30**	–0.08
Glucose (mmol/L)	0.02	–0.08	0.14	–0.02	–0.11	0.21

*Significantly different from zero: *, P<0.05; **, P<0.01.*

5.6 LIFESTYLE PATTERN AS A PREDICTOR OF CHD

Logistic regression analyses were performed to examine the predictive power of cigarette smoking, physical activity and stress level on CHD events. It was found that, after adjusting for age and education level, physical activity and stress were independent and strong predictors of CHD for the total population and for men. However, this was not the case for women, amongst whom cigarette smoking was the predictor of CHD (Table 5.16).

Table 5.16: Odd ratio (95% CI) of lifestyle variables

	Total	Men	Women
Smoking status	NA	NA	0.2 (0.04 – 0.7) **
Physical activity	0.4 (0.2 – 0.8) **	0.4 (0.1 – 0.8) *	NA
Stress	2.2 (1.3 – 3.7) **	2.6 (1.3 – 5.3) *	NA

Variables entered into model: age, gender (1=male; 2=female), education, smoking (0=non-smoker, 1=ex-smoker; 2=smoke), physical activity (1= low; 2=moderate; 3=high) and stress level (1=low; 2=moderate; 3=high)

*Significantly different from one: **, P<0.01, *, P<0.05.*

NA: data not available as the variable was removed from the model, as the significance level was higher than 0.15.

5.7 DISCUSSION

The present study examined demographic and lifestyle patterns in relation to CHD events and selected conventional CHD risk factors. Multivariate logistic analyses were used to identify the factors that may explain the association with CHD events.

5.7.1 AGE AND SELECTED CHD RISK FACTORS

In Western populations, weight and BMI increase during the ages of 20 to 65 (as a result of increasing fat mass) and thereafter decline (as a result of decreasing lean muscle mass). In contrast, in non-Westernised populations such the Kitavan in Papua New Guinea, weight, BMI, and triceps decreased linearly with age from 20 or 30 y onward. (Lindeberg et al., 1997b; Mancilha-Carvalho et al., 1989; Trowell and Burkitt, 1979). In a contemporary of rural Sarawak in Malaysian Borneo, BMI started to decline at the age of 40 y (Strickland and Ulijaszek, 1993). Results from this study show that weight and BMI increased during the ages of 40 to 50 and decrease thereafter, but triceps, biceps, subscapular and supra-iliac skinfold thickness decreased after 40 y. Furthermore, a weak inverse relationship between age and height was observed among men in the Case group, but absent in other groups. This suggests an absence of secular trend of peak height.

Diastolic blood pressure was not associated with age, which was common in traditional populations (Lindeberg et al., 1997b; Trowell & Burkitt, 1979). But systolic blood pressure increased linearly after 40 y in the Control group. This may be part of the normal aging process for healthy subjects. While amongst subjects in

the Case group, hypertension was similar across all age groups. Also, no association was found between age and serum lipid and glucose concentrations, while there were inverse relationships between age and serum total cholesterol and triglycerides for men, both in the Case and Control groups. It was reported in a study in the Honolulu Program that age was inversely related to total cholesterol and positively related to HDL (Abbott, 1998).

The observations of the relationships between age and CHD risk factors in the present study suggest the true effects of biological aging. While some relationships indicate the non-Westernised status, other results may possibly show the beginning of the 'Westernization' of lifestyle patterns in this study population.

5.7.2 Socio-Economic Factors

A strong association between CHD and high socio-economic levels, which was indicated by higher education and job, in developing countries has been repeatedly reported (Singh et al., 1999; INCEN, 1999). In the present study, although no significant difference was found in education levels and occupation between the Case and the Control groups, educational attainment of subjects in this study was higher from that of general population in West Sumatra. As subjects in the present study were mostly recruited from hospitals in West Sumatra, it was likely that they had a relative higher socio-economic levels and therefore they had more access to the health facilities.

It has been suggested that rapid nutrition transition in developing countries, which changed diet and lifestyle in the higher social class, was related to the increasing of coronary heart disease (INCEN, 1999). Studies in India, where the prevalence of CHD is increasing rapidly, showed that CHD risk factors, such as hypertension, diabetes mellitus, serum total cholesterol, obesity and a sedentary physical activity, were associated with higher social class (Singh et al., 1999). However, explanations of the relative importance of socio-economic factors as CHD risks in developing countries, are different from those in developed countries. In developing countries, people in the higher socio-economic class reside in the urban area, where obesity, central obesity hypertension, diabetes mellitus and total cholesterol are common (Singh et al., 1999). It has been suggested that urbanization is the cause of an increase in the CHD prevalence amongst the Malays in Malaysia (Khor, 1994; Khor et al., 1999). At the same time, people from lower a socio-economic class who cannot afford animal foods, consume more fruit and vegetables and usually have physically demanding occupations and lower serum cholesterol (Oenzil, 1997). In more developed countries, low socio-economic levels as indicated by low education attainment, are associated with increased risk for CHD (Chandola, 1998; Wamala et al., 1999 & 2001).

5.7.3 SMOKING, PHYSICAL ACTIVITY, AND STRESS

Results from the present study show that smoking was strongly associated with CHD amongst women. This is in line with several case-control and cohort studies, where women seemed to be more affected by smoking than men (Bosetti et al., 1999). It has been suggested that female smokers are more susceptible for CHD, and they are somewhat estrogen-deficient (Baron et al., 1990). Also smokers tend to have a lower HDL-cholesterol concentration (Criqui et al., 1980; Raftopoulos et al., 1999).

It was observed in the present study that higher physical activity and lower stress level were independently strong protective factors against CHD. Results of the Harvard Alumni Health Study, a prospective study, clearly indicated that physical activity was associated with decreased CHD risk even for shorter sessions of activity (Lee et al., 2000). Physical activity increased HDL-cholesterol concentration (Spate-Douglas & Keyser, 1999) and also has also been reported to decrease CHD risk (Eaton et al., 1995). Also in United States physical inactivity is associated in ethnics with higher susceptibility to CHD (Crespo et al., 2000).

The exact mechanism through which psychosocial stress relates to CHD is not definitely known. One hypothesis is that stress may have indirect influence on lipid and hemostatic profiles through smoking and physical inactivity (Brunner, 1997). Another hypothesis is that stress may result in pathogenic physiological mechanisms, through nervous and endocrine processes. Stress hormones, including cathecholamines, have pronounced effects on hemodynamics, lipid metabolism, hemostasis, and other aspects of metabolism (Bjontorp, 1976; Brindley et al., 1999).

5.7.4 WEIGHT HISTORY AND FAMILY HISTORY

A change in body weight is a potentially useful predictor of CHD, because increases in weight largely reflect fat mass. These increases may anticipate a risk within ranges of attained weights that are plausibly due to differences in muscle or bone mass (Willett et al., 1995). It has been suggested that middle age women who gain weight more than 5 kg from the age of 18 years, appear to have higher risk for CHD (Willett et al., 1995). Excessive weight change was also reported to promote atherosclerosis (Stevens et al., 1998). In the present study, no difference was found in weight changes between the Case and the Control groups, while the Case had significantly higher maximum weight.

Familial aggregation of CHD has been reported in several studies. (Allayee et al., 1998; Caicoya et al., 1999; Canani et al., 1998; Hotopf et al., 1999; Lagstrom et al., 1999; Zureik et al., 1999). The present study found that a familial aggregation of CVD is related to CHD. This was in agreement with other studies (Ciruzzi et al., 1997; Friedlander et al., 1998; Leander et al., 2001). The specific underlying mechanisms and the relative contribution of atherosclerosis to the subsequent CHD events in subjects with family history are not well established. It should be emphasised that genetic factors may be involved, but it cannot be ruled out that the observed relationship is confounded by other risk factors, especially family aggregation of lifestyles

5.8 CONCLUSION

The present study showed that certain lifestyle factors were important in CHD events in Minangkabau. Being physically active, at either a moderate or high level, was protective against CHD, while high stress level and cigarette smoking were predictors for CHD. Family history with at least one CHD risk factor was another predictor. For level of education as potential factor, since no differences between the case and control groups were found, no conclusion can be made in this point.

CHAPTER 6

A Case-Control Study of Nutrition and Coronary Heart Disease: Food and Nutrient Intakes

6.1 INTRODUCTION

The relationships between dietary factors, serum lipids and CHD, have been studied mostly amongst Western populations. Only a couple of epidemiological studies among Asian populations are available regarding the association between dietary factors and CHD risks (Oenzil, 1993 & 1997; Singh et al., 1993 & 1998).

Epidemiological studies in Western populations suggest a strong positive association between CHD and several dietary factors, such as total dietary fat, saturated fat, protein, dietary cholesterol and animal food intakes (Hu et al., 1997 & 1999; Keys et al., 1986b; Shekelle et al., 1981; Smit et al., 1999; Tzonou et al., 1993). Inverse associations have also been reported between CHD and polyunsaturated fat, total carbohydrate, and fish intakes (Abbott et al., 1990; Dreon et al., 1994; Grundy et al., 1982; Grundy, 1999; Krauss et al., 2000; Leaf and Weber, 1988; Schmidt and Dyerberg, 1994). To date, most studies have assessed Caucasian populations consuming high intakes of total fat and low intakes of fish with the risk of developing CHD. Only few studies are available exploring associations between CHD events in Asian populations with low total dietary fat and high fish and coconut intakes (Singh et al., 1998). Such a dietary pattern is found in Minangkabau Indonesia.

This chapter describes differences in food and nutrient intakes between CHD patients and healthy controls.

6.2 SUBJECTS AND METHODS

Subjects included in the present study were participants of the Case-Control Study. Eligible cases were diagnosed within 6 months prior to the participation in the study. Within this timeframe, the subjects in the Case group would have been able to recall their food habits and lifestyles before being diagnosed as having CHD. A

total of 93 eligible cases (62 men and 31 women) and 189 subjects (113 men and 76 women) in the Control group were recruited.

A questionnaire (Appendix 2) on demography, health status, lifestyle and general food habits and practices (questions 35-52) was developed and administered. The questionnaire was modified using the Nutrition and Metabolic Study of Indonesian Elderly (Purba, 2000). Information on the intakes of individual foods and dishes over the past 6 months was obtained using the Food Frequency questionnaire (Appendix 3). Nutrient intakes included carbohydrates, proteins, total fat, monounsaturated fat, polyunsaturated fat (n-6 and n-3), cholesterol, vitamins (A, B1, B2) and minerals (calcium, phosphorus, iron, sodium and potassium). To examine the association between CHD events and food and nutrient variables, all subjects were divided into four equal groups according to the quartile values of food and nutrient intakes. The odds ratio (OR) was computed as the rate in a specific quartile divided by the value in the group with the lowest intake. In multivariate analyses, energy intake, the percentage of energy derived from fat intake, and other potentially confounding variables (like stress, physical activity, smoking status) were simultaneously included into the models (Willett, 1998b).

6.3 RESULTS

6.3.1 FOOD HABITS

6.3.1.1 Type of Cooking Methods

Coconut milk and deep fried dishes were the most popular dishes in this study population. About 94% of the cases and 93% of the controls prepared beef with coconut milk. Deep-fried beef mixed with chilli and tomato ('balado') was the second most popular in both groups (86% in the cases and 81% in the controls). For chicken and fish, the 'balado' style cooking method was the most popular followed by the coconut milk dish in both groups.

For vegetables, stir-frying was the most popular cooking method in both groups (85% in the cases and 93% in controls), followed by boiling in coconut milk.

6.3.1.2 Animal Fats, Butter and Soy Sauce

In response to the question whether subjects ate fats in the meat, significantly

higher proportion of the Cases ate the fats compared to the controls (71% vs 55%, *P<0.05*). The use of animal fat as spreads was not common amongst the Minangkabau. For margarine, only 22% and 7% of the cases and controls respectively used margarine (1.5 teaspoons per day was consumed by the cases compared to 0.7 by the controls) (*P<0.001*). Also, soy sauce is not part of Minangkabau food culture - 49% of the cases and 40% of the controls never added soy sauce to their cooking.

6.3.1.3 Herbs and Spices

The Minangkabau use a wide variety of herbs in their cooking. Ginger, turmeric, galanga *(Languas galanga)*, onion and garlic were used at least once a day by more than 60% of the subjects in both groups. Less than half of the subjects in both groups used lemon grass, lime leaves, turmeric leaves, bay leaves and parsley once a day. Other herbs and spices such as coriander, nutmeg, clove, nutmeg, candle nut, pepper, tamarind, spring onion and cardamon, were used by most of the subjects less than once a week.

6.3.1.4 Dietary Change

Subjects in this study were asked whether they had changed their diet over the past 6 months. More than half of the cases (53%) had changed their diet compared to only 13% of the controls *(P<0.001)*. Amongst subjects in the Case group who reported changing their diet, interestingly, no significant differences were observed in weight, BMI, serum total cholesterol, HDL-cholesterol, LDL-cholesterol, triglyceride, or glucose concentrations.

6.3.2 FOOD INTAKE

Table 6.1 shows the mean intake of each food groups of the cases and the controls.

Table 6.1: Descriptive statistics of average daily food consumption (g/d) by food groups for the Case and Control groups

Food groups	Cases (n=93)	Controls (n=189)
	Mean ± SD	Mean ± SD
Fish, seafood and products	67.5 ± 33.8	58.4 ± 30.8
Meats and meat products	47.1 ± 40.2 **	34.5 ± 28.5
Eggs	37.7 ± 31.7 **	26.8 ± 19.3
Milk and milk products	20.0 ± 39.0	17.0 ± 30.4
Soy products	90.1 ± 82.5	100.7 ± 79.5
Legumes and nuts	81.1 ± 43.1	77.0 ± 54.1
Coconut milk and grated coconut	42.0 ± 21.7	38.2 ± 18.2
Rice and cereals	382.3 ± 108.9 ***	407.4 ± 116.0
Vegetables	338.0 ± 146.6	303.4 ± 108.4
Fruits	129.9 ± 73.7 **	114.9 ± 69.8
Non alcoholic beverages	131.1 ± 105.6 **	126.4 ± 78.6
Sugar	34.6 ± 28.8 *	31.6 ± 21.0
Palm oil	25.3 ± 14.2	23.3 ± 10.5

*Significantly different from the Control group: *, P<0.05; **, P<0.01; ***, P<0.001.*

6.3.2.1 Food Variety

Table 6.2 shows descriptive statistics of the total weekly food variety scores between the groups. Significant differences were not found between the cases and the controls. Except for women in the control group, the average weekly food variety score for all groups was below 20. As much as 57% of the cases and 50% of the controls had a food variety score above 20, and therefore were categorised to have a fair to good weekly food variety score (Savige et al., 1997).

Table 6.2: Descriptive statistics of weekly total food variety score of the Case and Control group

	Mean ± SD	Minimum	Median	Maximum
CASES	19.7 ± 3.3	12	20	29
Men	19.9 ± 3.0	12	20	25
Women	19.4 ± 3.9	13	20	29
CONTROLS	19.6 ± 2.8	11	19	27
Men	19.2 ± 2.9	11	19	26
Women	20.1 ± 2.7	14	20	27

6.3.1.6 Animal Foods

The animal food group included fish, eggs, meat, chicken and dairy foods. The Case group had significantly higher intakes of animal foods, compared to the Control group (247 g/d vs 187 g/d, *P<0.0001*). This was mainly due to the difference in the intakes of meat and eggs. Table 6.3 shows distribution of the cases and the controls according to animal foods consumed. The odds ratio for subjects who consumed animal foods in the highest quartile (above 210 grams) to those in the lowest quartile (below 108 grams) was 4.8 (95% CI 2.25–10.30, *P<0.0001*).

Table 6.3: Distribution of subjects according to their animal food consumption

Animal food intake (g/d)	Cases		Controls	
	n	%	n	%
< 108.1	14	15.1	67	30.1
108.1 – 149.8	18	19.4	52	23.5
149.8 – 210.4	27	29.0	43	22.8
> 210.4	34	36.6	37	19.6

6.3.1.7 Plant Foods

The plant food group included rice and cereal, tempeh and tofu, legumes and nuts, vegetables, fruits and coconut milk. There was no difference in plant food intake

between the two groups. Average intake of plant food was 1,061 g/d for the Case group and 1,028 g/d for the Control group.

6.3.3 MACRONUTRIENTS

The intake of macronutrients is presented as g/d; nutrient density is expressed as g/MJ.

6.3.3.1 Macronutrient Intakes

Table 6.4 shows the descriptive statistics of the macronutrients for the Case and Control groups. In total, the cases had significantly higher intakes of protein and cholesterol, but lower intakes of carbohydrate (*P<0.0001* for all cases). Between the men, the cases had significantly higher intakes of total energy, protein, cholesterol, but a lower intake of carbohydrate (*P<0.05*, *P<0.01*, *P<0.0001* and *P<0.001*, respectively). Between the women, the cases had a significantly lower intake of carbohydrate (*P<0.05*).

As expected, rice was the major food source of total energy intake in this population. Rice and cereals contributed as much as 32% and 36% of total energy for the Case and Control groups, respectively. Other major food sources of total energy were fish, vegetables and soy dishes for both groups.

Amongst the macronutrients included in the univariate logistic regression analysis, intakes of protein and cholesterol were found to be risk factors for CHD. More subjects in the Case group were in the highest quartiles of protein and total cholesterol (Table 6.5). The odds ratio for those with intakes of protein and cholesterol in the highest quartile were 1.01 (95% CI 1.01–1.02) and 1.005 (95% CI 1.003–1.007), respectively, compared to those in the lowest quartile.

Table 6.4: Macronutrient intakes of the Case and Control groups

	Total	Men	Women
	Mean ± SD	**Mean ± SD**	**Mean ± SD**
Cases	N = 93	n = 62	n = 31
Total energy (kcal)	1765 ± 534	1823 ± 548 *	1647 ± 491
Total energy (kJ)	7502 ± 2436	7658 ± 2303	6893 ± 2054
Protein (g)	92.0 ± 33.5 **	93.1 ± 34.2 **	88.5 ± 32.5 ****
% total energy	20.6 ± 3.1 ****	20.3 ± 3.3 **	21.1 ± 2.7 ****
Nutrient density (g/MJ)	12.3 ± 1.9	12.1 ± 1.9	12.6 ± 1.6
Carbohydrates (g)	204.1 ± 55.9 ***	213.2 ± 58.3 **	186.0 ± 46.1 *
% total energy	55.8 ± 7.5 ***	56.6 ± 7.5 **	55.3 ± 7.5 *
Nutrient density (g/MJ)	28.0 ± 4.6	28.4 ± 4.5	27.8 ± 4.5
Total fats (g)	47.2 ± 20.9	47.7 ± 21.2	46.4 ± 20.6
% total energy	23.6 ± 5.6	23.1 ± 5.5	24.6 ± 5.8
Nutrient density (g/MJ)	6.4 ± 1.4	6.1 ± 1.5	6.5 ± 1.5
Dietary cholesterol (mg)	296 ± 205 ****	327 ± 207 ****	235 ± 192
Fibre (g)	10.1 ± 4.0	10.1 ± 4.1	10.0 ± 3.6
Controls	N = 189	n = 113	n = 76
Total energy (kcal)	1657 ± 487	1666 ± 475	1643 ± 507
Total energy (kJ)	6934 ± 2038	6971 ± 1987	6877 ± 2122
Protein (g)	79.3 ± 28.2	80.4 ± 28.9	77.7 ± 27.1
% total energy	19.0 ± 2.7	19.1 ± 2.8	18.9 ± 2.5
Nutrient density (g/MJ)	11.4 ± 1.6	11.4 ± 1.7	11.3 ± 1.5
Carbohydrates (g)	204.7 ± 52.9	205.7 ± 50.0	203.1 ± 57.4
% total energy	57.6 ± 6.5	57.7 ± 6.7	57.5 ± 6.2
Nutrient density (g/MJ)	30.1 ± 3.9	30.1 ± 40.5	29.9 ± 3.7
Total fats (g)	44.0 ± 18.6	43.9 ± 18.1	44.2 ± 19.3
% total energy	23.4 ± 4.9	23.2 ± 4.8	23.6 ± 5.1
Nutrient density (g/MJ)	6.2 ± 1.3	6.2 ± 1.3	6.3 ± 1.4
Dietary cholesterol (mg)	187 ± 109	190 ± 113	182 ± 103
Fibre (g)	9.3 ± 3.6	9.2 ± 3.4	9.4 ± 3.9

*Significantly different from the Control group: *, P<0.05; **, P<0.01; ***, P<0.001; ****, P<0.0001.*

Table 6.5: Distribution of cases and controls in the highest and lowest quartiles, the odds ratio (95% confidence intervals) according to their protein and cholesterol intakes

	Cases	Controls	Odds ratio	95% CI
	n (%)	n (%)		
Protein (g/day)				
1st quartile (<61.7 g/day)	16 (17.2)	54 (28.6)	1.01	1.01 – 1.02
4th quartile (>100.2 g/d)	35 (37.6)	36 (19.0)		
Cholesterol (mg/day)				
1st quartile (<125.2 mg/d)	16 (17.2)	55 (29.1)	1.005	1.003 – 1.007
4th quartile (>271 mg/d)	40 (43.0)	30 (15.9)		

6.3.3.2 Dietary Fat and Fatty Acids

Table 6.6 shows consumption of dietary intake in gram/day. Table 6.7 shows percent energy contribution of dietary fat for the Case and Control groups. The tables show that there was no significant differences in the intakes of saturated and unsaturated fatty acids between the cases and controls, except for arachidonic acid (C20:4). The intakes of individual saturated fatty acids (SFAs) were similar in both groups. Lauric (C12:0), palmitic (C16:0) and myristic (C14:0) acids accounted for 43%, 25% and 17% of total fat intake for both the Case and Control groups. The average intake of marine and plant n-3 fatty acids was 1.9 g/d for the Case group and 1.7 g/d for the Control group.

Table 6.6: Fatty acid intakes (g/d) of the Case and Control groups

Dietary fat intake (g/d)	CASES	CONTROLS
	Mean ± SD	Mean ± SD
Saturated fatty acids	**29.4 ± 16.5**	**26.2 ± 11.0**
Short chain fatty acids	2.0 ± 1.2	1.8 ± 0.8
Capric acid (C10:0)	1.6 ± 0.9	1.4 ± 0.6
Lauric acid (C12:0)	12.4 ± 6.9	11.3 ± 4.8
Myristic acid (C14:0)	5.0 ± 2.8	4.6 ± 1.9
Palmitic acid (C16:0)	7.3 ± 4.0	6.5 ± 2.7
Stearic acid (C18:0)	3.1 ± 1.8	2.7 ± 1.1
Arachidic acid (C20:0)	0.03 ± 0.0	0.02 ± 0.02
Monounsaturated fatty acids	**10.8 ± 5.8**	**9.5 ± 4.0**
Palmitoleic (C16:1)	0.6 ± 0.3	0.5 ± 0.2
Oleic acid (C18:1)	10.6 ± 5.8	9.4 ± 4.0
Polyunsaturated fatty acids	**9.1 ± 6.7**	**8.3 ± 4.2**
n-3 fatty acids		
Linolenic acid (C18:3)	0.6 ± 0.6	0.6 ± 0.4
Eicosapentaenoic acid (C20:5, EPA)	0.3 ± 0.2	0.2 ± 0.1
Docosahexaenoic acid (C22:6, DHA)	0.9 ± 0.5	0.7 ± 0.4
n-6 fatty acids		
Linoleic acid (C18:2)	6.8 ± 5.5	6.5 ± 3.6
Arachidonic acid (C20:4)	0.2 ± 0.1 ****	0.1 ± 0.1

*Significantly different from the Control group: ****,P<0.0001*

Short chain fatty acids = C4:0 – C8:0

Table 6.7: % Energy contribution of dietary fat for the Case and Control groups

% Energy	Total	Men	Women
	Mean ± SD	Mean ± SD	Mean ± SD
CASES			
Total dietary fat	**23.6 ± 5.6**	**23.1 ± 5.5**	**24.6 ± 4.8**
Saturated fatty acids	**14.3 ± 3.6**	**14.0 ± 2.3**	**14.5 ± 3.3**
Short chain fatty acids	1.0 ± 0.3	0.9 ± 0.3	1.0 ± 0.3
Capric acid (C10:0)	0.8 ± 0.2	0.7 ± 0.2	0.8 ± 0.2
Lauric acid (C12:0)	6.1 ± 1.7	5.8 ± 1.5	6.3 ± 1.6
Myristic acid (C14:0)	2.4 ± 0.6	2.4 ± 0.6	2.5 ± 0.6
Palmitic acid (C16:0)	3.5 ± 0.9	3.5 ± 0.9	3.5 ± 0.9
Stearic acid (C18:0)	1.5 ± 0.4	1.5 ± 0.4	1.4 ± 0.4
Monounsaturated fatty acids	**5.3 ± 1.5**	**5.2 ± 1.5**	**5.3 ± 1.5**
Palmitoleic (C16:1)	0.3 ± 0.1	0.3 ± 0.1	0.3 ± 0.1
Oleic acid (C18:1)	5.1 ± 1.5	5.1 ± 1.5	5.2 ± 1.6
Polyunsaturated fatty acids	**4.4 ± 1.8**	**4.0 ± 1.4**	**4.8 ± 1.5**
n-6 fatty acids	3.4 ± 1.5	3.1 ± 1.3	3.8 ± 1.4
Linoleic acid (C18:2)	3.3 ± 1.5	3.0 ± 1.3	3.7 ± 1.4
Arachidonic acid (C20:4)	0.06 ± 0.03 ****	0.08 ± 0.03 ****	0.07 ± 0.03 *
n-3 fatty acids	0.7 ± 0.3	1.0 ± 0.3	1.1 ± 0.3
α-linolenic (C18:3)	0.3 ± 0.0	0.3 ± 0.1	0.4 ± 0.2
Eicosapentaenoic acid (C20:5, EPA)	0.2 ± 0.1	0.2 ± 0.1	0.2 ± 0.1
Docosahexaenoic acid (C22:6, DHA)	0.5 ± 0.2	0.5 ± 0.2	0.5 ± 0.2
n-3/n-6 fatty acid ratio	0.3 ± 0.2	0.3 ± 0.2	0.3 ± 0.1
P:S ratio	0.3 ± 0.1	0.3 ± 0.1	0.3 ± 0.1

*Significantly different from the Control group: *, P<0.05; **, P<0.01; ***, P<0.001; ****, P<0.0001.*

P:S ratio: the ratio of polyunsaturated and saturated fatty acid intakes

Tables continues on next page

Table 6.7: % Energy contribution of dietary fat for the Case and Control groups (continued)

% Energy	Total	Men	Women
	Mean ± SD	Mean ± SD	Mean ± SD
CONTROLS			
Total dietary fat	**23.4 ± 4.9**	**23.2 ± 4.8**	**23.6 ± 5.1**
Saturated fatty acids	**13.9 ± 3.0**	**13.8 ± 2.9**	**14.1 ± 3.1**
Short chain fatty acids	1.0 ± 0.2	1.0 ± 0.2	1.0 ± 0.3
Capric acid (C10:0)	0.8 ± 0.2	0.8 ± 0.2	0.8 ± 0.2
Lauric acid (C12:0)	6.0 ± 1.4	6.0 ± 1.4	6.0 ± 1.4
Myristic acid (C14:0)	2.4 ± 0.5	2.4 ± 0.5	2.5 ± 0.6
Palmitic acid (C16:0)	3.5 ± 0.8	3.4 ± 0.7	3.6 ± 0.8
Stearic acid (C18:0)	1.4 ± 0.3	1.4 ± 0.3	1.4 ± 0.4
Monounsaturated fatty acids	**5.1 ± 1.1**	**5.0 ± 1.1**	**5.1 ± 1.1**
Palmitoleic (C16:1)	0.3 ± 0.1	0.3 ± 0.1	0.3 ± 0.1
Oleic acid (C18:1)	5.0 ± 1.2	5.0 ± 1.2	5.1 ± 1.2
Polyunsaturated fatty acids	**4.4 ± 1.4**	**4.3 ± 1.3**	**4.4 ± 1.4**
n-6 fatty acids	3.5 ± 1.3	3.4 ± 1.3	3.6 ± 1.3
Linoleic acid (C18:2)	3.4 ± 1.3	3.4 ± 1.3	3.5 ± 1.3
Arachidonic acid (C20:4)	0.06 ± 0.02	0.06 ± 0.02	0.06 ± 0.02
n-3 fatty acids	0.9 ± 0.2	0.9 ± 0.3	0.9 ± 0.3
α-linolenic (C18:3)	0.4 ± 0.0	0.3 ± 0.1	0.4 ± 0.1
Eicosapentaenoic acid (C20:5, EPA)	0.1 ± 0.1	0.1 ± 0.1	0.1 ± 0.0
Docosahexaenoic acid (C22:6, DHA)	0.4 ± 0.2	0.4 ± 0.2	0.4 ± 0.1
n-3/n-6 fatty acid ratio	0.3 ± 0.1	0.30 ± 0.2	0.29 ± 0.1
P:S ratio	0.3 ± 0.1	0.3 ± 0.1	0.3 ± 0.1

*Significantly different from the Control group: *, P<0.05; **, P<0.01; ***, P<0.001; ****, P<0.0001.*

P:S ratio: the ratio of polyunsaturated and saturated fatty acid intakes

Table 6.8 shows the percentage of fat from fat groups. Fish dishes were the first sources for total fat in both groups, followed by soy dishes, rise and cereal dishes. Fish and soy dishes were the major food sources for MUFAs. The Case and Control groups were not different in terms of food sources for MUFAs, PUFAs and n-3/n-6 fatty acid ratio. As expected, fish was almost the only source of n-3 in the present study; it accounted for more than 90% of n-3 fatty acid intake in both

groups. The cases had significantly higher intakes of arachidonic acid (C20:4) due to the greater consumption of meat and eggs. The proportion of food sources for omega-6 was similar in both groups. Eggs, fish and beef were the most important sources of arachidonic acid, whilst soy, egg and beef were the most important sources of linoleic acid.

Table 6.8: Percentage of fat from food groups

Food groups	CASES	CONTROLS
	Mean ± SD	Mean ± SD
Fish, seafood and products	26.9 ± 10.7	24.0 ± 11.6
Soy products	18.1 ± 11.9 **	22.1 ± 12.5
Cereal and cereal products	11.8 ± 8.3	12.9 ± 7.5
Meats and meat products	11.5 ± 7.2 **	9.2 ± 6.4
Vegetables	9.8 ± 4.5	10.3 ± 4.3
Eggs	10.2 ± 7.3	9.0 ± 5.3
Nuts and seeds	4.4 ± 5.1	4.4 ± 4.4
Milk and milk products	4.1 ± 8.7	4.5 ± 9.3
Fruits	2.8 ± 2.4	2.8 ± 1.9
Confectionary	0.2 ± 0.7	0.2 ± 0.7
Non alcoholic beverages	0.4 ± 1.2	0.7 ± 1.7

*Significantly different from the Control group: **, P<0.01.*

As shown in Table 6.9, SFAs mainly came from fried and coconut dishes of fish and soy.

