EFFECT OF DIETS PREPARED BY USING SODIUM- REDUCED CONDIMENT ON LOWERING BLOOD PRESSURE IN HYPERTENSIVE PERSONS

WEERAWAN LIMMANON

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Thesis
Entitled

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....
Miss. Weerawan Limmanon
Candidate

....
Asst.Prof. Wantanee Kriengsinyos, Ph.D, RD.
Major-Advisor

....
Assoc.Prof. Prapaisri Sirichakwal, Ph.D
Co-Advisor

....
Prof. Surat Komindr, M.D.
Co-Advisor

....
Prof. M.R. Jisnuson Svasti, Ph.D
Dean
Faculty of Graduate Studies

....
Assoc.Prof. Emorn Wasantwisut, Ph.D.
Chair
Master of Science Programme in Nutrition
Faculty of Medicine, Ramathibodi Hospital
and Institute of Nutrition

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on
April 10, 2006

....
Miss. Weerawan Limmanon
Candidate

....
Asst.Prof. Wantanee Kriengsinyos, Ph.D., RD.
Chairman

....
Assoc.Prof. Prapaisri Sirichakwal, Ph.D.
Member

....
Prof. Surat Komindr, M.D.
Member

....
Sukit Yamwong, M.D.
Member

....
Assoc.Prof. Emorn Watsantwisut, Ph.D.
Chair
Master of Science Programme in Nutrition
Faculty of Medicine, Ramathibodi Hospital
and Institute of Nutrition

....
Prof. M.R. Jisnuson Svasti, Ph.D
Dean
Faculty of Graduate Studies
Mahidol University

....
Prof. Rajata Rajatanavin,M.D., F.A.C.E.
Dean
Faculty of Medicine, Ramathibodi Hospital
Mahidol University

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Weerawan Limmanon

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EFFECT OF DIETS PREPARED BY USING LOW SODIUM CONDIMENTS ON LOWERING BLOOD PRESSURE IN HYPERTENSIVE PERSONS

WEERAWAN LIMMANON 4436586 RANU/M

M.Sc. (NUTRITION)

THESIS ADVISORS: WANTANEE KRIENGSINYOS, Ph.D., RD., PRAPAISRI SIRICHAKWAL, Ph.D., SURAT KOMINDR, M.D.

ABSTRACT

Epidemiological research has reported that dietary sodium is an important contributor to the pathogenesis of hypertension. Reduced sodium intake is widely prescribed for hypertensive persons to control blood pressure. The primary objective of this thesis is to study the effect of diets prepared by using low sodium condiments on lowering blood pressure. Eighteen hypertensive men and women (25-56 yrs) were included in the study approved by the Committee on Human Research of Mahidol University. Baseline systolic and diastolic blood pressures were 147.9 ± 12.4 and 96.6 ± 9.0 mmHg, respectively. The study was a randomized crossover trial consisting of a 1-week run-in period, followed by two 4-week intervention periods, normal-sodium condiment (NSD) and low-sodium condiment diets (LSD), separated by a 1-week washout period. Sodium intake during the LSD (2,506.0 ± 343.8 mg/d), which was significantly lower than that of the NSD (3,840.8 ± 557.0 mg/d). Inversely, potassium intake was significantly higher during the LSD (3,567.5 ± 497.2 mg/d) compared with that of the NSD (2,090.7 ± 495.3 mg/d). Systolic and diastolic blood pressures were significantly decreased during the run-in period compared to the baseline. Further significant reduction in systolic blood pressure was observed either during the control (137.7 ± 18.0 mmHg) or low-sodium condiment (132.0 ± 17.0 mmHg) diet. A small decrease in diastolic blood pressure was observed during low-sodium condiment diet, but did not reach significance. Self-recorded home blood pressure measurement revealed that diurnal systolic blood pressure was approximately the same throughout the study. However, the diurnal diastolic blood pressures at just waking up was greater than that at just before sleeping during the run-in and NSD, but were not different during the LSD period. There were positive correlations between sodium and potassium intakes and urinary sodium and potassium excretions, corresponding to 75.6 % and 67.8 % of their intakes, respectively.

These results suggest that reduced sodium intake using low sodium condiments instead of the regular ones is capable of decreasing systolic blood pressure in hypertensive persons.

KEY WORDS : DIET / LOW SODIUM CONDIMENTS / BLOOD PRESSURE

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ประสิทธิผลของการจำกัดปริมาณการบริโภคโซเดียมโดยใช้เครื่องปรุงรสที่มีโซเดียมต่ำแทนเครื่องปรุงรสปกติต่อระดับความดันโลหิตในผู้ที่มีความดันโลหิตสูง (EFFECT OF DIETS PREPARED BY USING LOW SODIUM CONDIMENTS ON LOWERING BLOOD PRESSURE IN HYPERTENSIVE PERSONS)

วีรวรรณ ลิ้มมานนท์ 4436586 RANU/M

วท.ม. (โภชนศาสตร์)

คณะกรรมการควบคุมวิทยานิพนธ์ : วันทนีย์ เกรียงสินยศ, Ph.D., RD., ประไพศรี ศิริจักรวาล, Ph.D., สุรัตน์ โคมินทร์, M.D.

บทคัดย่อ

จากรายงานการศึกษาทางระบาดวิทยาพบว่าปริมาณโซเดียมจากอาหารเป็นสาเหตุสำคัญต่อการทำให้เกิดโรคความดันโลหิตสูง ดังนั้นคำแนะนำที่ใช้อย่างกว้างขวางในการควบคุมความดันโลหิต ก็คือ การลดการบริโภคโซเดียม ในการศึกษานี้มีวัตถุประสงค์ที่จะทำการศึกษาผลของการจำกัดปริมาณการบริโภคโซเดียมโดยใช้เครื่องปรุงรสที่มีโซเดียมต่ำแทนเครื่องปรุงรสปกติต่อระดับความดันโลหิตในผู้ที่มีความดันโลหิตสูง.

อาสาสมัครชายหญิงที่มีความดันโลหิตสูงจำนวน 18 คน (อายุระหว่าง 25-65 ปี) ค่าความดันซีสโตลิคและความดันไดแอสโตลิคก่อนเข้าการศึกษา คือ 147.9 ± 12.4 และ 96.6 ± 9.0 มิลลิเมตรปรอทตามลำดับ การศึกษานี้เป็นการศึกษาแบบ crossover ซึ่งประกอบด้วย ระยะ run-in 1 สัปดาห์ หลังจากนั้นเข้าสู่ระยะศึกษาซึ่งมีอาหาร 2 ชนิดคือ อาหารที่ปรุงด้วยเครื่องปรุงรสปกติ และอาหารที่ปรุงด้วยเครื่องปรุงรสที่มีโซเดียมต่ำ อาสาสมัครจะได้รับอาหารชนิดละ 4 สัปดาห์ โดยมีระยะพักระหว่างอาหารแต่ละชนิดเป็นเวลา 1 สัปดาห์ ปริมาณโซเดียมที่ได้รับในระหว่างการบริโภคอาหารที่ปรุงด้วยเครื่องปรุงรสที่มีโซเดียมต่ำ (2,506.0 ± 343.8 มิลลิกรัมต่อวัน) ต่ำกว่าการบริโภคอาหารที่ปรุงด้วยเครื่องปรุงรสปกติ (3,840.8 ± 557.0 มิลลิกรัมต่อวัน) ในทางกลับกันปริมาณโปแตสเซียมที่ได้รับในช่วงอาหารที่ปรุงด้วยเครื่องปรุงรสที่มีโซเดียมต่ำ (3,567.5 ± 497.2 มิลลิกรัมต่อวัน) สูงกว่าปริมาณโซเดียมที่ได้รับในช่วงอาหารที่ปรุงด้วยเครื่องปรุงรสปกติ(2,090.7 ± 495.3 มิลลิกรัมต่อวัน) ผลการศึกษาพบว่าภายหลังจากระยะ run-in ความดันทั้งซีสโตลิคและไดแอสโตลิคมีการลดลงอย่างมีนัยสำคัญเมื่อเปรียบเทียบกับความดันก่อนการศึกษา ความดันซีสโตลิคมีการลดลงอย่างมีนัยสำคัญอีก เมื่อบริโภคอาหารที่ปรุงด้วยเครื่องปรุงรสปกติ (137.7 ± 18.0 มิลลิเมตรปรอท) และอาหารที่ปรุงด้วยเครื่องปรุงรสที่มีโซเดียมต่ำ (132.0 ± 17.0 มิลลิเมตรปรอท) ในส่วนความดันไดแอสโตลิคมีการลดลงอีกเล็กน้อยในช่วงอาหารที่ปรุงด้วยเครื่องปรุงรสที่มีโซเดียมต่ำแต่ไม่มีนัยสำคัญทางสถิติ สำหรับความดันโลหิตในแต่ละช่วงเวลาของวันที่วัดโดยอาสาสมัครเองพบว่าไม่แตกต่างของความดันซีสโตลิคตลอดการศึกษา ส่วนความดันไดแอสโตลิคเวลาตื่นนอนมีค่าสูงกว่าตอนก่อนนอนในช่วง run-in และช่วงรับประทานอาหารที่ปรุงด้วยเครื่องปรุงรสที่มีโซเดียมปกติแต่ไม่มีความแตกต่างกันในช่วงรับประทานอาหารที่ปรุงด้วยเครื่องปรุงรสที่มีโซเดียมต่ำ การบริโภคโซเดียม โปแตสเซียมมีความสัมพันธ์ในเชิงบวกกับการขับออกของโซเดียมและโปแตสเซียมโดยการขับออกของโซเดียมและโปแสเซียมคิดเป็น 75.6 และ 67.8 เปอร์เซนต์ของปริมาณที่บริโภคตามลำดับ

ผลการศึกษานี้สรุปได้ว่าการจำกัดการบริโภคโซเดียมโดยใช้เครื่องปรุงรสที่มีโซเดียมต่ำแทนเครื่องปรุงรสปกติมีผลต่อการลดลงของความดันซีสโตลิคในคนที่มีความดันโลหิตสูง

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LIST OF ABBREVIATIONS

ABPM	Ambulatory blood pressure	**h**	hour
ACE	angiotensin-converting enzyme	**HT**	Hypertension
ALT	Alanin aminotransferase	**JNC VII**	Joint National Committee VII
ANP	Atrial natriuretic peptide	**K**	Potassium
AP	Alkaline phosphatase	**Kcal**	Kilicalories
ARBs	angiotensin-receptor blockers	**kg**	kilogram
AST	Aspartate aminotransferase	**L**	Liter
BMI	Body Mass Index	**LSD**	Low sodium condiment diet
BW	Body weight	**mg**	milligram
CCBs	calcium channel blockers	**mL**	milliliter
cm	centrimeter	**mmHg**	Millimeter mercury
CO	Cardiac output	**mmol**	millimole
CVD	Cardiovascular disease	**MSG**	monosodiumglutamate
d	day	**n**	number
DASH	Dietary Approaches to Stop Hypertension	**Na**	Sodium
BP	Blood pressure	**NAS/NRC**	National Academy of Science and National Research Council
dL	Deciliter	**NCP**	Non- communicable disease
DM	Diabetes milletus	**NHANES**	National Health and Nutritions Examination Survey
g	gram	**NSD**	Normal sodium condiment diet

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LIST OF ABBREVIATIONS (CONT.)

PABA	p-amino benzoic acid	**TPR**	Total peripheral resistance
RDI	Recommended Daily Intake	**WHO**	World Health Organization
SD	Standard deviation	**WHO-ISH**	World Health Organization-International Society of Hypertension
SV	Stroke volume	**y**	year
TC	Total cholesterol	**μL**	microliter
TOHP-I	Trail of Hypertension prevention, Phrase I		

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CHAPTER I

INTRODUCTION

The prevalence and incidence of hypertension (HT) has been increasing in both developed and developing countries. Data from The National Health and Nutrition Examination Surveys III (NHNES III) indicated that the prevalence of HT in U.S. adults increased from 24 percents (approximately 43 million people) in 1988-1991 to 28.7 percents in 1999-2000 (1). In some European countries e.g. Italy, Sweden, England, Spain, Finland, and Germany found that HT prevalence was average 44.2 percents (2). In Thailand, public health statistics from 1997 to 2004 showed that prevalence and mortality rate of HT were increasing every year (3-10). Recently, in 2004 the prevalence of hypertension people was 389.3 per 100,000 population, increased 2 fold when compared with that of 1999 (6, 10). A preliminary survey conducted by the Institute of Nutrition, Mahidol University (June, 2003) indicated that approximately 14 percents of Mahidol University staff has had HT.

The problem with HT is that most of the patients have no symptoms, which is why HT is known as the “Silent Disease” or “Silent Killer”. It can lead to sudden death and chronic disability, e.g. cardiovascular disease, myocardial infarction, stroke, congestive heart failure, and end-stage renal disease (11-15). Mortality rate of HT and stroke is one of the sixth priorities of non-communicable disease (NCD). It increased from 5.10 per 100,000 population in 2002 to 5.41 per 100,000 population in 2003 (9, 10). Therefore, prevention and control of HT becomes an important goal to reduce the incidence of HT, and its complications (16). In meta-analysis study, 10-12 mmHg decreased in systolic blood pressure (Systolic BP) and 5-10 mmHg decreased in diastolic blood pressure (Diastolic BP) reduced mortality rate from cardiac disease to 20 percents (17).

Epidemiological research during the past decades has reported that dietary sodium (Na) is an important contributor to the pathogenesis of HT (18). Higher blood pressure (BP) had been observed in population with higher Na intake. Furthermore, it has been found that Na restriction of hypertensive patients resulted in a significant

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reduction of BP (19-21). The current recommendation of World Health Organization (WHO) (22) and the seventh report of the Joint National Committee (JNC VII) (21) for prevention and control HT is to consume Na no more than 2,400 mg/d (approximately 6 g of sodiumchloride). In Thailand the suggestion of moderate Na intake has been endorsed as part of the food based dietary guidelines since 1995. To date, there is a little data of the actual intake of Na intake in Thai population. However, it is likely that Thai people consume higher Na than the recommendation. The relevant data from the Ministry of Public Health, Thailand indicated that using condiments in Thai population has been increased from 7 g/d/person in 1960 to 20.3 g/d/person in 1995 (23, 24). The regular condiments such as fish sauce, soy sauce, flavored sauce, oyster sauce, are high in Na, ranging from 1,000 to 1,600 mg per serving (15 g). Around 98 percent of Thai people add condiments everyday (24). Therefore, the suggestion of "do not add condiments during eating" has been provided during diet therapy especially for hypertensive individuals. Although this suggestion seems to be easy, it is likely difficult for many people to follow due to unpleasant-flavor foods. The low-tasty Na condiments may be an alternative successive way to reduce Na intake of Thai people, and perhaps reduce risks of hypertension and cardiovascular disease. Currently there is no data to support this issue.

Objective

To study the effect of diets prepared by using low-sodium condiments on lowering blood pressure in hypertensive persons.

Specific objectives

The specific objectives of the present study were as follow:

1. To determine sodium and potassium contents in diets prepared by using normal and low sodium condiments.
2. To investigate the effect of low sodium restriction on systolic and diastolic blood pressure by using low-sodium condiments.
3. To investigate diurnal blood pressure along the day during the intervention period.
4. To investigate the relationship between sodium, potassium intake and urinary sodium, potassium excretion.