Table 6.9: Top six food dishes contributing to specific saturated fatty acids in the diet (%)

SCFAs (C4:0-C8:0)	Capric acid (C10:0)	Lauric acid (C12:0)	Myristic acid (C14:0)	Palmitic acid (C16:0)	Stearic acid (C18:0)	Total SFAs
soy (31)	soy (30)	soy (28)	soy (27)	soy (25)	soy (25)	soy (28)
fish (27)	fish (26)	fish (24)	fish (26)	fish (16)	beef (17)	fish (25)
vegetables (15)	vegetables (18)	vegetables (22)	vegies (20)	beef (13)	fish (17)	vegetables (12)
chicken (6)	beef (6)	beef (8)	beef (8)	egg (13)	egg (12)	beef (8)
beef (6)	chicken (5)	chicken (5)	chicken (5)	vegetables (8)	vegetable (8)	egg (13)

SCFAs: short-chain fatty acids (C4:0-C8:0); SFAs: saturated fatty acids

Table 6.10 shows the relationships between individual SFAs. Since SFAs shared their food sources, the correlations amongst specific SFAs were high. The food sources of short-chain fatty acids (containing less than 10 carbon atoms) were similar to those of stearic acid (C18:0). All SFAs had strong positive associations with the consumption of soy, fish and coconut dishes ($P<0.0001$) both in the Case and Control groups.

Table 6.10: Pearson correlation coefficients (*r*) describing relationships between the intakes of individual saturated fatty acids

	SCFAs	Capric acid	Lauric acid	Myristic acid	Palmitic acid	Stearic acid
Short-chain fatty acids	1.00					
Capric acid	0.99	1.00				
Lauric acid	0.97	0.99	1.00			
Myristic acid	0.99	0.99	0.99	1.00		
Palmitic acid	0.91	0.90	0.88	0.92	1.00	
Stearic acid	0.89	0.89	0.88	0.92	0.99	1.00

SCFAs: Short-chain fatty acids (C4:0-C8:0)

6.3.4 MICRONUTRIENT INTAKES

The descriptive statistics of the micronutrients are shown in Table 6.11. The cases had significantly higher intakes of phosphate, iron, sodium, potassium, retinol, vitamin B-2, and niacin. Significant differences were not found for those who consumed below the recommended intakes in both men and women in the two groups. For calcium, 35% of men and 30% of the women in the Case group, and 39% of the men and 38% of the women in the Control group, consumed less than the recommended (500 mg). For phosphate, 14% of the men and 16% of the women in the Case group, and 13% of the men and 17% of the women in the Control group consumed less than the recommended (450 mg). For iron, 31% of the men and 35% of the women in the Case group, and 35% of the men and 41% of the women in the Control group consumed less than the recommended (13 mg for men and 14 mg for the women). For vitamin B-1, 52% of the men and 54% of the women in the Case group, and 55% of the men and 48% of the women from the Control group, consumed less than recommended (1.0 mg). For vitamin B-2, 25% of the men and 24% of the women in the Case group, and 27% of the men and the women in the Control group, consumed less than the recommended (1.0 mg for women and 1.2 mg for men). For vitamin C, 23% of the men and 22% of the women in the Case group, and 21% of the men and 26% of the women in the Control group, consumed less than the recommended (60 mg).

Table 6.11: Percentile distribution of micronutrients in cases and controls

Micronutrients	Total	Men	Women
	Mean ± SD	Mean ± SD	Mean ± SD
CASES	*N = 93*	*n = 62*	*n = 31*
Calcium (mg)	772.8 ± 429	731.9 ± 387	914.5 ± 602
Phosphate (mg)	1452.1 ± 574 **	1458 ± 579 *	1523.4 ± 776
Iron (mg)	19.6 ± 7 *	19.8 ± 8	20.6 ± 9
Sodium (mg)	697.6 ± 287 **	698.7 ± 274 **	703.5 ± 320
Potassium (mg)	1891.7 ± 701 **	1879.2 ± 742	1974.3 ± 691 *
Retinol (µg)	641.3 ± 322 ****	681.7 ± 332 ****	564.9 ± 293
Carotene (µg)	7071.3 ± 3847	6970.3 ± 4118	7670.4 ± 3518
Retinol equivalent (µg)	1739.6 ± 780 *	1756.9 ± 822	1780.4 ± 744
Vitamin B-1 (mg)	1.2 ± 0	1.2 ± 1 *	1.2 ± 0.5
Vitamin B-2 (mg)	2.1 ± 1.0 *	2 ± 1	2.3 ± 1
Niacin (mg)	12.1 ± 5.2 **	12.5 ± 5 *	11.9 ± 5.8
Vitamin C (mg)	131.1 ± 65	131.1 ± 70	135.7 ± 58
CONTROLS	*N = 189*	*n = 113*	*n = 76*
Calcium (mg)	716.2 ± 329	695.66 ± 335	746.8 ± 318
Phosphate (mg)	1250.7 ± 462	1251.84 ± 473	1248.9 ± 447
Iron (mg)	17.8 ± 6	17.63 ± 6.4	18.1 ± 7
Sodium (mg)	597.8 ± 233	585.32 237.7	616.3 ± 227
Potassium (mg)	1671.1 ± 641	1700.63 ± 673	1627.2 ± 590
Retinol (µg)	499.3 ± 233	501.29 ± 245	496.5 ± 215
Carotene (µg)	6518.3 ± 3270	6596.35 ± 3381	6402.2 ± 3118
Retinol equivalent (µg)	1517.4 ± 653	1542.43 ± 678	1480.3 ± 619
Vitamin B-1 (mg)	1.1 ± 0.4	1.06 ± 0.5	1.1 ± 0
Vitamin B-2 (mg)	1.8 ± 1	1.78 ± 0.8	1.9 ± 1
Niacin (mg)	10.6 ± 4	10.76 ± 4	10.3 ± 4
Vitamin C (mg)	120.9 ± 52	121.75 ± 55	119.8 ± 50

*Significantly different from the Control group: *, P<0.05; **, P<0.01; ****, P<0.0001.*

6.4 MULTIVARIATE ANALYSES

To determine the predictive power of food and nutrient variables for CHD events, regression of food and nutrient variables was performed. Results are presented in Table 6.12. Included in this model were food variables, such as animal intake, and nutrient variables such as total energy, protein, carbohydrate and cholesterol intakes. Because animal intakes are highly likely to be confounded by other aspects of diet, especially saturated fat intake and lifestyle factors such as physical activity and stress levels, the regression analysis adjusted for these variables. Presented in this table are the partial correlation coefficients for the variables included in the models.

Table 6.12 shows the odds ratio and 95% CI of CHD by food and nutrient intakes. For the total population, higher intakes of carbohydrate, higher physical activity level, lower animal intake and lower stress levels, were protective against CHD. For men, carbohydrate intake was no longer protective, but lower animal intake and stress, and higher physical activity level were protective. For women, total carbohydrate, animal intake, stress levels and smoking were predictors for CHD.

Table 6.12: Odds Ratio (95% CI) of CHD by food and nutrient variables

	Total	Men	Women
Total Carbohydrate (highest vs lowest quartile)	0.7 (0.36 – 1.47)	NA	0.98 ** (0.97 – 0.99)
Animal food intake (highest vs lowest quartile)	4.8 **** (2.25 – 10.30)	5.6 *** (1.99 – 16.89)	4.7 * (1.28 – 16.98)
Physical activity (highest vs lowest quartile)	0.4 ** (0.2 – 0.8)	0.3 * (0.1 – 0.7)	NA
Stress level (highest vs lowest quartile)	2.9 ** (1.6 – 6.5)	2.8 * (1.2 – 6.3)	3.6 ** (1.3 – 10.3)
Smoke (highest vs lowest quartile)	NA	NA	0.2 ** (0.04 – 0.7)

Variables entered into the model include the intakes of animal food, carbohydrate, protein, cholesterol, saturated fat, physical activity, stress level and smoking status.

NA: data not available as the variable was removed from the model (the significance level did not reach 0.15).

*Significantly different from the odds ratio 1.0: *, P<0.05; **, P<0.01; ***, P<0.001; ****, P<0.0001.*

6.5 DISCUSSION

One major concern in estimating food intake in a case-control study is the strong influence of current diet on recall of previous diet. In a case-control study, the cases tended to over- or under-report past dietary practices (Willett, 1998b). However, Willet suggested that diet may be recalled with acceptable levels up to approximately 10 years - beyond this period greater uncertainty exists.

The incidence of CHD in this population could not be explained by the introduction of Western foods, because the influence of Western food in this study population was nearly nil. Although the cases had significantly higher intakes of margarine (1.5 teaspoons/day) compared to the controls (0.7 teaspoon/day), the intake of dairy foods (other than milk) was nil and fast food was 0.7 g/day. A study in Japan suggested an association between the increase of Western style fat-rich foods such as butter, margarine, cheese, bread, ham and sausage with an increase in mortality from degenerative diseases (Kato et al., 1987).

This study showed that there were no significant differences in CHD risk between those who changed their diet (over the previous 6 months) and those who did not. Assessment of dietary change was performed on the cases only, Cases were first diagnosed in the previous six months before the study commenced, therefore the extent of dietary change in this study was difficult to assess and interpret quantitatively.

6.5.1 TOTAL FAT INTAKE

The present study did not find any significant differences in dietary fat intake between the Case and Control groups, and there was also no relationship with CHD. Although other studies have found positive correlations with CHD (Tzonou et al., 1993), total fat intake was not always associated with CHD risk or mortality. After reviewing a wide range of studies, there was a conclusion that only little evidence supported a claim that a high proportion of dietary fat predisposed to CHD (Grundy, 1999; Hooper et al., 2001; Ravnskov, 1995 & 1998). Several case-control studies compared total fat intake between the cases and the controls and found no significant differences (Ravnskov, 1995). However, most of these studies were done in populations with high fat intakes. Populations with low fat intakes tended to be at low risk of CHD (Dreon et al., 1999; Grundy et al., 1982), and had lower plasma lipid concentrations (Raeini-Sarjaz et al., 2000). In addition, a recent study found that consumption of a low fat diet altered plasma fatty acid patterns in

a similar manner to that observed with feeding of n-3 long-chain fatty acids (Raatz et al., 2001).

The increased intake of saturated fat in different populations has been confirmed to be associated with the elevation of serum cholesterol concentrations and CHD mortality (Barr et al., 1992; Keys, 1986a; Kromhout et al., 2000; Shekelle et al., 1981). It has been suggested that a high intake of coconut oil may contribute to this relationship (Anderson et al., 1976; Pronczuk et al., 1991; Rumla et al., 1991; Soma et al., 1985; Van and Zilversmit, 1988). In contrast, when studies were conducted within populations (Ascherio et al., 1996), it was not always possible to demonstrate significant relationships between intake of saturated fat and the incidence of CHD, as it was shown in the present study. Likewise, it has been difficult to demonstrate significant relationships between saturated fat intake and serum lipid levels in observational studies (Jacobs et al., 1990; Samuelson et al., 2001).

There are several possible explanations for this, such as insufficient precision of the methods used for dietary surveys, large intra-individual variations in food intake, genetic variation with low and high responders to changes in dietary fat, or a small and heterogenous sample with regard to age and gender. Another possibility is that relationships between dietary fat intake and serum lipid levels are more complex than that has been realised hitherto.

Subjects in the Case group were recruited from a medically diagnosed group of CHD patients from several hospitals in West Sumatra, and those in the Control group were recruited carefully to match the age and gender of the Cases. The use of a validated FFQ may have helped identify the similarities between the Case and Control groups in terms of total fat intake, the contribution to energy intake by total fat and individual fatty acids.

6.5.2 Fatty Acid Intakes

Only limited information is available regarding the association between individual SFAs and the risk of CHD. Experimental studies have found that different classes of SFAs have different effects on plasma lipid and lipoprotein concentrations (Kelly et al., 2001; Nicolosi, 1997). The differential effects of specific saturated fats on plasma lipids and lipoproteins imply that these fats may have different effects on CHD risk (Hu et al., 1999). But in the present study, there were no differences in saturated fatty acid intakes between the cases and the controls. Moreover, the

results do not support correlation between consumption of each saturated fatty acid and CHD events.

Results from several studies have suggested that diets high in monounsaturated fatty acids have a more favourable effect on total cholesterol (Mensink, 1994) and cardioprotection (de Lorgeril et al., 1999). However, in this study, no association was found between monounsaurated and polyunsaturated fat and CHD events, although the cases had a significantly higher intake of arachidonic acid than the controls.

6.5.3 Total Energy, Carbohydrates, Protein and Dietary Cholesterol Intakes

The present study provides no evidence that total energy intake has implications to CHD, although other studies have found a positive relationship between total energy intake and CHD (Tzonou et al., 1993; Kromhout et al., 1988).

Similar to otherstudies (Tzonou et al., 1993), it was confirmed in the present study that high carbohydrate intake was protective against CHD in the Minangkabau population, especially for women. After adjusting for other CHD risk factors, the odds ratio of high carbohydrate intake was 0.9 (95% CI 0.8–0.9) in the total population, and 0.98 (95% CI 0.97– 0.99) in women. High-carbohydrate, low-fat diets have been widely recommended as a way to reduce CHD risk. This is because populations with low intakes of fat tend to be at low risk (Grundy, 1999; Dreon et al., 1994; Vogel et al., 1997). A study among the Pima Indians reported that a high carbohydrate diet decreased the conversion of VLDL to LDL, and this would lead to a reduction in serum LDL-cholesterol concentrations (Abbott et al., 1990). Moreover, other studies confirmed that high carbohydrate diets lowered not only total cholesterol, but also LDL- and HDL-cholesterol without modifying the LDL:HDL ratio. And at the same time, triglyceride concentrations were unchanged. (Vidon et al., 1997 & 2001). In association with losing body weight, high carbohydrate intakes had a similar effect to diets high in MUFAs (Walker et al., 1996). But some studies found that high-carbohydrate diets can reduce HDL-cholesterol and raise triglyceride concentrations (Mensink and Katan, 1992; Nelson et al., 1995; Dreon et al., 1994), and reduced LDL particle size (Dreon et al., 1997; 1999 & 2000; Dreon and Krauss, 1997).

A few case-control studies examined the association of CHD with protein intake. Smit et al. (1999) found a significant positive association between CHD with

protein intake. Analyses of the association between protein intake and CHD risk are difficult to interpret because they only involved simple comparisons of means between cases and non-cases and the intake of specific types of dietary fat had not been adjusted for. In the present study, in univariate analysis, the odds ratio of subjects with total protein intake in the top 25% compared to the lowest 25% was 3.2 (95% CI 1.6–6.6). But after adjusting for other risk factors, total protein failed to enter the model, suggesting that it was not a risk factor for CHD.

Dietary cholesterol intake was associated with an increased risk of CHD in some studies but not in others (Addis et al., 1995; Simkin-Silverman et al., 1995). In the present study, the intake of cholesterol was positively associated with CHD in the total population, especially in men, but not in women. In univariate analysis, the odds ratio of subjects with cholesterol intakes in the highest quartile, compared to the lowest quartile, was 4.7 (95% CI 2.3–9.7). Dietary cholesterol has been reported to increase liver cholesterol synthesis, resulting in down-regulation of LDL receptor concentrations (Schaefer, 1997). The background quality of dietary intake, bound in Minangkabau people, as with other population, effects serum cholesterol and lipoprotein status.

6.5.4 Food Intakes

In general, the intakes of several food groups were different between the cases and the controls. Only total animal intake was found to be independently correlated with CHD events. A higher fish intake (which was also associated with higher n-3 fatty acid intake), has been recognised to reduce very-low-density lipoproteins, inhibit thromboxane production, increase prostacyclin synthesis, reduce the likelihood of thrombosis, risk of cardiac arrhythmias, and blood viscosity (Agren et al., 1997; Krauss et al., 2000; Leaf and Weber, 1988; Schmidt and Dyerberg, 1994). Other populations with higher fish intake, such as the Eskimos and the Japanese, have long been recognised to have low rates of CHD (Bang et al., 1980; Hirai et al., 1980; Kromann and Green, 1980). However, in the present study, it was observed that both the Case and Control groups had high intakes of n-3 fatty acids. One possible explanation for the apparently discordant results of fish consumption and CHD in the present study was that fish consumption was probably unrelated to the CHD incidence, but related to the reduction in CHD mortality, perhaps by reducing the risk of fatal arrhythmia. Some studies conducted in North America and European countries, where fish consumption was lower than in the present study, have not found a correlation between fish intake and CHD incidence (Ascherio et

al., 1995; Kromhout et al., 1996; Norell et al., 1986), and in CHD mortality (Kromhout et al., 1996).

6.6 CONCLUSIONS

Two conclusions can be drawn from this chapter. Firstly, intakes of Western foods in this study population were minimal, so the incidence of CHD cannot possibly be explained by the introduction of Western foods. Secondly, the results from this study showed that the intakes of total fat and saturated fat were not associated with CHD events and thus cannot be used to predict CHD. In contrast, intakes of animal food, total protein, dietary cholesterol and total carbohydrate were found to be predictors of CHD.

CHAPTER 7

A Case-Control Study of Nutrition and Coronary Heart Disease:

Body Composition and Nutritional Biomarkers

7.1 INTRODUCTION

Although obesity has not been a public health problem in Indonesia, its prevalence is increasing and comparable with other data in other countries in Southeast Asia. The prevalence of overweight and obesity ranges from 14.3% in a small city in Central Java (Winkvist et al., 2000) to 39% in Metropolitan Jakarta (Weta et al., 2000). Many studies have suggested that obesity is associated with the development of CHD (Anderson et al., 1976; Dowse et al., 1991; Peiris et al., 1991; Waaler, 1984). However, some studies have reported contradictory findings (Hodgson et al., 1994; Paffenbarger, et al., 1986).

Furthermore, relationships have been established between obesity and CHD risk factors including cigarette smoking, insulin resistance, hyperinsulinaemia, diabetes, hypertension, and abnormality in plasma lipoprotein lipids (DeFronzo & Ferrannini, 1991). In addition, it is also observed that android fat patterning is related to the increased risk for developing CVD (Dowse et al., 1991; Peiris et al., 1991). This suggests that increased risk among the obese might depend primarily on the influence of other associated risk factors, and possibly not on elevated body weight per se (Tavani et al., 1997). Clarification of previous controversial findings is important to accurately estimate the scale of the effect of weight gain and other anthropometric measurements on the risk of CHD.

Hyperlipidaemia and low HDL-cholesterol have been recognised as risk factors for CHD. Findings from many epidemiological studies suggest that the CHD risk increases with increasing total cholesterol concentrations (Gouldbourt & Medalie, 1979; Kannel et al., 1990 ; Nobili et al., 1994). And also, some studies indicate that triglycerides and LDL-cholesterol are independent predictors for CHD event (Assmann et al., 1998; Packard et al., 2000). Low HDL-cholesterol has been associated with CHD, even without elevated LDL-cholesterol or total cholesterol concentrations (Assmann et al., 1998; Castelli et al., 1986; Wilt et al., 1997), although other studies have found mortality is not increased in subjects with isolated low HDL-cholesterol, compared with those with both low HDL-

cholesterol and high total cholesterol (Goldbourt et al., 1997). The mechanisms whereby HDL-cholesterol is related to atherosclerosis are not completely understood, but the Reverse Cholesterol Transport (RCT) has been accepted to describe the metabolism and important anti-atherogenic function of HDL (von et al., 2001).

This chapter describes the anthropometric measurements, blood pressure, lipid and glucose concentrations, and their associations with CHD risk factors, of the cases and their controls.

7.2 SUBJECTS AND METHODS

Subjects included in the present study were participants of the Case-Control Study conducted in February to August 1999. Of the total of 108 cases and 220 controls, 93 cases (62 men and 31 women) and 189 controls (112 men and 77 women) completed anthropometric measurements and their blood samples were taken. Appendices 4 and 5 show examples of all measurements and assessments performed on the subjects.

7.3 RESULTS

7.3.1 ANTHROPOMETRIC AND BODY COMPOSITION CHARACTERISTICS

Male subjects in the Case group had an average BMI of 22.3 kg/m^2 (range 16.1–30.5 kg/m^2), which is similar to their age-matched control subjects who had an average BMI of 22.3 kg/m^2 (range 15.4–30.2 kg/m^2). For women, the mean BMI was 24.0 kg/m^2 (17.4–30.0 kg/m^2) for the Case group, and 23.1 kg/m^2 (14.1–30.4 kg/m^2) for the Control group. No significant difference was observed in BMI between the cases and the controls, both in men and women. This is also true for other anthropometric and body composition measurements, as shown in Table 7.1.

Table 7.1: Anthropometric measurements of the Case and Control groups

	CASES (n = 93)					CONTROLS (n = 189)				
	n	Mean ± SD	Minimum	Median	Maximum	n	Mean ± SD	Minimum	Median	Maximum
Weight (kg)										
Total	93	56.4 ± 1.0	36.0	58.0	80.0	189	55.3 ± 0.7	36.0	54.0	71.0
Men	62	57.6 ± 1.1	38.0	59.0	80.0	112	57.4 ± 0.8	40.0	57.0	81.0
Women	31	53.4 ± 1.7	29.5	55.0	81.0	77	52.4 ± 1.1	29.5	53.0	74.0
Height (cm)										
Total	93	156.7 ± 0.8	139.2	156.7	178.2	189	156.3 ± 0.5	139.8	156.2	172.2
Men	62	160.7 ± 0.7	148.5	159.8	178.2	112	160.4 ± 0.5	147.8	160.5	172.2
Women	31	148.8 ± 0.9	139.2	148.4	159.8	77	150.0 ± 0.6	139.8	151.0	160.2
BMI (kg/m^2)										
Total	93	23.0 ± 0.4	16.1	23.2	30.5	189	22.6 ± 0.2	14.1	22.7	30.4
Men	62	22.3 ± 0.4	16.1	22.7	30.5	112	22.3 ± 0.3	15.4	22.4	30.2
Women	31	24.0 ± 0.7	17.4	25.0	30.0	77	23.1 ± 0.4	14.1	23.4	30.4

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Table 7.1: Anthropometric measurements of the Case and Control groups (continued)

	CASES (n = 93)					CONTROLS (n = 189)				
	n	Mean ± SD	Minimum	Median	Maximum	n	Mean ± SD	Minimum	Median	Maximum
Triceps skinfold (mm)										
Total	93	19.7 ± 1.0	4.7	18.5	37.0	189	20.0 ± 0.7	4.0	18.7	37.0
Men	62	16.4 ± 1.1	4.7	14.7	37.0	112	16.2 ± 0.8	4.0	13.6	37.0
Women	31	25.7 ± 1.5	6.8	27.6	37.0	77	26.2 ± 0.9	4.9	27.5	37.0
Biceps skinfold (mm)										
Total	93	13.5 ± 0.8	3.7	12.3	28.0	189	12.9 ± 0.5	3.3	10.7	28.0
Men	62	11.4 ± 0.7	3.7	10.0	28.0	112	9.6 ± 0.5	3.3	8.0	28.0
Women	31	17.2 ± 1.2	4.3	18.7	28.0	77	18.0 ± 0.8	3.6	18.5	28.0
Subscapular skinfold (mm)										
Total	93	27.5 ± 1.1	6.5	30.5	39.0	189	26.2 ± 0.7	4.8	26.5	39.0
Men	62	25.0 ± 1.2	7.2	24.5	39.0	112	23.8 ± 0.9	7.1	23.5	39.0
Women	31	31.6 ± 1.6	6.5	36.3	39.0	77	30.3 ± 1.1	4.8	32.5	39.0
Supra-iliac skinfold (mm)										
Total	93	22.7 ± 1.2	5.4	21.7	39.0	189	24.2 ± 0.8	4.1	24.5	39.0
Men	62	19.3 ± 1.2	5.5	19.9	39.0	112	20.8 ± 0.9	4.1	19.3	39.0
Women	31	28.2 ± 1.8	5.4	30.5	39.0	77	29.9 ± 1.1	4.4	32.9	39.0

The prevalence of overweight and obesity was similar between the two groups (Table 7.2). The percentage of obese subjects with BMIs over 30 kg/m^2 was less than 2% in the controls, while in the cases the prevalence was higher. The percentage of obese subjects with BMIs ranging from 25.0– 29.9 kg/m^2 was similar between the two groups (26%). It was found that obesity grade I (BMI 25.0–29.9 kg/m2) was more prevalent amongst women in the Case group, compared to their counterpart controls (47 vs 36%), however, this difference was not significant (*P= 0.52*).

Table 7.2: Prevalence of obesity, according to BMI, of the Case and Control groups

Category	CASES (n = 93)			CONTROLS (n = 189)		
	Total	Men	Women	Total	Men	Women
Underweight (BMI < 18.5kg/m^2)	10 (10.8)	7 (11.5)	3 (9.4)	32 (17.1)	22(11.5)	10 (12.3)
Acceptable weight (BMI < 22.9 kg/m^2)	35 (37.6)	28 (45.9)	7 (21.9)	68(36.4)	46 (45.9)	23 (30.1)
Overweight (BMI 23-24.9 kg/m^2)	20 (21.5)	14 (22.9)	6 (18.8)	36 (19.3)	20 (22.9)	16 (20.5)
Obese I (BMI 25-29.9 kg/m^2)	26 (28.0)	11 (18.0)	15 (46.9)	49 (26.2)	23 (18.0)	27 (35.5)
Obese II (BMI ≥ 30 kg/m^2)	2 (2.2)	1 (1.6)	1 (3.1)	2 (1.1)	1 (1.6)	1 (1.4)

7.3.2 Body Mass Index and CHD Risk Factors

It was found in the present study that BMI was correlated with some CHD risk factors (Table 7.3). Age and HDL-cholesterol were inversely associated with BMI in both groups, except for women in the Control group. Those who had higher BMIs tended to have higher triglyceride concentrations. The associations of BMI with total and LDL-cholesterol, and glucose were seen in only the Control group, especially for men, but not in the Case group, or in the female controls.

Table 7.3: Spearman correlation coefficients (r_s) describing relationships between BMI and CHD risk factors

Variable	CASES (n = 93)			CONTROLS (n = 189)		
	Total	Men	Women	Total	Men	Women
Age (year)	−0.4 ****	−0.4 **	−0.4 *	−0.3 ****	−0.4 ****	−0.2
Total cholesterol (mmol/L)	0.2	0.3 *	−0.1	0.3 ***	0.3 **	0.2
HDL-cholesterol (mmol/L)	−0.2 *	−0.3	−0.3	−0.2 *	−0.2 *	−0.3 **
LDL-cholesterol (mmol/L)	0.1	0.3 *	−0.2	0.2 ***	0.2 *	0.2 *
Trigiyceride (mmol/L)	0.4 ****	0.5 ***	0.4 *	0.3 ****	0.4 ****	0.3 *
Glucose (mmol/L)	−0.03	−0.0	0.0	0.2 **	0.2*	0.1

*Significantly different from zero: *, P<0.05; **, P<0.01; ***, P<0.001; ****, P<0.0001.*

7.3.3 BLOOD PRESSURE

Table 7.4 shows descriptive statistics for blood pressure for the Case and Control groups. Subjects in the Case groups had significantly higher systolic and diastolic blood pressures, compared to their control counterparts, for both men and women. The average systolic blood pressure for the cases was 139 mmHg, and for the controls was 127 mmHg (*P<0.0001*), while the average diastolic blood pressure was 85 and 77 mmHg for the Case and Control groups (*P<0.0001*). Similarly, Table 7.5 shows that the Case group had a higher prevalence of high systolic (22% vs 11%) and diastolic (15% vs 5%) blood pressure, and combined high (17% vs 5%) blood pressure, compared to the Control group.

Table 7.4: Mean systolic and diastolic blood pressure of the Case and Control groups

	CASES (n = 93)					CONTROLS (n = 189)				
	n	Mean ± SD	Minimum	Median	Maximum	n	Mean ± SD	Minimum	Median	Maximum
Systolic BP (mmHg)										
Total	93	138.5 ± 2.2 ****	95	140	190	189	126.7 ± 1.6	80	120	200
Men	62	140.6 ± 2.7 ****	95	140	190	112	129.0 ± 2.0	90	125	180
Women	31	134.4 ± 3.8 *	100	130	190	77	123.1 ± 2.5	80	120	200
Diastolic BP (mmHg)										
Total	93	84.5 ± 1.1 ****	50	80	120	189	77.3 ± 0.8	50	80	110
Men	62	85.2 ± 1.4 ****	50	90	120	112	78.4 ± 1.0	50	80	110
Women	31	83.1 ± 2.0 **	60	80	120	77	75.5 ± 1.3	60	70	100

*Significantly different from the Control group: *, P<0.05; **, P<0.01; ****, P<0.0001.*

Table 7.5: Prevalence of high blood pressure of the Case and Control groups

	CASES		CONTROLS	
	n	%	n	%
High systolic blood pressure (≥ 160 mmHg)	21	22.3 *	21	11.1
High diastolic blood pressure (≥ 95 mmHg)	14	14.9 **	10	5.3
Combined high blood pressure	11	13.6 *	8	4.6

*Significantly different from the Control group: *, P<0.05; **, P<0.01.*

7.3.4 SERUM LIPID AND GLUCOSE CONCENTRATIONS

Descriptive statistics for serum lipid and glucose measurements are presented in Table 7.6. Significant differences were not observed in the means of total cholesterol, LDL-cholesterol and triglyceride between the Case and Control groups. However, more subjects in the Case group had hypercholesterolaemia ($P<0.05$) and combined hyperlipidaemia ($P<0.05$), compared to those in the Control group, as shown in Table 7.7. The mean HDL-cholesterol concentration was significantly lower in the Case group, especially for women. Similar glucose concentrations were observed between the Case and Control groups, except for the higher concentration in the male subjects in the Case group, compared to their control counterparts.

Table 7.6: Serum lipid and glucose measurements of the Case and Control groups

	CASES (n = 93)					CONTROLS (n = 189)				
	n	Mean ± SEM	Minimum	Median	Maximum	n	Mean ± SEM	Minimum	Median	Maximum
Total cholesterol (mmol/L)										
Total	93	5.4 ± 0.1	3.2	5.3	8.7	189	5.4 ± 0.1	3.1	5.3	8.4
Men	62	5.3 ± 0.1	3.6	5.3	8.7	112	5.2 ± 0.1	3.1	5.2	7.6
Women	31	5.8 ± 0.2	3.2	5.5	8.1	77	5.6 ± 0.2	4.0	5.4	8.4
HDL-cholesterol (mmol/L)										
Total	93	1.20 ± 0.03 *	0.63	1.16	1.91	189	1.30 ± 0.02	0.56	1.29	2.52
Men	62	1.15 ± 0.03	0.63	1.13	1.78	112	1.20 ± 0.03	0.56	1.18	2.52
Women	31	1.30 ± 0.05 *	0.94	1.33	1.91	77	1.44 ± 0.04	0.92	1.42	2.46
LDL-cholesterol (mmol/L)										
Total	93	3.5 ± 0.1	1.9	3.4	6.4	189	3.4 ± 0.1	1.8	3.3	6.5
Men	62	3.5 ± 0.1	1.9	3.3	6.4	112	3.3 ± 0.1	1.8	3.2	6.3
Women	31	3.6 ± 0.2	1.9	3.4	5.3	77	3.6 ± 0.1	2.0	3.6	6.5

*Significantly different from the Control group: *, P<0.05.*

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Table 7.6: Serum lipid and glucose measurements of the Case and Control groups (continued)

	CASES (n = 93)					CONTROLS (n = 189)				
	n	Mean ± SEM	Minimum	Median	Maximum	n	Mean ± SEM	Minimum	Median	Maximum
Triglyceride (mmol/L)										
Total	93	1.6 ± 0.1	0.5	1.3	4.0	189	1.4 ± 0.0	0.5	1.3	3.6
Men	62	1.6 ± 0.1	0.5	1.4	4.0	112	1.5 ± 0.1	0.5	1.3	3.8
Women	31	1.5 ± 0.1	0.7	1.2	3.2	77	1.3 ± 0.1	0.6	1.2	2.7
Glucose (mmol/L)										
Total	93	5.5 ± 0.2	3.1	5.0	15.6	189	5.2 ± 0.1	3.0	5.0	15.7
Men	62	5.5 ± 0.2 *	3.1	5.1	13.9	112	5.1 ± 0.1	2.9	5.0	10.0
Women	31	5.5 ± 0.3	3.8	4.8	15.6	77	5.3 ± 0.2	3.7	5.0	15.7

*Significantly different from the Control group: *, P<0.05.*

Table 7.7: Prevalence of hypercholesterolaemia and hypertriglyceridaemia between groups

	CASES		CONTROLS	
	n	%	n	%
Serum total cholesterol ≥ 5.5 mmol/L	36	38.3	80	42.2
Serum total cholesterol ≥ 6.5 mmol/L	15	16.0	24	12.7
Serum total cholesterol ≥ 7.8 mmol/L	5	5.3 *	1	0.5
Serum triglyceride ≥ 2.3 mmol/L	12	12.8	14	7.4
Combined hyperlipidaemia	11	13.8 *	7	4.6

Combined hyperlipidaemia: serum total cholesterol ≥ 6.5 mmol/L and triglyceride ≥ 2.0 mmol/L.

*Significantly different from the Control group: *, P<0.05.*

7.3.5 Serum Lipid and Glucose Concentrations and CHD Risk Factors

Table 7.8 presents results of univariate correlation analyses of serum lipid and glucose concentrations and selected nutrient intakes. It was found that dietary cholesterol intake was positively correlated with serum cholesterol concentration for the Case group only, and there were no other significant relationships observed with other nutrient intakes. For the Control group, dietary cholesterol intake was positively associated with serum LDL-cholesterol, while the intakes of MUFAs and PUFAs were positively correlated with serum cholesterol.

Table 7.8: Spearman correlation coefficient (r_s) describing the relationships between serum lipid and glucose measurements, and dietary fat intakes (g/d)

Serum concentration (mmol/L)	CASES (n = 93)				CONTROLS (n = 189)			
	Cholesterol	SFAs	MUFAs	PUFAs	Cholesterol	SFAs	MUFAs	PUFAs
Total cholesterol	0.21*	0.12	0.16	0.15	0.13	0.12	0.14 *	0.14 *
LDL-cholesterol	0.15	0.05	0.08	0.05	0.14 *	0.09	0.13	0.11
HDL-cholesterol	0.02	0.07	0.06	0.08	0.09	0.10	0.13	0.07
Triglyceride	0.16	0.05	0.08	0.03	0.01	0.02	0.02	0.05
Glucose	–0.07	–0.07	–0.16	–0.16	–0.03	0.00	0.02	0.04

SFAs: saturated fatty acids, MUFAs: monounsaturated fatty acids; PUFAs: polyunsaturated fatty acids.

*Significantly different from zero: *, P<0.05.*

7.3.6 PREDICTORS FOR CHD

To determine the predictive power for CHD of serum lipid and glucose concentrations, logistic regression analyses were performed. Table 7.9 shows the distribution of subjects, according to the quartiles of serum HDL-cholesterol, systolic and diastolic blood pressures. More subjects in the Case group were in the lowest quartile of HDL-cholesterol concentrations, compared to the Case group (38% vs 20%). About 31% of the controls and 49% of the cases had serum HDL-cholesterol below 1.05 mmol/L, and 24% of the controls and 12% of the cases had serum HDL-cholesterol above 1.45 mmol/L. The odds ratio for subjects with a HDL-cholesterol concentration in the highest quartile (above 1.45 mmol/L), compared to those in the lowest quartile (below 1.05 mmol/L) was 0.3 (95% CI 0.1–0.8).

Table 7.9: Distribution of subjects in the Case and Control groups according to quartiles of serum HDL-cholesterol concentration

	CASES		CONTROLS	
	n	%	n	%
Serum HDL-cholesterol				
1st Quartile (<1.05 mmol/L)	34	38	39	20
4th Quartile (>1.45 mmol/L)	18	20	56	29
Systolic blood pressure				
1st Quartile (<110 mmHg)	19	17.6	56	25.5
4th Quartile (>140 mmHg)	52	48.2	23	23.6
Diastolic blood pressure				
1st Quartile (<70 mmHg)	20	18.5	52	23.6
4th Quartile (>90 mmHg)	45	42.7	32	15.5

There were more subjects in the Case group who had systolic blood pressures over 140 mmHg (highest quartile). Moreover, 26% of the controls, compared to 18% of the cases had systolic blood pressures below 110 mmHg, while 48% of the CHD

cases had systolic blood pressures above 140 mmHg, compared to 24% of the controls (*P<0.0001*). The odds ratio for subjects who had systolic blood pressures in the highest quartile (above 140 mmHg), compared to those in the lowest quartile (below 110 mmHg) was 1.4 (95% CI 1.2–1.8). Similarly, the odds ratio for subjects who had diastolic blood pressures in the highest quartile (above 90 mmHg), compared to those in the lowest quartile (below 70 mmHg) was 1.5 (95% CI 1.2–1.9).

Multivariate logistic regression analyses were also performed, and the variables included in the model were serum total cholesterol, LDL- and HDL-cholesterol, triglyceride and glucose concentrations for nutritional biomarkers, and anthropometric measurements including body weight, height, BMI, triceps, biceps, subscapular and supra-iliac skinfold thicknesses. As it was observed earlier that physical activity and stress levels were significantly different between the Case and Control groups, they were included in the logistic regression model.

Multivariate logistic regression analyses showed that serum HDL-cholesterol concentration, along with physical activity and stress level, were found to be predictors for CHD for the whole study population. However, when analyses were made separately for men and women, it was found that only HDL-cholesterol was a predictor for women, and glucose concentration was for men (Table 7.10).

Table 7.10: Odds ratio (95% CI) of CHD by serum lipid and glucose concentrations

	Total	Men	Women
Model 1			
HDL-cholesterol	0.3 (0.1-0.8) *	NA	0.2 (0.04 – 0.99) *
Glucose	NA	1.3 (1.0 – 1.7)*	NA
Model 2			
HDL-cholesterol	0.3 (0.1-0.9) *	NA	0.2 (0.04 – 0.99) *
Glucose	NA	1.3 (1.0 – 1.7)*	NA
Physical activity	0.4 (0.2 – 0.8)	NA	NA
Stress	2.3 (1.3 – 4.2)	NA	NA

Variable entered into model 1 include serum total, LDL- and HDL-cholesterol, triglyceride and glucose concentrations.

Variable entered into model 2 include those in model 1 plus age, smoking, physical activity and stress level.

*Significantly different from the odds ratio of one: *, P<0.05.*

Table 7.11 shows results when anthropometric measurements were included in the logistic regression model. HDL-cholesterol was the only predictor of CHD for the total population, but none of the variables were found to be predictors of CHD for the male subjects. Amongst women, it was observed that higher HDL-cholesterol concentration (like in other statistical analyses performed earlier) and increased height are protective factors for CHD.

Table 7.11: Odds ratio (95% CI) of CHD by biochemical and anthropometric measurements

	Total	Men	Women
Serum HDL-cholesterol	0.3 (0.1 – 0.8) *	NA	0.1 (0.0 – 0.7) *
Height	NA	NA	0.9 (0.8 – 0.98) *
Systolic blood pressure	3.2 (1.7 – 6.2)**	3.8 (1.6 – 9.8)**	2.9 (1.1 – 7.9)*
Diastolic blood pressure	5.1 (2.6 – 9.9)***	5.8 (2.3 – 9.8)***	3.9 (1.4 – 10.1)**

Variables entered into model include serum total, LDL- and HDL-cholesterol, triglyceride and glucose concentrations, body weight, height, systolic, diastolic blood pressure.

NA: data not available as the variable was removed from the model (the significant level was higher than 0.15).

*Significantly different from the odds ratio of one: *, P<0.05; **, P<0.01; ***, P<0.001.*

When physical activity and stress level were added to the variables already in the model (adjusting for age and smoking), it was found that serum HDL-cholesterol, physical activity and stress level remained predictors of CHD in the total population (Table 7.12). When men and women were analysed separately, serum glucose and stress level were risk factors for CHD, while physical activity was a protective factor against CHD in men. And for women, increased HDL-cholesterol and less stress appeared to protect the subjects against CHD. No other variables were found to be predictors.

Table 7.12: Odds ratio (95% CI) of CHD by serum lipid and glucose concentrations and anthropometric measurements

	Total	Men	Women
HDL-cholesterol	0.3 (0.1 – 0.8)	NA	0.2 (0.0 – 0.9) *
Glucose	NA	1.3 (1.0 – 1.7) *	NA
Physical activity	0.4 (0.2 – 0.8) **	0.3 (0.1 – 0.8) *	NA
Stress level	2.3 (1.3 – 4.2) **	2.9 (1.2 – 6.5) *	3.1 (1.2 – 8.2) *

Variable entered into model include serum total, LDL- and HDL-cholesterol, triglyceride and glucose concentrations, body weight, height, BMI, biceps, triceps, subscapular and supra-iliac skinfold thicknesses, physical activity and stress level. Age and smoking status are adjusted for.

*Significantly different from the odds ratio of one: *, P<0.05; **, P<0.01.*

NA: not available as the the variable was removed ferom the model, and the significant level was higher than 0.15

7.4 DISCUSSION

7.4.1 BODY COMPOSITION

No significant differences were found in all anthropometric measurements between the Case and Control groups in the present study. Furthermore, the anthropometric measurements, except for height, failed to be predictors for CHD. Although BMI was positively associated with some CHD risk factors such as triglycerides, and inversely related to serum HDL-cholesterol, BMI failed to enter into the model in the logistic regression analysis. These results confirm those reported by other studies, where no statistically significant association was found between some anthropometric measurements with CHD (Paffenbarger, et al., 1986; Stevens et al., 1998). Positive associations were reported in some case-control and prospective studies between excessive BMI or body weight and CHD (Kim et al., 2000; Rosengren et al., 1999; Dowse et al., 1991; Peiris et al., 1991).

Nevertheless, the associations of CHD risk with excessive body weight or obesity and its possible independent role, is still open to debate. There has been suggestion that increased risk amongst the obese might depend primarily on the influence of the associated risk factors and not on elevated body weight per se (Tavani et al.,

1997). Previous studies have confirmed correlations between obesity and risk factors for CHD (DeFronzo & Ferrannini, 1991).

No relationship was found between overweight and CHD events in this study. The low prevalence of overweight and obesity amongst subjects in the present study may explain the inconsistency data in this study compared to other studies. The WHO established the MONICA Project in the early 1980s in many Centres around the world to MONItor trends in CArdiovascular diseases and to relate these to risk factor changes in the population over a ten-year period. From the WHO MONICA Project, it was found that the sensitivity of measurement to identify subjects with overweight or obesity was generally lower in populations in which overweight was relatively uncommon {Molarius, Seidell, et al. 1999 92 /id}. The prevalence of overweight in the present study was 23%, compared to 13% from a study in Metropolitan Jakarta Indonesia (Boedi-Darmojo, 1990), while obesity was less prevalent in the present study (1%), compared to 5% in Jakarta.

Although the odds ratio was low (OR 0.9, 95% CI 0.8–0.98), the results suggest that, in women, increasing height was protective against CHD. Forsen et al. reported that height was inversely related to fatal CHD and non-fatal CHD, and these relationships persisted after other major cardiovascular risk factors were adjusted for (Forsen et al., 2000). Lower stature reflected in small size at birth is linked to an increased risk of coronary heart disease and its major biological risk factors. This has led to the hypothesis that coronary heart disease originates in utero through the persistence of adaptation made by the fetus in response to undernutrition during specific stages of gestation (Barker et al., 1989).

7.4.2 Blood Pressure

The present study showed that high systolic and diastolic blood pressures increased the risk of CHD. Some studies have demonstrated a strong association between elevated blood pressure and CHD. In a follow-up study of the original Framingham Study subjects and their offspring, it was observed that hypertension increased the risk of developing CHD by three fold (Levy et al., 1996). The mechanisms by which high blood pressure is causative for CHD have been established. It is related to the response to injury hypothesis (Ross, 1985). An increased blood pressure may place higher stress on the arterial wall, which may result in endothelial damage, initiating atherosclerosis. It is also possible that atherosclerotic changes in the arterial wall

result in an elevation of blood pressure. Therefore, there may be synergy between atherosclerosis and hypertension.

7.4.3 SERUM LIPID AND GLUCOSE CONCENTRATIONS

Serum total cholesterol failed to be a predictor for CHD in the present study, although more subjects in the Case group were hypercholesterolemic, compared to those in the Control group. Hypercholesterolaemia has been recognised as a major risk factor for CHD for several decades. Findings from many epidemiological studies showed that the risk of CHD increased sharply when the total cholesterol concentration exceeded 6.5 mmol/L (Gouldbourt & Medalie, 1979; Kannel et al., 1971; Nobili et al., 1994; Verschuren et al., 1995). However, the present study found that the only difference in total cholesterol between the two groups, were amongst those who had total cholesterol exceeding 7.8 mmol/L. It has been reported that there is a moderate and not always clear relation with CHD events, for total cholesterol concentrations between 5.2 and 6.5 mmol/L (Deslypere, 2000; Kannel et al., 1971; Pooling Project Research Group, 1978).

The present study consistently showed that serum HDL-cholesterol was an independent factor for CHD events. In univariate analyses, the odds ratio of CHD for those in the lowest quartile of HDL-cholesterol, compared to those in the highest quartile, was 0.7 (95% CI, 0.5–0.9). Again, in multivariate logistic regression analyses, HDL-cholesterol was found to be a significant predictor of CHD event, both before and after controlling for other CHD risk factors. The results confirmed that increased HDL-cholesterol concentration is protective against CHD. Many studies have reported that low HDL-cholesterol is associated with CHD, even without elevated total or LDL-cholesterol concentrations (Assmann et al., 1998; Castelli et al., 1986; Wilt et al., 1997). In addition, the ratio of total and HDL-cholesterol has been reported to be associated with CHD (Brochu et al., 2000). However, other studies found that mortality was not increased in subjects who had isolated low HDL-cholesterol, compared to those with both low HDL-cholesterol and high total cholesterol (Goldbourt et al., 1997).

The reverse cholesterol transport (RCT) has been thought to be involved in the metabolism and anti-atherogenic function of HDL (von Eckardstein et al., 2001). HDL helps to mediate the efflux of cholesterol from non-hepatic cells and to deliver it to the liver and steroidogenic organs, where it is used for the synthesis of lipoproteins, bile acids, vitamin D, and steroid hormones (Genest, 1999; Stein and

Stein, 1999). Distortion of RCT can favour the deposition of cholesterol within the arterial wall, andtherefore promote the development of arteriosclerosis (Glomset,1998). Apart from HDL-cholesterol, some studies have suggested that triglycerides and LDL-cholesterol are independent predictors for CHD events. (Assmann et al., 1998; Packard et al., 2000). The present study, however, failed to confirm this observation.

No difference was found in the mean concentration of serum glucose between the Case and Control groups. But it was found to be a significant predictor for CHD, especially for men. After controlling for other CHD risk factors, higher glucose concentration increased the risk for CHD, with the odds ratio of 1.3 (95% CI, 1.0–1.7). Epidemiological data supported by three large prospective surveys (Bjontorp, 1976; Depres et al., 1996) suggested a role of hyperglycaemia and hyperinsulinaemia as independent risk factors for CHD.

Comparisons of serum lipidconcentrations were made between subjects in the Control group of the present study and the Kitavan, a traditional Melanesian population and coconut consumer. It was found that male subjects in the present study had higher total and HDL-cholesterol and triglyceride concentrations, while the female subjects had lower total and LDL-cholesterol and triglycerides and higher HDL-cholesterol concentrations. In a study conducted by Boedi-Darmojo, it was found that about 13% of subjects recruited from the Metropolitan Jakarta were hyperlicholesterolaemia (total cholesterol ≥ 6.5 mmol/L) (Boedhi-Darmojo et al., 1990; Boedhi-Darmojo, 1993). This was comparable to the prevalence of 13% in the Controls and 16% in the Cases found in the present study.

7.5 CONCLUSION

Results of the present study confirmed and further quantified the association of increased CHD risk with higher blood pressure, lower HDL-cholesterol, higher fasting glucose, and lower stature. However, the present study failed to demonstrate an increase in CHD risk for those with higher total and LDL-cholesterol and triglyceride concentrations, and obesity, as indicated by increased body weight, BMI and skinfold thicknesses.

CHAPTER 8

Food Intervention with Coconut:

Food and Nutrient Intakes

8.1 INTRODUCTION

Historically, coconuts have been important foods throughout the tropical lowlands for thousands of years (Child, 1964; Piggott, 1964). Populations along the coastal area in Indian and Pacific oceans such as Madagascar, Sri Lanka, India, Philippines, Indonesia, and the Pacific islanders are high consumers of coconuts. In 1957, Keys et al. and then followed by Hegsted et al. (1965) demonstrated the quantitative relationship between saturated fat in the diet and serum cholesterol (Hegsted et al., 1965; Keys et al., 1957). Since then, coconuts have been regarded a major culprit for atherogenesis (Pehowich et al., 2000; Remla et al., 1991; Rodriguez-Vico et al., 1993; Van and Zilversmit, 1988; Van and Zilversmit, 1988). However, there is still only limited and inconclusive evidence about the effects of various food sources of saturated fat, such as saturated fat from coconut meat, milk and oil on cardiovascular morbidity and mortality (Hooper et al., 2001; Ravnskov, 1995; 1998 & 2000). Other saturated food such as chocolate has not been found to raise blood cholesterol levels or to increase the risk of CHD (Kelly et al., 2001).

A series of studies on some traditional populations who consume coconut food found that stroke and ischaemic heart disease were nearly absent among the populations, and that they have significantly lower serum cholesterol, lower diastolic blood pressure and leanness (Lindeberg et al., 1993; 1994; 1996; 1997a & 1997b; 1999; Ravnskov, 2000). Prior et al. (1981) concluded that there was no reason for populations whose dietary patterns include coconut foods to alter their diet in order to reduce coronary risk.

Focus group discussions (Chapter 4) showed that the consumption of coconut foods has been decreasing in some parts of West Sumatra. This study sought to understand what effect coconut consumption had on CVD risk in two semi-urban populations in West Sumatra.

This chapter describes the study methods, socio-economic characteristics of the subjects, the food and nutrient intakes and the correlations between food intakes. The effects of the coconut foods on intervention nutritional biomarkers and body composition are discussed in Chapter 9.

8.2 METHODS

8.2.1 STUDY POPULATION

The participants were recruited from two villages located in West Sumatra. Both were in a semi-urban area. Villagers from Nareh in the southern part of Padang Pariaman Municipality were assigned as the Intervention (Coconut) group. They were either fishermen (mainly men) or embroidered for a living (mainly women). The Control (Non-coconut) group was chosen from Kapalo Koto village, District of Pauh in Padang Municipality, where the villagers were, in the majority, farmers. Although the main occupation was different, both Pariaman and Padang were coastal and had similar food habits.

8.2.2 SELECTION OF PARTICIPANTS

A screening interview was conducted on individuals from the two villages who responded to the announcement to participate in the study. Invitations to participate in the study were disseminated by the village mayor, nurses and midwives of the two Community Health Centres and also by the word of mouth. Selection of study subjects was based on the following criteria: a) commitment to the program and adherence to the protocol, b) no health problems, c) cannot be or become pregnant. Forty-six participants were selected from Nareh, whereas in the control village 42 participants were selected.

8.2.3 CONDUCT OF THE STUDY

After the subjects agreed to participate in the study and before an interview took place, they were given an introductory information package in the Indonesian language, and the informed consent forms were signed. For the subjects in the Coconut group, all interviews and clinical examinations were done in the Community Health Centre of Southern Pariaman in Nareh. For the Non-coconut group (controls) the assessments were performed in the Community Health Centre of Pauh V in Kapalo Koto. All participants were invited to the Community Health

Centres. They were interviewed by two dietitians using Health and Demography questionnaires and Food Frequency Questionnaire (FFQ). Prior to blood collection, they were asked to fast overnight. All blood samples were collected by trained health professionals. Anthropometric, bio-impedance, blood pressure and electrocardiogram measurements were performed by a single examiner (the candidate) in order to eliminate inter-observer variation and to standardise measurements.

8.2.4 STUDY DESIGN

This study was designed as a culturally appropriate, community-based intervention model with special consideration to accessibility. The study was focused on the effect of coconut consumption to the whole food pattern. In the intervention village (the Coconut group), one raw coconut was distributed to one household every day for 6 weeks. They were asked to use the coconut provided with their other food materials as they used to consume. On the other hand, participants in the control village (the Non-coconut group) were asked to continue their usual diet.

In the second and the fifth weeks of the study, the candidate and the dietitians visited all participants. They were asked about their food intake in the last week. For the coconut group, questions were focused not only on their food intake, but also on coconut intake.

This study was approved by the Monash University Standing Committee on Ethics in Research Involving Humans in November 1998 (Project Number 98/458). It was also approved by the Socio-Political Office of West Sumatra, the Head of Community Health Centres of the two districts and at the community level by the inhabitants and their chiefs.

8.3 RESULTS

8.3.1 SOCIO-DEMOGRAPHIC CHARACTERISTICS OF STUDY SUBJECTS

A significant difference in mean age was not observed between the Coconut (mean 49.9 years, range 28–81 years) and the Control (50.3 years, 29–75 years old) groups. Table 8.1 shows the number of subjects by gender and their highest education level. Compared with the Non-coconut group, a higher proportion of subjects in the Coconut group were males (22% vs 7%), but this failed to reach significance

(p=0.054). More than 51% and 80% of the Coconut and Non-coconut groups respectively either never attended school or had been in elementary school.

Table 8.1: Distribution of subjects according to their gender and highest education level

	Coconut group		Non-coconut group	
	n	%	n	%
Gender				
Male	10	21.7	3	7.1
Female	36	78.3	39	92.9
Highest education level				
No formal education	3	6.5	13	31.0
Elementary school	21	45.6	21	50.0
Junior high school	7	15.2	5	11.9
Senior high school	12	26.1	3	7.1
College or university	3	6.5	0	0.0

8.3.2 LIFESTYLE

None of the women in both groups were smokers. Among the males, all three males in the Non-coconut group and 4 out of 10 subjects in the Coconut group were smokers. Most of the subjects in both groups were rarely or never under stress and had a moderate level of physical activity, as shown in Table 8.2 below.

Table 8.2: Distribution of subjects according to their stress and physical activity level

Lifestyle factors	Coconut group		Non-coconut group	
	n	%	n	%
Stress level				
Rarely or never under stress	45	97.8	39	92.9
Under a little stress	1	2.2	3	7.1
Physical activity level				
Low level	3	6.5	4	9.5
Moderate level	43	93.5	38	90.5

8.3.3 FOOD INTAKES

8.3.3.1 Food Intakes at Baseline

Before the food intervention, differences were not observed in total food consumption between the two groups (Table 8.3). Daily intake of total food (not including beverages) was 1273 g/d for the Coconut group and 1311 g/d for the Non-coconut group. Beverages, mainly tea and coffee, would have added 109 g/d and 74 g/d to total food intake for the Coconut and Non-coconut group, respectively. Some subjects did not report their intake of plain water.

Table 8.3: Comparison of total food intake (g/d) between the Coconut and Non-coconut group

	Coconut group	Non-coconut group
Number of subjects	43	40
Mean ± SD	1375 ± 314	1268 ± 426
Minimum	841	584
Median	1394	1140
Maximum	2402	3042

Table 8.4 shows the differences in the intakes of various food groups between the Coconut and Non-coconut groups. At baseline, subjects in the Coconut group consumed more cereal and beverages (mostly tea), and less soy foods, compared to those in the Non-coconut group. The intakes of other food groups were similar between the two groups. Only one subject in the Coconut group reported using coconut oil, whilst all other subjects in the present study regularly used palm kernel oil, instead of coconut oil.

In both groups, cereal was the major food group, accounting for 36 to 40% of total food intakes. Rice was the major type of cereal in the study population; it accounted for 56% and 63% of cereal intake in the Coconut and Non-coconut groups, respectively. Other major food groups were vegetables, fruits, coconut milk and fish.

Table 8.4: Comparison of food intakes at baseline between the Coconut and Non-coconut groups

	Coconut group			Non-coconut group		
	Mean ± SD	**Median**	**Range**	**Mean ± SD**	**Median**	**Range**
Fish	54 ± 20	51	20–114	51 ± 46	20	15–113
Meat	29 ± 41	16	1–73	31 ± 31	19	1–122
Egg	28 ± 18	23	1–70	31 ± 21	25	2–87
Coconut #	45 ± 19	40	23–94	39 ± 28	30	9–83
Palm oil	27 ± 10	24	6–51	30 ± 11	29	10–63
Soy and soy products	112 ± 83 *	102	3–299	167 ± 148	116	0–214
Legumes and nuts	31 ± 24	20	0–73	28 ± 24	21	0–100
Vegetables	254 ± 112	245	100–345	30 ± 103	210	67–408
Fruits	110 ± 41	89	20–195	97 ± 56	75	10–214
Dairy products	19 ± 25	3	0–100	14 ± 26	0	0–103
Beverages	100 ± 54 *	103	0–207	69 ± 52	71	0–200
Rice and other cereals	512 ± 86 *	499	357–658	467 ± 87	458	249–741
Sugar and sugar products	30 ± 17	27	2–58	17 ± 17	21	0–143

*Significantly different from the Non-coconut group: *, $P<0.05$.*

 # All coconut products except for coconut oil

Comparisons of animal and plant food intakes between the two groups are shown in Table 8.5. The ratio of animal to plant food (15:85) was observed in both groups.

Table 8.5: Comparison of animal and plant food intake (g/d) between groups

	Coconut group (n = 44)	**Non-coconut group (n = 43)**
	Mean ± SD	Mean ± SD
Plant food (g/d)	1062 ± 252	1084 ± 504
Animal food (g/d)	111 ± 61	113 ± 72

8.3.3.2 Food Intakes during the Food Intervention

8.3.3.2.1 Animal Foods

Significant differences were not found in animal food intakes between the Coconut and Non-coconut groups during the intervention phase. Within the coconut group, animal food intake increased significantly after three and six weeks during the intervention phase, mainly due to an increased intake of fish.

The fish group included fresh water fish, seafood, and shellfish. Fish intake after three weeks intervention increased in the Coconut group from 54 g/d to 66 g/d, and to 86 g/d by the end of the intervention. Whereas in the Non-coconut group, the average consumption was steady from 49 to 56 g/d.

Fifty-five per cent of subjects in the coconut group and 78% in the non-coconut group consumed less than 25 g/d fish. The most popular cooking methods for fish included deep fried with chillies and tomatoes (called "balado") , cooked with coconut milk, and boiled fish cooked with herbs and chillies.

b) Meat group (beef, lamb and chicken)

During the intervention phase, the average meat intake in the Coconut group was between 29 g/d in the first assessment to 25 g/d in the third assessment. Whereas in the Non-coconut group the average intake was from 32/d to 36 g/d. Sixty four per cent of the subjects in the Coconut group, and 63% in the Non-coconut group consumed less than 25 g/d.

The most popular cooking method for meat amongst the Coconut group was cooking with coconut milk. Deep-fried with chillies was the favourite cooking method amongst the Non-coconut group.

c) Egg group (chicken eggs, duck eggs, quail eggs)

The average egg intake was 28 - 35 g/d and 30 - 31 g/d for the Coconut and Non-coconut groups respectively during the intervention phase. Sixty-four per cent of subjects in the Coconut group, and 55 % in the Non-coconut group consumed less than 25 g/d.