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CHAPTER II

LITERATURE REVIEW

2.1 Definition of hypertension

HT is usually defined as a systolic BP of 140 mmHg or greater and/or a diastolic BP of 90 mmHg or greater (22).

2.2 Classification of hypertension

HT can be categorized on the basis of severity and etiology as follow.

2.2.1 Classification by severity

According to the JNC VII, BP can be classified as normal, prehypertension and HT stage 1 and 2 as shown in Table 2.1 (21). The diagnosis of HT is made when diastolic BP on at least 2 subsequent visits is $\geq$ 90 mmHg or systolic BP is consistently $\geq$ 140 mmHg (average of two or more BP measurements). Individuals with prehypertension tend to maintain pressures that are above average for the general population and are at greater risk for development of definite HT events than the general population.

Table 2.1 Classification of blood pressure for adults aged 18 years and older by JNC VII.

Category	Systolic BP (mmHg)		Diastolic BP (mmHg)
Normal	<120	and	<80
Prehypertension	120-139	or	80-89
Hypertension			
Stage 1	140-159	or	90-99
Stage 2	$\geq$160	or	$\geq$100

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World Health Organization- International Society of Hypertension (WHO-ISH) recommended and classified HT in adults, as optimal, normal, high-normal, grade 1, 2, 3 and isolated systolic HT as shown in Table 2.2 (22). BP must be measured at least twice and found that it is high before diagnosis of hypertension. Borderline HT becomes a subgroup within grade 1 and isolated systolic HT. Isolated systolic HT is defined as systolic BP ≥ 140 mmHg and diastolic BP < 90 mmHg. When patient's systolic and diastolic blood pressure fall into different categories, the higher category should be applied.

Table 2.2 Classification of blood pressure for adults aged 18 years and older by WHO-ISH

Category	**Systolic BP (mmHg)**	**Diastolic BP (mmHg)**
Optimal	<120	<80
Normal	<130	<85
High-normal	130-139	85-89
Grade 1 hypertension (mild)	140-159	90-99
Subgroup : borderline	140-149	90-94
Grade 2 hypertension (moderate)	160-179	100-109
Grade 3 hypertension (severe)	≥180	≥110
Isolated systolic hypertension	≥140	<90
Subgroup : borderline	140-149	<90

2.2.2 Classification by etiology

HT is classified according to etiology as primary or essential HT, in which the cause is unknown; or secondary to a specific disorder, such as renal disease, neoplasia, or pregnancy. More than 90 % of the cases of HT are essential HT (25).

2.3 Pathophysiology of hypertension

BP is the force of blood against the arterial blood vessel. BP is the product of the cardiac output (CO) and the total peripheral vascular resistance (TPR). This relationship can be written as the equation: BP = CO X TPR. As shown in Figure 2-1,

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the pathophysiology of HT is the consequence of increased cardiac output and/or peripheral vascular resistance, resulting from the alteration of sympathetic nervous system, renal function, renin-angiotensin system, hormone and mechanism of other factors.

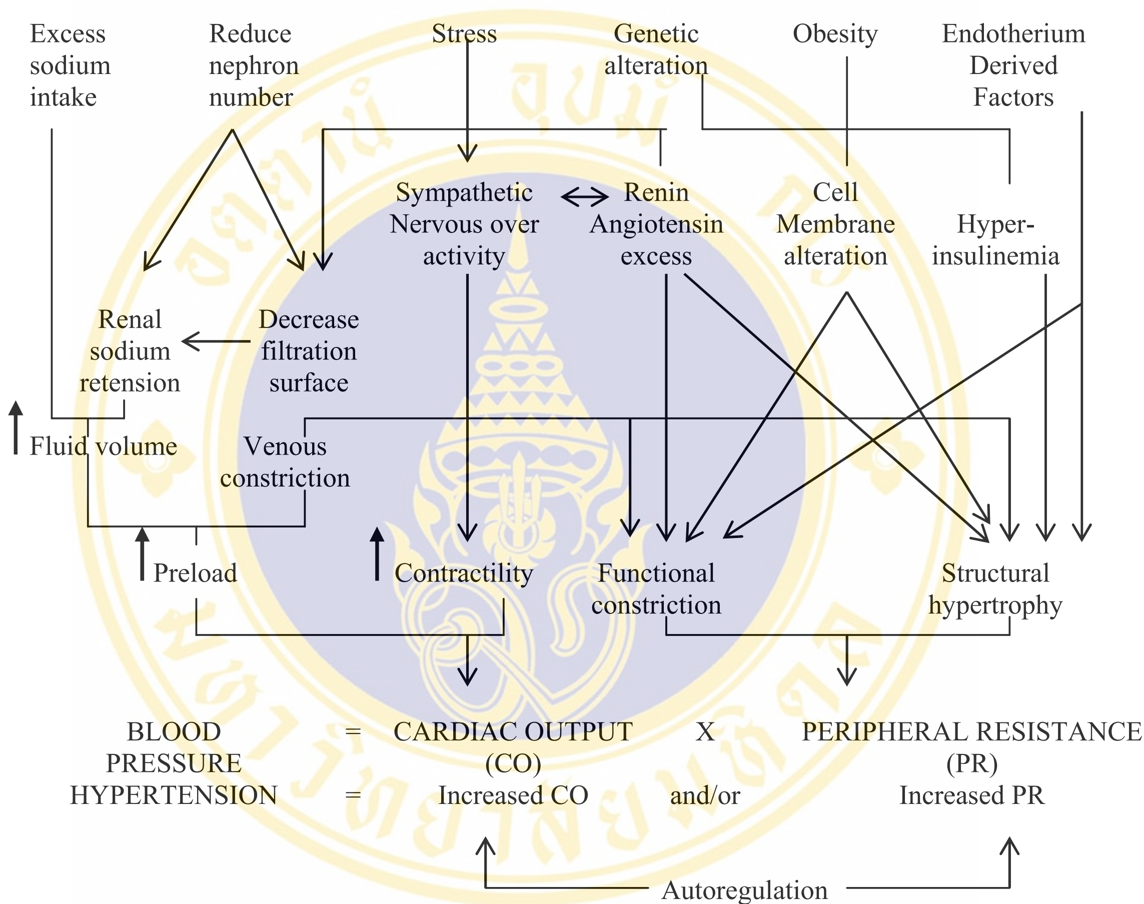

Figure 2-1. Pathophysiology of hypertension adapted from Kaplan's clinical hypertension (25).

CO is the blood volume that the heart pumps to the human body in one minute. CO increases if stroke volume and the heart contractility increase. Stroke volume is blood volume before the heart pumps. Stroke volume increases as the effect of vasoconstriction and fluid retention consequently from high Na intake and Na retention of renal. In a stress situation, heart contractility increases and peripheral vascular resistance increases from deterioration of vessels and other factors such as renin-angiotensin excess, cell membrane alteration, hyperinsulinemia that affect from

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stress, genetic alteration, obesity, and hyperthelium derived factors. The autoregulation can be described that with increased CO, more blood flows through the tissue than is required, and the increased flow delivers extra nutrients or removes additional metabolic products; in response, the vessels constrict, decreasing blood flow and returning the balance of supply and demand to normal. Thus, PR increases and remains high by the rapid induction of structural thickening of the resistance vessels (25).

2.4 Factors influencing blood pressure

The factors influencing BP in primary HT are divided to 2 main factors.

2.4.1. Non-modifiable factors: age, gender, ethnicity, and genetic

Aging

Arterial pressure tends to increase with aging in most populations, thus effecting to increase the incidence and prevalence of HT in the elderly. These increases in pressure most often involve systolic and pulse pressure, whereas diastolic pressure tends to decrease or remains at the same level. The relation between age and BP has been demonstrated in many cross-sectional or longitudinal studies (1, 26, 27). In the Framingham Heart Study, the incidence of HT was shown to increase from 3.3 % in men between 30 and 39 years of age to 6.2 % in men between 70 and 79, and from 1.5% to 8.6 % in women of comparable ages (28). The prevalence of HT also increased with age; it was about 25 % in 40- to 50-year-old men and increased to well over 50% after the age of 60 years (28).

Ethnicity

Ethnicity also plays an important role in the pathogenesis of HT. The prevalence of HT is higher in African-Americans than in other ethnic groups. The incidence of HT has been reported to be comparable in Mexican-Americans and non-Hispanic whites but is lower in Asian-Americans (29).

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Gender

The prevalence and incidence of HT are slightly higher in men compared to those of women. This gender difference is greater in the young and middle-aged population, but it decreases or even reverses in older subjects (28).

2.4.2. Modifiable factors: body mass index (BMI), smoking, alcohol intake and dietary intake

Body mass index

Relationship between BMI and BP has been well established in both cross-sectional and longitudinal studies. Large intervention trials of Hypertension Prevention Collaborative Research Group demonstrated a decrease in the incidence of HT and reduction in systolic and diastolic BP after reducing body weight (30). A 4 % reduction in body weight over 3 years resulted in a decrease in both systolic and diastolic pressure of 2.4 and 1.8 mmHg, respectively (31). Similarly, the Trial of Hypertension Prevention, Phrase I (TOHP-I) study, conducted in a cohort of 35 to 54 year old men and women demonstrated a reduction of 2.9 and 2.3 mmHg in systolic and diastolic BP, respectively, after weight loss of 3.9 kg over an 18-month period of follow-up (32).

Smoking

The relation between smoking and HT still remains to be established (21). Nevertheless, both systolic and diastolic BP are increased after a single cigarette (33). In the heavy smoker consuming two or three or more packages per day, arterial pressure may be increased most of the time (33).

Alcohol consumption

Fuchs et al (34) studied effect of alcohol consumption on the incidence of HT in 8,334 black and white American men and women in a 6-year follow up. The risk of developing HT was found to be higher in all subjects when the alcohol consumption was greater than 210 g/wk. Ethanol intake lesser than 210 g per week was a risk factor for HT only in black men (34). Moreover, reduction in alcohol

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consumption was associated with a mean decrease of 3.31 mmHg for the systolic BP and 2.04 mmHg for the diastolic BP (35).

Dietary factors

Dietary factors are among the most potentially modifiable determinants of BP level and the occurrence of HT. The relationship between electrolyte and BP level is complex and controversial. So far, most research in this area has focused on effect of Na and K intakes on BP level. More recently, interest has broadened to include the effect on BP of dietary calcium (Ca) and magnesium (Mg) intakes. The relationships between dietary factor and BP have been described in next session.

2.5 Dietary factors and hypertension

2.5.1 Sodium and blood pressure

There is considerably evidence relating Na intake to BP. Analysis of the international study of electrolyte excretion and blood pressure (INTERSALT)(36-38), which included more than 10,000 subjects across 52 centers around the world, showed a significant relation between salt intake, BP, the upward slop of BP with age, and the prevalence of high BP. For individuals, the within-population analyses demonstrated that the relationship between Na excretion and BP was similar for nonhypertensive and all participants, indicating that varying degrees of salt sensitivity of BP occur throughout the population. Overall the principle observations in INTERSALT were that (1) for individuals, a difference of 100 mmol (equivalent to 2,300 mg or 5.9 g sodiumchloride) per day in Na intake was associated with a difference of 3 to 6 mmHg in systolic BP; and (2) for populations, a 100 mmol/d lower Na intake was associated with attenuation of the rise in systolic BP by 10 mmHg in person aged 25 to 55 years. It was also found that in several isolated, preliterate population in 4 remote INTERSALT samples in which Na excretion was low, there was little of no HT, and no upward slope of HT with age.

A positive relation between dietary Na and BP has been shown from observational studies in humans. Comparison of BP between Yi farmers who had or had not migrated to an urban area and a group of Han residents of the same urban area

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found that BP raised very little with age in the Yi farmers but increased with age in Yi migrants and Han residents. (39)

Randomized, controlled trials provide compelling evidence for a causal relationship between dietary Na and BP. Despite reservations about the limitations of meta-analyses, 2 recent meta-analyses document consistent reductions in BP in response to lowered intake of Na (13, 40). In one meta-analysis of 32 trials, estimated median reductions of 24-h urine Na excretion across trials were 76 mmol (1,748 mg) and 106 mmol (2,430 mg) in hypertensive and nonhypertensive subjects, respectively; overall reductions of systolic and diastolic BP were 1.9/1.1 mmHg in nonhypertensive subjects and 4.8/2.5 mmHg in hypertensive subjects (13). Similarly results were found in a second meta-analysis of 52 trials. The estimated median reductions of Na excretion across trials were 79 mmol (1,817 mg) and 133 mmol (3,059 mg) for nonhypertensive and hypertensive subjects, respectively; overall reductions of BP were 1.6/0.5 mmHg in nonhypertensive subjects and 5.9/3.8 mmHg in hypertensive subjects (40). The reductions in BP by a diet lower in Na are more prominent in hypertensive than in nonhypertensive individuals. In a recent 18-month study, He et al demonstrated a net reduction (compare with the control group) of 3.3 mmHg and 1.7 mmHg for systolic and diastolic BP, respectively. Results form He et al (41) and Benetos et al (42) and showed that low dietary Na intake significantly lowered BP when compared with normal dietary Na intake. This report is in line with the Cappuccio study (15) which found that decreased Na intake to 80 mmol (1,840 mg) effected both SBP and DBP reduction of 7.2/3.2 mmHg, respectively.

The Trials of Hypertension Prevention (TOHP) evaluated the effects of nonpharmacological therapy BP in adults with high-normal BP. In phase I, systolic and diastolic BP were significantly reduced in separate groups treated with weight loss or reduction of NaCl intake over 18 months (32, 43). The results of phase I were the basis for phase II of TOHP, which more extensively evaluated the effects of weight loss and reduced Na intake, alone and in combination, on BP over 3 to 4 years in overweight adults with high-normal BP (30). Compared with BP in a usual-care control group, at 6 months, systolic and diastolic BP were both significantly reduced by weight loss alone (6.0/5.5 mmHg) and lowered Na intake alone (5.1/4.4 mmHg).

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At 6 months, hypertension incidence was 7.3% in the usual care group, 4.2% in the weight loss group, 4.5% in the reduced-Na group, and 2.7% in the combined weight loss-reduced-Na group. Beyond 6 months, the interventions were less effective for maintaining both weight loss and lowered Na intake, and the impact on BP was also lessened. At termination of the study there were small but significant reductions in systolic BP in the weight loss and low-Na group, whereas reduction of systolic BP in the combined-intervention group (weight loss and low Na) did not achieve statistical significance. Reduction of diastolic BP was significant only in the weight loss group. The incidence of hypertension during the entire course of the study was significantly lower in each of the 3 intervention groups (approximately 38%) than in the usual-care group (44%).

There is considerable evidence that NaCl, rather than sodium per se, is responsible for the known adverse effects of dietary salt. Many people doubted whether other form of Na such as sodium citrate, sodium bicarbonate or sodium phosphate could effect to raise BP. McCarty (44) concluded that many studies in both animal and human indicated that, other sodium salt such as sodium citrate, sodium bicarbonate or sodium phosphate have not effects to increase in vascular volume, and BP by Na-retention since the retention of Na and subsequent development of HT depend on the presence of an appropriate chloride (Cl). Indeed, if dietary Cl is severely restricted, the renal tubular Na K-ATPase must do disproportionate part of the work required to retain Na, whereas when Cl intake are more ample, Na, K, 2Cl co-transport can play the role in this regard (44). However, sodium citrate and other sodium salt do share one important property with NaCl; that when administered to humans consuming a low salt diet, they markly suppress the production of rennin and angiotensin II, and dampen the activity of the sympathetic nervous system. The available evidence suggests that their impact in this regard may be somewhat less dramatic than that of NaCl.