Table 8.6: Consumption of selected food groups (g/d) during the food intervention

	Baseline	Week 3	Week 6
	Mean ± SD	**Mean ± SD**	**Mean ± SD**
COCONUT GROUP			
Animal foods	**111 ± 60**	**136 ± 60** [eh]	**156 ± 53** [ce]
Fish	54 ± 20	66 ± 25 [a]	86 ± 37 [bcde]
Meat	29 ± 41	28 ± 44	25 ± 26
Egg	28 ± 18	28 ± 19	35 ± 30
Plants foods	**1062 ± 251**	**987 ± 314**	**937 ± 241**
Legumes and nuts	32 ± 24	30 ± 23	30 ± 22
Soy and soy products	112[a] ± 83	123 ± 85	111 ± 84 [a]
Cereal	512 ± 86 [a]	496 ± 182 [a]	458 ± 101
Coconut #	45 ± 19	59 ± 30 [fg]	57 ± 23 [ag]
Vegetables	254 ± 112	191 ± 83	193 ± 80
Fruits	93 ± 37	88 ± 37	85 ± 31
Sugar	30 ± 17	29 ± 17	29 ± 18
Vegetable oil	28 ± 12	26 ± 15	23 ± 10
Beverages	100 ± 54 [b]	100 ± 52 [a]	101 ± 54 [b]

a: Significant differences from the Non-coconut group (P<0.05)

b: Significant differences from the Non-coconut group (P<0.01)

c: Significant differences from the Non-coconut group in the changes from Baseline (P<0.0001)

d: Significant differences from Week 3 within the group (P<0.01)

e: Significant differences from Baseline within the group (P<0.001)

f: Significant differences from the Non-coconut group (P<0.001)

g: Significant differences from Baseline within the group (P<0.05)

h: Significant differences from the Non-coconut group in the changes from Baseline (P<0.05)

 # All coconut products except for coconut oil

Table continues on next page

Table 8.6: Consumption of selected food groups (g/d) during the food intervention (continued)

	Baseline	Week 3	Week 6
	Mean ± SD	**Mean ± SD**	**Mean ± SD**
NON-COCONUT GROUP			
Animal Foods	**114 ± 71**	**120 ± 65**	**134 ± 80**
Fish	51 ± 46	49 ± 32	56 ± 39
Meat	32 ± 31	30 ± 27	36 ± 34
Egg	31 ± 21	31 ± 22	30 ± 21
Plants foods	**1084 ± 504**	**906 ± 292**	**997 ± 480**
Legumes and nuts	28 ± 24	24 ± 20	31 ± 25
Soy and soy products	167 ± 148	157 ± 123	181 ± 162
Cereal	473 ± 87	410 ± 127	399 ± 131
Coconut #	39 ± 28	36 ± 26	41 ± 29
Vegetables	261 ± 179	195 ± 95	223 ± 129
Fruits	105 ± 140	85 ± 47	119 ± 158
Sugar	26 ± 29	25 ± 29	27 ± 33
Vegetable oil	32 ± 16	28 ± 12	29 ± 12
Beverages	62 ± 54	66 ± 55	62 ± 55

a: Significant differences from the Non-coconut group (P<0.05)

b: Significant differences from the Non-coconut group (P<0.01)

c: Significant differences from the Non-coconut group in the changes from Baseline (P<0.0001)

d: Significant differences from Week 3 within groups (P<0.01)

e: Significant differences from Baseline within groups (P<0.001)

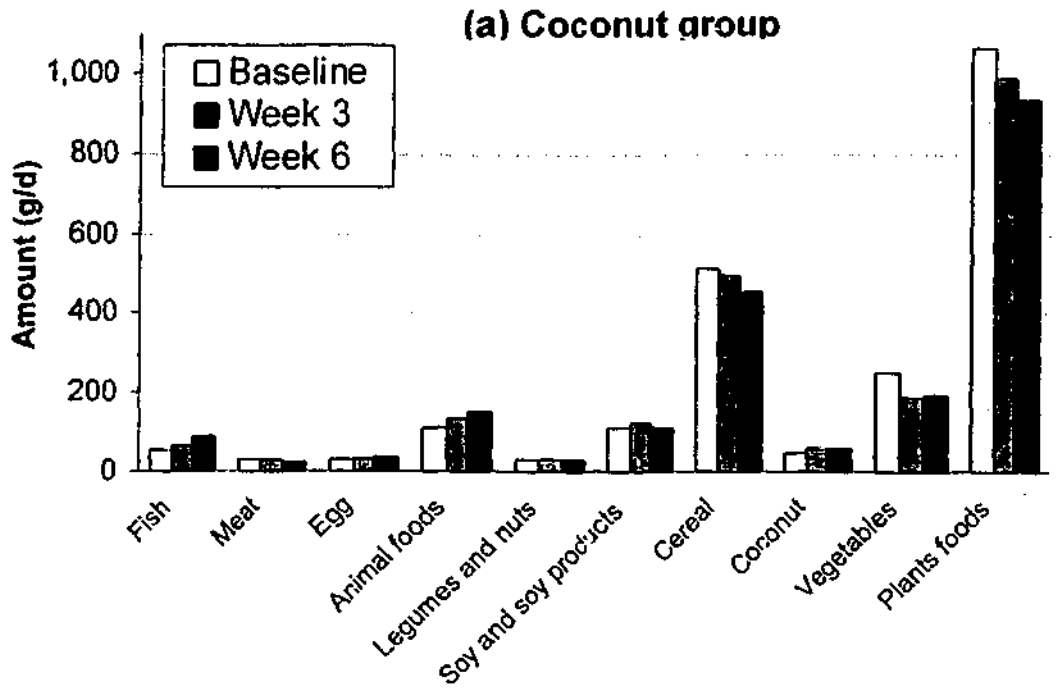
f: Significant differences from the Non-coconut group (P<0.001)

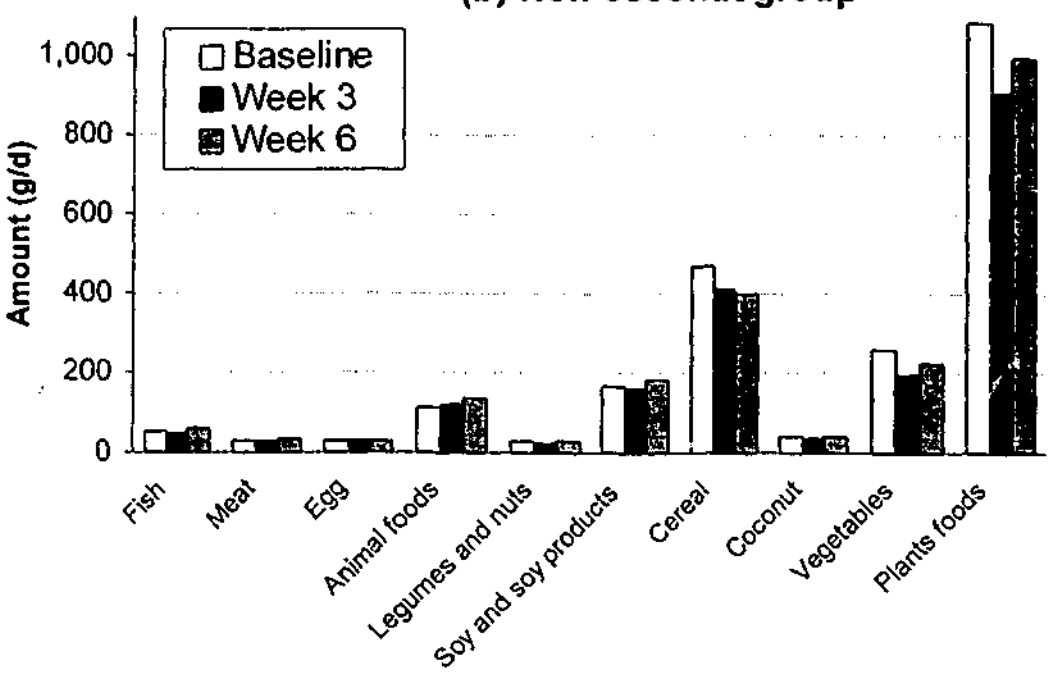
g: Significant differences from Baseline within groups (P<0.05)

h: Significant differences from the Non-coconut group in the changes from Baseline (P<0.05)

 # All coconut products except for coconut oil

Figure 8.1 Consumption of selected food groups (g/d) during the food intervention for the Coconut (a) and Non-coconut (b) groups





8.3.3.2.2 Plant Foods

The plant food group included legumes, nuts, soy products, vegetables, fruits, rice and cereals, and coconut milk.

a) Legumes and nuts

During the intervention phase, legume and nut consumption were similar between the two groups. It was found that 57% and 47% of subjects in the Coconut and Non-coconut groups, respectively, consumed less than 25 g/d legumes and nuts. Legumes and nuts were mainly boiled or deep-fried in both groups.

b) Soy and soy products

Prior and during the intervention phase, the Non-coconut group had a higher intake of tempeh and tofu compared to the Coconut group. In the sixth week, 49% of subjects in the Coconut group consumed less than 25 g/d, but 78% of subjects in the Non-coconut group consumed more than 75 g/d. No significant changes from baseline after 3 and 6 weeks were observed in the consumption of soy and soy products for both Coconut and Non-coconut groups.

c) Rice and cereal group

Before and during the intervention phase, subjects in the Coconut group had higher intakes of cereals. The average intakes were 458– 512g/d and 399–473g/d for the Coconut and Non-coconut group, respectively. More than 60% of subjects in both groups had more than 300g/d of rice and cereals. Rice contributed 58% of total cereal intake in both groups. Although there was a trend that both Coconut and Non-coconut groups consumed less cereals over the intervention period, no significant changes from baseline were observed.

d) Fruits group

At the end of the intervention, the Non-coconut group had a slightly higher intake of fruits, although not significant. Most of the subjects in both groups had fruit intake more than 75 g/d; 63% and 56% for Coconut and Non-coconut subjects respectively. The most popular fruits were bananas, oranges, papaya and watermelons, all of which were available all year round, whilst other fruits such as rambutan, mango and durian were seasonal.

e) Vegetables

During the intervention, differences were not observed in vegetable consumption between subjects in both groups. The average intake after 6 weeks was 85 g/d in

the Coconut group and 119 g/d in the Non-coconut group. Forty-six per cent of subjects in both groups had an average intake of more than 200 g/d. The most popular vegetables were swamp cabbage, cassava leaves and spinach, which were stewed or cooked with coconut milk.

f) Coconut products

Coconut is an important food ingredient in Minangkabau food culture (Chapter 2). Coconut-products used by the Minangkabau are coconut milk and coconut meat. During the intervention phase, coconut consumption significantly increased within the Coconut group, whereas no difference was found in the Non-coconut group. Average coconut intake after six weeks was 57 g/d and 41 g/d for the Coconut and Non-coconut group respectively ($p<0.05$). It was found that 67% of subjects in the Coconut group consumed more than 50 g/d, whereas 78% subjects in the Non-coconut group consumed less than 50 g/d. This difference in coconut intake was significant at $P = 0.002$.

g) Sugar and sugar products

Average intake of sugar and sugar products were similar in both groups. Most of the subjects in both groups (90%) consumed less than 50g/d sugar products.

h) Oils and spreads

Subjects in this study consumed mainly palm and coconut oils. They were used mainly for cooking. Other fat sources, such as other vegetable oils were never used; a small amount of spreads were used in both groups (0.25 and 0.43 g/d by subjects in the Coconut and Non-coconut groups, respectively). Most of the subjects in both groups consumed less than 25 g/d palm oil.

i) Beverages

Intakes of tea and coffee were low in both groups. Average daily intakes of tea and coffee were 68mL in the Coconut group and 53mL in the Non-coconut group. The total consumption of beverages remained unchanged during the intervention for both the Coconut and Non-coconut groups.

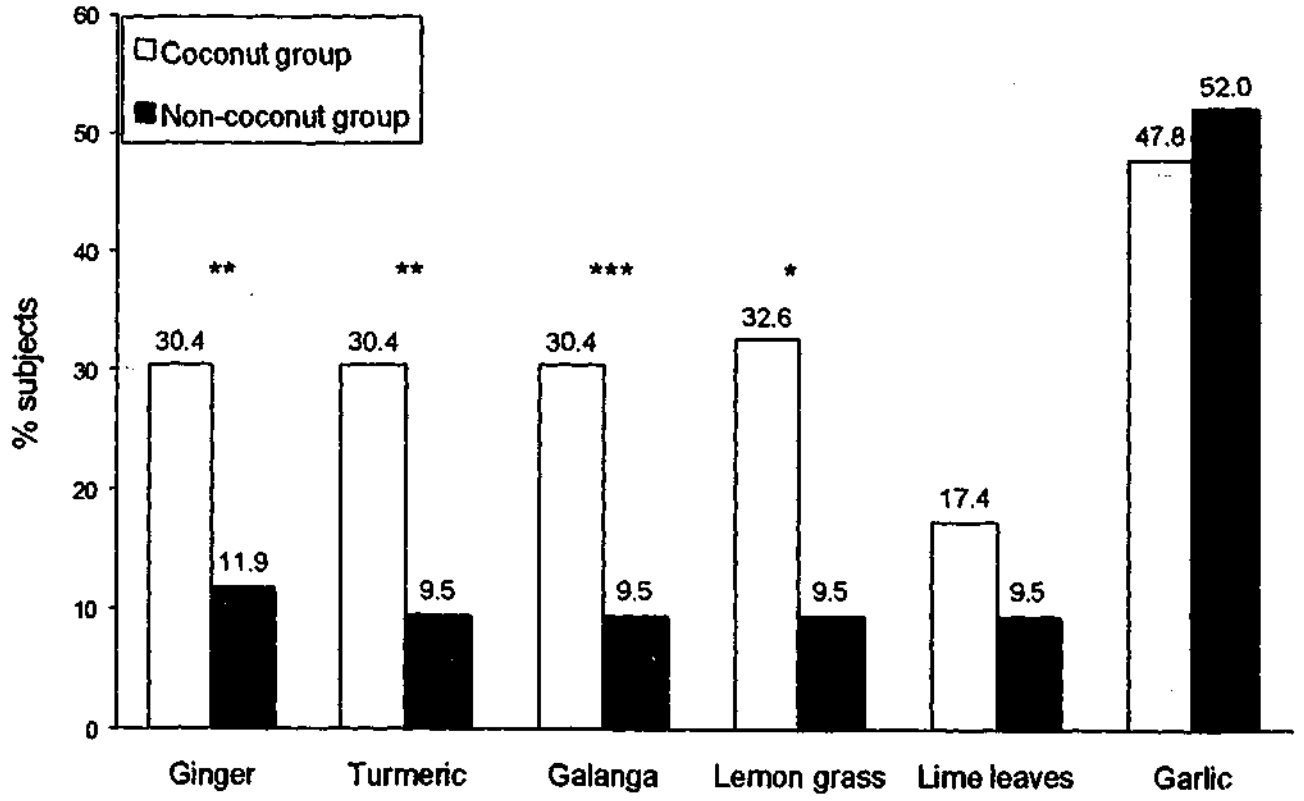
8.3.4 FOOD VARIETY

Differences were not found in weekly food variety scores between the two groups. The food variety score in the Coconut group was 24.6 and ranged from 19 to 31 and in the Non-coconut group the score was 24.5 and ranged from 14 to 30.

8.3.5 HERBS AND SPICES

The Coconut group had significantly higher intakes of herbs that were related to coconut milk dishes. Consumption of ginger, turmeric, galanga, onion, lemon grass, turmeric leaves, salam leaves and bay leaves were significantly higher in the Coconut group.

Figure 8.2: % subjects reporting use of selected herbs and spices at least once a day



*Significant differences between two groups: *, P<0.05; **, P<0.01; ***, P<0.001.*

8.3.6 NUTRIENT INTAKE PATTERN

8.3.6.1 Energy Intake

Although not significant, women in both groups had higher energy intakes than the men. In total, average energy intake was 2028 kcal/day, which is lower than the recommended daily intakes for adult Indonesians (2250 kcal for women and 2500 for men). A greater proportion of subjects in both groups (60%) had energy intakes between 1500-2500 kcal.

Table 8.7: Energy intake of the subjects by gender and groups

	Coconut group	Non-coconut group
Mean ± SD	2001 ± 471	1892 ± 518
Minimum	1142	781
Median	2009	1812
Maximum	3163	3097

8.3.6.2 Macronutrient Intakes

Table 8.8 shows macronutrient intakes in grams per day, percentage from total energy intake and nutrient density. Significant differences were not found in macronutrient intakes between the two groups. Cholesterol intake was 214 mg/d in the Coconut group and 220 mg in the Non-coconut group. Dietary fibre intake was low in both groups; 11 g/d and 10 g/d for the Coconut and Non-coconut groups respectively.

Table 8.8: Macronutrient intakes of the study population

	Amount (g/d)	% Contribution to total energy	Nutrient density
	Mean ± SD	Mean ± SD	Mean ± SD
COCONUT GROUP			
Carbohydrate	234.3 ± 45.7	56.6 ± 7.6	28.1 ± 3.0
Protein	98.2 ± 27.4	19.6 ± 2.4	11.7 ± 1.4
Fat	55.0 ± 22.9	23.8 ± 6.1	6.3 ± 1.6
NON-COCONUT GROUP			
Carbohydrate	209.9 ± 46.8	53.1 ± 6.8	27.2 ± 3.2
Protein	95.7 ± 30.1	20.8 ± 2.5	12.2 ± 1.5
Fat	61.0 ± 21.2	26.1 ± 5.0	6.9 ± 1.3

8.3.6.3 Protein Intake

Protein intake was not significantly different between the two groups. Protein intakes in this study were higher than the recommended intake. In both groups, more than 40% of total protein was from fish, and another 40% were from plant protein, such as soy, cereal, vegetables and legumes. In total, the ratio of animal to vegetable protein was 60:40 in both groups, as shown in Table 8.9.

Table 8.9: Percentage of animal and vegetable protein consumption

	Coconut group	Non-coconut group
	Mean ± SD	Mean ± SD
Animal protein (% of total protein)	59.7 ± 8.4	56.5 ± 8.8
Vegetable protein (% of total protein)	40.3 ± 7.6	43.5 ± 7.1

8.3.6.4 Total Fat

The Non-coconut group had a higher intake of total fat than the Coconut group. Daily fat intake of subjects in the Coconut group was 55 g/d, and 61 g/d in the Non-coconut group. Total fat intakes were 24% and 26% of total energy intake in the Coconut and the Non-coconut group respectively. More than 57% of subjects in the Coconut group consumed between 15 to 25% of total energy intake as fat, whilst 68% of the Non-coconut group consumed between 20 to 30% of total energy intake as fat. This difference in fat intake was significant ($P<0.05$). Table 8.10 shows food sources for total fat. The most important sources for fat in both groups were fish, soy, cereals, and vegetable dishes.

Table 8.10: Percentage contribution of food groups (as dishes) to dietary fat intake

	Coconut group	Non-coconut group
	%	%
Fish	30.0	26.9
Soy	15.3 ***	23.9
Rice and cereals	14.2 ***	10.2
Vegetables	12.1	11.6
Egg	6.9	8.8
Legumes and nuts	6.1	6.7
Meat	5.4	6.0
Dairy products	4.2	3.2
Fruit	2.5	2.0
Others	0.4	0.7

*Significantly different from the Non-coconut group: ***, P<0.001.*

8.3.6.4.1 Saturated Fat

Saturated fat accounted for 60% of total fat intake in the Coconut group and 58% in the Non-coconut groups. Daily mean intakes were 33 g/d in the Coconut group and 36 g/d in the Non-coconut group, which accounted for 14% and 15% of energy intakes respectively. As coconut is the main source of fat, lauric acid (C12:0) was the main contributor to saturated fatty acid intakes in both groups. Other fatty acid contributors, in order, were palmitic, myristic, stearic and short chain fatty acids with less than 10 C-atoms. In the Coconut group, most of the saturated fat

was from fish dishes - mostly deep fried fish in palm oil. In the Non-coconut group, the most important saturated fatty acid source was from soy dishes which included deep fried tempeh and tofu fried in palm oil.

Intake of lauric acid was significantly higher in the Non-coconut group due to their regular inclusion of deep fried dishes using palm oil. In contrast, the Coconut group consumed more coconut dishes, many of which replaced deep fried foods in palm oil. Total intake of coconut dishes in the Coconut group was 195 g/d and 145 g/d in the Non-coconut group ($P<0.01$).

8.3.6.4.2 Monounsaturated Fat

The mean intake of monounsaturated fat was 13 g/d in the Non-coconut group and 12 g/d in the Coconut group, which accounted for 6% and 5% of total energy intake respectively. Monounsaturated fat was 21% of total fat in both groups. The main contributor to monounsaturated fat in both groups was linoleic acid (C18:2); the consumption of linoleic acid was significantly higher in the Non-coconut group than the Coconut group. The most important food sources of MUFAs in both groups were from deep fried soy and fish dishes (deep fried in palm oil).

8.3.6.4.3 Polyunsaturated Fat

Percent total energy from PUFA was significantly higher in the Non-coconut group than the Coconut group. Daily mean intakes were 13 g/d in the Non-coconut group and 10 g/d in the Coconut group, which accounted for 5% and 4% of total energy intake respectively. PUFAs accounted for 18% and 20% of total fat intake for the Coconut and Non-coconut groups, respectively. More than 70% and 68% of PUFAs, in the Non-coconut group and the Coconut group, were contributed by soy and fish dishes. Top contributors for PUFAs in soy dishes were deep-fried and 'balado' tofu, whereas the top contributors in fish dishes were boiled and grilled fish. The main polyunsaturated fats consumed from these dishes were n-3 linolenic acid, EPA and DHA, as opposed to n-6 fatty acids.

Table 8.11: Dietary fat intakes as % of total energy

	Coconut group	Non-coconut group
	Mean ± SD	Mean ± SD
Saturated fat	14.3 ± 3.45	15.2 ± 2.65
Monounsaturated fat	5.06 ± 1.25	5.52 ± 1.04
Polyunsaturated Fat	4.44 ± 1.63 *	5.27 ± 1.60
P:S ratio	0.30 ± 0.01	0.34 ± 0.06

*Significance different from the Non-coconut group: * P<0.05*

P:S ratio = polyunsaturated fatty acid intake/saturated fatty acid intake

8.3.7 CORRELATION ANALYSES

Table 8.12 shows correlation between consumption of coconut products and some selective food groups. In the Coconut group, coconut intake was positively related to consumption of fish, chicken and vegetables. In contrast, in the Non-coconut group it was strongly associated with the consumption of meat, vegetables and fruits.

Table 8.12: Spearman correlation coefficient (r_s) describing relationships between coconut consumption and selected food groups

	Coconut group	Non-coconut group	Total
Fish	0.54 ***	0.35 *	0.48 ****
Soy	0.36 *	0.42 **	0.32 **
Chicken	0.42 **	0.38 *	0.39 ***
Meat	0.32 *	0.54 ***	0.32 **
Vegetables	0.71 ***	0.60 ***	0.65 ****
Fruits	0.27	0.69 ***	0.45 ****

*Significantly different from zero: *, P<0.05; **, P<0.01; ***, P<0.001; ****, P<0.0001.*

8.4 DISCUSSION

Dietary pattern in this population is unique. Both groups consumed low energy from fat (24% and 26% of total energy in the Coconut and the Non-coconut group respectively). On the other hand, 60% and 58% of the fat was from saturated fatty acid, which was mainly derived from coconut products (namely meat and milk) and palm oil. Although coconut consumption was higher in the Coconut group, intakes of saturated fatty acids were not different between the groups. Consumption of palm oil, which was higher in the Non-coconut group, contributed to the higher intake of saturated fat in this study population. The dietary cholesterol intake of both groups was low, as was their intake of polyunsaturated fatty acids. These were largely the long-chain highly unsaturated fatty acids from fish lipids.

After 6 weeks of the intervention study, with one additional raw coconut for each family, the Coconut group increased its consumption of coconut and fish. Intake of other food groups remained unchanged. However, within the Coconut group, subjects who had a high intake of coconut also had significantly higher intakes of fish and vegetable. In the Non-coconut group, consumption of coconut had a strong correlation with intakes of vegetables, fruits and meat. Thus if people in Indonesia had enough coconut, this may facilitate an increased intake of fish.

Coconut, in traditional populations, has always been consumed with fish (Lindeberg et al., 1994; Prior et al., 1981). In such populations, stroke and ischaemic heart disease are usually absent. In South Asia, such as India and Sri Lanka, where populations consume a low fat diet and large amounts of coconuts, consumption of coconut and coconut oil has not been correlated with CHD and CHD risk factors(Atukorala and Jayawardene, 1991; Kumar, 1997).

The Coconut group was also found to have consume more often of spices, such as curcumin (turmeric), ginger (*Zingiber officinale*), galanga (*Linguas gallanga)*, and some Minangkabau traditional herbs. Other spices commonly used by the Minangkabau include red chillies (capsaicin), onion (*Alium cepa*), ginger (*Allium sativum*), cloves (*Syzigium aromaticum*), cinnamon (*Cinnamommum zeylanicum*), pepper (*Piper nigrum*), nutmeg (*Myristica fragrans*), coriander (*linalool*). Several studies have consistently demonstrated these spice principles prevent oxidation of oils and fats, and inhibit lipid peroxidation(Shobana and Naidu, 2000; Nagababu and Lakshmaiah, 1992). Spices or aromatic vegetable products are used to impart flavour, aroma and taste to

foods, and also some have medicinal aspects (Van and Cox, 1994). Indonesia has been widely known as the source of spices for hundreds of years.

Vegetables and fruit intakes were correlated with coconut consumption in this study. Vegetables and fruits have been recognised to contribute to reduced risk of CVD (Ness and Powles, 1997). Vegetables and fruits are sources of a variety of nutrients, including vitamins, trace minerals, dietary fibre, and many other biologically active compounds. These phytochemicals can have complimentary and overlapping mechanisms of action, including antioxidant activity, decrease in platelet aggregation, alteration in cholesterol metabolism, and blood pressure reduction (Lampe, 1999).

Coconut has been used not only for food and drink, but it has also been part of life for people throughout the wet tropical lowlands for thousand of years. Indonesia has been suggested as the original home for the coconut (Child, 1964; Piggott, 1964). However, in the last 50 years there has been major concern about coconut consumption due to its saturated fatty acid content. But recent review papers began to question the recommendation to reduce saturated fat intake, as reducing intake of saturated fatty acids has little or no effect on CHD mortality (Ascherio et al., 1996; Hooper et al., 2001; Ravnskov, 1995; 1998 & 2000; Samuelson et al., 2001; Taubes, 2001).

8.5 CONCLUSION

The present study aimed to evaluate the effect of increasing coconut intake on the whole food pattern. No differences were found in individual characteristics or lifestyle between the Coconut and the Non-coconut groups. At baseline, although not significant, the Coconut group had a higher intake of coconut products, other than coconut oil. They also had significantly higher intakes of cereals, beverages, herbs and spices, but lower intakes of soy products. The Non-coconut group had a significantly higher fat intake, which was mostly contributed by deep fried tofu in palm oil. During the intervention study, the Coconut group increased the intake of coconut products (mainly coconut meat and coconut milk) and fish. The increased intake of coconut products was positively correlated with intakes of fish, vegetables, fruit and meat, showing the broader dietary intake of coconut consumption.

CHAPTER 9

Food Intervention with Coconut:

Coronary Heart Disease Risk Factors

9.1 INTRODUCTION

A growing body of literature suggests that medium chain triglycerides (MCTs), containing medium chain fatty acids (MCFAs) composed of chains of 8-14 carbon atoms, are preferentially oxidised as compared to long chain triglycerides (LCTs) containing long-chain fatty acids (LCFAs) (Bray et al., 1980; DeLany et al., 2000; Geliebter et al., 1983; Hill et al., 1989; Jiang et al., 1993; Papamandjaris et al., 2000; Seaton et al., 1986). The capacity of MCTs to be oxidised easily and also to increase endogenous oxidation of other fatty acids suggests that diets containing MCTs are less likely to lead to obesity. The most available food for MCTs is coconut. About 60% of the fatty acid composition of coconut are dominated by chains of less than 14 carbon atoms. Furthermore, epidemiological studies have shown that populations with high consumption of coconut tend to be very slim with a low risk of CVD (Lindeberg et al., 1994; 1996; 1997a & 1997b; 1999; Lindeberg and Lundh, 1993; Prior et al., 1981). These studies also showed that their consumption of fish was high.

Diets rich in fish have been reported to protect against cardiovascular disease. The beneficial effect is attributed to the n-3 fatty acids, eicosapentaenoic acid (EPA C20:5n-3), and docosahexaenoic acid (DHA C22:6n-3), for which the main dietary sources are fish. The imbalance in the ratio of n-6 to n-3 PUFAs (i.e. increased arachidonic acid to EPA ratio) in tissue membranes may promote production of thromboxane A2, a potent platelet aggregation agent and vasoconstrictor, leading to increased thrombosis tendency (Dolecek, 1992).

Analysis of platelet/plasma lipid fatty acids has been used as an indication of the quality of dietary fat intake (Zock et al., 1997). The serum /plasma lipid fatty acid profiles may be influenced by diet in the short-term, whereas a relatively longer period of time is required for the diet to influence the platelet phospholipid fatty acid compositions (Sinclair and Mann, 1996). This chapter describes the effect of coconut consumption in the food intervention study to anthropometric, body

composition and nutritional biochemistry measurements. Plasma phospholipid measurements are also presented.

9.2 RESULTS

9.2.1 SERUM LIPIDS AND GLUCOSE

9.2.1.1 Serum Cholesterol

At baseline, subjects in the Non-coconut group did not have a significant difference in total serum cholesterol concentration to those in the Coconut group. At the end of the study, the level was higher in the Non-coconut group. Although the cholesterol concentration increased in the second assessment and no difference from baseline was found in the third assessment for the Coconut group, there was no significant difference in the trend over the intervention period between the Coconut and Non-coconut groups. When hypercholesterolaemia was diagnosed as total cholesterol more than 5.5 mmol/l, the prevalence of hypercholesterolaemia was significantly higher in the Non-coconut group *(P=0.025)*.

HDL-cholesterol concentration was similar between the groups during the study. However, within the Coconut group, HDL-cholesterol concentration was significantly higher in the second assessment, but no other difference was found. Within the Non-coconut group, HDL-cholesterol concentration was increased significantly in the third assessment. Furthermore, no difference was found in LDL-cholesterol concentration between the groups during the study. Within the Coconut group, LDL-cholesterol concentration increased in the second assessment. The ratio of LDL/HDL-cholesterol, greater than 4, was found in 26% and 24% of subjects in the Coconut and Non-coconut groups respectively, but the prevalence decreased after the 6th week of the intervention.

9.2.1.2 Triglycerides and Lipoprotein (a)

Triglyceride concentrations were higher in the Non-coconut group in the first assessment. This difference, however, no longer existed after the intervention for 6 weeks, although no significant changes were observed in both groups.

When hypertriglyceridaemia was defined as triglyceride concentration ≥ 2.3 mmol/L, 4% and 10% of the subjects in the Coconut and Non-coconut group respectively, were found to be hypertriglyceridaemic. However, the prevalence decreased by the end of the study to 2% in the Coconut and 3% in Non-coconut groups. Combined hyperlipidaemia (cholesterol concentration ≥ 6.5 mmol/L and triglyceride concentration ≥ 2.0

mmol/L in a subject) was found in one subject in each group in the first assessment. In the third assessment, combined hyperlipidaemia was still found in one subject (2%) in the Coconut group, but none in the Non-coconut group.

9.2.1.3 Glucose and Insulin

Mean fasting serum glucose in the Non-coconut group at baseline was 5.9 mmol/L, which was significantly higher than the Coconut group ($P<0.01$). After three and six weeks of intervention, no difference was found between the first, second and third assessment in each group. No significant difference was observed between groups after the baseline values were adjusted for.

The prevalence of hyperglycaemia (fasting serum glucose $\geq$ 7.0 mmol/L, WHO, 1985) was found to be 6% and 18% at baseline, and 4% and 9% at the end of the intervention for the Coconut and Non-coconut groups, respectively.

No significant difference was found in insulin concentration at baseline between groups. However, the insulin concentration increased remarkably in the second and third assessments in the Coconut group. Such increase was not observed in the Non-coconut group.

Table 9.1: Serum lipid and glucose concentrations during the food intervention

	Baseline	Week 3	Week 6
	Mean ± SD	Mean ± SD	Mean ± SD
COCONUT GROUP			
Total cholesterol (mmol/L)	4.9 ± 0.1	5.2 ± 0.1 [c]	4.9 ± 0.2
HDL-cholesterol (mmol/L)	1.1 ± 0.0	1.2 ± 0.0 [d]	1.1 ± 0.1
LDL-cholesterol (mmol/L)	3.3 ± 0.1	3.4 ± 0.1 [c]	3.1 ± 0.12
LDL/HDL-cholesterol ratio	3.3 ± 0.2	3.0 ± 0.2	3.0 ± 0.2
Cholesterol/HDL-cholesterol ratio	4.9 ± 0.2	4.5 ± 0.2	4.6 ± 0.2
Lipoprotein (a) (mg/L)	100.4 ± 24.4	97.3 ± 22.0	100.2 ± 24.2
Triglyceride (mmol/L)	1.2 ± 0.1	1.2 ± 0.1	1.4 ± 0.1
Insulin (µmol/L)	10.1 ± 3.2	14.2 ± 2.8 [c]	20.2 ± 2.2 [d]
Glucose (mmol/L)	4.9 ± 0.26	5.2 ± 0.2	5.1 ± 0.3

a: Significant differences between groups at P<0.05

b: Significant differences between groups at P<0.01

c: Significant differences to the third assessment within the group at P<0.05

d: Significant differences to the first assessment within the group at P<0.01

e: Significant differences to the first assessment within the group at P<0.05

Table continues on next page.