Salt sensitivity and salt resistance.

One importance reason for difference in findings across both observational and experimental studies may be that individuals vary in their susceptibility to the

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effect of Na on BP. Usually, salt sensitivity is considered a categorical parameter, with salt-sensitive individuals being defined as those with a difference in mean arterial pressure (MAP) between low and high-Na diet >10%, and salt-resistant subjects those in whom MAP does not increase or shows an increase <5% under Na loading (45-47).

Blacks have been consistently shown to have a greater frequency of salt sensitivity than whites. Weinberger et al (48)observed that 73% of black hypertensive patients were salt sensitive compared with 56% of a white hypertensive group; but in the normotensive population, the frequency of salt sensitivity among blacks (36%) was similar to that seen among whites (29%). Similar observations of a greater frequency of salt sensitivity among blacks have been reported in other studies. Increasing salt sensitivity has been noted with increasing age in several such studies (48-51). This relationship appears to be stronger in hypertensive than in normotensive individuals (49). In the large epidemiological INTERSALT study (52), the relationship between Na excretion and BP was most notable when examined on the basis of age. Moreover, Sullivan et al (49) has recently reported observations in subjects who were followed for at least 10 years after the initial classification of salt sensitivity. They found that salt-sensitive individuals had a rise in BP over time, which was significantly greater than in those who were salt resistant. An influence of sex has been suggested by some investigators (53) who found salt sensitivity among female hypertensive patients but not in males when changed from a diet containing 15 g/d salt to one containing 3 g/d. Other investigators have not confirmed this effect of sex on salt sensitivity (48, 51). In view of the age-related effect cited by many investigators (54-56), such a separate sex effect may be difficult to discern. Another possible explanation is that since women are typically lighter in weight than men and since all of the studies examining salt sensitivity have administered a uniform amount of Na to all subjects in each phase of the study, women may have received a greater salt load on a body weight basis than did men, thus making it more likely that a response would be observed in women.

Mechanisms of sodium and hypertension.

The mechanisms by which Na intake may increase BP still not completely understood. Blaustein and grim (57) explained that when more Na is ingested than the

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kidneys are able to handle (excrete), there is a (transient) slight positive Na balance; as a result Na, chloride, and water are retained, resulting in an expansion of plasma volume (Fig. 2-2). The initial physiologic responses include the increased secretion of atrial natriuretic peptides (ANP) and the digitalis-like substance (natriuretic hormone), and inhibition of vasopressin and aldosterone secretion. The net effect directly enhanced natriuresis and diuresis, and a reduction in plasma volume, with no significant effect on BP. However, if there is a continuing tendency to Na retention and volume expansion, the capacity of the aforementioned mechanisms to control plasma volume will be exceeded; then, the chronically elevated level of natriuretic hormone will inhibit the sodium pumps in the arterial and venous smooth muscle cells and in the sympathetic neurons. The increased venous tone will help to reduce plasma volume directly by reducing central venous volume. Arterial tone will be increased by direct action of natriuretic hormone on the arterial smooth muscle and, indirectly, via the hormone's action on the sympathetic neurons. Initially, of course, BP will be maintained in the normal range (but will be labile) because of the compensating cardiovascular reflexes. Once the capacity of these reflexes to control BP is exceeded, however, the BP will begin to rise; this will induce a pressure natriuresis to help restore plasma volume to normal (Fig. 2-3).

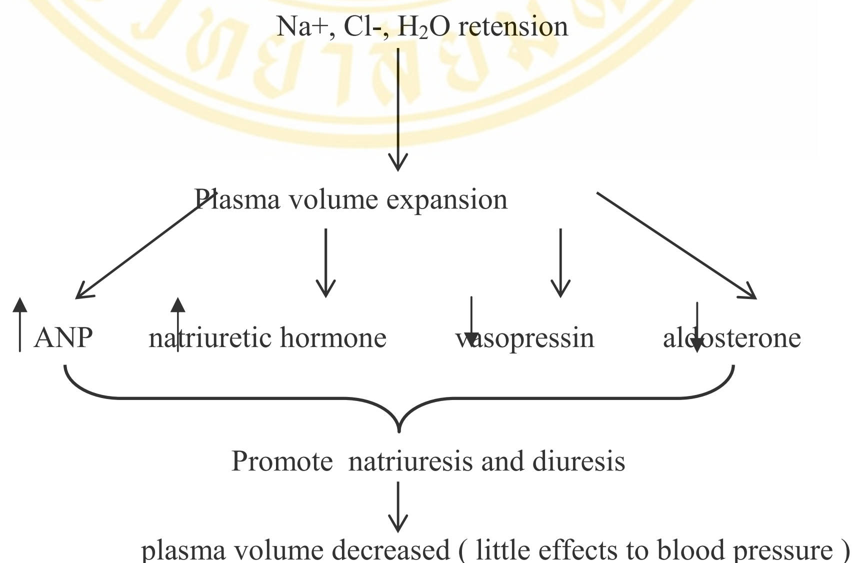

Figure 2-2 Mechanism of blood pressure increasing when sodium load (57).

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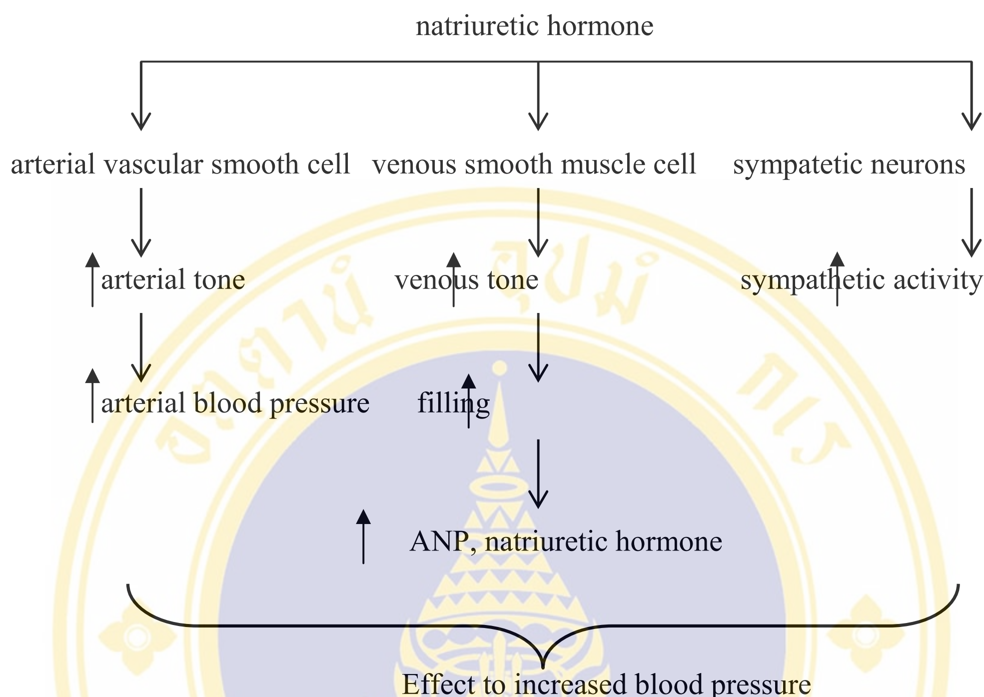

Figure 2-3. Effect of natriuretic hormone to increased blood pressure (57).

From plasma rennin activity study in 20 essential hypertension, definded subjects into 2 groups: salt sensitive and salt resistance. The study found that Na load from 25 to 250 mmol/d in salt sensitive effected to increased BP by increased CO, change in distribution of local blood flow (blood circulated into liver and kidney was constant whereas blood circulated into skin was increased) and difference in vascular resistance (contraction of abdominal organ, tight of hemodynamic characteristic) (58, 59)). The change in vascular resistance similar to the defense response of sympathetic overactivity (58, 59). In salt sensitive people when they were on high salt diet (360 mmol/d), the increase in MAP was observe in accordance with the increase in norepinephrine and angiotensin II (60).

2.5.2. Potassium and blood pressure.

Both epidemiological and clinical studies have strongly suggested that an increase in K intake also lowered BP (38, 61-63).

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In INTERSALT, the relationship between urinary K and BP was explored in 52 centers (38). Results demonstrated that 40 mmol increase in urinary K excretion was associated with 1.8 and 1.2 mmHg lower levels of systolic and diastolic BP, respectively.

In a clinical study, Franzoni et al (61) demonstrated that supplement potassium aspartate 30 mmol/d in hypertensive subjects was significantly lower systolic and diastolic BP 12/8 mmHg. This was supported by the conclusion of 2 recent meta-analyses of clinical trials that oral K supplement (60 to 120 mmol/d) lowered BP by 1.8/1.0 mmHg in nonhypertensive subjects and 4.4-8.2/2.5-4.5 mmHg in hypertensive subjects (62, 63). This suggested that the magnitude of the BP-lowering effect was greater in hypertensive than in nonhypertensive persons and more pronouced in persons consuming a diet high in Na.

2.5.3. Dietary Approaches to Stop Hypertension and blood pressure

The Dietary Approaches to Stop Hypertension (DASH) trial demonstrated that a diet that emphasizes fruits, vegetables, and low-fat dairy products, that includes whole grains, poultry, fish, and nuts, that contains only small amounts of red meat, sweets, and sugar-containing beverages, and that contains decreased amount of total and saturated fat and cholesterol lowered BP substantially both in normotensive and hypertensive persons (64, 65).

In a randomized, multicenter study, the DASH trial (65) evaluated the effects on BP of 3 dietary patterns over 8 wk in 459 adults with high-normal BP or mild HT. The dietary interventions were (1) a control diet with K, Ca, and Mg levels close to the 25th percentile of US consumption; (2) a diet rich in fruits, vegetables; and (3) a "combination" diet rich in fruits, vegetables, and fat-free or low-fat dairy products or DASH diet. The Na intakes in 3 these dietary pattern were about 3,000 mg/d. The study diets were relatively high in K, Ca, and Mg content. Compared with the control diet, the study diets also were higher in fiber, protein, carotenoid, and folate, and lower in total fat, saturated fat, and cholesterol. Systolic and diastolic BP were significantly reduced by the diet enrich with fruits and vegetables (2.8/1.1 mmHg) compared with the control diet. Reduction in BP was even greater extent by

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the DASH diet (5.5/3.0 mmHg). The greater effect of the DASH diet was manifested in both hypertensive (11.4/5.5 mmHg) and normotensive (3.5/2.1 mmHg) persons.

One DASH trial (64) including 8-wk feeding study the average BP reduction in systolic and diastolic BP were 11/6 mmHg among those with HT and 4/2 mmHg among those without HT with the greatest reduction in black HT persons. The response of BP almost certainly is not attributable to the influence of a single nutrient. Compared with the control diet, the DASH diet has an increase in Ca content, a lower Na content (3,000 mg/d), 173 % higher Mg, 150 % higher K, 240 % higher fiber, and 30% higher protein. Other non-targeted nutrients were also higher e.g. vitamin A, B (66), C, and E, folate, riboflavin, phosphorus, and zinc (67). Although it was not designed to identify the effective nutrients of the diets on BP, the DASH trial convincing reaffirmed the importance of multiple factors in the diet for BP control.

2.5.4. Other dietary factors and blood pressure

The relation between Ca intake and BP has also been examined in meta-analyses. One meta-analysis pooled data from 22 clinical trials involving Ca supplements of 400 to 2,160 mg/d (66). It showed a decrease in systolic BP of 0.5 mmHg for trials involving normotensive persons and 1.7 mmHg for trials involving hypertensive persons. A second meta-analysis of 33 trials involving Ca supplements of 1,000 to 2,000 mg/d showed a reduction in systolic BP of 1.3 mmHg in normotensive participants (68). These data indicate that Ca supplementation has a small effect on SBP level in hypertensive persons and an uncertain effect in normotensive individuals.

There are inconsistent findings associating Mg with BP level. A recent review of 20 observational studies concluded that the evidence suggested an inverse association between Mg intake and BP level (69). However, recent controlled clinical studies showed no significant effect of Mg on BP (70).

2.6 Management of hypertension

The primary goal of managing HT is to prevent morbidity and mortality from cardiovascular disease (CVD). Treating SBP and DBP to target that are less than 140/90 mmHg is associated with a decrease in CVD complications. In patients with

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HT with diabetes or renal disease, the BP goal is less than 130/80 mmHg (Figure 2-4)(21, 71). Management of HT includes lifestyle modifications and pharmacological treatments.

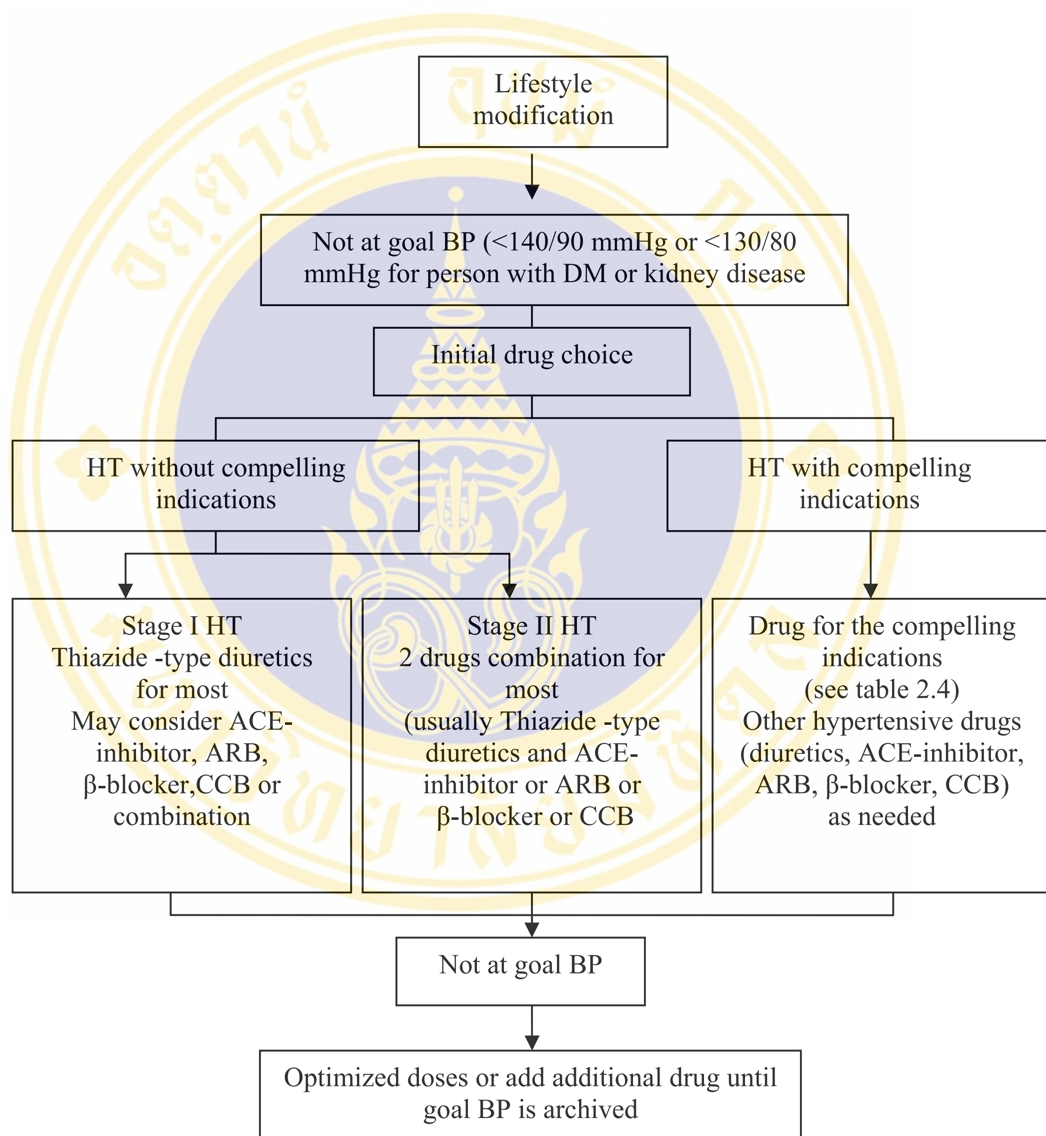

Figure 2-4 Algorithm for treatment of hypertension adapted from the seventh report of JNC (21).

2.6.1 Lifestyle modifications

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Adoption of healthy lifestyles by all individuals is critical for the prevention of high BP and an indispensable part of the management of those with HT. Major lifestyle modifications shown to lower BP include weight reduction in those individuals who are overweight or obese (30, 41); adoption of DASH eating plan (31), which rich in K and Ca (72); dietary Na reduction (31, 72, 73); physical activity (74, 75); and moderation of alcohol consumption (35) (Table 2.3). Lifestyle modifications decrease BP, enhance antihypertensive drug efficacy, and decrease cardiovascular risk. Combinations of 2 or more lifestyle modifications can achieve even better results.

Table 2.3 Lifestyle modifications to manage hypertension.

Modification	Recommendation	Approximate SBP reduction, range
Weight reduction	Maintain normal body weight (Body mass index 18.5-24.9 kg/m^2)	5-20 mmHg/ 10 kg weight loss
Adopt DASH eating plan	Consume a diet rich in fruits, vegetables, and low-fat dairy products with reduced contents of saturated and total fat	8-14 mmHg
Dietary sodium reduction	Reduced dietary sodium intake to no more than 100 mmol (2.4 g sodium or 6 g sodium chloride)	2-8 mmHg
Physical activity	Engage in regular aerobic physical activity such as brisk walking (at least 30 minutes pre day, most day of the wk)	4-9 mmHg
Moderation of alcohol consumption	Limit consumption to no more than 2 drinks per day (1 Oz. or 30 mL ethanol (e.g. 24 Oz. beer, 10 Oz wine, or 3 Oz 80-proof whiskey) in most men and no more than 1 drink per day in women and lighter-weight persons	2-4 mmHg

2.6.2 Pharmacological Treatment

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Excellent clinical trial outcome data prove that lowering BP with several classes of drugs, including angiotensin-converting enzyme (ACE) inhibitors, angiotensin-receptor blockers (ARBs), β-blockers, calcium channel blockers (CCBs), and thiazide-type diuretics, will all reduce the complications of HT (17, 76-82).

Thiazide-type diuretics have been the basis of antihypertensive therapy in most outcome trials (17). In these trials, including the recently published Antihypertensive and Lowering Treatment to Prevent Heart Attack Trial (79), diuretic have been virtually unsurpassed in preventing the CVD complications of HT. Diuretics enhance the antihypertensive efficacy of multi-drug regimens and are more affordable than other antihypertensive agents. Despite these findings, diuretics remain underused (83).

Thiazide-type diuretics should be used as initial therapy for most patients with HT, either alone or in combination with one of the other classes (ACE inhibitors, ARBs, β-blockers, CCBs). The list of compelling indications requiring the use of other antihypertensive drugs as initial therapy are listed in Table 2.4. If a drug is not tolerated or is contraindicated, then one of the other classes proven to reduce cardiovascular events should be used instead. Most patients with HT will require 2 or more antihypertensive medications to achieve their BP goal. Addition of a second drug from a different class should be initiated when use of a single drug in adequate doses fails to achieve the BP goal. When BP is more than 20/10 mmHg above goal, consideration should be given to initiating therapy with 2 drugs, either as separate prescriptions or in fixed dose combinations (Figure 2-3). The initiation of drug therapy with more than 1 agent may increase the likelihood of achieving the BP goal in a more timely fashion, but particular caution is advised in those at risk for orthostatic hypotension, such as patients with diabetes, autonomic dysfunction, and some older persons. Use of generic drugs or combination drugs should be considered to reduce prescription costs (21).

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Table 2.4 Clinical trial and guideline basis for compelling indications for individual drug classes.

High risk conditions with compelling indication*	**Recommended drugs**					
	Diuretic	**β-blockers**	**ACE inhibitor**	**ARB**	**CCB**	**Aldosterone antagonist**
Heart failure	x	x	x	x		x
Post myocardial infraction		x	x			x
High coronary disease risk	x	x	x		x	
Diabetes	x	x	x	x	x	
Chronic kidney disease			x	x		
Recurrent stroke prevention	x		x			

* Compelling indications for antihypertensive drugs are based on benefits form outcome studies or existing clinical guidelines.

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CHAPTER III
MATERIALS AND METHODS

3.1 Study population

The hypertensive subjects were recruited from the screening BP of staffs at Mahidol University (Salaya campus), Ampol food factory and Thepphadungporn factory staff. BP measurement was repeated twice for anyone who was high and interested to join the study. Study subjects were enrolled sequentially in groups; the first group began in June 2004, and last group started in January 2005.

The inclusion criteria were:

1. Age between 20 and 60 years.
2. Essential hypertension newly diagnosed or previously diagnosed according to JNC VII criteria (systolic BP is in the range of 140-160 mmHg, and/or diastolic BP 90-95 mmHg on more than three separate occasions).
3. They were not taking any vitamin or mineral supplements.

The exclusion criteria were:

1. Heart disease.
2. Kidney's disease or impaired renal function (mean plasma creatinine concentration >200 mmol/l).
3. Edema from whatever cause.
4. Pregnancy.
5. Person, who were on a special diet.

The study protocol was approved by the Committee on Human Research of Mahidol University. Written inform consent was obtained from all subjects after an oral explanation of the study.

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3.2 Study design

The study consisted of 2 phases including 1) a 3-wk pre-experimental phase and 2) a 9-wk experimental phase.

1. **Pre-study phase** (3 wk): This phase was divided into a 2-wk baseline and 1 wk run-in period.

Baseline (2-wk period): Subjects consumed their own usual diets. The following activities were performed:

- In the first week, subjects were asked to record their usual intakes for 3 days. The example of 24-h dietary record is in Appendix A. The 24-h dietary records were calculated for energy, macronutrient, Na and K intakes by IMMUCAL program version ND 2. These data were used to estimate energy of diets provided during the study period.
- In the last week, subjects collected 24-h duplicate meals along with 24-h urine collection for 3 days. The duplicate meals and 24-h urine were analyzed for Na and K.
- Menu items used during the experimental period were selected by individual subject based on their merit. The example of the menu item questionnaire is in Appendix B.

Run-in (1-wk period): Subjects consumed diet (using regular condiments) prepared by the researchers. The diet was a seven-day rotating menu. This was done in order to allow all subjects to adjust to the regimen and establish energy need. Estimation of energy requirement was calculated by using the Harris Benedict equation (85) plus an activity factor. Amount of foods provided was also changed or adjusted proportionately to have enough food and maintain body weight of individual subject. Lunches were consumed in the Institute of Nutrition or their workplace. Subjects picked up dinner, breakfast at lunch for home consumption. Weekend meals were picked up on Friday and kept in refrigerator. Subjects warmed their foods before eat followed the instructions given by a researcher.

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2. Study phase

The study was a randomized crossover controlled feeding trial consisting of two 4-wk feeding periods, separated by a 1-wk washout period, in which subjects were free to consume self-selected diets. Subjects were randomly assigned to receive either the normal sodium condiment diet (NSD), or low-sodium condiment diet (LSD) for 4 wk. At the end of this period, the study diet was stopped for 1 wk; a crossover was then made, with subjects in group I take the NSD before LSD and subjects in group II take LSD before NSD. Based on the randomization, there were eight subjects who were in group 1, and the rest were in group 2. The diagram of study protocol is shown in Figure 3-1.

Lunches were consumed in the Institute of Nutrition or their workplace. Subjects picked up their food the same protocol as that of the run-in period. Researcher provided fish sauce or soy sauce for individual subject day to day in which they could add to their foods based on their merit. Amount of condiments added by individual subject was recorded every day.

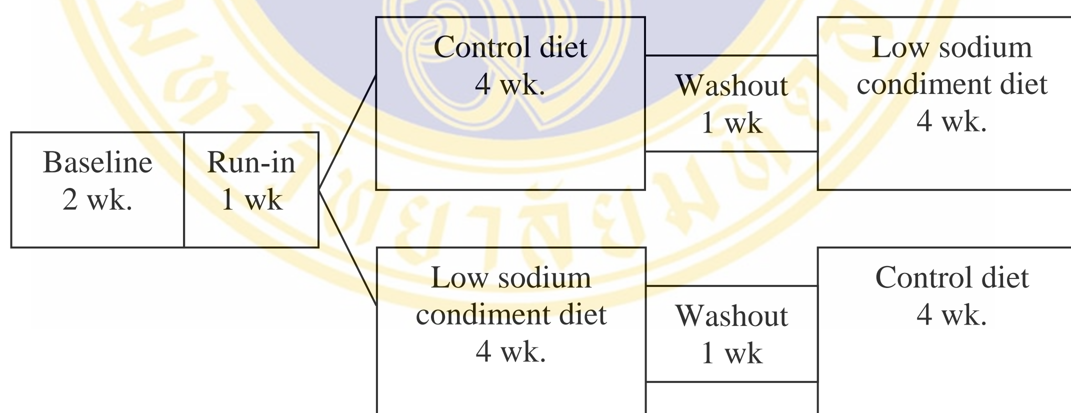

Figure 3-1. Study protocol

3.3 Study diet

A fourteen-day rotating menu was developed for individual subject based on their preference and energy need. The information obtained during the run-in period was the basis for the developing the study menu. Examples of the menus used in this study are shown in Appendix C. Major nutrient composition (energy, protein, fat

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carbohydrate and fiber) of each menu was calculated by using INMUCAL program version ND 2.

Na and K contents of all condiments used in this study were analyzed. These values (appendix D) were used in the INMUCAL program to calculate Na and K contents in each menu.

The preliminary study of sensory evaluation of the developed menus using normal-Na condiments was conducted in order to obtain food menus that would be well-accepted by the subjects. The same 14-day rotating menu was given to the subjects either in the NSD or LSD. The only difference was the use of low-Na condiments to substitute for the regular ones.

All subjects agreed to consume only and all the foods presented to them. The only exceptions were water, juice (no added Na), tea, coffee and fresh fruits, depending on individual eating habit. The amount of beverage (except water) or fruits taken was recorded daily. In order to make foods more favorable to them, subjects were allowed to add extra condiments provided by the researcher into their experimental diets while the amount of all added condiments were recorded and calculated.

Raw ingredients used during the study period were purchased in bulk from the same market to decrease variation due to processing batches.

Compliance to diets was evaluated in detail by the researcher during each appointment and also by regular 24-h urinary Na excretion analysis at the middle and at the end of each period.

3.4 Measurement

1. Height and body weight

Measurement for height (Ht) was done with a microtoise. Body weight (BW) was measured using beam balance scale with light clothing and without shoes at the beginning of the study. BW was recorded weekly by using the same beam balance scale. Readings of Ht and BW were taken to the nearest 1-centimeter and 0.1 kg, respectively.

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2. Blood pressure

Prior to the study, each sphygmomanometer was calibrated by a specialist in The Medical Instrument Department, Ramathibodi Hospital. The digital sphygmomanometer used by individual subject was validated with a mercury sphygmomanometer every Monday. Subjects were asked to measure BP themselves by using their own digital sphygmomanometers after the researcher measured BP by using mercury sphygmomanometer. The readings from these two instruments were compared.

The standard BP was measured by the same researcher after lunch everyday on the right arm in a sitting position using the same mercury sphygmomanometer (after rest for 5 minutes). Korotokoff sound 1 was used for systolic and sound 4 for diastolic. Home BP was measured by the subjects themselves using NISSEI digital BP measurement model 157. All subjects were well instructed and standardized the way to measure BP by this instrument. For each record the mean from 3 measurements was used. Stressful occurrences such as illness, accidents or crises were recorded and these readings were excluded.

Baseline BP was defined as the average of the three preintervention measurements taken on three separate occasions at least 2 days apart. BP at each week during the intervention period was defined as the average of the BP measured on Tuesday, Wednesday and Thursday.

Diurnal blood pressures were self measured for 3 times a day: just after waking up, before dinner, and just before sleeping on the right arm and sitting position (after resting for 5 minutes). Diurnal BP at each week during the intervention period were taken in the same manner as the standard BP.

3. 24-h urine collection

Subjects were asked to collect complete 24-h urine sample for continuous 3 days, every two weeks. Subjects received verbal and written instruction (Appendix E) on the technique of collecting 24-h urine sample. On the first morning of the urine collection, subjects were asked to discard their first urine specimen and from then on to collect all specimens for the next 24 h, including the first urine specimen of the next day. The urine was collected in plastic containers without any preservative for 24-h and kept in cool area. Prior to the urinary collection, every subject was given a urine

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container which had a name tag written on it. The tight lid was rechecked to prevent any leak that may happen. In addition, the urine container was kept upright position all the time and put inside a fixed fabric container during the transportation.

Urinary volume was measured on each collection day, and 30 mL of urine was aliquoted in five plastic bottles and kept frozen at -20 °c for further analysis of Na and K. During each collection period, the subjects were asked whether urine collection was completed, or any leaking during the 24-h collection. Completeness of urine excretion was ascertained on the basis of the 24-h creatinine excretion. Na and K levels in urine were analyzed by AOAC method (86) using flame Atomic Absorption Spectrometry (Model Spectr AA-20, Varian Associates, Australia). Creatinine concentrations in urine were analyzed by the Jaffe method (87).

4. Blood collection

Blood screening was done during the first week of pre-study phase. If there were any abnormal values that affected the study, subjects were informed and excluded from the study. Blood samples were collected at the end of each study diet for routine analysis. Blood samples were drawn from 7:00 A.M. to 8:00 A.M. after an 8-h overnight fast. An 11 mL blood sample was taken by venipuncture. Blood was drawn into two safety tubes: 4 mL containing EDTA for plasma and 7 mL without anticoagulant for serum. Blood samples were centrifuged within ½ to 1 hr of the blood drawn to separate plasma and serum. All samples were stored at 4-7 ^{o}C during transportation to the Pathophysiology Department of Ramathibodi Hospital for further analysis.

5. Stress status

Stress status of individual subject was evaluated by a stress level questionnaire (Appendix F) developed by Psychiatry Department, Ministry of Public Health at the end of each study period.

3.6 Statistical analysis

Statistical for the Social Science (SPSS) for window Release 13.0, Standard Version was used to perform statistical analyzes. The data of each variable was tested the normality of the distribution using the Kolmogorov-Smirnov test before further analyses. The statistical significance was considered at P-value less than 0.05.