Table 9.1: Serum lipid and glucose concentrations during the food intervention (continued)

	Baseline	Week 3	Week 6
	Mean ± SD	Mean ± SD	Mean ± SD
NON-COCONUT GROUP			
Total cholesterol (mmol/L)	5.3 ± 0.1	5.4 ± 0.1	5.4 ± 0.2 [a]
HDL-cholesterol (mmol/L)	1.1 ± 0.0	1.2 ± 0.0	1.2 ± 0.1 [d]
LDL-cholesterol (mmol/L)	3.6 ± 0.1	3.4 ± 0.1	3.4 ± 0.1
LDL/HDL-cholesterol ratio	3.5 ± 0.2	3.0 ± 0.2	3.0 ± 0.2
Cholesterol/HDL-cholesterol ratio	5.2 ± 0.2	4.6 ± 0.2 [a]	4.6 ± 0.2
Lipoprotein (a) (mg/L)	120.7 ± 24.4	108.9 ± 22.4	109.8 ± 23.8
Triglyceride (mmol/L)	1.5 ± 0.1 [a]	1.4 ± 0.1	1.6 ± 0.1
Insulin (µmol/L)	18.8 ± 3.2	15.1 ± 2.8	14.5 ± 2.1
Glucose (mmol/L)	5.9 ± 0.26 [b]	5.7 ± 0.2	5.4 ± 0.3

a: Significant differences between groups at P<0.05

b: Significant differences between groups at P<0.01

c: Significant differences to the third assessment within the group at P<0.05

d: Significant differences to the first assessment within the group at P<0.01

e: Significant differences to the first assessment within the group at P<0.05

Table 9.2: Prevalence (%) of hypercholesterolaemia, hypertriglyceridaemia and hyperglycaemia

	Coconut group		Non-coconut group	
	Baseline	Week 6	Baseline	Week 6
Hypercholesterolamia				
Serum cholesterol ≥ 5.5 mmol/L	24.4	17.0	43.9	39.0
Serum cholesterol ≥ 6.5 mmol/L	4.3	4.3	12.2	4.9
Serum cholesterol ≥ 7.8 mmol/L	0.0	0.0	1.0	1.0
Hyperglyceridaemia Serum triglyceride ≥ 2.3 mmol/L	4.3	2.3	9.8	2.6
Combined hyperlipidaemia*	2.3	3.2	2.4	0
LDL/HDL ratio > 4	25.5	6.4	23.8	7.1
Hyperglycaemia ≥ 7.0 mmol/L	6.4	4.3	18.6	9.3

* *Combined hyperlipidaemia: Serum cholesterol ≥ 6.5 mmol/L and serum triglyceride ≥ 2.0 mmol/L*

9.2.2 PLASMA PHOSPHOLIPID FATTY ACIDS

Table 9.3 shows plasma phospholipid fatty acid composition and its changes during the study. At baseline, the Coconut group had significantly higher phospholipid concentrations of myristic acid (C14:0), n-3 EPA (C20:5), total n-3 fatty acids and n-6/n-3 ratio, but lower concentrations of palmitic (C16:0) and n-6 homo γ linolenic acid (20:4) than the Non-coconut group. No significant changes in these fatty acids were observed in both groups after three and six weeks of the intervention, except for α-linoleic acid (C18:3n-3). The increasing of α-linoleic acid in third assessment from baseline was significantly higher in the Coconut group, compared to the Non Coconut group.

However, it was found that, during the last three week of the intervention, there was a significant increase in stearic (C18:0), linoleic (C18:2n-6), homo γ linolenic (20:3n-6), docosatetraenoic (C22:4n-6), α-linolenic (C18:3n-3) acids for the Coconut group. In addition, n-3 docosapentaenoic acid (C22:5n-3) at Week 6 was significantly higher than baseline. In contrast, there was a fall in homo γ linolenic (C20:3n-6), docosapentaenoic (C22:5n-6), and DHA (C22:6n-3), and an increase in α-linolenic (C18:3n-3) acid during the first three weeks of the intervention for the Non-coconut group. However, none of these changes existed in the following three weeks.

Table 9.3: Phospholipid fatty acid composition (% of total fatty acid) during the food intervention

	Baseline	Week 3	Week 6
	Mean ± SD	Mean ± SD	Mean ± SD
COCONUT GROUP			
C14:0	0.55 ± 0.05 [a]	0.50 ± 0.04 [a]	0.50 ± 0.03 [a]
C16:0	31.06 ± 0.34	31.98 ± 0.51	31.61 ± 0.29
C18:0	13.55 ± 0.31	13.55 ± 0.58	13.61 ± 0.3 [e]
Cis-C18:0	12.64 ± 0.28	12.85 ± 0.60	12.33 ± 0.4
Trans-C18:0	0.08 ± 0.00	0.07 ± 0.05	0.06 ± 0.01
Total SFAs	45.33 ± 0.36	46.19 ± 0.98	45.86 ± 0.24
C16:1	1.01 ± 0.11	0.98 ± 0.08	1.04 ± 0.06
C18:2n-6	20.94 ± 0.55	20.31 ± 0.88	21.39 ± 0.6 [f]
C20:4n-6	7.21 ± 0.29	7.15 ± 0.21 [c]	7.06 ± 0.2
C20:3n-6	2.76 ± 0.01	2.84 ± 0.01	3.06 ± 0.01 [e]
C22:4 n-6	0.29 ± 0.01	0.28 ± 0.02	0.29 ± 0.01 [e]
C22:5n-6	0.43 ± 0.03	0.43 ± 0.02 [b]	0.44 ± 0.02 [b]
Total n-6 fatty acids	31.64 ± 0.4	31.01 ± 0.9	32.24 ± 0.4
C18:3n-3	0.13 ± 0.01	0.12 ± 0.01	0.17 ± 0.01 [af g]
C20:5n-3	0.76 ± 0.05 [a]	0.86 ± 0.05 [d]	0.81 ± 0.06 [a]
C22:5n-3	0.62 ± 0.03	0.67 ± 0.02 [d]	0.70 ± 0.03 [ch]
C22:6 n-3	6.51 ± 0.227	6.95 ± 0.26 [d]	6.53 ± 0.26 [b]
Total n-3 fatty acids	8.00 ± 0.3 [a]	8.77 ± 0.3 [c]	8.31 ± 0.3 [b]
Total PUFAs	40.58 ± 0.38	39.65 ± 0.84	40.55 ± 0.38
DHA/EPA ratio	10.66 ± 0.27	10.35 ± 0.31 [a]	9.45 ± 0.31
n-6/n-3 ratio	3.73 ± 0.23 [a]	3.69 ± 0.23 [d]	4.07 ± 0.20 [b]

a: Significant differences between groups at P<0.05

b: Significant differences between groups at P<0.01

c: Significant differences between groups at P<0.001

d: Significant differences between groups at P<0.0001

e: Significant differences from Week 3 within the group at P<0.05

f: Significant differences from Week 3 within the group at P<0.01

g: Significant differences from Baseline within the group at P<0.05

h: Significant differences from Baseline within the group at P<0.01

Table continues on next page.

Table 9.3: Phospholipid fatty acid composition (% of total fatty acid) during the food intervention (continued)

	Baseline	Week 3	Week 6
	Mean ± SD	Mean ± SD	Mean ± SD
NON-COCONUT GROUP			
C14:0	0.38 ± 0.05	0.37 ± 0.04	0.40 ± 0.03
C16:0	32.51 ± 0.34 [b]	33.04 ± 0.52	32.12 ± 0.30
C18:0	13.83 ± 0.31	14.88 ± 0.59	13.99 ± 0.31
Cis-C18:0	12.93 ± 0.28	13.25 ± 0.62	13.04 ± 0.41
Trans-C18:0	0.07 ± 0.00	0.07 ± 0.01	0.07 ± 0.01
Total SFAs	46.19 ± 0.37	47.14 ± 0.77	46.54 ± 0.54
C16:1	0.94 ± 0.11	1.01 ± 0.08	0.98 ± 0.06
C18:2n-6	21.16 ± 0.55	21.29 ± 0.9	21.73 ± 0.60
C20:4n-6	3.37 ± 0.16 [b]	3.21 ± 0.15	3.53 ± 0.01
C20:3n-6	6.68 ± 0.29	5.90 ± 0.22 [g]	6.61 ± 0.3
C22:4 n-6	0.31 ± 0.01	0.31 ± 0.02	0.30 ± 0.01
C22:5n-6	0.39 ± 0.02	0.35 ± 0.02 [h]	0.37 ± 0.03
Total n-6 fatty acids	31.92 ± 0.40	31.07 ± 0.90	32.54 ± 0.50
C18:3n-3	0.13 ± 0.01	0.14 ± 0.01 [ag]	0.13 ± 0.01
C20:5n-3	0.56 ± 0.05	0.48 ± 0.05	0.59 ± 0.06
C22:5n-3	0.58 ± 0.03	0.52 ± 0.02	0.56 ± 0.03
C22:6 n-3	5.72 ± 0.27	4.78 ± 0.27 [h]	5.19 ± 0.28
Total n-3 fatty acids	7.43 ± 0.40	6.17 ± 0.40	6.87 ± 0.40
Total PUFAs	39.99 ± 0.28	38.48 ± 0.80	39.22 ± 0.64
DHA/EPA ratio	9.97 ± 0.42	9.32 ± 0.40	9.40 ± 0.45
n-6/n-3 ratio	4.59 ± 0.24	5.48 ± 0.27	5.05 ± 0.31

a: Significant differences between groups at P<0.05

b: Significant differences between groups at P<0.01

c: Significant differences between groups at P<0.001

d: Significant differences between groups at P<0.0001

e: Significant differences from Week 3 within the group at P<0.05

f: Significant differences from Week 3 within the group at P<0.01

g: Significant differences from Baseline within the group at P<0.05

h: Significant differences from Baseline within the group at P<0.01

9.2.3 ANTHROPOMETRIC MEASUREMENTS

9.2.3.1 Height

In the Coconut group, mean height for women was 147 cm and 162 cm for men, whereas in the Non-coconut group, mean height for women was 146 cm and 163 cm for men. Group differences were significant ($P<0.05$). It was found that 57% of subjects in the Coconut group compared to 71% in the Non-coconut group had heights <150cm.

9.2.3.2 Weight

No differences were found in mean weight of the subjects from the two groups throughout the study. Mean weight of the Coconut group was 53 kg in the first assessment and 55 kg in the third assessment, but the increase was of no significance. No change was observed in mean weight in the Non-coconut group from the first assessment to the third assessment.

9.2.3.3 Skinfold Thicknesses

Although not of significance, the skinfold thickness was less in subjects in the Coconut group, compared to that of the Non-coconut group. The average biceps skinfold thicknesses at baseline and study exit were 14.4 mm and 14.5 for the Coconut group, whilst those of the Non-coconut group were 16.6 mm and 16.1 mm. Similarly, the average triceps skinfold thicknesses of the subjects in the Coconut group were markedly lower than the Non-coconut group (19 mm vs 21 mm), subscapular skinfold (25mm vs 30 mm), and supra-iliac skinfold (21mm vs 25mm).

9.2.3.4 Abdominal Circumference

No differences were found in waist and umbilical circumferences between subjects in both groups. Waist circumferences of the Coconut group were 79 mm, 79 mm, and 78 mm compared to 81 mm, 81 mm and 80 mm for the Non-coconut group for the first, second and third assessments respectively. Furthermore, umbilical circumferences were 83 mm, 83 mm, and 82 mm for the Coconut group and 84 mm, 85 mm and 85 mm for the Non-coconut group.

Abdominal circumference of ≥ 100 cm can also be used to indicate the presence of abdominal adipose tissue (Pouliot et al., 1994), apart from BMI and AHR. Most of the subjects in both groups had abdominal circumferences < 100 cm.

Although not of significance, the average hip circumference of the Coconut group was smaller than the Non-coconut group. During the study, the range of the average hip circumference in the Coconut group was 91 mm to 92 mm, whereas in the Non-coconut group the range was between 93 mm to 94 mm.

Table 9.4: Anthropometric measurements during the food intervention period

	Baseline	Week 3	Week 6
	Mean ± SD	Mean ± SD	Mean ± SD
COCONUT GROUP			
Weight (kg)	53.3 ± 9.4	53.9 ± 9.5	54.7 ± 9.6
BMI (kg/m^2)	23.3 ± 4.1	23.6 ± 4.0	23.7 ± 3.8
Triceps skinfold thickness (mm)	18.2 ± 10.3	18.9 ± 7.7	19.3 ± 11.3
Biceps skinfold thickness (mm)	14.4 ± 10.3	14.1 ± 10.6	14.5 ± 10.3
Subscapular skinfold thickness (mm)	27.2 ± 14.1	24.9 ± 12.9	25.0 ± 12.5
Suprailiac skinfold thickness (mm)	21.7 ± 10.6	21.2 ± 11.0	20.7 ± 9.6
Waist circumference (cm)	79.1 ± 9.6	78.9 ± 9.7	77.5 ± 7.9
Umbilicus circumference (cm)	82.8 ± 10.9	83.3 ± 10.1	81.8 ± 9.5
Hip circumference (cm)	91.0 ± 7.6	91.6 ± 7.1	91.7 ± 6.8
NON-COCONUT GROUP			
Weight (kg)	53.4 ± 11.0	53.0 ± 10.6	52.3 ± 10.3
BMI (kg/m^2)	24.7 ± 4.8	24.5 ± 4.7	24.3 ± 4.8
Triceps skinfold thickness (mm)	20.5 ± 12.8	20.5 ± 12.7	21.2 ± 11.8
Biceps skinfold thickness (mm)	16.6 ± 10.4	16.8 ± 10.2	16.1 ± 10.7
Subscapular skinfold thickness (mm)	30.1 ± 15.6	30.3 ± 15.2	29.2 ± 15.7
Suprailiac skinfold thickness (mm)	24.8 ± 14.1	24.3 ± 13.1	24.4 ± 14.5
Waist circumference (cm)	81.2 ± 11.1	81.3 ± 9.9	79.7 ± 9.8
Umbilicus circumference (cm)	84.3 ± 11.0	85.4 ± 11.0	84.5 ± 11.0
Hip circumference (cm)	93.3 ± 10.3	93.5 ± 9.3	93.1 ± 9.2

9.2.4 PREVALENCE OF OBESITY

9.2.4.1 Body Mass Index

Along with weight, body mass index was similar during the study in both groups. In the first assessment, the prevalence of subjects who had BMIs < 23 kg/m² was 67% in the Coconut group compared to 46% in the Non-coconut group. In the third assessment 4% of subjects in the Coconut group had BMIs > 30 kg/m² compared to 12% in the Non-coconut group. However, no differences were found in the prevalence of obesity by BMI.

Table 9.5: Prevalence of obesity by BMI

	Coconut group			Non-coconut group		
	Baseline	Week 3	Week 6	Baseline	Week 3	Week 6
BMI <22.9 kg/m² (%)	59.6	61.7	63.8	46.3	48.8	53.7
BMI 23.0 –24.9 kg/m² (%)	14.9	10.6	12.8	14.6	14.6	12.2
BMI 25.0–29.9 kg/m² (%)	19.2	23.4	19.2	22.0	17.1	22.0
BMI ≥ 30.0 kg/m² (%)	6.4	4.3	4.3	17.1	19.5	12.2

9.2.4.2 Central Obesity

Unlike BMI, there was no cut-off point for abdominal-hip ratio (AHR). However, it was found that AHRs ≥ 0.95 for men and ≥ 0.85 for women, were associated with abnormal glucose and lipid levels (Bjontorp, 1992; Depres, 1992; Sinclair and Mann, 1996). None of the male subjects in both groups had AHRs ≥ 0.95. However, 39% and 62% of female subjects in the Coconut and Non-coconut groups respectively had higher AHRs than normal.

Table 9.6: Abdominal obesity according to the abdominal–hip ratio *

	Coconut group		Non-coconut group	
	Normal	Obese	Normal	Obese
Male	10 (100.0)	0 (0.0)	3 (100.0)	0 (0.0)
Female	22 (61.1)	14 (38.9)	15 (38.5)	24 (61.5)

** Abdominal obesity: abdominal-hip ratio ≥ 0.95 for men, and ≥ 0.85 for women.*

9.2.5 BLOOD PRESSURE

Table 9.7 shows the differences in blood pressure between the two groups during the study. Significant differences were not observed in systolic and diastolic blood pressures between groups during the intervention, as well as within the groups.

Table 9.7: Blood pressure measurements during the food intervention period

	Baseline	Week 3	Week 6
	Mean ± SD	Mean ± SD	Mean ± SD
COCONUT GROUP			
Systolic blood pressure (mmHg)	127.4 ± 24.6	121.0 ± 19.1	126.4 ± 25.5
Diastolic blood pressure (mmHg)	79.8 ± 11.8	77.4 ± 8.3	77.9 ± 9.6
NON-COCONUT GROUP			
Systolic blood pressure (mmHg)	126.4 ± 18.0	121.3 ± 19.6	115.5 ± 18.6
Diastolic blood pressure (mmHg)	77.7 ± 8.8	77.9 ± 8.2	74.8 ± 7.6

9.2.6 CORRELATION ANALYSES

Table 9.8 shows that BMI was positively associated with insulin and AHR, but was negatively association with HDL-cholesterol in the Coconut group. In the Non-coconut group, BMI had a negative association with age. AHR had a strong positive association with triglycerides and BMIs in all groups.

Table 9.8: Spearman correlation coefficient (r_s) describing relationships between body fatness indices (BMI and AHR) and age, serum lipid and glucose concentrations

	Coconut Group (n = 46)		Non-coconut Group (n = 42)		Total (n = 88)	
	BMI	AHR	BMI	AHR	BMI	AHR
Age (years)	–0.26	0.30	–0.38 *	0.04	–0.31 **	0.19
Total cholesterol (mmol/L)	–0.12	0.29	0.20	0.20	0.08	0.24 *
LDL-cholesterol (mmol/L)	0.08	0.28	0.15	0.13	0.14	0.22
HDL-cholesterol (mmol/L)	–0.34 *	-0.21	–0.01	–0.05	–0.17	–0.15
Triglyceride (mmol/L)	0.08	0.48 **	0.26	0.35 *	0.21	0.40 ***
Lp(a) (mg/L)	0.03	0.11	–0.18	–0.12	–0.07	–0.01
Insulin (µmol/L)	0.39 *	0.29	0.38	0.08	0.39 **	0.12
Glucose (mmol/L)	0.04	0.40 *	0.23	0.18	0.21	0.20

*Significantly different from zero: *, P<0.05; **, P<0.01; ***, P<0.001; ****, P<0.0001.*

BMI: body mass index; AHR: abdominal hip ratio

None of the selective food groups in Table 9.9 were significantly correlated with lipid and glucose measurements.

Table 9.9: Spearman correlation coefficient (r_s) describing relationships between selected food group intakes and serum lipid and glucose concentrations

Food group intake (g/d)	Coconut Group			Non-coconut Group		
	HDL/LDL ratio	Triglyceride	Glucose	HDL/LDL ratio	Triglyceride	Glucose
Fish	0.00	0.08	–0.00	0.05	0.06	–0.10
Meat	0.05	–0.07	–0.28	–0.00	0.04	–0.15
Egg	0.07	–0.05	–0.02	0.26	0.08	0.27
Fruits	0.06	0.08	–0.13	–0.17	0.09	–0.10
Vegetables	0.06	–0.14	–0.08	–0.13	0.07	–0.02
Coconut milk	–0.20	–0.23	–0.10	–0.02	0.19	0.08
Palm and coconut Oil	0.13	–0.16	–0.19	–0.05	0.10	–0.08

Table 9.10 shows that fish consumption was negatively correlated with arachidonic (C20:4n-6), docosatetraenoic (C22:4n-6), and total n-6 fatty acids, but was positively associated with EPA/AA ratio. Coconut consumption was negatively associated with arachidonic acid (C20:4n-6), docosatetraenoic (C22:4n-6), but was positively correlated with alpha-linolenic acid (C18:3n-3). Meat consumption was positively correlated with linoleic (C18:2n-6) as well as with α-linolenic (C18:3n-3) acids.

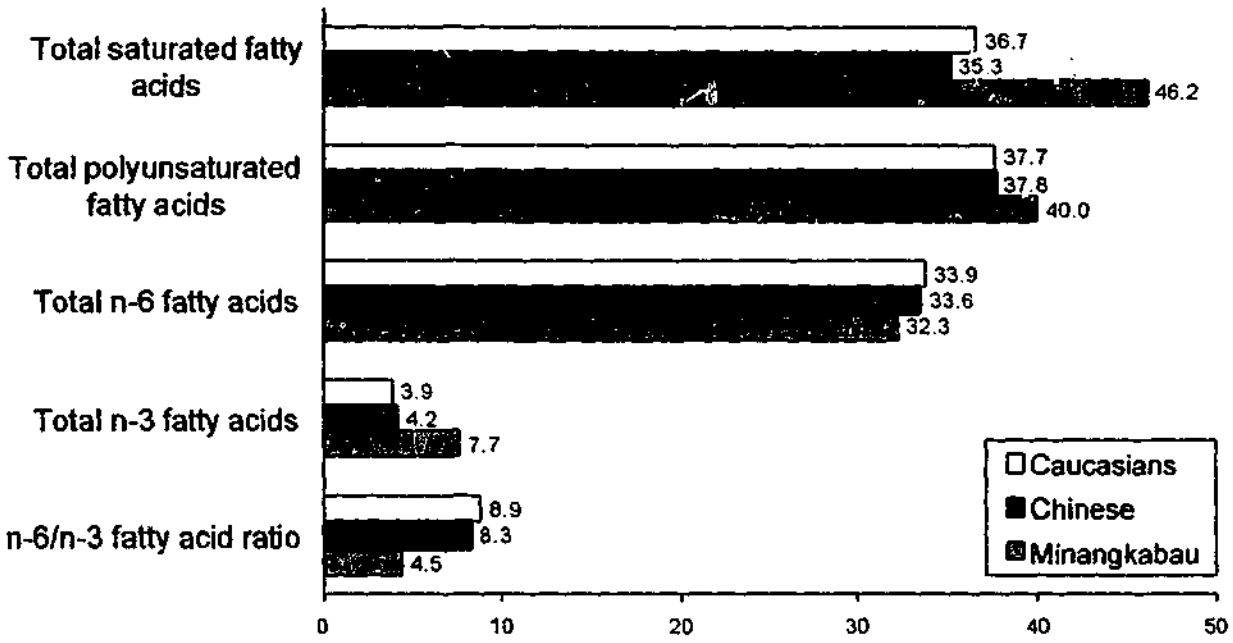
Table 9.10: Spearman correlation coefficient (r_s) describing relationships between selected food intakes (g/d) and individual phospholipid fatty acids (%)

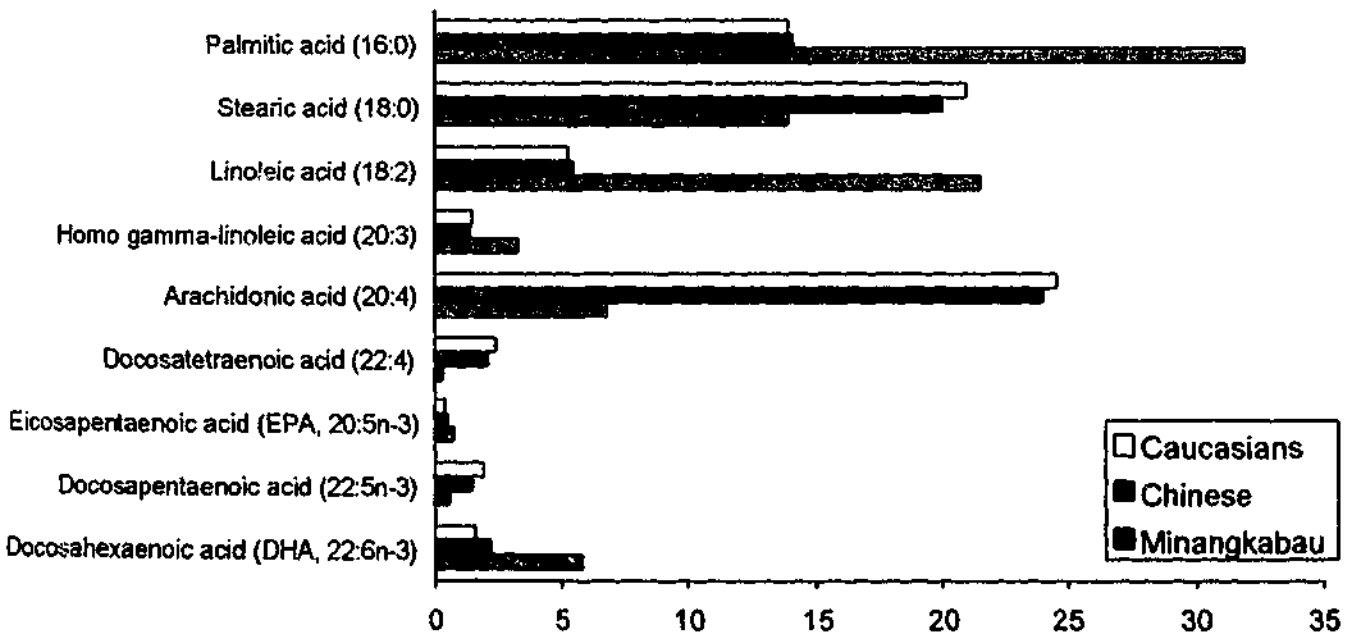
	Fish	Fish & coconut	Coconut	Meat	PUFA
C18:2n-6	−0.16	−0.03	0.13	0.32 *	−0.06
C20:3n-6	0.03	−0.16	−0.05	−0.22	0.14
C20:4n-6	−0.29 *	−0.26 *	−0.28 *	−0.04	−0.01
C22:4n-6	−0.29 *	−0.28 *	−0.28 *	−0.06	0.02
C22:5n-6	−0.10	−0.03	−0.12	−0.11	−0.03
Total n-6 fatty acids	−0.35 *	−0.27 *	−0.23	−0.06	−0.30 *
C18:3n-3	0.06	0.19	0.46 **	0.38 **	0.03
C20:5n-3	0.06	0.19	0.03	−0.2	−0.02
C22:5n-3	−0.13	−0.05	−0.23	−0.24	−0.09
C22:6n-3	−0.03	0.09	−0.01	−0.07	−0.05
Total n-3 fatty acids	−0.06	−0.00	−0.11	−0.28	−0.19
EPA/DHA ratio	−0.04	0.18	0.08	0.20	0.05
EPA/arachidonic acid ratio	0.08	0.27 *	0.22	-0.09	0.03

*Significantly different from zero: *, P<0.05; **, P<0.01.*

Figure 9.1 shows the differences in plasma phospholipid composition between the Minangkabau and the Melbourne Chinese and Caucasians. The Minangkabau plasma phospholipid composition was taken from the mean fatty acid composition of the second assessment in this study. The plasma phospholipids of the Melbourne Chinese and Caucasians were reported by Li et al. (2000).

Figure 9.1: Comparison of fatty acid profile (as % of total fatty acid) between the Minangkabau (in plasma phospholipids) reported in this Chapter, the Chinese (in platelets) and the Caucasians (in platelets), reported by Li et al. (2001)





Saturated fatty acid composition of plasma phospholipids of the Minangkabau was different to the Melbourne Caucasians. The Minangkabau had higher palmitic acid but lower stearic acid concentrations compared to the Melbournians. Moreover, polyunsaturated fatty acid composition of plasma phospholipids was different between the populations. The main source of n-6 fatty acids among the Minangkabau was linoleic acid, while arachidonic acid was the most important source of n-6 fatty acids for the Melbournians. All the n-3 fatty acids were higher in the Minangkabau. Total n-3 fatty acid concentrations of the Minangkabau were nearly double that of the concentrations found in Melbournians, with DHA concentrations being 3.6 times greater.

9.3 DISCUSSION

9.3.1 SERUM LIPIDS

During the food intervention, there was an elevation of total cholesterol, LDL-cholesterol as well as HDL-cholesterol concentrations in the second assessment. It has been confirmed by previous metabolic studies that coconut oil increases total cholesterol and LDL-cholesterol (Castillo et al., 1996; Cox et al., 1995; Rodriguez-Vico et al., 1993; Soelaiman et al., 1996; Van and Zilversmit, 1988). Other metabolic studies have shown that lauric and myristic acids increase HDL and decrease LDL/HDL (Hajri et al., 1998; Mensink et al., 1994)

However, the increase in serum lipid concentrations was temporary in the current study. Despite lack of statistical significance, the concentration of serum lipids tended to decrease towards the end of the intervention. Moreover, when compared to the Non-coconut group, at the third assessment, the Coconut group had lower cholesterol concentrations, while the concentrations of LDL- and HDL-cholesterol were not significantly different between the two groups during the study.

In accordance with previous results in other case-control studies, increased coconut consumption (which is dominated by medium chain saturated fatty acids) did not alter lipid concentrations in this study. A recent study in Sweden showed that the dietary content of saturated fatty acids with a chain length of four to fifteen carbon atoms had an inverse association with serum cholesterol and ApoB (Samuelson et al., 2001). Another clinical trial on healthy subjects comparing consumption of lauric acids with trans fatty acids found that consumption of lauric acid resulted in a more favourable serum lipid profile (de Roos et al., 2001). When compared to

butterfat, coconut oil has been reported to be less atherogenic (Hajri et al., 1998; Mensink et al., 1994; Soelaiman et al., 1996).

The serum cholesterol concentration of the Coconut group in the present study was comparable with other populations. The average serum cholesterol of the Kitavan, a traditional Melanesian society in Papua New Guinea was 4.8 mmol/L (Lindeberg et al., 1994). In another traditional population on Polynesian atolls the cholesterol concentration was 4.7 mmol/L (Prior et al., 1981), whereas in the Coconut group in this study the concentration was 4.9 mmol/L. On the contrary, the average serum cholesterol concentrations from Western Caucasian societies from the IUNS Food Habits in Later Life study were 6.2, 6.3 6.5 and 6.7 mmol/L for Anglo-Celtic Australians, Greek-born Australians, Greeks in rural Greece and Swedes in Sweden, respectively (Purba, 2000).

Increased consumption of coconut could explain the relatively lower triglyceride concentration of the Coconut group compared to the Non-coconut group in the present study. A number of studies indicate that MCTs are metabolised differently compared to LCTs. LCTs are not completely oxidised and some long-chain fatty acids continue to be re-esterified to triglycerides in the liver (Goodenough and Wolfe, 1984). On the other hand, MCTs are more rapidly cleared from the circulation (Dawes et al., 1986) and metabolised faster (Deckelbaum et al., 1990). MCTs are thought by some to enter into the cell mitochondria for metabolism relatively independently of the carnitine acyltransferase transportation system required for long-chain fatty acids (Bach and Babayan, 1982; Cotter et al., 1989; Jiang et al., 1993; Seaton et al., 1986). Due to the higher degree of oxidation and minimal reesterification, MCTs are cleared more efficiently and lead to a lower concentration of triglycerides (Cotter et al., 1989; Jiang et al., 1993; Seaton et al., 1986).

The Coconut group tended to have lower plasma Lp(a) concentrations than the Non-coconut group, although it was not statistically different. No relationship was found between Lp(a) and other heart disease risk factors. This is not in line with other studies, which found a positive relationship between Lp(a) and risk of heart disease (Kostner, 1989; Rhoads et al., 1986; Sandkamp et al., 1990; Rhoads et al., 1986).