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Comparison of dietary nutrients intake, Na and K intakes between the NSD and LSD were analyzed by using paired t-test. The average BP on Tuesday, Wednesday and Thursday during run-in, wk 1, wk 2, wk 3 and wk 4 of the NSD and LSD were used to represent BP in each week. Repeated measure of ANOVA models adjusted for age and BMI was used to test BP responded at the end of each intervention diet, weekly change in both systolic and diastolic BP during the NSD and LSD, the change of diurnal BP between time of the day and the difference of urinary Na and K excretion between each intervention diet. Least significant difference was used to compare multiple comparisons. Comparison of the changes in both systolic and diastolic BP at the same time of each intervention diet was analyzed by using paired t-test. In addition, carry over effect was determined by comparison of the initial BP between the NSD and LSD using pair t-test. The average of 3-d Na, K intake and urinary Na, K excretion at wk 2 and wk 4 of each diet were used to test the correlation. Pearson]s correlation was used to determine correlations between Na or K intake and urinary Na or K excretion.

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CHAPTER IV
RESULTS

Eighteen hypertensive subjects according to the criteria of JNC VII were enrolled in the study. There were 15 stage I hypertensive subjects who had recently been diagnosed, one stage I hypertensive with diabetes mellitus also took diabetes mellitus medication (glybenclamide 5 mg in the morning and evening) and 2 hypertensive subjects took antihypertensive medications. One person took prazosin 1 mg and hydrochlothiazide 50 mg in the morning and the other took nifedipine 30 mg in the morning. There was no change in doses or types of medication before starting study for at least 3 months and during the study. All subjects completed the study. No one took any supplements or drink any alcohol during the study. There was one subject who smoked a curl of cigarette after meal everyday.

Part I General characteristics of participants at baseline.

General characteristics of the participants at baseline of the study are shown in table 4.1. The subjects consisted of 9 females and 9 males. Their mean age, BW and body mass index (BMI) with standard deviation were 44.4 $\pm$ 8.2 years, 64.6 $\pm$ 11.1 kg and 24.6 $\pm$ 3.0 kg/m^2, respectively. Systolic and diastolic BP were 147.9 $\pm$ 12.4 mmHg and 96.6 $\pm$ 9.0 mmHg, respectively. Mean baseline energy, Na and K intakes of all subjects, which was calculated from 3-day 24 h dietary records before beginning the study were 1,456.4 $\pm$ 312.91 kcal/d, 2,907.9 $\pm$ 966.1 mg/d and 1,230.2 $\pm$ 413.4 mg/d, respectively. Cholesterol intakes from the same dietary records of male subjects was 301.2 $\pm$ 174.4 mg/d, which was significantly higher than that of female (103.6 $\pm$ 96.1 mg/d).

Baseline stress score and blood biochemistry of all participants were in normal ranges except that plasma cholesterol of all subjects and triglyceride in male were slightly higher than the reference ranges (Table 4.2). Baseline creatinine and uric acid values of male were also slightly higher than that of female.

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Table 4.1 General characteristics of subjects at baseline.

Parameters	Male	Female	Total
Sex: Female/Male	9	9	18
Age (yrs)	43.8 ± 9.5	45.0± 7.1	44.4 ± 8.2
Height (cm)	164.8± 6.7	157.4 ± 6.7	161.1 ± 7.5
Weight (kg)	71.4 ± 11.1	57.8 ± 6.0	64.6 ± 11.1
BMI (kg/m^2)	26.0 ± 3.0	23.3 ± 2.6	24.6 ± 3.0
Stress score (unit)	9.3 ± 5.5	12.0 ± 7.4	10.7 ± 6.5
Systolic blood pressure (mmHg) [1]	149.4 ± 13.6	146.4 ± 11.6	147.9 ± 12.4
Diastolic blood pressure (mmHg) [1]	98.7 ± 8.3	94.4 ± 9.5	96.6 ± 9.0
Energy intake (Kcal/d) [2]	1434.5± 366.6	1481.4 ± 294.2	1456.4 ± 312.9
Energy distribution (% of total calories) [2]			
Carbohydrate	58.9 ± 9.3	63.9 ± 9.8	61.2 ± 9.2
Protein	16.0 ± 3.6	13.3 ± 3.7	14.7 ± 3.7
Fat	25.1 ± 6.1	22.9 ± 6.9	24.1 ± 6.1
Dietary fiber (g/d) [2]	8.0 ± 4.3	9.6 ± 4.4	8.8 ± 4.1
cholesterol (mg/d) [2]	301.2 ± 174.4 [a]	103.6 ± 96.1 [b]	209.0 ± 166.1
Sodium intake (mg/d) [2]	2921.9 ± 1251.3	2892.0 ± 711.4	2907.9 ± 966.1
Potassium intake (mg/d) [2]	1346.8 ± 423.6	1097.0± 423.2	1230.2 ± 413.4

Data are presented as means ± SD.

[1] average of 3 measurements at screening using a standard sphygmomanometer.

[2] calculation from 24-h dietary record for 3 days.(n=15)

The different superscripts mean significant difference at P<0.05, paired t-test

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Table 4.2 Blood biochemistry of subjects at baseline.

Parameter	Male	Female	Total	Normal range*
Sodium (mmol/L)	141.9 ± 2.7	141.1 ± 1.5	141.5 ± 2.1	136.0 - 145.0
Potassium (mmol/L)	4.3 ± 0.1	3.9 ± 0.4	4.1 ± 0.4	3.5 - 5.5
Cholride (mmol/L)	104.4 ± 3.0	103.9 ± 2.6	104.2 ± 2.7	98.0 - 107.0
Bicarbonate (mmol/L)	25.1 ± 2.0	24.9 ± 3.8	25.0 ± 3.0	22.0 - 29.0
Calcium (mmol/L)	2.4 ± 0.1	2.3 ± 0.1	2.4 ± 0.1	2.2 - 2.62
Glucose (mmol/L)	5.0 ± 0.6	5.6 ± 2.1	5.3 ± 1.5	4.2 - 6.4
Total protein (g/L)	78.1 ± 4.0	77.8 ± 4.1	77.9 ± 3.9	64.0 - 82.0
Albumin (g/L)	46.8 ± 3.4	45.6 ± 3.1	46.2 ± 3.2	43.1 - 53.3
Alkaline phosphatase (U/L)	74.7 ± 15.3	70.3 ± 32.4	72.5 ± 24.7	50 - 136
AST (U/L)	31.1 ± 16.5	21.1 ± 4.4	26.1 ± 12.8	8.0 - 40.0
ALT (U/L)	62.6 ± 41.2	39.4 ± 6.7	51.0 ± 31.0	6.0 - 48.0
BUN (mmol/L)	4.4 ± 1.1	3.6 ± 0.8	4.0 ± 1.0	2.5 - 6.4
Creatinine (µmol/L)	85.2 ± 13.8 [a]	53.6 ± 7.8 [b]	69.4 ± 19.6	53.0 - 115.0
Uric acid (mg/dL)	7.3 ± 1.7 [a]	4.6 ± 1.0 [b]	6.0 ± 1.9	2.0 - 7.0
Cholesterol (µmol/L)	5.9 ± 1.0	5.7 ± 1.0	5.8 ± 1.0	3.8 - 5.2
Triglyceride (mmol/L)	2.1 ± 1.0	1.4 ± 0.7	1.8 ± 0.9	0.34 - 1.7

Data are presented as means ± SD.
The different superscripts mean significant difference at $P<0.05$, paired t-test
*normal range based on Harrison's Internal Medicine (88).

Part II. Diet adherence and dietary intake.

All subjects ate intervention diets, the NSD and LSD, provided by researcher every day without any problems. They also ate extra fruits and milks in which they normally ate before participating in this study. There were three subjects who consumed other kinds of foods, including bread, glutinous rice streamed with banana and coconut milk, fish ball, fried fermented pork, noodle, salapao sai moosub (Thai), etc. during both study periods. Extra food items eaten by subjects were recorded daily and calculated for their nutritive values including Na and K contents by using INMUCAL program version ND 2 and reported in Table 4.3.

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Average actual dietary nutrient intakes consumed by subjects during each intervention period are shown in table 4.3. There were no significant differences in energy intake, percentage of energy distribution, dietary fiber and cholesterol intake between the NSD and LSD. Na intake during the LSD was approximately 2,500 mg (108.7 mmol), which was lower than that of the NSD (3,840 mg or 167.0 mmol). Inversely, K intake was approximately 1.5 times significantly higher during the LSD compared with that of NSD.

Table 4.3 Actual daily nutrient intakes of subjects during the study periods.

Variables	**NSD**	**LSD**
Energy (kcal)		
Experimental diet	1663.9 ± 306.2	1676.5 ± 315.5
Extra foods eaten by subjects	215.5 ± 255.4	225.0 ± 247.4
Total	1879.4 ± 435.6	1900.1 ± 416.5
Energy distribution (% of total calories)		
Carbohydrate	48.8 ± 10.9	50.7 ± 9.9
Protein	21.4 ± 11.1	20.3 ± 11.4
Fat	33.5 ± 5.8	32.8 ± 7.9
Dietary fiber (g)	10.1 ± 8.0	10.8 ± 9.8
Cholesterol (mg)	210.9 ± 94.2	200.5 ± 101.3
Sodium (mg)		
Experimental diet	3343.4 ± 313.9 ^{a}	2104.0 ± 205.8 ^{b}
Extra foods eaten by subjects	497.5 ± 353.3	402.1 ± 218.6
Total	3840.8 ± 557.0 ^{a}	2506.0 ± 343.8 ^{b}
Potassium (mg)		
Experimental diet	1815.7 ± 229.1 ^{b}	3043.3 ± 351.4 ^{a}
Extra foods eaten by subjects	275.0 ± 375.9	524.9 ± 236.8
Total	2090.7 ± 495.3 ^{b}	3567.5 ± 497.2 ^{a}
Sodium:potassium ratio	1.9 ± 0.3 ^{a}	0.7 ± 0.1 ^{b}

Data are presented as means ± SD.

The different superscripts mean significant difference at P<0.05.

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Na intake during both the NSD and LSD mainly came from the condiments used during the cooking process and added during eating. Fifteen subjects added fish sauce, whereas the rest (two subjects) added soy sauce. Amounts of condiments added during the NSD (5.7 ± 5.0 g.) and LSD (6.2 ± 4.5 g.) were not different.

Table 4.4 Amount of condiments including their sodium and potassium contents added in the experimental diets during eating.

Variables	**NSD**	**LSD**
Table condiments added during eating (g)	5.7 ± 5.0	6.2 ± 4.5
sodium content (mg)	404.8 ± 355.7	303.6 ± 211.2
potassium content (mg)	62.0 ± 96.2	308.4 ± 235.6

Data are presented as means ± SD.

Food ingredients contributed approximately 18.7 % and 29.1 % of total Na intake in the NSD and LSD, respectively. K intake during the NSD mainly (88.9 %) came from the food ingredients, whereas K intake during the LSD derived approximately equal amounts from both food ingredients and condiments used during the cooking process (Figure 4.1). However, total Na and K intakes either in the NSD or LSD were less than the upper limit of recommendation. In the present study, using low Na condiments substitute for the regular ones could decrease Na intake by 1,340 mg/d (42.6%) and increase K intake by 1,463 mg/d.

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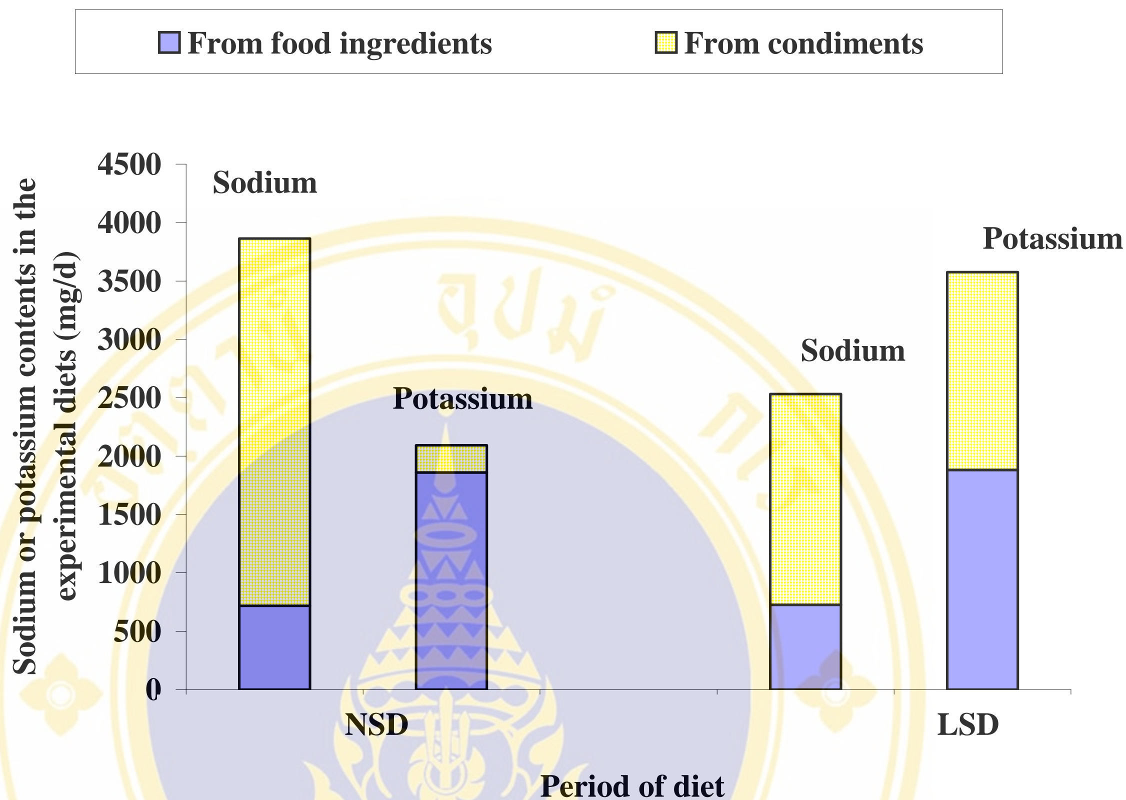

Figure 4-1 The proportions of sodium and potassium intakes derived from food ingredients and condiments used during the cooking and eating

Part III. Effects of sodium restriction by using low sodium condiments substitute for normal sodium condiments.

1. Body weight and stress score

Body weight did not significantly change and stress scores were in a normal range throughout the study (Table 4.5).

Table 4.5 Body weight and stress score of subjects at the end of each intervention diet.

Variables	NSD	LSD
Body weight (kg)	64.6 ± 11.0	64.2 ± 10.8
Stress score (units)	9.7 ± 4.1	9.7 ± 5.4

Data are presented as means ± SD.

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2. Blood biochemistry

All blood biochemical profiles were within normal ranges except that plasma cholesterol and triglyceride were slightly higher than the reference ranges. However, these values were not different from the baseline. All blood biochemical profiles did not differ between the NSD and LSD (Table 4.6).

Table 4.6 Blood biochemistry of subjects each intervention diet.

Variables	NSD	LSD	Normal range*
Sodium (mmol/L)	141.1 ± 1.6	141.6 ± 2.3	136.0 - 145.0
Potassium (mmol/L)	4.2 ± 0.4	4.2 ± 0.4	3.5 - 5.5
Cholride (mmol/L)	103.8 ± 1.8	104.1 ± 2.1	98.0 - 107.0
Bicarbonate (mmol/L)	25.0 ± 3.0	25.0 ± 2.2	22.0 - 29.0
Calcium (mmol/L)	2.4 ± 0.1	2.2 ± 0.3	2.2 - 2.62
Glucose (mmol/L)	5.6 ± 1.3	5.1 ± 1.8	4.2 - 6.4
Total protein (g/L)	77.8 ± 5.1	77.8 ± 3.9	64.0 - 82.0
Albumin (g/L)	46.2 ± 2.6	48.3 ± 9.4	43.1 - 53.3
Alkaline phosphatase (U/L)	72.1 ± 32.1	76.5 ± 29.5	50 - 136
AST (U/L)	24.7 ± 17.7	22.6 ± 12.4	8.0 - 40.0
ALT (U/L)	49.3 ± 35.7	46 ± 25.8	6.0 - 48.0
BUN (mmol/L)	4.5 ± 0.7	4.6 ± 0.9	2.5 - 6.4
Creatinine (?mol/L)	73.8 ± 20.	73.9 ± 25.2	53.0 - 115.0
Uric acid (mg/dL)	6.2 ± 2.2	6.2 ± 2.1	2.0 - 7.0
Cholesterol (?mol/L)	6.1 ± 0.9	5.9 ± 0.9	3.8 - 5.2
Triglyceride (mmol/L)	2.0 ± 2.0	1.6 ± 0.8	0.34 - 1.7

Data are presented as means ± SD.

* normal range based on Harrison 's Internal Medicine (88).

3. Blood pressure

Comparisons of BP from baseline until the end of the study are shown in table 4.7. Both systolic and diastolic BP were significantly lower at run-in compared with initial period. After 4 wk of either NSD or LSD, systolic BP decreased by 5.3

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mmHg (95% CI, -1.6 to –9.0) and by 5.7 mmHg (95% CI, -1.7 to –9.5), respectively. Diastolic BP decreased from run-in period slightly by 1.4 mmHg (95% CI, 0.6 to –3.4) and 2.7 mmHg (95% CI, 0.3 to –5.8), respectively. Similarly, mean arterial pressures (MAP) were significantly lower at run-in compared with baseline. Further reduction in MAP was observed either in the NSD or LSD. However, MAP during the NSD and LSD was not significantly different.

Table 4.7 Blood pressure response at the end of each intervention diet.

Blood pressure (mmHg)	Baseline	Run-in	NSD	LSD
Systolic	147.9 ± 12.4 [a]	143.0 ± 15.9 [b]	137.7 ± 18.0 [c]	132.0 ± 17.0 [d]
Diastolic	96.6 ± 9.0 [a]	91.4 ± 11.1 [b]	90.0 ± 12.4 [b]	88.7 ± 9.0 [b]
Mean arterial *	113.7 ± 2.0 [a]	108.6 ± 3.0 [b]	105.9 ± 3.1 [c]	103.1 ± 2.8 [c]

Data are presented as means ± SD.
The different superscripts mean significant difference at P<0.05, repeated measure ANOVA adjusted for age and body mass index.
*MAP calculated from (SBP+2DBP)/3

Changes in the BP during each week of each intervention diet were investigated as shown in figure 4.2. BP of the first day of each intervention diet was used as the initial value for the comparison. Using pair t-test, the initial systolic BP of the NSD (138.4 ± 18.6) and LSD (139.1 ± 19.5) was not different. However, the initial diastolic BP of the LSD (94.5 ± 11.8) was significantly higher than that of NSD (90.1 ± 83.1).

There was no change in either systolic or diastolic blood pressure in any week during the NSD diet. For the LSD, both systolic and diastolic BP significantly reduced by the end of wk 1 compared with the initial value, with no further change in these parameters was observed after that. Pattern of change in MAP during the NSD and LSD was the same as that of diastolic BP. Differences in the reduction of systolic BP between the NSD and LSD were significant at wk 1, wk 3 and wk 4, whereas the difference in the reduction of diastolic BP between two diets was observed only at wk 1.

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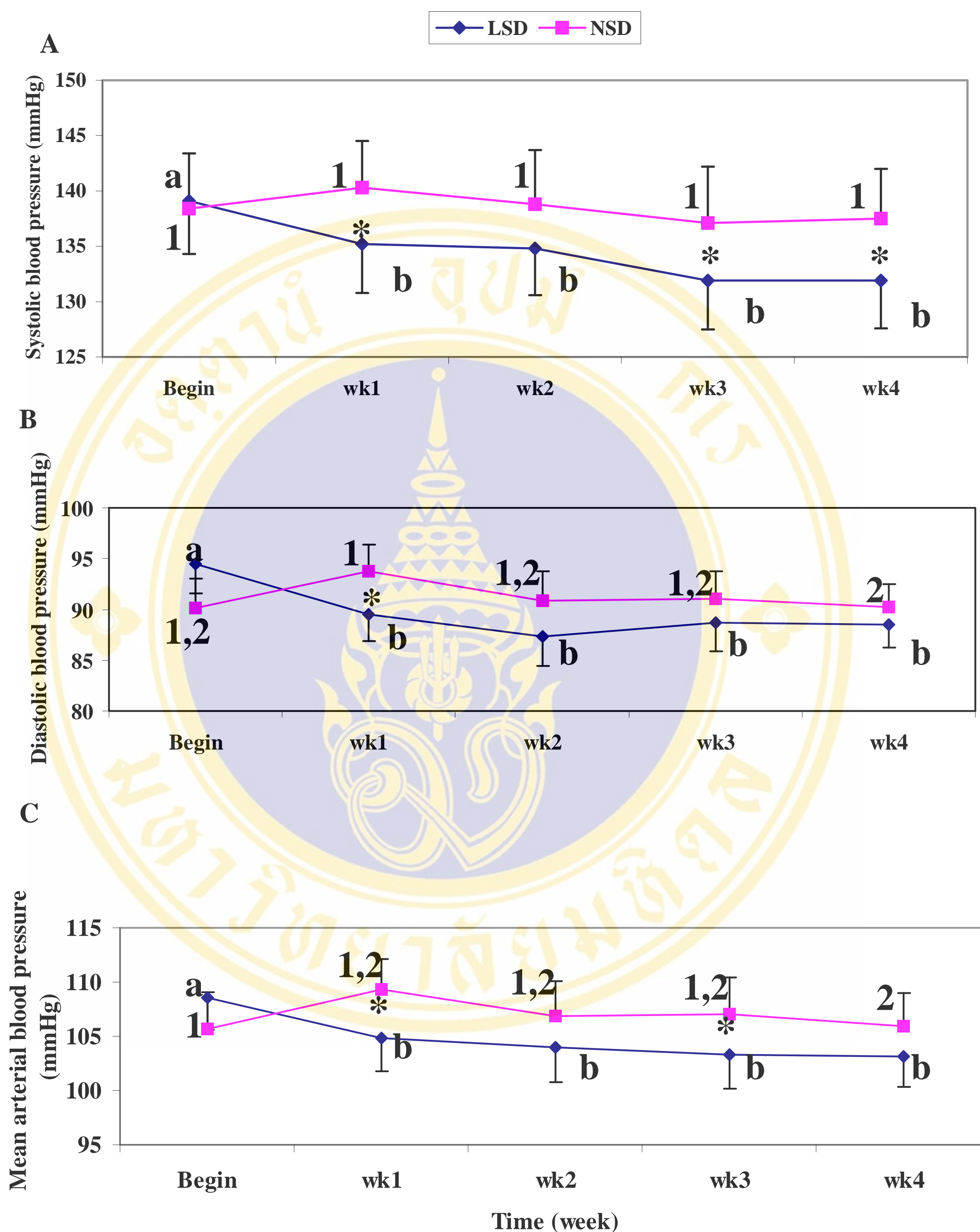

Data are presented as means ± SEM.
Different superscripts mean difference within the same diet (P < 0.05), repeated measure ANOVA adjusted for age and body mass index.
* significant difference from NSD at the same time (P<0.05), paired t-test.

Figure 4-2 Systolic (A), diastolic (B), and mean arterial (C) blood pressure from the initial until the end of each intervention diet.

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The change in category of BP in these subjects after on NSD or LSD was observed and demonstrated in Table 4.8. Based on the JNC VII criteria and classified by systolic BP, there were approximately 50 % of subjects (4 out of 8) in group II changing from stage I hypertension to prehypertension after completing the NSD diet, and increased to 75 % when they continued with the LSD. Moreover, systolic BP of one subject in this group turned to normal during the LSD. When classified the category of hypertension by using the diastolic BP, there were still 50 % of subjects in this group changing from the stage I hypertension to prehypertension after the NSD, but there was no further change in the category of hypertension when they continued with the LSD.

For the group I, 50 % of subjects improved from stage I hypertension to prehypertension during the run-in phase either classified by systolic or diastolic BP. When they completed the first phase (LSD) and classified the category of hypertension by using systolic BP, around 40 % of subjects were still in the stage I hypertension, whereas the rest of subjects improved to prehypertension (30%) and normal blood pressure (30%). When they returned to NSD during phase 2, there was one subject who was normal blood pressure during the LSD turned to prehypertension, otherwise were the same. Similar trend was observed when classified category of hypertension using the value of diastolic BP.

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Table 4.8 Change in category of blood pressure classified by systolic (A) and diastolic (B) blood pressure after completing each phase of the study

Group	Category of BP	Phase of the study			
		Baseline	Run-in	Phase 1*	Phase 2*
A. Classified by systolic BP					
Group I	Stage I hypertension	10	5	4	4
	Prehypertension	-	5	3	4
	Normal	-	-	3	2
Group II	Stage I hypertension	8	3	4	1
	Prehypertension	-	5	4	6
	Normal	-	-	-	1
B. Classified by diastolic BP					
Group I	Stage I hypertension	10	5	5	4
	Prehypertension	-	4	3	4
	Normal	-	1	2	2
Group II	Stage I hypertension	8	4	3	3
	Prehypertension	-	3	4	4
	Normal	-	1	1	1

*Phase 1 and 2 were the study phase based on group of the subjects;
Group I: subjects were on LSD at phase 1 and NSD at phase 2
Group II: subjects were on NSD at phase 1 and LSD at phase 2

4. Diurnal blood pressure

Diurnal BP during the intervention studies were measured and recorded by individual subjects using NISSEI digital BP measurement. The validation of this measurement with the mercury sphygmomanometer was done and found that the values of BP reading by these two devices did not differ (Data not shown). Systolic BP was approximately the same throughout the day. However, the diastolic BP were different between just after waking up and just before sleeping during the run-in and NSD, but there was no difference in these measurements during the LSD.

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Table 4.9 Diurnal systolic and diastolic blood pressures at each intervention diet

Variable	Time		
	Morning	Evening	Before bed
Blood pressure (mmHg)			
Systolic			
Run-in	137.7 ± 11.8	137.5 ± 16.1	135.3 ± 15.9
NSD	134.2 ± 16.0	134.3 ± 15.7	131.8 ± 16.5
LSD	130.8 ± 16.3	131.4 ± 17.4	129.7 ± 17.1
Diastolic			
Run-in	94.6 ± 9.6 [a]	89.7 ±11.6 [b]	87.7 ± 12.1 [b]
NSD	91.3 ± 10.9 [a]	89.0 ± 11.8 [ab]	87.2 ± 12.4 [b]
LSD	87.7 ± 12.7	86.0 ± 12.4	85.6 ±13.5

Data presented as means ± SD.
Different superscripts mean significant difference within the same diet period (P<0.05), repeated measure ANOVA adjusted for age and body mass index.

Part IV. Urinary sodium and potassium excretion

Urinary creatinine excretions of all subjects were in the normal range indicated completeness of 24-h urine collection. Mean 24-h urinary Na and K excretions during the second and fourth week of each diet were not different. Thus, the average of excretions between week two and four were used for the comparison. Mean urinary Na excretion at baseline was 2,899 mg/24 h (126.0 mmol/24 h), a value higher than that found while participants ate NSD. During the LSD phase, 24-h Na excretion decreased from the NSD phase by 558 mg/24 h (24.3 mmol/24 h). Mean urinary K excretion during the NSD was 1,180 mg/24 h (30.3 mmol/24 h), and increased to 1,802 mg/24 h (46.2 mmol/24 h) during the LSD. The mean urinary Na to K ratio was 3.6 at baseline, and decreased significantly during the NSD and LSD.

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Table 4.10 Daily urinary sodium, potassium and creatinine excretions of subjects during each intervention diet [1]

Time	Na (mmol/d)	K (mmol/d)	Na/K ratio	Cr (mmol/L)
Baseline	2899.4 ± 1008.0 [a]	939.1 ± 477.1 [c]	3.6 ± 1.4 [a]	8.4 ± 3.5
NSD				
wk 2	2486.4 ± 856.6	1133.8 ± 671.4	2.7 ± 1.3	8.2 ± 3.9
wk 4	2675.4 ± 1083.7	1226 ± 622.3	2.5 ± 1.0	9.2 ± 4.3
average	2580.9 ± 967.5 [a]	1179.9 ± 367.9 [b]	2.6 ± 1.1 [b]	8.7 ± 4.1
LSD				
wk 2	2093.6 ± 746.1	1904.3 ± 1003.2	1.3 ± 0.6	8.5 ± 3.9
wk 4	1984.2 ± 583.8	1698.7 ± 765.7	1.4 ± 0.7	8.7 ± 3.4
average	2038.9 ± 662.5 [b]	1801.5 ± 885.7 [a]	1.4 ± 0.6 [c]	8.6 ± 3.6

[1] Average of three 24-h urine collection.
Data are presented as means ± SD.
Different superscripts mean significant difference within the same column ($P < 0.05$), repeated measure ANOVA adjusted for age and body mass index.

Na intake and K intakes were positively correlated to the 24-h urinary Na and K excretions (Figure 4-3 and 4-4). Excretion of Na and K were 75.6 % and 67.8 % of their intakes, respectively.