9.3.2 PHOSPHOLIPID FATTY ACIDS

Increasing coconut and fish consumption in the Coconut group during the intervention phase did not seem to alter the concentration of total SFA and PUFA in phospholipids. It has been discussed in many studies whether the presence of MCTs in the diet can change the metabolism of other meal fatty acids. Cotter et al. (1989) conducted a study to assess the potential metabolic competitive interactions of intravenous MCTs and LCTs. LCTs emulsions were added to an intravenous dose of MCTs emulsions. Their results showed a strong competitive interaction between MCTs and LCTs, which appeared to favour LCTs for removal and metabolism over MCTs. However, in this study, total PUFAs did not increase and SFAs did not decrease in phospholipids.

From the baseline data, the Coconut group tended to have a higher intake of coconut and fish (Chapter 8) which was reflected by their higher intakes of n-3 fatty acids. In the Coconut group, the promotion of coconuts during this study not only increased n-3 α-linolenic and docosapentaenoic acids, but also n-6 linoleic, homo γ linolenic and docosatetraenoic acids. The increasing phospholipid concentrations of n-3 fatty acids could be explained by the increasing intake of fish. A study by Li et al. (2001) showed that higher phospholipid concentrations of n-3 fatty acids amongst Chinese living in Melbourne was positively correlated with dietary intake of fish compared to their Caucasian counterparts.

The increasing in the concentration of n-6 fatty acids in the present study, such as linoleic acid, could not be explained by diet. Linoleic acid is a major component of plasma phospholipids in this study population, as shown in Figure 9.1. More than 50% of the linoleic acid in the present study was from soy dishes, even though soy consumption was not found to increase. So, the increasing concentration of linoleic acid profiles in the present study may not relate to intake, but it may be influenced by factors in the metabolism of fatty acids in vivo.

In the present study, intake and plasma linoleic acid profile was not correlated with CHD risk factors. Some studies have suggested that a high intake of linoleic acid is a risk factor for heart disease events (Blankenhorn et al., 1990; Hodgson et al., 1993), although some have found an inverse relationship (Rimmrsma et al., 1986). The mean concentration of linoleic acid in the present study was exceptionally high (21.5%). Further studies are needed to explain the contradictory findings in linoleic acid and heart disease risk.

The comparison of fatty acid composition between Minangkabau and Melbourne Caucasians show that the main difference between the groups was in n-3 fatty acids. All the n-3 fatty acids were higher in the Minangkabau. The difference in fish consumption could explain this, fish consumption in the Minangkabau was 58 g/day compared to 26 g/day in the Melbourne Caucasians (Li et al., 2001).

9.3.3 Insulin and Glucose

Infusion of MCTs has been found to increase plasma insulin concentrations in previous studies (Cotter et al., 1989; Sanbar et al., 1967; Seaton, 1986). This is in line with the present study, where increased insulin was only observed in the Coconut group, but not in the Non-coconut group. The elevation of insulin with increased coconut consumption could resemble the incorporation of MCFAs into pancreatic islet cell membranes, which activates the insulin secretion system (Zawalich et al., 1983). However, the increase in insulin concentrations in the Coconut group was almost doubled in the sixth week compared to the baseline, which was followed by the increase in glucose concentrations (although it was not significant). No increase in sugar consumption was observed in the Coconut group. Further study is needed as to whether the increase in coconut consumption could be relevant to diabetes risk in this population.

It has been suggested that saturated fatty acids can be deleterious with respect to fat-induced insulin insensitivity, relative to monounsaturated and polyunsaturated fat (Lichtenstein and Schwab, 2000). However, this suggestion may not be applicable in this study population. Previous results show that MCTs from coconut activates the insulin secretion system. Furthermore, the hypothesis of "genetically unknown foods" was suggested as the strongest reason for the increasing incidence of diabetes mellitus in some "newly modernised" Asian-Pacific populations who have recently adopted Western dietary habits. These populations, which are believed to have the original genotype of humankind, are metabolically unable to cope with high-fat meals and sucrose in solid form or in solution with concentrations > 4.18 MJ/L (Baschetti, 1999). Unless the Minangkabau change their diet to a Western diet, this hypothesis does not apply.

9.3.4 Body Composition

The mean weight, BMI, skinfold thickness and abdominal circumference did not change significantly during the intervention study in both groups. A growing number of studies have confirmed that the MCFAs, which are most available in coconut, are more prone to oxidation. This suggests that coconut fat may be less likely to lead to obesity (Baba et al., 1982; Bray et al., 1980; Cotter et al., 1989;

DeLany et al., 2000; Hill et al., 1989; Geliebter et al., 1983; Papamandjaris et al., 2000; Seaton et al., 1986). Among the fatty acids, MCTs were highly oxidised, whereas the polyunsaturated and monounsaturated fatty acids are fairly well oxidised. Oxidation of the saturated fatty acids decreases with increasing carbon length (lauric acid > myristic acid > pamitate acid > stearic acid). MCTs are rapidly cleared from the circulation and are more likely to be oxidised than to be stored in adipse tissue. When MCTs are given, it results in diminished deposition of fat (Baba et al., 1982; Geliebter et al., 1983). The reduction in the deposition of fat could be explained by the obligatory oxidation of MCT–derived fatty acids in the liver after being transported there via the portal vein, leaving almost no MCT for incorporation into body fat. Further explanation to the decreased body fat in an MCT-enriched diet is related to the increased metabolic rate and thermogenesis (Baba et al., 1982; Hill et al., 1989; Seaton et al., 1986). In another study, the results showed that the presence of MCTs in the diet increased endogenous oxidation of LCFAs (Papamandjaris et al., 2000).

During the study, fat intake was not calculated. It is therefore not known whether the increased intake of coconut altered the fat composition of the diet. The similarity of all anthropometric measurements during this study, however, suggests there was only little, if any changes, in dietary fat composition. It has also been suggested that dietary fat was not a major determinant of body fat (Willett, 1998d).

However, compared to other coconut consuming populations, body weight and fat mass of the Coconut group in the present study were relatively higher. The mean BMI in the present study was 23.8 kg/m^2, whereas BMI in the Kitavan study was 20 kg/m^2 (age range 40–59 years). Waist circumference in this study was 77.8 cm compared to 74 cm in the Kitavan. These differences could be partly explained by the fact that the present study dominated by women. However, it also important to note the differences in coconut consumption. The Minangkabau had reduced their coconut oil consumption to nearly nil and the intake of coconut products (other than coconut oil) was only 45 g/d. In contrast, intake of coconut products by the Kitavan was 110 g/day (Lindeberg et al., 1994).

9.4 CONCLUSIONS

This study was conducted on all Minangkabau subjects. Dietary patterns may differ between gender, ethnic, and cultural groups. An advantage of the present study is

that it is the first intervention study on the consumption of coconut. The measurements were repeated three times on 87 healthy subjects.

Temporary increased concentrations of plasma total cholesterol, HDL-cholesterol and LDL-cholesterol were observed during the study in the Coconut group. However, at the end of the coconut intervention, the concentrations decreased, and plasma cholesterol concentrations were found to be significantly lower in the Coconut group compared to the Non-coconut group.

Increasing consumption of coconut and fish were possibly the main explanations for the differences in phospholipid fatty acid patterns between the two groups. The Coconut group also had a higher insulin concentration, but similar body composition to the Non-coconut group during the intervention phase.

CHAPTER 10

The Minangkabau, Coconut and Coronary Heart Disease: General Discussion and Conclusions

10.1 INTRODUCTION

This is the first study about food culture and its relationship with coronary heart disease amongst the traditional consumers of coconut. Minangkabau food culture provides a unique opportunity to look at the impact of economic development on a traditional food supply centred on women. West Sumatra, as is the case in many developing communities, is experiencing a nutrition transition reflected in rapid changes in nutrient intakes and dramatic shifts in the causes of death, although many characteristics of the traditional diet may still be preserved. This thesis has found through focus group discussions and compilation of historical data, that the methods of food preparation and taste preferences have remained remarkably consistent in recent generations. This is despite the finding that the amount of food eaten has changed and that novel foods from other parts of Indonesia have become integral to Minangkabau food culture.

10.2 FOOD AND COCONUT INTAKE

The dietary pattern of the Minangkabau people is unique. The influence of Western foods on the population studied has been negligible. Considering both the Case-control Study and the Intervention study, energy intake from fat has risen from about 11% to a still relatively low 23% - 26% of total energy intake. Now, nearly 60% of the fat is from saturated fatty acids, derived from both coconut products and palm oil. In the past, it was almost exclusively from coconut. The intakes of monounsaturated and polyunsaturated fatty acids were 25% and 15% of total fat respectively, with fish and soy dishes, the most important sources for these fatty acids, as judged from the subjects studied in the Case-control and Intervention Studies.

Between the cases and the controls, there was little difference either in the amount and the quality of fat consumed. Moreover, with the increased coconut consumption in the Intervention study, the overall nutritional quality of the coconut

diet improved, especially in relation to fish and plant food intake, including the relationship amongst the fatty acids.

The Case-control Study showed that total fat and saturated fat consumption were unlikely to be predictors for CHD in this food culture. However, the intakes of more animal foods, total protein, dietary cholesterol, and less plant-derived food were predictors of CHD. Of food intake factors, animal food intake was the strongest independent predictor of CHD. Subjects in the highest quartile of animal intake had more than double the risk for CHD compared to those in the lowest quartile. However, whilst on univariate analysis the intake of total protein and cholesterol were predictors for CHD, they failed to enter the model in multivariate analysis.

It seems that a traditional diet, based on coconut, might actually be protective for the Minangkabau. Study participants who had a high intake of coconut had significantly higher intakes of fish and vegetables. In the intervention study, the Coconut group was found to have a higher intake of spices such as turmeric (*Curcuma domestica*), ginger (*Zingiber officinale*), galanga (*Laguas gallanga*) and some Minangkabau traditional herbs. Several studies consistently demonstrated that these spices have the capacity to prevent oxidation of oils and fats and inhibit lipid peroxidation (Nagababu and Lakshmaiah, 1992; Shobana and Naidu, 2000). However, the increase in coconut consumption also increased insulin concentration, although more study is needed as to whether this could be relevant to diabetes risk in this population. Thus, if coconut consumption were to be discouraged, the health profile of the Minangkabau diet may be less favourable in several respects (fish, vegetables and spices intakes).

10.3 LIFESTYLE FACTORS

Some lifestyle factors emerged as important for CHD risk in the Minangkabau (Chapter 5). Physical inactivity was the strongest predictor of CHD of all variables considered. It seems that perceived stress doubled the risk for CHD in the total population and for men in particular. Among women, smoking was a strong predictor for CHD, although few smoked. Family history, for one of several CHD risk factors, was a consideration.

On the question of educational attainment, this is steadily improving in West Sumatra. This might support a significant negative association between CHD and higher socio-economic class in developing countries observed by other authors (Singh et al., 1999; INCEN, 1999). We are unable to make any conclusion o n this point, since no differences were found between the cases and the controls.

10.4 BLOOD PRESSURE

Higher blood pressure was a strong predictor for CHD (Chapter 7). In the case-control study, the odds ratio for "high systolic blood pressure" was 1.4 (CI 95% 1.2–1.8), and for "diastolic blood pressure" was 1.5 (CI 95% 1.2–1.9). In the coconut intervention study, the blood pressure of the coconut group remained unchanged during the study, so that coconut intake had no observable impact on blood pressure (Chapter 9).

10.5 LIPIDS AND GLUCOSE MEASUREMENTS

No differences were found in the average serum total or LDL-cholesterol, and triglyceride concentrations between CHD cases and the controls. However, the frequency of marked hypercholesterolaemia (serum cholesterol ≥ 7.8 mmol/L) and combined hyperlipidaemia (serum total cholesterol ≥ 6.5 mmol/L and triglyceride ≥ 2.0 mmol/L) was greater in the cases (Chapter 7). HDL-cholesterol concentration was lower in the cases. The case-control study showed that HDL-cholesterol was an independent predictor for CHD event by both univariate as well as multivariate analysis. The dietary contributed to these differences in serum lipid status between cases and controls are by no means clear. Although dietary cholesterol is a weak determinant of serum cholesterol and of LDL cholesterol in controls. No foods studied were found to be determinant of blood lipids. It might have been expected that food with components known to affect blood lipids (like fat, fibre and soy protein) would emerge with predictive value, but they did not.

In the intervention study, increasing coconut intake prevented the increase in total cholesterol concentration in the coconut group (Chapter 9). There were no differences found in HDL and LDL-cholesterol, Lp(a) or triglycerides at the sixth week of the study between the Coconut and the Non-coconut group. Also, no significant differences were found in the concentrations of glucose or insulin.

For plasma phospholipid fatty acid composition, at the end of the study, the Coconut group had significantly higher proportions of myristic (C14:0), docosapentaenoic acid (C22:5n-6), α-linolenic (C18:3n-3), EPA (C20:3n-3), docosapentonoic (C22:5n-3), DHA (C22:6n-3), DHA/EPA ratio and a lower n-6/n-3 fatty acid ratio. There is increasing interest and recognition that such fatty acid changes may be favourable, not only to CHD risk, but also to other aspects of health linked to inflammatory diseases (Sinclair and Li, 2000). Comparison between

the Minangkabau and other populations resident in Western settings (the Chinese and the Anglo-Celtic in Melbourne), where the arachidonic status is much higher than for the Minangkabau, add further interest to the role of coconut in health protection. It is interesting, even with increasing coconut intake in the intervention study, and food intake changes like the increase in fish consumption, that the overall composition of fatty acid intake did not change detectably.

10.6 BODY COMPOSITION

No significant differences were found in any anthropometric or body composition indices between the CHD cases and their controls. Amongst these measurements, only height was able to predict for CHD events - taller stature in women was protective. Lower stature reflects childhood nutrition status; the finding that increased stature was cardioprotective is consistent with the Barker hypothesis on fetal and childhood nutrition and cardiovascular disease (Barker et al., 1989). In the intervention study, mean weight, BMI, skinfold thickness, abdominal circumference or WHR did not change during the intervention. Other studies have suggested that the MCT in the coconut is less likely to lead to obesity (DeLany et al., 2000; Papamandjaris et al., 2000). In this study, increasing coconut consumption did not result in changes to weight or other anthropometric measurements of body fat.

10.7 LIMITATIONS OF THIS STUDY

Although all subjects in this thesis were Minangkabau, dietary patterns do differ with intra-cultural demographic differences for gender, age, socio-economic status, dwelling and work arrangements.

In the case-control study, as in others, there may have been selection bias. To minimise this bias, hospital and population-based approaches were used. The control subjects were selected from the same hospital and from the same population from which patients were obtained. Nevertheless, subjects in this study may have not represented the actual population structure in West Sumatra. Most subjects in the case-control study came from big cities in West Sumatra from where most of the CHD cases came.

Another possible source of bias may have been less access of individuals with lower socio-economic and lower education to the study because of less likely admission to hospitals.

In the intervention study, changes in food intake, body composition and biochemistry measurements may have been confounded by other factors not assessable in this study.

10.8 CONCLUSIONS

1. The Minangkabau in West Sumatra are experiencing nutrition transition reflected in rapid changes in dietary composition and dramatic shifts in causes of death, but the traditional diet has been remarkably preserved.

2. The influence of Western food on Minangkabau food culture is still negligible. Total fat and saturated fat consumption were not different between the cases and the controls and were not predictors of CHD. But the shift towards more animal and less plant foods is predictive; the indicators of these include nutrients like dietary cholesterol, total protein, and lower intake of total carbohydrate. Being physically inactive, having higher levels of perceived stress, smoking, higher systolic and diastolic blood pressures, lower serum HDL-cholesterol and having higher glucose and lower stature increase the risk of CHD events significantly.

3. Increased coconut consumption significantly increased fish consumption and was positively associated with the consumption of vegetables, fruits and meat. For all intents and purposes, there were no biologically meaningful differences in body composition or serum lipid measurements. After 6 weeks of increased coconut consumption, associated higher fish and plant food intakes would have been the explanation for the higher plasma n-3 α-linolenic and docosapentaeonoic and n-6 linoleic, homo γ-linoleic and docosatetranoic acids in the Coconut group.

10.9 SUGGESTIONS AND RECOMMENDATIONS

Epidemiological studies have indicated that deviation from traditional ways of eating and living may account for the rise of cardiovascular risk (Bell et al., 1999; Boyce and Swinburn, 1993; Shintani and Hughes, 1994). It is important for health authorities to emphasise traditional culture in their dietary recommendations. The

recent dietary recommendations for Indonesia about fat consumption could be more culturally sensitive (Muhilal et al., 2001). The recommendation not to consume more than 25% of energy intake from food, may be supported (Muhilal et al., 2001), but a recommendation to reduce saturated fatty acids, in particular from coconut, requires re-evaluation. Of considerable importance, in any dietary recommendation must be about food variety, encompassing food cultural difference and advantage.

Future efforts to advance health among the Indonesian people will require attention to traditional diets.

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APPENDICES

Appendix 1	Focus Group Discussion questionnaire
Appendix 2	Health status and lifestyle questionnaire
Appendix 3	Food intake questionnaire
Appendix 4	Clinical Examination
Appendix 5	Consent forms
Appendix 6	Letters of invitation to participate in the study
Appendix 7	Letter of recommendations

IN CONFIDENCE

FOCUS GROUP DISCUSSION QUESTIONNAIRE

MINANGKABAU TRADITIONAL DIET AND CARDIOVASCULAR DISEASE RISK IN WEST SUMATRA, INDONESIA

Investigators

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Notes of the Recorder

Date: Time: Start:

End:

Duration:

Community:

Brief description:

Meeting place:

Brief description:

Number of participants: Women

Men

Total

Name and characteristic of the participants

Name	Sex	Age	Occupation
1			
2			
3			
4			
5			
6			

Group dynamics:

Basic foods:

1. What do most people eat for breakfast?
2. What do most people eat for lunch?
3. What do most people eat for dinner?
4. What do most people eat for morning snack?
5. What do most people eat for afternoon snack?
6. Are there any changes in what people eat between now and 30 or more years ago?, for breakfast, lunch, dinner, snack?

Cooking techniques:

1. How do people cook coconut and meat?
2. How do people cook coconut and fish?
3. How do people cook coconut and vegetable?
4. Do people mix/cook coconut with fruit?
5. Do people cook coconut with staple foods (rice, corn, sago)
6. How important coconut in our food?
7. How often do people use coconut oil in cooking?
8. Do you think people prefer coconut oil rather than other oils or vice versa?, why?
9. Are there any changes in cooking techniques between now and 30 or more years ago?
10. Are there any changes in flavour principles between now and 30 or more years ago?

RAHASIA

KUESIONER DISKUSI KELOMPOK

MAKANAN TRADISIONAL MINANGKABAU DAN RESIKO PENYAKIT KARDIOVASKULER DI SUMATRA BARAT, INDONESIA

TIM PENELITI

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Macam dan jenis makanan:

1. Menurut ibu/bapak, makanan apa saja yang biasa dimakan pada waktu:
   Sarapan:

   Snack pagi:

   Makanan siang:

   Snack sore:

   Makan malam:

2. Apakah sudah ada perubahan jenis makanan yang dimakan pada waktu ibu/bapak masih muda dengan sekarang?

Cara mengolah dan memasak makanan:

1. Bagaimana cara ibu/bapak mengolah dan memasak gulai daging, rendang?

2. Bagaimana cara ibu/bapak mengolah dan memasak gulai ayam?

3. Bagaimana cara ibu/bapak mengolah dan memasak gulai ikan?

4. Jenis sayur apa saja yang dimasak dengan santan/kelapa?

   Bagaimana cara ibu/bapak mengolah dan memasaknya?

5. Jenis buah apa saja yang dimasak dengan santan/kelapa?

   Bagaimana cara mengolah dan memasaknya?

6. Apakah ibu/bapak memasak makanan pokok (nasi, jagung, ubi, sagu) dengan santan/kelapa?

   Bagaimana cara mengolah dan memasaknya?

7. Menurut ibu/bapak seberapa pentingkah kelapa dalam makanan kita?

8. Seberapa seringkah penggunanaan kelapa/minyak kelapa dibanding jenis minyak goreng lain dalam kehidupan sehari-hari ibu/bapak?
   m. sawit : sejak ± 4 th,

9. Apakah menurut ibu/bapak telah ada perubahan cara mengolah dan memasak makanan pada saat sekarang dibanding dengan keadaan pada waktu ibu/bapak masih muda?

Selera:

1. Jenis dan rasa makanan seperti apa yang lebih ibu/bapak sukai?

2. Menurut ibu/bapak, apakah telah terjadi perubahan kecenderungan selera antara ibu/bapak dibanding dengan orang-orang yang lebih muda?

IN CONFIDENCE

HEALTH STATUS AND LIFESTYLE QUESTIONNAIRE

MINANGKABAU TRADITIONAL DIET AND CARDIOVASCULAR DISEASE RISK IN WEST SUMATRA, INDONESIA

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BACKGROUND

1. **Date of birth:** _____/_____/_____
   day month year

2. **Sex:** (circle one number)

   1. Male
   2. Female

3. **Marital status**: (circle one number)

   1. Single (never married)
   2. Married
   3. Divorced
   4. Widowed

4. **What is the highest level of education you have completed**: (circle one number)

   1. Never attended school
   2. Primary school
   3. Junior High School
   4. Senior High School
   5. University

5. **Which of the following best describes your usual occupational category, or if retired, your past occupational category?** (circle one number)

   1. Government officer
   2. Member of the armed forcer
   3. Tradesman
   4. Farmer, fisherman
   5. Housewife
   6. Not employed

6. **Which of the following range best describes your household total gross income per month?** (please give total sum, circle one number)

   1. Less than Rp. 250.000,-
   2. Rp. 250.000 - 500.000,-
   3. Rp. 500.000 - 1.000.000,-
   4. Rp. 1.000.000,- - 2.000.000,-

5. More than Rp. 2.000.000,-

7. **How many times did you visit a medical doctor during the past 12 months?** (circle one number)

1. Not at all
2. Once or twice
3. 3 - 12 times
4. More than 13 times

8. **If you have been hospitalised for reasons other than pregnancy/childbirth, how many days have you spent in hospital during the past 12 months?** (circle one number)

1. None
2. 1 - 14 days
3. More than 14 days

9. **About how many days during the past 12 months have you been sick in bed all or most of the day?** (circle one number)

1. None
2. 1 - 14 days
3. More than 14 days

10. **How would you rate your overall health at the present time?** (circle one number)

1. Excellent
2. Good
3. Fair
4. Poor

11. **How many times have you suffered from a cold or flu in the past 12 months?** (please write number in space provided)

Number of times: _____

12. **Do your health problems stands in the way of you doing things you want to do?** (circle one number)

1. I do not have any health problems
2. No at all
3. A little
4. A great deal

13. **Are you currently taking any dietary supplements (usually tablets bought from the chemist or health food stores, for example vitamins, minerals, fish oil tablets, dietary fibre, garlic)?** (circle one number)

1. Yes
2. No

If **Yes,** please write the name of the supplement from all packets or bottle in space provided below and indicate what this supplement is used for:

Name of supplement (write name from packet/bottle)	Why do you take this
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____

LIFESTYLE

14. Physical Activities

We are interested in knowing about your physical activities in the present time.

If you engage in any of the following regularly (ie. weekly), please estimate how many hours per week (and months per year if activity is seasonal) you spend in these types of. Place numbers in space provided.

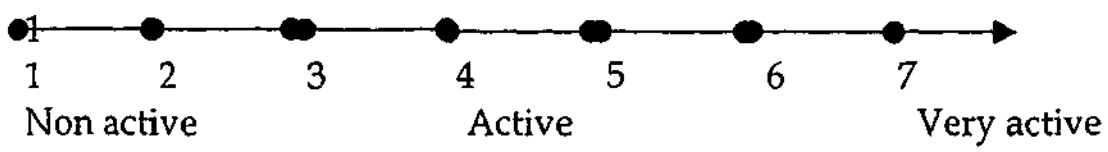
Currently:

Activities	**Hours per week**	**Months per year**
Walking	_____	_____
Jogging	_____	_____
Warm-up/toning exercises eg. stretching, push-ups	_____	_____
Swimming	_____	_____
Cycling	_____	_____
Team sports eg. football	_____	_____
Playing tennis	_____	_____

House cleaning	_____	_____
House repairs	_____	_____
Gardening	_____	_____

For interviewers:
Exercise scores are:

1= Non active, sitting or lying all days
2= Non active, sitting most of the times
3= Non active, sitting most of times with mild walking
4= Active, walking or gardening (for 1 hour) or house cleaning for a couple of hours, 3-4 times a day
5= Active, walking or gardening (more than 1 hour) or house cleaning or repairs for hours
6= Active, farming or aerobic
7= Very active, farming, mining, heavy sport for hours a day



Stress:

15. **Do you think that stress is adversely affecting either your health or the quality of your life?** (circle one number)

    1. Yes
    2. No

16. **This question about stress levels during every day life. Would you say that you are**: (circle one number)

    1. Rarely or never under much stress
    2. Sometimes under a little stress
    3. Often under a little stress
    4. Sometimes under a lot of stress
    5. Often under a lot of stress
    6. Almost always under a lot stress

Cigarette smoking:

17. **Have you ever smoked regularly, at least one cigarette a day?** (circle one number)

    1. Yes
    2. No (please go to question 29)

A) At which age did you start smoking _____

B) If you have given up smoking completely

a) At what age did you give up smoking _____
b) How many cigarettes did you smoke a day _____

C) If you currently smoking:

a) How many cigarettes do you smoke a day _____

b) For how many years have you been smoking this much? _____

c) If you smoked more or less in the pass, how many cigarettes did you smoke _____

d) If you smoked more or less in the pass, how many years did you smoke amount stated in (c)? _____

Food Habits and Cooking Methods:
We are interested in knowing your food habits and cooking methods in the present time and in the last ten years.

18. **What are the usual time that your meals and snacks are eaten?**

Meal	Time	
	Present	Last
Breakfast		
Morning tea		
Lunch		
Afternoon tea		

Dinner		
Supper		

19. How do you usually cook meat, chicken or fish?
(please tick one or more cooking method for each item)
Present time:

	Beef/lamb	Chicken	Fish
Boiled	_____	_____	_____
Steamed	_____	_____	_____
Casseroled	_____	_____	_____
Stirred	_____	_____	_____
Fried	_____	_____	_____
Grilled	_____	_____	_____
Roaste	_____	_____	_____

20. Do you use coconut milk?

1. Rarely or never
2. Sometimes
3. Often
4. Always

21. What kind of oil do you usually use in your household?
(circle one number or two)

1. Coconut oil
2. Palm oil
3. Corn oil
4. Other polyunsaturated vegetable oil
5. Butter
6. Margarine
7. Other, please specify .....

22. On average, how many teaspoon of coconut oil would you eat per day?
(there are about 4 teaspoon in a tablespoon, please write the number)

Number of teaspoons coconut oil per day (currently): _____

23. On average, how many teaspoon of other oils would you eat per day?

(there are about 4 teaspoon in a tablespoon, please write the number)

Number of teaspoons oils per day (currently):_____
Number of teaspoons oils per day (last time) : _____

24. On average, how many teaspoon of butter/margarine would you eat per day? (there are about 4 teaspoon in a tablespoon, please write the number)

Number of teaspoon butter/margarine per day (currently):_____
Number of teaspoon butter/margarine per day (last time):_____

25. How do you usually cook or eat your vegetables?

1. Boiled
2. Steamed
3. Casseroled
4. Stirred
5. Roasted
6. Raw, salad

26. If salt is added to the cooking, are the foods? (circle one number)

1. I never add salt to cooking
2. Lightly salted
3. Medium salted
4. Heavily salted
5. Salting is highly varied

27. Do you drink tea?

1. Rarely
2. Sometimes
3. Often
4. Always

28. Do you drink coffee?

1. Rarely
2. Sometimes
3. Often
4. Always

29. On average, how many teaspoons of sugar do you have per day which is added to food and drinks? (please write number)

Number of teaspoons per day : _____

30. Do you eat vegetables/fruits grown in your backyard? (circle one number)

1. Yes, please specify_____
2. No

31. How often do you use the following herbs and spices (please tick one or more)

Currently:

	Always	Often	Sometimes	Rarely
Chilli	_____	_____	_____	_____
Ginger	_____	_____	_____	_____
Turmeric	_____	_____	_____	_____
Galangal	_____	_____	_____	_____
Cinnamon	_____	_____	_____	_____
Cloves	_____	_____	_____	_____
Coriander	_____	_____	_____	_____
Nutmeg	_____	_____	_____	_____
Candle nut	_____	_____	_____	_____
White pepper	_____	_____	_____	_____
Black pepper	_____	_____	_____	_____
Garlic	_____	_____	_____	_____
Onion	_____	_____	_____	_____
Lemon grass	_____	_____	_____	_____
Lime leaves	_____	_____	_____	_____
Turmeric Leaves	_____	_____	_____	_____
Salam leaves	_____	_____	_____	_____
Kemangi Leaves	_____	_____	_____	_____
Celery	_____	_____	_____	_____
Onion leaves	_____	_____	_____	_____
Ketchup (salty)	_____	_____	_____	_____
Ketchup (sweet)	_____	_____	_____	_____

Notes:

Always:	3-7 days a week
Often :	1-3 days a week
Sometimes:	less than once a week
Rare:	less than once a month

32. Have you ever drunk alcohol regularly?

1. Yes, and I currently drink (Please answer Q33,)
2. Yes, but I have given up drinking (Please go to Q33)
3. Yes, but I drink less than once a

RAHASIA

KUESIONER DEMOGRAFI DAN STATUS KESEHATAN

MAKANAN TRADISIONAL MINANGKABAU DAN RESIKO PENYAKIT KARDIOVASKULER DI SUMATRA BARAT, INDONESIA

TIM PENELITI

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Berapakah usia anda
pada saat itu: _____kg 10a. ¡

11. Berapa berat badan anda terendah: _____kg 11a. ¡

Berapakah usia anda
pada saat itu: _____kg 11b. ¡

12. Berapakah berat badan anda sekarang? _____kg 12. ¡

13. Berapa berat badan yang anda inginkan: _____kg 13. ¡

14. Apakah anda pernah dinasehati dokter/petugas kesehatan untuk menurunkan berat badan?:

1. Ya
2. Tidak 14a. ¡

Jika ya, berapa banyak disarankan
harus dikurangi _____kg 14b. ¡

15. Adakah kejadian penting dalam hidup anda yang menyebabkan perubahan berat badan meningkat secara drastis? (Pilih satu jawaban) 15. ¡

1. berat badan saya tak pernah meningkat secara drastis
2. Ketika saya menikah
3. Setelah kehamilan pertama
4. Ketika saya berhenti berolahraga
5. Ketika saya berhenti merokok
6. Ketika saya memakai alat kontrasepsi
7. Setelah stress besar seperti: perceraian, kematian, ganti pekerjaan
8. Setelah pensiun
9. Setelah tamat SMA atau kuliah

16. Apakah anda harus tetap memperhatikan berat badan anda karena berat badan anda mudah naik?

1. Ya 16. ¡
2. Tidak

Jika tidak langsung ke pertanyaan 18

17. Jika ya, langkah apa yang anda lakukan untuk menjaga agar berat badan anda memuaskan? (pilih satu jawaban) 17. ¡

1. Diet khusus

23. Bagaimana anda menilai tingkat kesahatan anda pada saat ini?

1. Sangat baik
2. Baik
3. Cukup
4. Kurang

23. j

24. Apakah dokter pernah memberitahu bahwa anda dalam keadaan seperti tersebut dibawah ini dan kapan anda diberitahu?

No	Keadaan	ya	tahun
A	Tekanan darah tinggi		
B	Kolesterol tinggi		
C	Nyeri jantung (angina)		
D	Serangan jantung (trombosis koroner, Infark miokard)		
E	Stroke		
F	Sangat gemuk		
G	Diabetes		
H	Osteoporosis		
I	Gangguan kandung kemih		
J	Gangguan lambung		
K	Radang usus kronis		
L	Haemoroid		
M	Kurang darah		
N	Artritis/rematik		
O	Asma		
P	Gangguan ginjal		
Q	Gangguan tiroid		
R	Infeksi saluran kemih		
S	Gangguan hati		
T	Penyakit parkinson		
U	Gangguan prostat		
V	Gangguan syaraf		
X	Kanker payudara		
Y	Kanker rahim		
Z	Kanker usus besar		
Aa	Keadaan lain, sebutkan:		

25. Apakah anda pada saat ini makan obat?

1. Ya
2. Tidak

25. j

Jika ya, sebutkan obat nama obat (tidak termasuk makanan tambahan, lihat pertanyaan 26) dan kegunaannya:

Nama obat Kegunaan

_____ _____

_____ _____

_____ _____

beberapa jam tiap hari atau sering jalan
6= aktif, berkebun (berat) atau bertani atau olahraga aerobik atau jalan selama beberapa jam 3-4 kali sehari
7= sangat aktif, berkebun (berat) atau bertani atau olahraga aerobik atau jalan selama beberapa jam tiap hari.