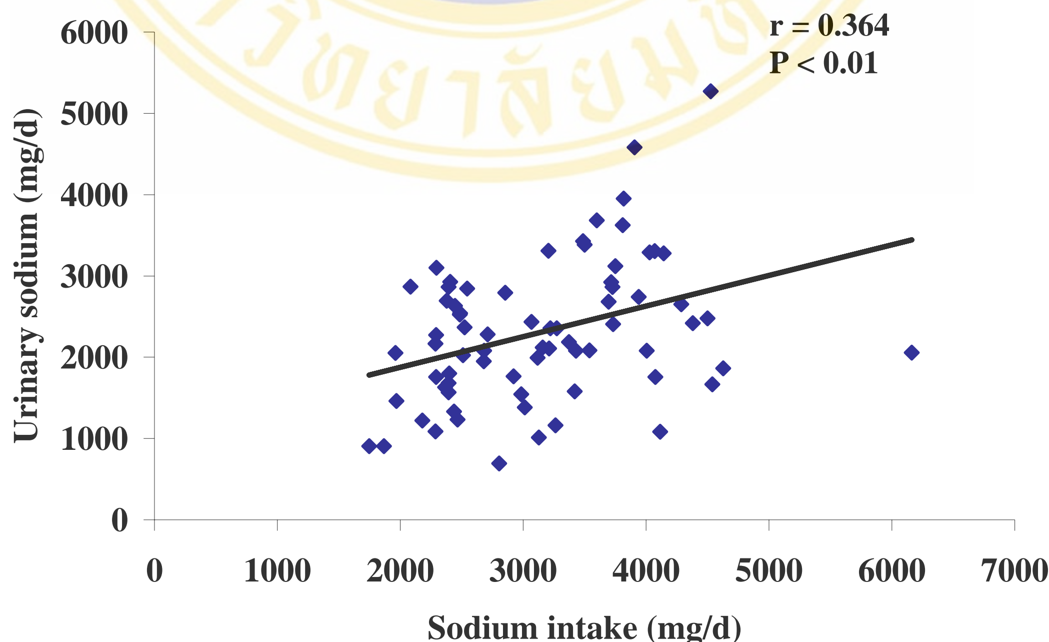

Figure 4-3 Correlation of sodium intake and 24- h urinary sodium excretion

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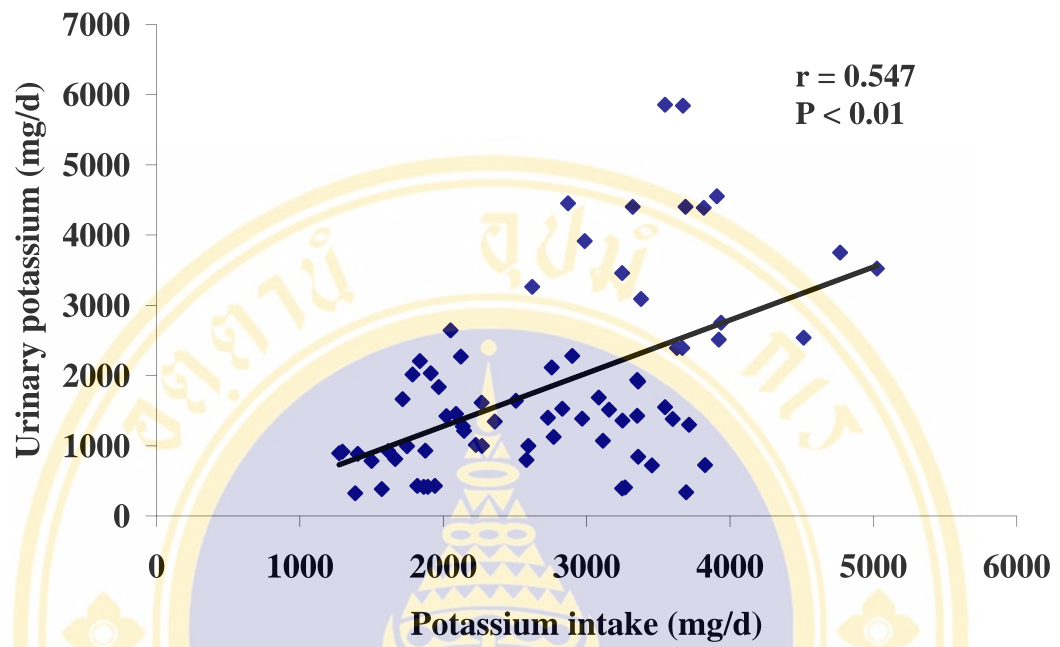

Figure 4-4 Correlation of potassium intake and 24- h urinary potassium excretion

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CHAPTER V
DISCUSSION

This present study investigated the effect of Na restriction by using low Na condiments on blood pressure in hypertensive persons. The randomized crossover design was used in order to prevent the sequence of intervention diet (NSD or LSD) on outcome parameters. However, it is likely that there was a carry over effect in the present study. The diastolic BP of the first day (used as the initial BP) of the LSD was significantly higher than that of the NSD (Figure 4.2 B), whereas there was no difference in the initial systolic BP of these two diets. Seven of ten subjects in group I (who started with the LSD first and followed with the NSD after washout) had lower diastolic BP at the initial of the NSD compared with that of LSD (see raw data in appendix G). This phenomenon demonstrated that the one-wk washout period in this study was not enough. The possible carry over effect in the present study may be apparently smaller treatment effect.

One limitation of this study was that the diet during the washout period was subject's self selected diet, whereas the diet during the run-in period was the controlled normal sodium condiment diet prepared by the researcher. These two different diets prior to the NSD or LSD may affect the initial BP differently.

There was no change in body weight and level of stress between the NSD and LSD. Energy intake and distribution between these two diets were also the same. Only significant difference was the amounts of Na and K intakes. Therefore, the changes in either systolic or diastolic blood pressure observed in the present study came from the amount of Na and K intakes.

Although one subject of this study smoked a curl of cigarette after meal, it is unlikely to affect his BP because pattern of his smoking did not change throughout the study.

Using low Na condiments substitute for the regular ones in the present study could reduce Na intake approximately 1,300 mg (58 mmol) per day. The low Na condiment diet also reduced in systolic BP (5.7 mmHg) and DBP (1.3 mmHg).

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Systolic BP in the LSD was lower than that in the NSD, and diastolic BP tended to downward in the LSD (table 4.7). From this study, using low Na condiments substitute for normal Na condiments during the cooking process effect to reduce in daily Na intake from 3,143 mg to 1,803 mg and increase in daily K intake from 232 mg to 1,695 mg.

According to our results, systolic BP and diastolic BP decreased 5.7/1.3 mmHg with a reduction of 1,300 mg (58 mmol) of Na. In consistent with results of Cutler et al (13), who reviewed 32 randomized trials and concluded that 2,300 mg (100 mmol) reduction of Na intake was associated with a BP fall of 5.8/2.5 mmHg systolic and 2.3/1.4 mmHg diastolic in hypertensive and normotensive subjects, respectively. Cuppucio (15) found in his study that a modest reduction in Na intake of 1,840 mg (80 mmol) produced a reduction in both systolic and diastolic BP of 7.2/3.2 mmHg. Korhonen's study (89) also showed that during the salt restriction diet, urinary Na excretion decreased 1,700 mg (74 mmol), and resulted in significant decrease of 7.1 and 4.2 mmHg in systolic and diastolic BP, respectively.

Furthermore, dietary intervention in the present study included not only a reduction in dietary Na, but also a marked increase in dietary K. This combined approach exerted a greater BP lowering effect than Na reduction alone (63, 90). K intake in this study increased about 1,400 mg (36 mmol). This was consistent with the Franzoni's study (62) who found that supplementing 30 mmol of potassium aspartate per day in hypertensive subjects significantly reduced systolic and diastolic BP of 12/8 mmHg. A meta-analysis of 33 trials by Whelton and He (64) reported a fall in supine BP of 3.1 mmHg systolic and 2.0 mmHg diastolic with a 1.5 to 1.6 higher increase in K excretion than in this study. In Gelegnse study (91) , which combined the effect of Na, K and Mg found that when daily Na intake was lower by 870 mg (38 mmol), K intake was raised by 700 mg (18 mmol), and Mg intake was estimated to be raised by 245 mg (7 mmol) affected to reduction in SBP 7.6 mmHg and DBP 3.3 mmHg.

The present study found that both systolic and diastolic BP decreased during the run-in period. The reason for the reduction in BP in the run-in period was not clear. The possible explanation was the lower amount of Na intake during the run-in period comparing to that during the baseline. Subjects were on the normal sodium condiments diet prepared by the researcher during the run-in period in order to allow

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all subjects to adjust to the regimen and establish energy need. However, this diet was likely to have lower sodium content comparing to their habitual intake in which the consumed at baseline. The baseline urinary Na excretion (2,899 mg/d) was greater than urinary Na excretion in the NSD (2,580 mg/d). Urinary Na excretion during the run-in period was not collected. However, the diet during the run-in period was the same as that of the NSD. Another possible explanation was that there might be some difference in the dietary pattern between the run-in period and the habitual diet at baseline. The researcher tried to set daily menus with all balanced food groups recommended in the Thai food based dietary guideline. This dietary pattern may be better than the original pattern of individual subjects consumed before entering the study, and this may improve their blood pressure.

Most of the participants demonstrated excellent adherence to low Na diets provided in this study. They may readily accept the rationale for the reduced Na intake and the taste of low Na diets was acceptable. However, some participants had difficulty adherence to the experimental diet. This may be due to the limited variation of food choices.

According to the eating pattern of Thai people, condiments are the major source of daily Na intake (92). The proportions of daily Na intake from canned food, instant food and pickled food in Thai are small (92). Daily Na intake in the present study, either the NSD or LSD, also mainly came from condiments. Na intakes from condiments in the NSD and LSD were 3,143 and 1,803 mg, (82 and 71 % of total Na intake), respectively. The condiments were used mainly during the cooking process, approximately 71 and 59 % in the NSD and LSD, respectively. Around 11-12 % of condiments either the NSD or LSD was added by subjects at the table during eating. Daily Na intake from food ingredients was 719 mg (18% of total Na intake) and 727 mg (29% of total Na intake) during the NSD and LSD, respectively. The proportions of Na intake from different sources in the present study reflected the common sodium pattern eating in general Thai populations since all menus used in this study were commonly consumed. However, the sources of daily sodium intake in this study were somewhat different from the western style. The major source of Na in western diet is processed food or preserved food such as bread, ham, sausage, and pizza. On average, the natural Na content of food is about 10 %, while discretionary Na use provides

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another 5 to 10 % and approximately 75 % of total Na intake is from addition of Na during product manufacturing (93).

Most Thai people usually consume foods that are prepared from fresh products such as vegetables, meats and cooked with condiments. The use of low Na condiments substitute for the regular Na condiments seemed to be practical and effective in improving the compliance in BP control. The major advantage is that it does not require substantial change in dietary habits of an individual. In addition, the use of low Na condiments helps controlling the Na intake within the recommendation (94). However, there is also a limitation of this approach for a person who has problem with K excretion such as kidney disease. The use of low Na condiments could increase K intake. Therefore, the amount of low Na condiments used should be aware. In this study, average daily K intake was about 2,100 mg and 3,600 mg in the NSD and LSD, respectively. Although the K intake was increased from using low Na condiments, the available K intake was not more than the current recommendation (94).

The amount of extra condiments in which provided by the researcher during the experimental diets did not differ during the NSD and LSD (Table 4.4). This demonstrated that the salty taste of the LSD was not different. The food leftover either in the NSD or LSD was small and not different indicated the subjects' well acceptability of these two diets.

The diurnal BP was used to show BP of participant in the other time because BP at lunch which detected by researcher may be affected by the white coat situation (25). This study is first to show diurnal variation in BP from self-recorded home BP measurement. Several studies (95-98) from ambulatory blood pressure monitoring (ABPM) showed a similar diurnal variation, reaching the highest level during the morning and declining to reach a lowest at about midnight. A recent study that examined the 24-h ABPM of 379 hypertensive patients found 2 peaks in BP, one in the morning and one in the evening. Assessment of individual patient's sleeping times showed that 75% of participants had an afternoon nap while undergoing 24-h ABPM. This period of time was associated with a significant drop in systolic and diastolic BP close to the levels of the nighttime period (98). Thus, both the morning's peaks of BP seemed to be activity dependent because of all occurred on awaking. White (99)

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concluded that the majority of patients with essential HT, the BP rise steeply at the time of awakening in association with mental and physical activity. BP remains at its highest levels during the 8-h post awakening and then will ordinarily decline with rest, relaxation, or sleep. The mechanism responsible for the phenomenon cannot be classified by the nature of the present investigation.

24-h urinary Na excretion is the most accurate way to estimate dietary Na intake (100). However, it is important to have complete 24-h urine collection. Two methods have been suggested for this assessment: the creatinine and p-amino benzoic acid test (PABA). The use of creatinine is based on the assumption that excretion per kg body mass is constant. This method is often used in clinical and population-based studies because the sample collection procedure and laboratory measurement are simple. The consistency of daily creatinine excretion is, however, still a matter of debate. Several studies indicated that the coefficient of variation of day-to-day creatinine excretion in well-motivated healthy subjects ranged from 5% up to 13% (101, 102). A new method, the PABA test was developed by Bingham et al (102, 103). This test is based on recovery in the urine of three oral 80-mg capsules or tablets of PABA taken with meals. The use of the PABA check test was suggested to be a more sensitive and reliable verification of the completeness of 24-h urine collection than creatinine.

Nevertheless, many studies (101, 104) suggested that when urine was consciously collected, creatinine excretion would be remarkably constant, and could be used as a reliable marker for the completeness of 24-h urine collection. Therefore, we decided to use 24-h creatinine excretion in this study. Before the study, full information on the technique for 24-h urine collection was carefully given in oral and written instructions as well as demonstration to each participant. Creatinine excretion of subject in the present study was in a normal range (8.2 to 9.2 mmol/d). The coefficient of variation in the creatinine excretion in this study was approximately 25.5 %. The possible explanation about the variation of urinary creatinine of this study was variation in body weight, surface area and day to day variations in meat consumption may also partly contribute to the variation in creatinine excretion in individuals.

The percentage of dietary Na was excreted in 24-h urine in the present study was 75.6 %. Other studies reported that 83% (100) and 86% (105) of Na intake,

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estimated from dietary records, was excreted in the urine, whereas apparent absorption of Na was 98.5% (105). The percentage of K excreted in the urine was less than urinary Na excretion both in this study (67.8%) and other study (77%) (105). Urinary K excretion was lower than urinary Na excretion because apparent absorption of K was less than Na (84.5%) (105) and about 15 % of K intake is lost in the feces (106) whereas, about 90 to 98 % of Na intake excreted via urine (94). Probably the insensible losses, which we did not assess, rather than a physiological conservation or retention of Na, may explain the possible balances in our subjects but this method is more expensive.

One possible reason to explain the lower of urinary Na and K excretion found in this study was the difference in the climate. The current study was in tropical country, whereas others were in cold countries. Na and K excretion via sweat depended on heat acclimation (94). For this reason, sweat Na and K losses in this study may be higher than other studies, hence reduced Na and K excretions in urine were observed.

According to our result, urinary Na/K ratio was lower during the LSD (1.4 $\pm$ 0.6) compared with NSD (2.6 $\pm$ 1.1). The use of low Na condiments can lower urinary Na/K ratio to nearest 1. This reduction of urinary Na/K ratio would help to lower BP. Data from the INTERSALT study have identified the relationship between BP and urinary Na/K ratio that BP has been more closely related to the ratio of urinary Na to K. The INTERSALT analysis also showed that a reduction of the 24-h urinary Na to K ratio from 3:1 (170 mmol of Na/ 55 mmol of K) to 1:1 (70 mmol of Na/70 mmol of K) was associated with a 3.4 mmHg reduction in systolic BP (107).

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CHAPTER VI
CONCLUSION

This study demonstrated that restricted Na intake by using low Na condiments substitute for the regular condiments augmented lowering BP in hypertensive persons. BW throughout the study was stable. The energy intake, energy distribution during the run-in, low Na diet (LSD) and normal sodium diet (NSD) periods were not significantly different. The only change was the amount of Na and K intake. The diet during the run-in period was the same as that of NSD. Na intake during low Na and normal Na period were about 2,500 mg/d (109 mmol) and 3,800 mg/d (165 mmol), respectively. K intake during low Na and normal Na period were about 2,100 mg/d (54 mmol) and 3,600 mg/d (92 mmol), respectively. The data showed that using low Na condiments substitute for the regular condiments contributed to decrease in Na intake from condiments about 1,300 mg/d (57 mmol), and increased K intake from condiments about 1,500 mg/d (38 mmol). However, Na and K intake derived from food ingredients during both NSD and LSD periods did not change. The results showed that both systolic and diastolic BP were significantly decreased during the run-in period. Further significant decrease in systolic BP was observed either on normal or low Na condiments. Small decrease in diastolic BP during LSD was observed but did not reach significance.