1	2	3	4	5	6	7
Tidak aktif			Aktif			Sangat aktif

Stress

28. Apakah menurut anda stress mempengaruhi kesehatan atau kualitas hidup anda?
    1. Ya
    2. Tidak — 28. ¡

29. Pertanyaan dibawah ini menanyakan tentang tingkat stress yang anda hadapi dalam hidup sehari-hari. Apakah menurut anda, anda: (pilih satu jawaban)
    1. Tidak pernah atau jarang stress — 29. ¡
    2. Kadang-kadang stress sedikit
    3. Sering berada dalam keadaan stress sedikit
    4. Kadang-kadang berada dalam keadaan stress besar
    5. Sering berada dalam keadaan stress besar
    6. Selalu berada dalam keadaan stress besar

30. Berapa jam rata-rata anda tidur dalam satu hari? (termasuk tidur siang): (pilih satu jawaban)
    1. kurang dari 4 jam — 30. ¡
    2. 4 - 7 jam
    3. 7 - 9 jam
    4. 9 - 12 jam
    5. lebih dari 12 jam

Merokok

31. Apakah anda merokok paling sedikit satu batang sehari?
    1. ya
    2. tidak — 31. ¡

31A. pada umur berapa anda mulai merokok?
    Umur: -------- tahun — 31A. ¡¡

32 B. Jika anda telah berhenti merokok pada saat ini,

37. apakah anda memakai santan kelapa? (pilih satu jawaban)

1. Jarang atau tidak pernah
2. Kadang-kadang
3. Sering
4. Selalu 37. ¡

37. Minyak/lemak apakah yang biasa anda gunakan?: (pilih satu jawaban)

1. Minyak kelapa 37. ¡
2. Minyak kelapa sawit
3. Minyak jagung
4. Jenis minyak lemak tak jenuh lain, sebutkan:.....
5. Mentega
6. Mentega dari lemak nabati
7. Jenis lemak lain, sebutkan:.....

38. Berapa banyak minyak kelapa yang anda konsumsi rata-rata perhari? (satu sendok makan kira-kira sama dengan 4 sendok teh) 38. ¡

--------sendok teh

39. Berapa banyak minyak lain yang anda konsumsi rata-rata perhari? (satu sendok makan kira-kira sama dengan 4 sendok teh) 39. ¡

--------sendok teh

40. Berapa banyak mentega yang anda konsumsi rata-rata perhari? (satu sendok makan kira-kira sama dengan 4 sendok teh) 40. ¡

--------sendok teh

41. Apakah anda makan daging yang mengandung lemak didalamnya? (pilih satu jawaban)

1. tidak atau sedikit 41. ¡
2. setengah dari lemak tersebut
3. hampir semua lemak tersebut
4. semua lemak tersebut

42. Bagaimana anda biasanya memasak sayur-sayuran: (pilih satu atau lebih jawaban)

1. rebus
2. tumis
3. gulai
4. goreng
5. kukus
6. mentah

43. Apakah anda suka makanan yang asin:(pilih satu jawaban)

1. Tidak pernah pakai garam 43. ¡
2. Sedikit asin
3. Cukup asin
4. Asin

N. Serai	_____	_____	_____	_____
O.Daun jeruk	_____	_____	_____	_____
P. D. kunyit	_____	_____	_____	_____
Q. Daun salam	_____	_____	_____	_____
R. D. kemangi	_____	_____	_____	_____
S. D. seledri	_____	_____	_____	_____
T. D. bawang	_____	_____	_____	_____
U. Terasi	_____	_____	_____	_____

Catatan :
Selalu : 3-7 hari perminggu
Sering : 1-3 hari perminggu
Kadang: Kurang dari 1 kali seminggu
Jarang: Kurang dari 1 kali sebulan

50. Apakah anda pada saat ini telah merubah kebiasaan diet anda?
    1. Diet khusus mengurangi berat badan — 50. ¡
    2. Diet rendah lemak
    3. Diet diabetes
    4. Diet khusus, sebutkan:.....
    5. Tidak merubah kebiasaan diet

51. Berapa lama anda telah merubah kebiasaan diet anda:
    1. Kurang dari I bulan — 51. ¡
    2. 1 sampai 3 bulan
    3. 3 sampai 6 bulan
    4. 6 sampai 12 bulan
    5. Lebih dari 12 bulan

52. Dapatkah anda menilai seberapa jauh anda telah melakukan perubahan diet tersebut. Nilai 1 berarti "hanya merubah sedikit" dan nilai maksimum 6 berarti "perubahan total"

1	2	3	4	5	6	
sedikit			sedang	total		52. ¡

Konsumsi Alkohol:

53. Apakah anda meminum alkohol pada saat ini?
    1. ya
    2. tidak — 53. ¡

54. Jika anda telah berhenti minum sama sekali, berapa sering Biasanya anda meminumnya?:
    1. kira-kira satu kali sebulan — 54. ¡
    2. sekali setiap 2 minggu
    3. 1-2 kali seminggu
    4. 3-4 kali seminggu
    5. 5-6 kali seminggu
    6. setiap hari.

IN CONFIDENCE

FOOD FREQUENCY QUESTIONNAIRE

MINANGKABAU TRADITIONAL DIET AND CARDIOVASCULAR DISEASE RISK IN WEST SUMATRA, INDONESIA

Investigators

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YOUR FOOD INTAKE

This questionnaire covers food items that you ate/drank at different times of the day and how often you ate/drank them for the last ten years. It is important that your answers are based upon the usual foods/drinks consumed by you (not your whole family) during the period. Careful answers will enable us to give you an accurate analysis for more than 20 nutrients in your diet and how many it may relate to your long-term health status.

ABOUT THE QUESTIONNAIRE

We would like to know how much and how often you ate specific foods in the last 10 years.

To ANSWER THEM YOU NEED TO KNOW THE FOLLOWING:

How many times eaten?

If you eat the food once or more per month when it is available we would like you to indicate how often you eat the food by placing a number in one of the three columns provided. We want you to recall your food intake in terms of these three categories shown here:

Daily food items that you eat at least once every day. Write how many times per day.

Weekly that is not everyday but at least once per week. Write how many times per week

Monthly that is not every week, but at least once a month. Write how many times per month.

We would like you also to estimate the amount of food you ate.
In the column "Reference Serving" there is a reference serving of the food you are to refer to when estimating your serving size.
A Reference Serving is usually given in household measures or as a unit eg. 2 chops or 1 cup of milk.

a) If your serving size is the same as the reference serve: place a tick (√) *in* the column named "your serve".
b) If your serving size is not the same as the reference : for example the reference serve maybe "2 chops", but your serving size maybe 3 chops.

You would therefore write "3" in the "your serve" column ie. the actual number of chops you normally eat.

Important note: The reference serve given is not necessarily an average or normal serving size. It is there to be used as a guide for determining your serving size.

Example:

FOOD ITEM	HOW MANY TIMES EATEN (on average from the last 10y) (place a number in one of the columns below if you have ticked the first column) PER DAY	PER WEEK	PER MONTH	Reference Serving	Yours Serve
Fruits Banana		3		1 mediu	√
Orange	1			1 medi	1/2
Papaya			3	1 chop	2
Rambutan				1 piece	
Other, speci fy: Durian		3		1 piece	5

Banana	This person eats 1 medium banana on average three (3) times a week,
Orange	Half medium orange is eaten about once a day
Papaya	Two chops papaya are eaten about 3 times a
Rambutan	Rambutan is not eaten. All spaces for this food have therefore been left blank
Other	Five pieces of durian are eaten 3 times a week when in season. Durian is not listed as a food item, so they have written it in "other" section, together with how often it is eaten and the serving size.

INDIVIDUAL FOOD AND DRINKS ITEMS

FOOD ITEM	HOW MANY TIMES EATEN (on average from the last 10 y) (place a number in one of the columns below if you have ticked the first column) PER DAY	PER WEEK	PER MONTH	Reference Serving	Yours Serve
SEAFOOD **Fresh fish** -fried with chilli					
-deep fried					
-grilled					
-boiled					
-b w/ coconut					
Tinned sardines					
Other:					
Other sea food: -fried w/chilli					
-deep fried					
- b w/coconut					
Other:					
Shrimp					
-deep fried					
- fried w/ ch					
- b w/ coconut					
Other					
Salty small fish					
-deep fried					
-fried w/ ch					
-grilled w/coconut					
Other:					
Eggs					
-Fried w/ch					
-omelette					

-boiled					
-b w/ coconut					
FOOD ITEM	HOW MANY TIMES EATEN (on average from the last 10 y) (place a number in one of the columns below if you have ticked the first column) PER DAY / PER WEEK / PER MONTH			Reference Serving	Yours Serve
-drinks					
-quailed eggs: soup					
- - stirred					
Other:					
Tofu					
-deep fried					
-fried w/ch					
-stirred					
-b w/ coconut					
-boiled					
Other:					
Tempe:					
-deep fried					
-fried w/ch					
-stirred					
-b w/coconut					
-boiled					
Others:					
Peanut:					
-fried					
-fried w/ch					
-stirred					
-fr w/ flour					
-boiled					
-roasted					
-peanut butter					
-others:					
Mung beans					
-boiled					

-boiled w/coconut					
-other:					
FOOD ITEM	HOW MANY TIMES EATEN (on average from the last 10 y) (place a number in one of the columns below if you have ticked the first column) PER DAY	PER WEEK	PER MONTH	Reference Serving	Yours Serve
Chicken					
-fried					
-fried w/chilli					
-b w/ coconut					
-soup w/ ve					
-soto					
-satay/grilled					
-semur					
-rendang					
Other					
Beef:					
-fried					
-fried w/chilli					
-b w/ coconut					
-soup w/ vegetable					
-soto					
-satay/grille					
-semur					
-rendang					
-corned beef					
-others:					
Liver:					
-fried					
-fried w/chi					
-b w/ coconut					
-soup w/ vegetable					
-soto					
-satay/grilled					
-semur					
-rendang					
-other					

FOOD ITEM	HOW MANY TIMES EATEN (on average from the last 10y) (place a number in one of the columns below if you have ticked the first column) PER DAY	PER WEEK	PER MONTH	Reference Serving	Yours Serve
Brain:					
-b w/coconut					
-other					
Offal					
-b w/ coconut					
-rendang					
-other					
Fast food					
Hamburger					
Kentucky					
Chips					
Milk					
-wholemilk					
-skim milk					
-Sweetened condensed milk					
-fresh cow milk					
-yoghurt					
-other					
Cheese					
Vegetables:					
Swamp cabbage:					
-stirred					
-boiled					
-b w/ coco					
Spinach					
-stirred					

-boiled					
FOOD ITEM	HOW MANY TIMES EATEN (on average from the last 10 y) (place a number in one of the columns below if you have ticked the first column) PER DAY / PER WEEK / PER MONTH			Reference serving	Your serve
Cassava leaves:					
-boiled					
-b w/ coco					
Beansprout					
Cabbage					
Carrots					
Cauliflower					
Eggplant					
Cucumber					
Tomatoes					
Lettuce					
Pumpkins					
Kidney beans					
Bean curd					
Long beans					
Sprout					
Potatoes:					
-soup					
-boiled					
-fried					
-'pergedel'					
Other:					
FRUITS					
Bananas					
Papaya					
Orange					
Mangoes					
Spinach					
Rambutan					
Duku					
Durian					
Apel					

Grapefruit					
Avocado					
FOOD ITEM	HOW MANY TIMES EATEN (on average from the last 10 y) (place a number in one of the columns below if you have ticked the first column) PER DAY / PER WEEK / PER MONTH			Reference serving	Your serve
Pear					
Melon					
Watermelon					
Salak					
Jambu air					
Jambu bol					
Marquisa					
Sawo					
Pineapple					
Jackfruit					
Dried/preserved fruit					
Others:					
Carbohydrate					
Rice: boiled					
fried					
Corn: boiled					
Fried					
Noodle:soup					
fried					
Bread					
Cassava:boiled					
-fried					
Sweet potato:-boiled					
-fried					
Sago					
Refined sugar					
Palm sugar					
Jam					
Sweets					
Chocolate bars					
Other,					
Drinks					

Tea					
Coffee					
FOOD ITEM	HOW MANY TIMES EATEN (on average from the last 10 y) (place a number in one of the columns below if you have ticked the first column) PER DAY / PER WEEK / PER MONTH			Reference serving	Your serve
Chocolate					
Orange juice					
Other fruit juice					
Syrup					
Energy drin					
Herb tea					
Cocacola					
Alcohol					
Ice cream					
Other:					
Snacks					
Lontong					
Sticky rice: boiled					
-b w/coconut					
-eat w/ cascade co					
-lemper					
-lemang					
-fermented					
Cassava crackers					
Cassava cake					
Fermented cassava					
Nagasari					
Fried banana					
Bakwan					
Biscuits					
Gado-gado					
Noddle w/ meat ball					
Martabak					
Rempeyek					
Kue bolu					
Kue bolu coklat					

RAHASIA

KUESIONER FREKUENSI KONSUMSI MAKANAN

MAKANAN TRADISIONAL MINANGKABAU DAN RESIKO PENYAKIT KARDIOVASKULER DI SUMATRA BARAT, INDONESIA

TIM PENELITI

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PETUNJUK PENGISIAN KUSIONER ASUPAN MAKANAN

Kuesioner ini bertujuan untuk mengetahui asupan makanan. Kuesioner ini terdiri dari dua bagian, yaitu:

Bagian A Kuesioner tentang asupan makanan/minuman selama 48 jam (2 hari) terakhir dan dirinci menurut waktu makan (makan pagi, snack pagi, makan siang, snack siang, makan malam dan snack malam).

Bagian B Kuesioner ini menanyakan tentang frekuensi dan jumlah asupan makanan/minuman selama 12 BULAN TERAKHIR.

Perlu diketahui bahwa yang kami ingin ketahui adalah makanan yang ANDA konsumsi (bukan anggota keluarga yang lain).

Untuk menjawab kuesioner ini ada beberapa hal yang perlu anda ketahui:

Frekuensi:
Mohon anda mengingat berapa kali anda makan makanan tertentu, jika makanan itu tersedia (dalam musimnya) dalam kategori dibawah ini:

Harian: jika anda makan makanan tertentu minimal satu kali sehari. Jawablah berapa kali anda memakan makanan tersebut dalam satu hari.
Mingguan: jika anda makan makanan tertentu minimal sekali seminggu. Jawablah berapa kali anda memakan makanan tersebut dalam satu minggu.
Bulanan: jika anda memakan makanan tertentu minimal sekali sebulan. Jawablah berapa kali anda memakan makanan tersebut dalam satu bulan.

Kami juga ingin anda memperkirakan berapa banyak yang anda makan.

BAGIAN A

Hari : ----------
Tanggal: ----------

Waktu makan	Jenis makanan	Ukuran	Keterangan
Makan pagi			
Snack pagi			
Makan siang			
Snack sore			
Makan malam			
Snack malam			

BAGIAN A

Hari : ----------
Tanggal: ----------

Waktu makan	Jenis makanan	Ukuran	Keterangan
Makan pagi			
Snack pagi			
Makan siang			
Snack sore			
Makan malam			
Snack malam			

BAGIAN B

Jenis makanan	FREKUENSI (rata-rata dalam 12 bulan terakhir) PER HARI	PER MINGGU	PER BULAN	PORSI	KETERANGAN
MAKANAN LAUT **Ikan segar (laut/tawar)** -goreng balado					
-goreng					
-gulai					
-rebus (asampadeh)					
-bakar					
-ikan kaleng					
Lain-lain:					
Makanan laut lain: -goreng balado					
-goreng					
- gulai					
Lain-lain:					
Udang					
-Tumis					
-Goreng balado					
-Gulai					
Lain-lain:					
Ikan teri/ikan asin					
-Tumis					
-goreng balado					
-gulai					
-palai					
-lain-lain:					
Telur					
-Goreng balado					
-Dadar/mata sapi					
-rebus					
-gulai					
-teh telur					
-telur burung puyuh:-sup					
-tumis					
-lain-lain					

Jenis makanan	FREKUENSI (rata-rata dalam 12 bulan terakhir) PER HARI	PER MINGGU	PER BULAN	PORSI	KETERANGAN
Tahu					
-goreng					
-goreng balado					
-tumis					
-gulai					
-rebus/asampadeh					
Lain-lain:					
Tempe:					
-goreng					
-goreng balado					
-tumis					
-gulai					
-rebus/asampadeh					
Lain-lain:					
Kacang tanah:					
-goreng					
-goreng balado					
-tumis					
-kacang bogor					
-rebus					
-bakar					
-selai kacang					
-lain-lain:					
Kacang hijau					
-rebus					
-kolak					
- lain-lain:					
Kacang-kacangan lain:					
-rebus					
-goreng					
-santan					
Lain-lain:					

Jenis makanan	FREKUENSI (rata-rata dalam 12 bulan terakhir) PER HARI	PER MINGGU	PER BULAN	PORSI	KETERANGAN
Ayam					
-goreng					
-goreng balado					
-gulai					
-sup sayuran					
-soto					
-sate/bakar					
-semur					
-rendang					
Lain-lain:					
Daging sapi:					
-goreng					
-goreng balado					
-gulai					
-sup sayuran					
-soto					
-sate/bakar					
-semur					
-rendang					
-kornet					
-lain-lain:					
Hati:					
-goreng					
-goreng balado					
-gulai					
-sup sayuran					
-soto					
-sate/bakar					
-semur					
-rendang					
-lain-lain:					

Jenis makanan	FREKUENSI (rata-rata dalam 12 bulan terakhir) PER HARI	PER MINGGU	PER BULAN	PORSI	KETERANGAN
Otak:					
-gulai					
-lain-lain:					
Jerohan lain					
-gulai					
-rendang					
-lain-lain:					
Makanan siap saji					
Hamburger					
Ayam Kentucky					
Kentang Kentucky					
Susu					
-full cream					
-rendah lemak					
-susu kental manis					
-susu sapi segar					
-yoghurt					
-lain-lain					
keju					
Sayur:					
Kangkung:					
-tumis					
-rebus					
-gulai					
-pecal					
Bayam					
-tumis					
-rebus					

Jenis makanan	FREKUENSI (rata-rata dalam 12 bulan terakhir) PER HARI	PER MINGGU	PER BULAN	PORSI	KETERANGAN
Daun ubi					
-rebus					
-gulai					
-palai					
Kol					
Wortel					
Bunga kol					
Terong					
Ketimun					
Tomat					
Slada					
Labu siam					
Sawi					
Tauge					
Kacang panjang					
Kentang:					
-sup					
-rebus					
-goreng					
-'pergedel'					
Lain-lain:					
Buah					
Pisang					
Pepaya					
Jeruk					
Mangga					
Nenas					
Rambutan					
Duku					
Durian					
Apel					
Anggur					
Alpokat					

Jenis makanan	FREKUENSI (rata-rata dalam 12 bulan terakhir) PER HARI	PER MINGGU	PER BULAN	PORSI	KETERANGAN
Pir					
Melon					
Semangka					
Salak					
Jambu air					
Jambu bol					
Markisa					
Sawo					
Nangka					
Buah kering					
Buah kaleng					
Lain-lain:					
Sumber karbohidrat					
Beras: rebus					
goreng					
Jagung: rebus					
Goreng					
Bakar					
Mie: rebus					
goreng					
Roti: + mentega					
+ sele					
+ mentega+sele					
Ubi: rebus					
goreng					
Ubi jalar: rebus					
Goreng					
Sagu					
Permen					
Coklat					
Jam					
Minuman					
Teh + gula					
Kopi + gula					
+ gula + susu					

Jenis makanan	FREKUENSI (rata-rata dalam 12 bulan terakhir) PER HARI	PER MINGGU	PER BULAN	PORSI	KETERANGAN
Coklat + susu + gula					
Orange juice					
Juice buah lain:					
Sirup					
Minuman berenergi					
Jamu					
Cocacola/sprite					
Alkohol					
Es krem					
Lain lain:					
Snacks					
Lontong					
Pulut: rebus					
-+ santan/n. kuning					
-+ kelapa					
-lemper					
-lemang					
-tape					
Kerupuk ubi					
Kue ubi					
Tape					
Nagasari					
Pisang goreng					
Bakwan					
Biskuit					
Gado-gado					
Mie bakso					
Martabak					
Rempeyek					
Kue bolu					
Kue bolu coklat					
Pecal					
Siomay					

IN CONFIDENCE

CLINICAL EXAMINATIONS

MINANGKABAU TRADITIONAL DIET AND CARDIOVASCULAR DISEASE RISK IN WEST SUMATRA, INDONESIA

Investigators

Dr Nur Indrawaty Lipoeto
Prof. Mark L Wahlqvist, B.Med.Sc, MD BS (Adelaide), MD (Uppsala), FRACP, FAIFST, FACN
Dr Naiyana Wattanapenpaiboon

Monash University, Medical School, Department of Medicine, Monash Medical Centre
246 Clayton Rd, Clayton, VIC 3168, AUSTRALIA

ASSESSMENTS

	Assessment I	Assessment II
Blood Pressure	_____	_____
Body Composition		
Weight (kg)	_____	_____
Height (cm)	_____	_____
BMI (kg/m2)	_____	_____
Waist (cm)	_____	_____
Abdominal (cm)	_____	_____
Hip (cm)	_____	_____
Skinfold thickness		
Biceps skinfold (mm)	_____	_____
Triceps skinfold (mm)	_____	_____
Subscapular skinfold (mm)	_____	_____
Suprailiac (mm)	_____	_____
Biochemistry assessment		
Total cholesterol (mg/dl)	_____	_____
LDL cholesterol (mg/dl)	_____	_____
HDL cholesterol (mg/dl)	_____	_____
Triglyceride (mg/dl)	_____	_____
Lipoprotein (a) (mg/l)	_____	_____
Plasma Phospholipid composition		
Glucose (mg/dl)	_____	_____
Insulin (µmol/L)	_____	_____

Informed Consent Form

Project Title: Traditional coconut usage and cardiovascular disease in West Sumatra, Indonesia
(Group discussion on coconut usage)

I agree to take part in the above Monash University research project. I have had the project explained to me, and I have read the Explanatory Statement, which I keep for my records. I understand that agreeing to take part means that I am willing to *participate in a group discussion* to discuss how we prepare traditional foods at present and in the last times.

I understand that any information I provide will be tape recorded and kept confidential, and that no information that could lead to the identification of any individual will be disclosed in any reports on the project, or to any other party.

I also understand that my participation is voluntary, that I can choose not to participate in part or all of the project, and that I can withdraw at any stage of the project without being penalised or disadvantaged in any way.

Please tick the appropriate box

- ☐ The information I provide can be used by other researchers as long as my name and contact information is removed before it is given to them
- ☐ The information I provide cannot be used by other researchers without asking me first
- ☐ The information I provide cannot be used except for this project

Name: ..... (please print)

Signature: ..... Date: .....

Independent witness to participant's voluntary and informed consent

I believe that ..... understands the above project and gives her/his consent voluntarily

Name: ..... (please print)

Signature: ..... Date: .....

Address .....

(Note: the Consent statement will be translated into Indonesian.)

SURAT PERNYATAAN PERSETUJUAN

Judul Penelitian: **Makanan Tradisional Minang dan Penyakit Kardiovaskuler di Sumatra Barat, Indonesia**
(Diskusi kelompok)

Dengan ini saya menyatakan bahwa saya setuju untuk ikut serta dalam penelitian dari Universitas Monash, Australia. Peneliti telah menerangkan pada saya maksud dan tujuan penelitian, saya juga telah membaca surat keterangan tentang penelitian yang akan saya simpan. Saya sadar bahwa dengan kesediaan saya, berarti saya setuju ikut dalam diskusi kelompok yang akan membicarakan tentang makanan tradisional Minang pada saat dulu dan sekarang.

Saya setuju bahwa diskusi akan direkam dan hasilnya akan disimpan oleh peneliti, dan tidak akan ada informasi yang dapat mengidentifikasi seseorang yang akan tercantum dalam laporan penelitian ini atau diberikan pada kelompok lain.

Saya setuju bahwa keikutsertaan saya adalah sukarela, saya dapat memilih tidak akan mengikuti sebagian atau seluruh bagian penelitian, dan saya boleh berhenti kapan saja tanpa didenda atau dikenai bentuk hukuman lain.

Pilih kolom yang sesuai:

- Keterangan yang saya berikan dapat dipakai oleh peneliti lain jika nama dan alamat saya dihapus sebelum diberikan.
- Keterangan yang saya berikan tidak dapat dipakai oleh peneliti lain tanpa menanyakan kesedian saya terlebih dahulu.
- Keterangan yang saya berikan hanya untuk penelitian ini dan tidak dapat dipakai oleh peneliti lain.

Nama:.....(mohon diketik)
Tandatangan:.....Tanggal:.....

Saksi:
Saya percaya bahwa.....telah mengerti tentang penelitian ini dan saya setuju bersaksi untuknya:

Nama:.....(mohon diketik)
Tandatangan:.....Tanggal:.....
Alamat:.....

Informed Consent Form

Project Title: Traditional coconut usage and cardiovascular disease in West Sumatra, Indonesia
(Case-Control Study)

I agree to take part in the above Monash University research project. I have had the project explained to me, and I have read the Explanatory Statement, which I keep for my records. I understand that agreeing to take part means that I am willing to:

- be interviewed by the researcher
- provide samples of blood
- provide samples of 24 hour urine collection
- participate in body composition assessment
- make myself available for a further interview should that be required
- allow the researchers to have access to my medical records

I understand that any information I provide is confidential, and that no information that could lead to the identification of any individual will be disclosed in any reports on the project, or to any other party.

I also understand that my participation is voluntary, that I can choose not to participate in part or all of the project, and that I can withdraw at any stage of the project without being penalised or disadvantaged in any way.

Please tick the appropriate box

- ☐ The information I provide can be used by other researchers as long as my name and contact information is removed before it is given to them
- ☐ The information I provide cannot be used by other researchers without asking me first
- ☐ The information I provide cannot be used except for this project

Name: ..... (please print)

Signature: ..... Date: .....

Independent witness to participant's voluntary and informed consent

I believe that ..... understands the above project and gives her/his consent voluntarily

Name: ..... (please print)

Signature: ..... Date: .....

Address .....

(Note: the Consent statement will be translated into Indonesian.)

Informed Consent Form

Project Title: Traditional coconut usage and cardiovascular disease in West Sumatra, Indonesia
(Intervention Study)

I agree to take part in the above Monash University research project. I have had the project explained to me, and I have read the Explanatory Statement, which I keep for my records. I understand that agreeing to take part means that I am willing to:

- To follow the diet pattern as instructions.
- provide samples of blood
- provide samples of 24 hour urine collection
- participate in body composition assessment

I understand that any information I provide is confidential, and that no information that could lead to the identification of any individual will be disclosed in any reports on the project, or to any other party.

I also understand that my participation is voluntary, that I can choose not to participate in part or all of the project, and that I can withdraw at any stage of the project without being penalised or disadvantaged in any way.

Please tick the appropriate box

- ☐ The information I provide can be used by other researchers as long as my name and contact information is removed before it is given to them
- ☐ The information I provide cannot be used by other researchers without asking me first
- ☐ The information I provide cannot be used except for this project

Name: ..... (please print)

Signature: ..... Date: .....

Independent witness to participant's voluntary and informed consent

I believe that ..... understands the above project and gives her/his consent voluntarily

Name: ..... (please print)

Signature: ..... Date: .....

Address .....

(Note: the Consent statement will be translated into Indonesian.)

SURAT PERNYATAAN PERSETUJUAN

Judul Penelitian: **Makanan Tradisional Minang dan Penyakit Kardiovaskuler di Sumatra Barat, Indonesia**
(Studi Intervensi)

Dengan ini saya menyatakan bahwa saya setuju untuk ikut serta dalam penelitian dari Universitas Monash, Australia. Peneliti telah menerangkan pada saya maksud dan tujuan penelitian, saya juga telah membaca surat keterangan tentang penelitian yang akan saya simpan. Saya sadar bahwa dengan kesediaan saya, berarti saya setuju untuk:

- Mengikuti menjalankan petunjuk diet yang diberikan
- Diambil sampel darah
- Menyediakan urin 24 jam
- Ikut dalam pengukuran komposisi tubuh

Saya setuju bahwa segala informasi yang saya berikan sifatnya rahasia, dan tidak akan ada informasi yang dapat mengidentifikasi seseorang yang akan tercantum dalam laporan penelitian ini atau diberikan pada kelompok lain.

Saya setuju bahwa keikutsertaan saya adalah sukarela, saya dapat memilih tidak akan mengikuti sebagian atau seluruh bagian penelitian, dan saya boleh berhenti kapan saja tanpa didenda atau dikenai bentuk hukuman lain.

Pilih kolom yang sesuai:

- Keterangan yang saya berikan dapat dipakai oleh peneliti lain jika nama dan alamat saya dihapus sebelum diberikan.
- Keterangan yang saya berikan tidak dapat dipakai oleh peneliti lain tanpa menanyakan kesedian saya terlebih dahulu.
- Keterangan yang saya berikan hanya untuk penelitian ini dan tidak dapat dipakai oleh peneliti lain.

Nama:.....(mohon diketik)
Tandatangan:.....Tanggal:.....

Saksi:
Saya percaya bahwa.....telah mengerti tentang penelitian ini dan saya setuju bersaksi untuknya:

Nama:.....(mohon diketik)
Tandatangan:.....Tanggal:.....
Alamat:.....

Project Title: Traditional coconut usage and cardiovascular disease in West Sumatra, Indonesia
(Group discussion on coconut usage)

My name is ***Dr Nur Indrawaty Lipoeto*** and I am doing research under the supervision of ***Prof Mark L Wahlqvist*** the Head of *Department of Medicine in Monash University* and **Dr Zulkarnain Agus** the Head of *Department of Nutrition of Medical Faculty of Andalas University* in Padang towards a PhD at Monash University in Melbourne, Australia.

The incidence of cardiovascular disease (CVD) is increasing in Indonesia including in West Sumatra. This study aims to ascertain incidence of CVD in West Sumatra, to document Minangkabau food culture, to investigate pattern of coconut and non coconut consumption and to identify changes of life style that relates to the development of CVD. I hope the results of this research project will improve nutrition planning in relation with CVD in Indonesia and internationally.

Men and women aged 65 years or over who live in this village, are eligible for this study. If you agree to be involved in this study, you will be invited to come to ..... <<venue>> ..... where we will have a group discussion. We will discuss about how we cook foods at present and in the past. The discussion will be tape-recorded and will take about 1-2 hours.

The confidentiality of your personal information will be safeguarded. No findings which could identify any individual participant will be published. The anonymity of your participation is assured by our procedure, in which only the combined results of participants will be published. Access to data is restricted to the investigation team. Coded data are stored for five years at the Department of Medicine, as prescribed by Monash University regulations.

Participation in this research is entirely voluntary. If you agree to participate you may withdraw at any time. You may also choose not to answer some of the questions.

If you have any queries or would like to be informed of the aggregate research finding, please contact Department of Nutrition at the Medical Faculty of Andalas University by phone 0751-39223 or fax: 0751-32838; or write to Prof Mark L Wahlqvist, Monash University Department of Medicine, Monash Medical Centre, 246 Clayton Road, Clayton VIC 3168, Australia, phone (001) 61 3 9550 5525, fax (001) 61 3 9550 5437

DEPARTMENT OF MEDICINE
MONASH MEDICAL CENTRE

Makanan Tradisional Minangkabau
dan Penyakit Kardiovaskuler di Sumatra Barat, Indonesia
(Diskusi Kelompok)

Nama saya Dr. Nur Indrawaty Lipoeto, saya adalah staf pengajar Bagian Gizi Fakultas Kedokteran Universitas Andalas yang dipimpin oleh Dr. Zulkarnain Agus MPH, MSc . Pada saat sekarang saya sedang mengikuti pendidikan S3 di Monash University, Australia dibawah bimbingan Prof. Mark L. Wahlqvist, Kepala Departemen Penyakit Dalam di Monash University, Melbourne, Australia.

Pada saat sekarang penyakit Jantung dan pembuluh darah sedang meningkat kejadiannya di Indonesia dan khususnya di Sumatra Barat. Penelitian ini dilaksanakan untuk mengetahui kejadian penyakit tersebut di Sumatra Barat, untuk mengetahui budaya makan orang Minangkabau, menganalisa pola konsumsi kelapa dan jenis makanan lain dan perubahan pola hidup yang dapat mempengaruhi perkembangan terjadinya penyakit jantung dan pembuluh darah tersebut. Hasil penelitian ini diharapkan dapat memperbaiki program penanggulangan gizi terutama penanggulangan penyakit jantung dan pembuluh darah di Indonesia dan di dunia Internasional.

Penelitian ini membutuhkan laki-laki dan perempuan dari desa ini yang berumur diatas 65 tahun. Jika Ibu/Bapak bersedia ikut dalam penelitian ini, Ibu/Bapak diundang untuk datang ke:

Tempat :.....
Hari/tanggal :.....
Jam :.....

untuk mengikuti diskusi kelompok. Diskusi akan membicarakan masakan Minangkabau. Diskusi akan direkam dan akan berjalan selama lebih kurang 1 - 2 jam.

Kerahasiaan data Ibu/Bapak akan kami jaga. Hasil penelitian ini tidak akan menampilkan identitas Ibu/Bapak. Kerahasiaan itu dijamin oleh karena hanya data kombinasi saja yang akan muncul dalam laporan penelitian. Data hanya dapat dilihat oleh tim peneliti. Data yang telah diberi kode akan disimpan selama lima tahun di Departemen Penyakit Dalam Monash University, sesuai peraturan di Monash University.

Keikursertaan Bapak dan Ibu dalam penelitian ini adalah sukarela. Jika Bapak/ibu ikut serta, ibu/bapak dapat keluar kapan saja. Ibu/bapak juga boleh saja tidak menjawab pertanyaan-pertanyaan kami.

Jika ingin mengetahui lebih lanjut informasi tentang penelitian ini, mohon menghubungi Bagian Gizi, Fakultas Kedokteran Universitas Andalas, tilpon 0751-39223 atau fax: 0751-32838, atau menulis surat ke Prof Mark L. Wahlqvist, di Monash University Department of Medicine, Monash Medical Centre, 246 Clayton rd, Clayton, VIC 3168, Australia, tilpon: 001-61-3-9550 5525, fax 001-61-3-95505437

Project Title: Traditional coconut usage and cardiovascular disease in West Sumatra, Indonesia
(Case-Control Study)

My name is ***Dr Nur Indrawaty Lipoeto*** and I am doing research under the supervision of ***Prof Mark L Wahlqvist*** the Head of *Department of Medicine in Monash University* and **Dr Zulkarnain Agus** the Head of *Department of Nutrition of Medical Faculty of Andalas University* in Padang towards a PhD at Monash University in Melbourne, Australia.

The incidence of cardiovascular disease (CVD) is increasing in Indonesia including in West Sumatra. This study aims to ascertain incidence of CVD in West Sumatra, to document Mināṅgkabau food culture, to investigate pattern of coconut and non coconut consumption and to identify changes of life style that relates to the development of CVD. I hope the results of this research project will improve nutrition planning in relation with CVD in Indonesia and internationally.

Men and women aged 35 years or over, who have been admitted to hospital and diagnosed suffering from CVD are eligible for this study. Pregnant or lactating women will be excluded. If you agree to be involved in this study, I will ask you questions about your life style, health status, usual food consumption and cooking methods. This interview will take about 1-2 hours. Then an appointment will be made for you to have a physical examination and have your blood taken at a health centre.

On the night before the appointment you will be asked to fast from 10.00 pm, and not to have breakfast on the following morning. About 30 millilitres of blood will be taken by a qualified nurse. Your physical examination including weight, height and body composition measurements will be performed by me or one of our research assistants. You will also be asked to collect your urine over a 24-hour period in a plastic bottle provided to you. The inconvenience or discomfort may occur during blood or urine collection.

If you are suffering from CVD, you will be assigned as *a case group*. You will be asked to nominate two of your friends or neighbours who are at the same age, with the same gender, and do not have CVD or any CVD-related disorders, to serve as *a control group*. They will undergo all the same procedures as you do. We will also require to get access to medical records of you and your nominees.

The confidentiality of your personal information will be safeguarded. No findings which could identify any individual participant will be published. The anonymity of your participation is assured by our procedures, in which only the combined results of participants will be published. Access to data is restricted to the investigation team. Coded data are stored for five years at the Department of Medicine, as prescribed by Monash University regulations.

Your participation and your nominees' in this study is entirely voluntary. If you (or your nominees) agree to participate you may withdraw at any time. You may also choose not to answer some of the questions.

If you have any queries or would like to be informed of the aggregate research finding, please contact Department of Nutrition at the Medical Faculty of Andalas University by phone 0751-39223 or fax: 0751-32838; or write to Prof Mark L Wahlqvist, Monash University Department of Medicine, Monash Medical Centre, 246 Clayton Road, Clayton VIC 3168, Australia, phone (001) 61 3 9550 5525, fax (001) 61 3 9550 5437.

Should you have any complaint concerning the manner in which this research (project number) is conducted, please do not hesitate to contact The Standing Committee on Ethics in Research on Humans at the following address:

The Secretary
The Standing Committee on Ethics in Research on Humans
Monash University
Wellington Road
Clayton Victoria 3168
Australia
Telephone (001) 61 - 3 - 9905 2052; Fax (001) 61 - 3 - 9905 1420

Thank you

_____(signature)

Dr Nur Indrawaty Lipoeto
(001) 61-3-9550 5549

(Note: the Explanation statement will be translated into Indonesian.)

Judul penelitian: **Makanan Tradisional Minang dan Penyakit Kardiovaskuler di Sumatra Barat, Indonesia**
(Studi Kasus Kelola)

Nama saya Dr. Nur Indrawaty Lipoeto, saya adalah staf pengajar Bagian Gizi Fakultas Kedokteran Universitas Andalas yang dipimpin oleh Dr. Zulkarnain Agus MPH, MSc . Pada saat sekarang saya sedang mengikuti pendidikan S3 di Monash University, Australia dibawah bimbingan Prof. Mark L. Wahlqvist, Kepala Departemen Penyakit Dalam di Monash University, Melbourne, Australia

Pada saat sekarang penyakit Jantung dan pembuluh darah sedang meningkat kejadiannya di Indonesia dan khususnya di Sumatra Barat. Penelitian ini dilaksanakan untuk mengetahui secara pasti kejadian penyakit tersebut di Sumatra Barat, untuk mengetahui budaya makan orang Minangkabau, menganalisa pola konsumsi kelapa dan jenis makanan lain dan perubahan pola hidup terhadap perkembangan terjadinya penyakit jantung dan pembuluh darah tersebut. Hasil penelitian ini diharapkan dapat memperbaiki program penanggulangan gizi terutama penanggulangan penyakit jantung dan pembuluh darah di Indonesia dan di dunia Internasional.

Penelitian ini membutuhkan laki-laki dan perempuan yang berumur diatas 35 tahun yang telah pernah dirawat di Rumahsakit dengan diagnosa penyakit Jantung dan pembuluh darah. Ibu hamil, atau menyusui tidak termasuk. Jika anda bersedia ikut dalam penelitian ini, saya akan menanyakan beberapa pertanyaan tentang keadaan kesehatan, aktifitas sehari-hari, makanan yang dimakan dan cara memasaknya. Interview itu akan memakan waktu kira-kira 1-2 jam. Kemuadian anda akan diundang datang ke puskesmas untuk pemeriksaan fisik dan pengambilan darah.

Pada malam sebelum kepuskesmas, anda diminta agar berpuasa dari jam 22.00, dan menunda sarapan pagi esok harinya sampai kami melakukan pemeriksaan darah. Kira-kira 30 mililiter darah akan kami ambil. Pemeriksaan fisik yang kami lakukan meliputi pengukuran tinggi, berat, komposisi tubuh, pemeriksaan akan dilakukan oleh tim peneliti. Anda juga dimohon untuk dapat mengumpulkan urin selama 24 jam dalam botol yang telah kami siapkan. Dalam pengambilan darah atau pengumpulan urin mungkin akan ada rasa sakit atau dapat mengganggu kenyamanan anda, untuk itu kami mohon maaf terlebih dahulu.

Jika anda penderita penyakit jantung dan pembuluh darah, maka anda termasuk dalam *Kelompok Kasus*, anda kemudian kami minta untuk mengajukan dua nama kawan atau tetangga anda yang mempunyai umur dan jenis kelamin yang sama dengan anda tapi diketahui sehat dan tidak pernah menderita penykait jantung dan pembuluh darah untuk diminta ikut serta dalam penelitian ini sebagai anggota *Kelompok Kelola* . Kelompok ini juga akan melalui semua prosedur yang sama dengan anda. Untuk mengetahui riwayat penyakit anda, untuk itu kami mohon izin dari anda agar kami dapat memeriksa catatan kesehatan anda yang ada di Rumahsakit.

Jika anda mempunyai keluhan tentang pelaksaan penelitian ini (nomor proyek:), mohon menghubungi Komite Etik Penelotian pada Manusia Monash University pada alamat dibawah ini:

The Secretary
The Standing Committee on Ethics in Research on Humans
Monash University
Wellington Road
Clayton Victoria 3168
Australia
Telepon (001) 61 - 3 - 9905 2052; Fax (001) 61 - 3 - 9905 1420

Terima kasih

_____(tandatangan)

Dr Nur Indrawaty Lipoeto
(001) 61-3-9550 5549

Project Title: Traditional coconut usage and cardiovascular disease in West Sumatra, Indonesia
(Intervention Study)

My name is ***Dr Nur Indrawaty Lipoeto*** and I am doing research under the supervision of ***Prof Mark L Wahlqvist*** the Head of *Department of Medicine in Monash University* and **Dr Zulkarnain Agus** the Head of *Department of Nutrition of Medical Faculty of Andalas University* in Padang towards a PhD at Monash University in Melbourne, Australia.

The incidence of cardiovascular disease (CVD) is increasing in Indonesia including in West Sumatra. This study aims to ascertain incidence of CVD in West Sumatra, to document Minangkabau food culture, to investigate pattern of coconut and non coconut consumption and to identify changes of life style that relates to the development of CVD. I hope the results of this research project will improve nutrition planning in relation with CVD in Indonesia and internationally.

Men and women aged 35-55 years without CVD are eligible for this study. Pregnant or lactating women will be excluded. People who agree to be involved in this study will be assigned into one of three groups. The first group will be asked to maintain their usual consumption of coconut products, the second group to replace coconut oil in cooking with other vegetable oil, and the third group to reduce consumption of coconut products by half over a period of 6 months.

At study entry (month 0), you will be asked questions about your life style, health status, usual food consumption and cooking methods. This interview will take about 1-2 hours. Then an appointment will be made for you to have a physical examination and have your blood taken at a health centre.

On the night before the appointment you will be asked to fast from 10.00 pm, and not to have breakfast the following morning. About 30 millilitres of blood will be taken by a qualified nurse. Your physical examination including weight, height and body composition measurements will be performed by me or one of our research assistants. You will also be asked to collect your urine over a 24-hour period in a plastic bottle provided to you. The inconvenience or discomfort may occur during blood or urine collection.

During the intervention period, you will be asked to provide blood and urine samples at months 3 and 6, and the physical examination will be repeated at the end of the study (month 6).

The confidentiality of your personal information will be safeguarded. No findings which could identify any individual participant will be published. The anonymity of your participation is assured by our procedures, in which only the combined results of participants will be published. Access to data is restricted to the investigation team. Coded data are stored for five years at the Department of Medicine, as prescribed by Monash University regulations.

Participation in this research is entirely voluntary. If you agree to participate you may withdraw at any time. You may also choose not to answer some of the questions.

If you have any queries or would like to be informed of the aggregate research finding, please contact Department of Nutrition at the Medical Faculty of Andalas University by phone 0751-39223 or fax: 0751-32838; or write to Prof Mark L Wahlqvist, Monash University Department of Medicine, Monash Medical Centre, 246 Clayton Road, Clayton VIC 3168, Australia, phone (001) 61 3 9550 5525, fax (001) 61 3 9550 5437.

Should you have any complaint concerning the manner in which this research (project number) is conducted, please do not hesitate to contact The Standing Committee on Ethics in Research on Humans at the following address:

The Secretary
The Standing Committee on Ethics in Research on Humans
Monash University
Wellington Road
Clayton Victoria 3168
Australia
Telephone (001) 61 - 3 - 9905 2052; Fax (001) 61 - 3 - 9905 1420

Thank you

_____(signature)

Dr Nur Indrawaty Lipoeto
(001) 61-3-9550 5549

(Note: the Explanation statement will be translated into Indonesian.)

Judul penelitian: **Makanan Tradisional Minang dan Penyakit Kardiovaskuler di Sumatra Barat, Indonesia**
(Studi Intervensi)

Nama saya Dr. Nur Indrawaty Lipoeto, saya adalah staf pengajar Bagian Gizi Fakultas Kedokteran Universitas Andalas yang dipimpin oleh Dr. Zulkarnain Agus MPH, MSc . Pada saat sekarang saya sedang mengikuti pendidikan S3 di Monash University, Australia dibawah bimbingan Prof. Mark L. Wahlqvist, Kepala Departemen Penyakit Dalam di Monash University, Melbourne, Australia.

Pada saat sekarang penyakit Jantung dan pembuluh darah sedang meningkat kejadiannya di Indonesia dan khususnya di Sumatra Barat. Penelitian ini dilaksanakan untuk mengetahui secara pasti kejadian penyakit tersebut di Sumatra Barat, untuk mengetahui budaya makan orang Minangkabau, menganalisa pola konsumsi kelapa dan jenis makanan lain dan perubahan pola hidup terhadap perkembangan terjadinya penyakit jantung dan pembuluh darah tersebut. Hasil penelitian ini diharapkan dapat memperbaiki program penanggulangan gizi terutama penanggulangan penyakit jantung dan pembuluh darah di Indonesia dan di dunia Internasional.

Penelitian ini membutuhkan laki-laki dan perempuan yang berumur diatas 35 tahun yang sehat dan tidak menderita penyakit jantung dan pembuluh darah. Ibu hamil, atau menyusui tidak termasuk. Jika anda bersedia ikut dalam penelitian ini, peserta akan kami bagi dalam tiga kelompok. Kelompok pertama akan diminta untuk tetap mengkonsumsi makanan seperti biasa. Kelompok keduaakan diminta untuk mengganti minyak kelapa dengan minyak goreng lain. Kelompok ketigas akan diminta untuk mengurangi konsulsi kelapa hingga setengah dan menggantinya dengan makanan tradisional lain selama kira-kira enam bulan.

Pada waktu studi dimulai, saya akan menanyakan beberapa pertanyaan tentang keadaan kesehatan, aktifitas sehari-hari, makanan yang dimakan dan cara memasaknya. Interview itu akan memakan waktu kira-kira 1-2 jam. Kemudian anda akan diundang datang ke puskesmas untuk pemeriksaan fisik dan pengambilan darah.

Pada malam sebelum kepuskesmas anda diminta agar berpuasa dari jam 22.00, dan menunda sarapan pagi esok harinya sampai kami melakukan pemeriksaan darah. Kira-kira 30 mililiter darah akan kami ambil. Pemeriksaan fisik yang kami lakukan meliputi pengukuran tinggi badan, komposisi tubuh, pemeriksaan akan dilakukan oleh tim peneliti. Anda juga dimohon untuk dapat mengumpulkan urin selama 24 jam dalam botol yang telah kami siapkan. Dalam pengambilan darah atau pengumpulan urin mungkin akan ada rasa sakit atau dapat mengganggu kenyamanan anda, untuk itu kami mohon maaf terlebih dahulu. Pemeriksaan akan diulang pada bulan ketiga dan keenam.

Kerahasiaan data anda akan kami jaga. Hasil penelitian ini tidak akan menampilkan identitas anda. Kerahasiaan itu dijamin oleh karena hanya data kombinasi saja yang akan muncul dalam laporan penelitian. Data hanya dapat dilihat oleh tim peneliti. Data yang telah diberi kode akan disimpan selama lima tahun di Departemen Penyakit Dalam Monash University, sebagaimana yang diatur oleh pihak Monash University.

Keikursertaan anda dalam penelitian ini adalah sukarela. Jika telah bersedia, anda dapat keluar kapan saja. Anda juga dapat memilih tidak menjawab pertanyaan-pertanyaan kami.

Jika ingin mengetahui lebih lanjut informasi tentang penelitian ini, mohon menghubungi Bagian Gizi, Fakultas Kedokteran Universitas Andalas, tilpon 0751-39223 atau fax: 0751-32838, atau menulis surat ke Prof Mark L. Wahlqvist, di Monash University Department of Medicine, Monash Medical Centre, 246 Clayton rd, Clayton, VIC 3168, Australia, tilpon: 001-61-3-9550 5525, fax 001-61-3-95505437

Jika anda mempunyai keluhan tentang pelaksaan penelitian ini (nomor proyek:), mohon menghubungi Komite Etik Penelotian pada Manusia Monash University pada alamat dibawah ini:

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Telepon (001) 61 - 3 - 9905 2052; Fax (001) 61 - 3 - 9905 1420

Terima kasih

_____(tandatangan)

Dr Nur Indrawaty Lipoeto
(001) 61-3-9550 5549

PEMERINTAH PROPINSI DAERAH TINGKAT I SUMATERA BARAT

DIREKTORAT SOSIAL POLITIK

Jln. Jend. Sudirman No. 51 Telp. 34224, 34475 Padang

REKOMENDASI

No. B. 070/791 /Sospol/ X /19 98.-

Tentang

Izin Melaksanakan Penelitian/Survey

Kami Gubernur Kepala Daerah Tingkat I Sumatera Barat, setelah mempelajari surat : Dekan Fakultas Kedokteran Universitas Andalas Padang No.2552/J.16.2/PL/1998 tanggal 13 Oktober 1998 perihal mohon izin-penelitian,
dengan ini menyatakan tidak keberatan atas maksud melaksanakan penelitian di Daerah Sumatera Barat yang dilakukan oleh :

Nama	:	Dr. NUR INDRAWATI LIPOTO MSc.
Tempat/Tanggal Lahir	:	Tapak Tuan, 7 Mei 1963.
Pekerjaan	:	Dosen Fak.Kedokteran Univ.Andalas Padang.
Alamat	:	Jl. Gajah No.7 Padang.
Nomor Kartu Identitas	:	NIP. 131873970.
Maksud Judul Penelitian	:	" POLA KONSUMSI DAN PENYAKIT KARDIOVASKULER - DI SUMATERA BARAT."
Lokasi/Tempat Penelitian	:	Kabupaten dan Kotamadya Se-Sumbar.
Waktu Penelitian	:	17 Oktober 1998 s/d. 20 Januari 1999.
Anggota	:	-.-

dengan ketentuan sebagai berikut :

1. Tidak boleh menyimpang dari kerangka serta tujuan penelitian
2. Memberitahukan kedatangan serta maksud penelitian yang akan dilaksanakan dengan menunjukan surat-surat keterangan yang berhubungan dengan itu, serta melaporkan diri sebelum meninggalkan Daerah penelitiannya kepada PEMDA setempat
3. Mematuhi semua peraturan yang berlaku dan menghormati adat istiadat serta kebijaksanaan Masyarakat setempat
4. Mengirimkan laporan hasil penelitiannya sebanyak 1 (satu) Eks kepada Gubernur KDH Tk. I Sumbar Cq. Kadit Sospol
5. Bila terjadi penyimpangan/pelanggaran terhadap ketentuan tersebut diatas, maka surat rekomendasi ini akan dicabut kembali

Demikianlah rekomendasi izin Penelitian/Survey ini diberikan kepada yang bersangkutan untuk dapat dipergunakan oleh yang berkepentingan dimana perlu

Padang, 16 Oktober 1998.-

An. GUBERNUR KDH TINGKAT I SUMATERA BARAT
KEPALA DIREKTORAT SOSIAL POLITIK
UB. KABID PENGAMANAN

[redacted]

NIP. 010074417.-

Kepada Yhs.
TEMBUSAN
1. Mendagri Cq. Dirjen Sospol di Jakarta
2. Bupati Kdh Tk.II Se-Sumbar.
3. Walikotamadya Kdh Tk.II Se-Sumbar.
4. Dekan Fak.Kedokteran UNAND Padang.
5. Arsip. —

DEPARTEMEN KESEHATAN RI
KANTOR WILAYAH PROVINSI
SUMATERA BARAT

Jalan Perintis Kemerdekaan No. 65 A
Padang 25001

Telepon : 25642 (Kepala) 25874 (Umum)
Telex : 55239
Facsimile : 33437

Nomor : DI 01 03. 1244 98 Padang 18 Februari 1999
Lampiran : -
Perihal : Izin Melakukan Penelitian

Kepada Yth :

Cdr Direktur Rumah Sakit :

PUSAT Dr. M. DJAMIL PADANG

di

P a d a n g

Sehubungan dengan surat dari Fakultas Kedokteran Universitas Andalas Padang Nomor : 42/J16.2/PL./1999, tanggal 6 Januari 1999, perihal sama dengan pokok surat diatas, pada prinsipnya kami dapat menyetujuinya, staf Pengajar Fakultas Kedokteran Unand Padang tersebut adalah :

N a m a : Dr.Nur Indrawati Lipoto M.Sc
N I P : 133873970
Jabatan : Staf Pengajar Bagian Ilmu Gizi Fakultas Kedokteran Unand Padang
Alamat : Jl Gajah Mada No 7 Padang

untuk melaksanakan penelitian dengan judul :

" Makanan Tradisional Minangkabau dan Penyakit Kardio vaskuler di Sumatera Barat "

Untuk itu kami harapkan bantuan saudara agar penelitian tersebut dapat berjalan dengan lancar

Demikianlah disampaikan, atas bantuan dan kerja samanya diucapkan terima kasih

Kepala Kantor Wilayah
Koordinator Administrasi

[redacted]

Dr H. [redacted] f Abdullah, SKM)
NIP : 410 002 749

Tembusan kepada Yth :
1. Cdr Dekan Fakultas Kedokteran Unand Padang
2. Perinnggal (File D4)

DEPARTEMEN KESEHATAN R.I
DIREKTORAT JENDERAL PELAYANAN MEDIK
RSUP. Dr. M. DJAMIL
Jl. Perintis Kemerdekaan Padang - 25127
Direktur - Telp. & Fac. : 32371
SLTO (Seluruh Bagian) : 32373 Hunting System / 6 Saluran

Padang, 3 Maret 1999

Nomor : LB.00.02.07. 306 .
Lamp : --
Perihal : Izin Mengadakan Penelitian
A/n. Dr.Nur Indrawati Liputo, MSc

Kepada Yth :
Sdr. Pembantu Dekan I
Fakultas Kedokteran Unand
Di -
PADANG
- -

Dengan hormat,
Membalas surat Saudara nomor : 41/J.16.2/PL/1998 tanggal 6 Januari 1999 Perihal tersebut pada pokok surat diatas, dengan ini disampaikan bahwa pada prinsipnya kami tidak keberatan memberi izin kepada :

N a m a : Dr.Nur Indrawati Liputo, MSc
N I P : 131 873 970
Jabatan : Staf Pengajar di Bagian Ilmu Gizi FK-Unand

Untuk pengambilan data di RSUP Dr.M.Djamil Padang dalam rangka pembuatan Karya Tulis yang berjudul :

" MAKANAN TRADISIONAL MINANG KABAU DAN PENYAKIT KARDIOVASKULER DI SUMATERA BARAT "

Dengan catatan sebagai berikut :
1. Semua data yang diperoleh di RSUP Dr.M.Djamil Padang semata-mata digunakan untuk perkembangan ilmu pengetahuan dan tidak disebar luaskan pada pihak lain
2. Penulisan karya tulis sebelum diketik bersih harus diketahui/disetujui oleh Kabid Diklit RSUP Dr.M.Djamil Padang
3. Harus menyerahkan 1 (satu) eksemplar karya tulisnya ke Perpustakaan RSUP Dr.M.Djamil Padang
4. Segala hal yang menyangkut pembiayaan penelitian adalah tanggung jawab sipeneliti

Demikianlah untuk dimaklumi, atas perhatian Saudara diucapkan terima kasih.

a.n. Direktur
Wadir Jang Medik & Pendidikan

RSUP. Dr. M. DJAMIL PADANG

Dr.H.Syahvar Iskan
NIP. 140 123 09[redacted]

Tembusan :
1. Ka.Kanwil Dep.Kes RI Prop.Sumbar di Padang
2. Ka.Subag Medical Record RSUP Dr.M.Djamil Padang
3. Ka.Instalasi Rawat Inap C (Penyakit Dalam) RSUP Dr.M.Djamil Padang
4. Yang bersangkutan
5. Arsip.

RUMAH SAKIT YOS SUDARSO

JALAN SITUJUH No. 1 PADANG - 25129
Telp. 33230 - 33231

Nomor : 136/K/III/RSYS/1999
Perihal : *Ijin Mengadakan Penelitian*

Padang, 15 Pebruari 1999

Kepada Yth.
Dr. Fadil Oenzil, Ph.D
Dekan Fakultas Kedokteran
Universitas Andalas Padang
di
P a d a n g

Dengan hormat,

Surat Saudara No. 225/J 16.2/PL/1999 perihal di atas telah kami terima. Terima kasih atas kepercayaan yang diberikan kepada rumah sakit kami untuk menjadi tempat penelitian Staff Pengajar Saudara.

Pada Prinsipnya kami tidak berkeberatan atas kegiatan tersebut, bilamana dapat memenuhi ketentuan sebagai berikut :

1. Data yang kami berikan semata-mata hanya untuk kepentingan ilmiah.
2. Data tersebut tidak untuk dipublikasikan.
3. Satu salinan karya tulis tersebut diserahkan ke RS Yos Sudarso untuk mendapat pengesahan atas data yang telah ditulis, sebelum diserahkan ke lembaga pendidikan.
4. Pembiayaan yang timbul atas kegiatan tersebut menjadi tanggungan yang besangkutan.
5. Kesepakatan atas ketentuan yang kami berikan dituangkan dalam surat perjanjian yang ditandatangani kedua belah fihak dan dibubuhi dengan meterai yang cukup.
6. Penelitian dapat dilaksanakan setelah selesai ditandatanganinya perjanjian tersebut.

Kami menunggu informasi selanjutnya atas ketentuan tersebut, berikut jadwal penelitian serta informasi yang lain yang dianggap perlu.

Demikian kami sampaikan. Atas perhatian dan kerjasamanya yang baik, kami ucapkan terima kasih.

[redacted]

Tembusan :
1. Dr. Nur Indrawati Lipoto, MSc
2. Arsip.

II-Wp\Direktur-Srt Kel\Dir.178

BIDANG PENDIDIKAN DAN PENELITIAN
RSUD. Dr. ACHMAD MOCHTAR BUKITTINGGI

Bukittinggi, 28 April 1999

No. : 131.2.2.3.RSAM.1999
Lamp.: -
Hal. : Mohon Bantuan Data.

K e p a d a :

Yth. Sdr. Bpk. Dr. H. ASRI
RSUD. Dr. Achmad Mochtar Bukittinggi
di-
Bukittinggi.

Dengan hormat,

Sehubungan dengan Surat dari Fakultas Kedokteran Universitas Andalas Padang No. 43/J16.2/PL/1999 tanggal 6 Januari 1999 yang ditujukan kepada Direktur RSUD. Dr. Achmad Mochtar Bukittinggi Perihal Mohon izin melaksanakan penelitian, maka bersama ini kami mohon kepada saudara untuk dapat membantu yang bersangkutan :

N a m a : Dr. NUR INDRAWATI L, MSc.
N i p : 131 837 970
Pekerjaan : Dosen Fakultas Kedokteran Unand Padang.
J u d u l : Makanan Tradisional Minang Kabau dan Penyakit Kardiovaskuler di sumatera barat.

Demikianlah kami sampaikan atas perhatian dan bantuan saudara diucapkan terima kasih.

An. KEPALA BIDANG PENDIDIKAN & PENELITIAN
RSUD. Dr. ACHMAD MOCHTAR BUKITTINGGI
KASIE. PENELITIAN & PENGEMBANGAN

[redacted]

= BENNI PRISDON, SKM =
NIP. 140 200 698

TEMBUSAN : Disampaikan kepada Yth.
1. Bpk. Wadir Penunjang Medis & Pendidikan RSAM Bukittinggi.
2. Ka. Subag. Catatan Medik RSAM Bukittinggi.
3. A r s i p.