Self-recorded home blood pressure measurement showed that, systolic BP was approximately the same throughout the study. However, the diastolic BP at just waking up was greater than that at just before sleeping during the run-in and the NSD, but there was no difference during the LSD period.

The relationships between Na, K intake and urinary Na, K excretion of this study were positive correlation. Urinary Na and K excretions were 75.6 % and 67.8 % of their intakes, respectively.

In conclusion, the uses of low Na condiments substitute for the regular ones in this study could modulate reduction of Na intake to the recommended level (2,400

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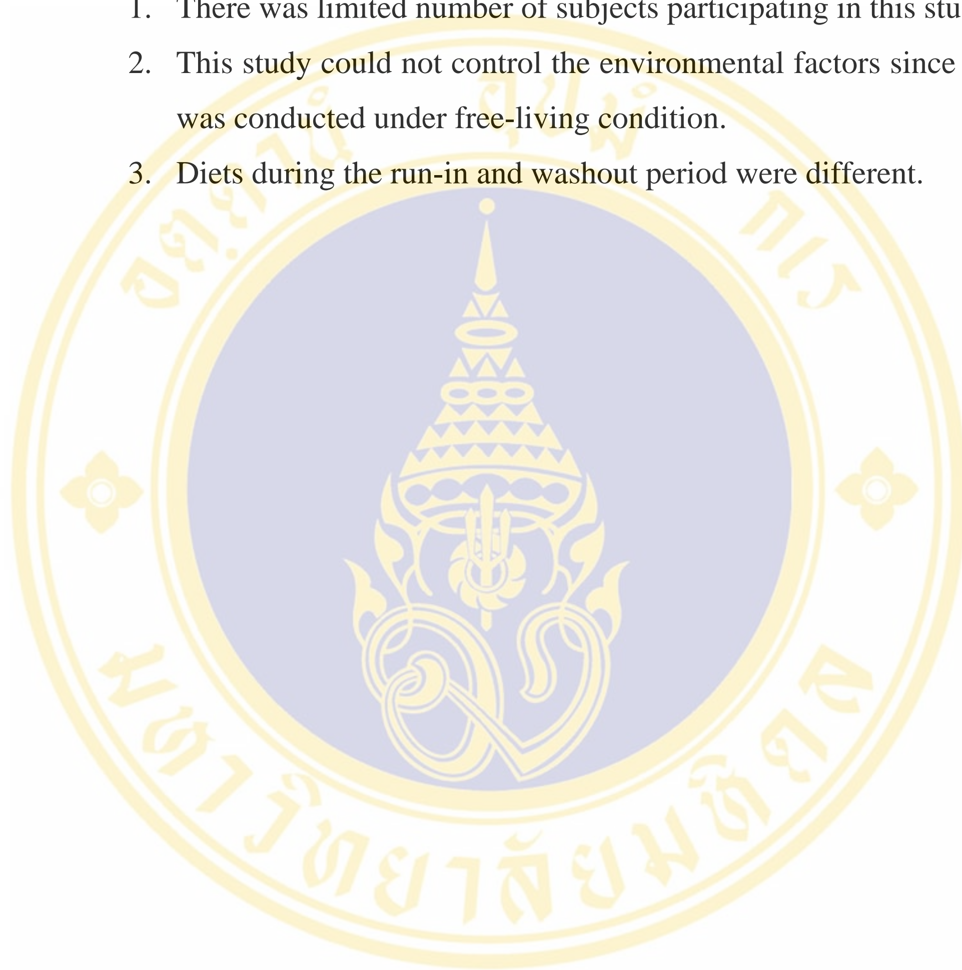
mg/d) and was able to decrease BP in hypertensive people. Therefore using low-Na condiments could serve as an effective choice for a person who needs to control BP.

Limitation of this study.

1. There was limited number of subjects participating in this study.
2. This study could not control the environmental factors since the study was conducted under free-living condition.
3. Diets during the run-in and washout period were different.



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APPENDIX

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Appendix A

Food record form ID……………….

แบบบันทึกรายการอาหารที่รับประทานใน 3 วัน

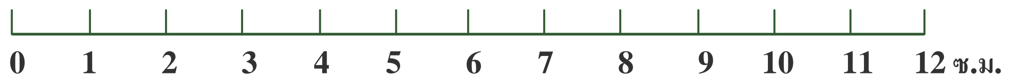
ข้อแนะนำในการบันทึก

1. บันทึกอาหารทุกมื้อทุกชนิดรวมทั้งขนม และเครื่องดื่มที่ท่านรับประทานตลอดวัน ตั้งแต่ท่านตื่นนอนจนเข้านอน (เฉพาะส่วนที่ท่านรับประทานเท่านั้น)
2. บันทึกอาหารที่รับประทานทั้งที่บ้านและนอกบ้าน ถ้าเป็นมื้อพิเศษให้ระบุด้วย เช่น งานเลี้ยงแต่งงาน งานทำบุญ เป็นต้น
3. ข้อความต่อไปนี้เป็นสิ่งจำเป็นในการบันทึก

ก. ระบุเครื่องประกอบของอาหารแต่ละชนิดพร้อมทั้งปริมาตร โดยของแข็งให้ระบุเป็นช้อนตวงหรือทัพพี ส่วนของเหลวให้ระบุเป็น ซี.ซี.หรือระบุตามที่สิ่งตวง-วัดอยู่ในบ้าน ถ้าไม่สามารถประมาณได้ให้พยายามบันทึกในรูปขนาด เช่น ขนาดเล็ก กลาง ใหญ่ หรือขนาดกว้างยาวของอาหารที่ใช้ ซึ่งสามารถประมาณความกว้าง-ยาวของอาหารได้จากไม้บรรทัดที่อยู่ส่วนล่างของแบบบันทึกเช่น ผัดเปรี้ยวหวานต้องระบุว่า รับประทานแตงกวาประมาณ 4 ช้อนโต๊ะ มะเขือเทศ 2 ช้อนโต๊ะ เนื้อหมู 2 ช้อนโต๊ะ หรือระบุว่ารับประทานแตงกวาประมาณครึ่งลูกใหญ่ มะเขือเทศ 1 ลูกเล็ก เนื้อหมู 5 ชิ้นขนาดชิ้นละ 1x2 ซ.ม. ถ้าเป็นเครื่องดื่มควรระบุเป็นปริมาตรหรือขนาดเช่น โค้ก 1 ขวดกลางหรือ 290 ซี.ซี. เป็นต้น

ข. วิธีการปรุงอาหารที่รับประทาน เช่น ปลาทูทอด ไก่ย่าง เป็นต้น

ค. การเติมน้ำตาล น้ำเชื่อม เกลือ หรือน้ำปลา ลงในอาหารหรือเครื่องดื่มกรุณาระบุปริมาณด้วยโดยบันทึกเป็น ช้อนชา ช้อนโต๊ะ ช้อนกินข้าว ดังแสดงในภาพ เช่น น้ำตาล 2 ช้อนชา, คอฟฟี่เมต 1 ช้อนชาในกาแฟ 1 แก้ว



ไม้บรรทัดสำหรับวัดความกว้าง-ยาว-หนาของอาหารหรือขนมที่เป็นชิ้น

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ตัวอย่างการบันทึกอาหาร ID............

ชื่อ-สกุล......นายขยัน หมั่นเพียร..............

วันที่บันทึก...3 กันยายน 2546......☐ วันธรรมดา (จันทร์-ศุกร์) ☐ วันหยุด (เสาร์-อาทิตย์)

มื้ออาหารและสถานที่	เวลา	ประเภท	อาหาร	เครื่องปรุงรส		ปริมาณ	สำหรับเจ้าหน้าที่	
				ใส่ขณะทำอาหาร	ใส่เพิ่มในอาหารปรุงเสร็จ		code	น.น
เช้า	7.00 น.	โจ๊กหมู	โจ๊ก (เฉพาะเนื้อโจ๊ก)			2 ทัพพี		
ที่บ้าน			หมูสับ			2 ช้อนโต๊ะ		
				ซีอิ๊ว		1 ช้อนชา		
			ปาท่องโก๋			1 ตัวขนาดใหญ่		
		กาแฟ	กาแฟ			1 ช้อนชา		
			น้ำตาล			2 ช้อนชา		
			นมสด (ตราหมี)			1 ช้อนโต๊ะ		
		น้ำเปล่า	น้ำเปล่า			1 แก้ว (150 ซี.ซี.)		
กลางวัน	12.00 น.	ก๋วยเตี๋ยวเนื้อ	เส้นก๋วยเตี๋ยว			1 ทัพพี		
ที่ทำงาน			ถั่วงอก			2 ช้อนโต๊ะ		
			เนื้อสด			2 ช้อนโต๊ะ		
			ลูกชิ้นขนาดเส้นผ่าศูนย์กลาง 2 ซม.			5 ลูก		
			น้ำมันกระเทียมเจียว			2 ช้อนชา		
			น้ำก๋วยเตี๋ยว			½ ถ้วยตวง (120 ซีซี.)		
					น้ำตาลทราย (ปรุงรส)	2 ช้อนชา		

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มื้ออาหาร และ สถานที่	เวลา	ประเภท	อาหาร	เครื่องปรุงรส		ปริมาณ	สำหรับเจ้าหน้าที่	
				ใส่ขณะทำอาหาร	ใส่เพิ่มในอาหารปรุงเสร็จ		code	น.น
		ปอเปี๊ยะสด	แป้งปอเปี๊ยะ			3 แผ่น		
			กุนเชียง			2 ช้อนชา		
			เต้าหู้			2 ช้อนโต๊ะ		
			น้ำราด (รสออกค่อนข้างหวาน)			1 ช้อนโต๊ะ		
		สับปะรด	สับปะรดขนาด 2x3 นิ้ว			1 ชิ้น		
					น้ำตาล	1 ช้อนชา		
		น้ำเปล่า	น้ำเปล่า			1 แก้ว (150 ซี.ซี.)		
อาหารเย็น	18.00 น.	ข้าวสวย	ข้าว			2 ทัพพี		
ที่บ้าน		ต้มยำโป๊ะแตก	เนื้อไก่			1 ช้อนโต๊ะ		
			เนื้อหมู			1 ช้อนโต๊ะ		
			น้ำต้มยำ			¼ ถ้วยตวง (60 ซีซี.)		
				มะนาว		1 ช้อนโต๊ะ		
				น้ำตาล		1ช้อนชา		
		ไข่ดาวทอด	ไข่ดาว			1 ฟอง		
			น้ำปลา			2 ช้อนชา		
		ผัดผักบุ้ง	ผักบุ้งจีน			½ ถ้วยตวง		
				เต้าเจี้ยว		1 ช้อนชา		
			น้ำมันพืช (องุ่น)			1 ช้อนโต๊ะ		
ก่อนนอน	22.00 น.	นมสด	นมสดหนองโพรสจืด			1 กล่อง (200 ซีซี.)		

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แบบบันทึกอาหาร ID.............

โครงการการใช้เครื่องปรุงรสโซเดียมต่ำแทนเครื่องปรุงรสปกติต่อระดับความดันโลหิต

ชื่อ-สกุล...

วันที่บันทึก........................... ☐ วันธรรมดา (จันทร์-ศุกร์) ☐ วันหยุด (เสาร์-อาทิตย์)

มื้ออาหารและสถานที่	เวลา	ประเภท	อาหาร	เครื่องปรุงรส		ปริมาณ	สำหรับเจ้าหน้าที่	
				ใส่ขณะทำอาหาร	ใส่เพิ่มในอาหารปรุงเสร็จ		code	น.น

0 1 2 3 4 5 6 7 8 9 10 11 12 ซ.ม.

ไม้บรรทัดสำหรับวัดความกว้าง-ยาว-หนาของอาหารหรือขนมที่เป็นชิ้น

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Appendix B

ชื่อ...

แบบสอบถามเกี่ยวกับอาหารที่รับประทาน

กรุณาเขียนเครื่องหมาย X ลงในช่องว่างที่กำหนดให้ตามความเป็นจริง

1. รายการอาหารต่อไปนี้ท่านรับประทานหรือไม่

รายการอาหาร	รับประทาน	ไม่รับประทาน	ไม่รู้จัก	อื่นๆ ระบุ.....
อาหารประเภทผัด				
1. ผัดถั่วพริกเผา				
2. ผัดเปรี้ยวหวาน				
3. ผัดวุ้นเส้นกะหล่ำปลี				
4. ผัดวุ้นเส้นใส่ไข่				
5. ไก่ผัดพริกสด				
6. ผัดผักบุ้งน้ำมันหอย				
7. ผัดผักกะหล่ำดอก				
8. ผัดผักกาดขาว				
9. ผัดผักรวมมิตร				
10. ผัดบรอคโคลีหมูสับ				
11. ผัดผักคะน้า				
12. กระเพราไก่				
13. หมูผัดซีอิ๊ว				
14. หมูผัดขิง				
15. ปลาหมึกผัดพริกสด				
16. ผัดเผ็ดหมูป่า				

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รายการอาหาร	รับประทาน	ไม่รับประทาน	ไม่รู้จัก	อื่นๆ ระบุ.....
อาหารประเภทผัด				
17. ผัดสามสหาย				
18. หมูผัดพริกไทยดำ				
อาหารประเภททอด				
1. หมูทอด				
2. ไก่ทอด				
3. ปลาทูทอด				
4. ปลาช่อนทอด				
5. ทอดมันปลา				
6. ไข่เจียว				
7. หมูสับทอด				
8. ปีกไก่หมักซอส				
อาหารประเภทแกงและแกงจืด				
1. แกงจืดเต้าหู้หมูสับ				
2. แกงจืดแตงกวาหมูสับ				
3. แกงจืดตำลึงหมูสับ				
4. แกงจืดเต้าหู้หมูแผ่น				
5. แกงเลียง				
6. แกงส้มผักรวม				
อาหารประเภทแกงและแกงจืด				
1. แกงเขียวหวาน				
2 เกี๊ยวน้ำ				
3. พะโล้				
4. พะแนงหมู				
5. ซี่โครงหมูต้มยำ				

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รายการอาหาร	รับประทาน	ไม่รับประทาน	ไม่รู้จัก	อื่นๆ ระบุ.....
อาหารประเภทแกงและแกงจืด				
6. ต้มยำกุ้ง				
7. ต้มข่าไก่				
8. มัสมั่นไก่				
9. ต้มจับฉ่ายไก่				
10. แกงคั่วฟักทอง				
11. แกงจืดมะระหมูสับ				
12. ต้มโคล้งปลาทูสด				
อาหารจานเดียว				
1. เกาเหลาลูกชิ้น				
2. เส้นใหญ่ขี้เมา				
3. เส้นหมี่หมูน้ำ				
4. เส้นหมี่หมูตุ๋น				
5. เส้นเล็กผัดซีอิ๊ว				
6. ก๋วยเตี๋ยวหมูสับผัด				
7. ก๋วยเตี๋ยวคั่วไก่				
8. ราดหนพริกเผา				
9. ราดหน้าหมูหมัก				
10. ข้าวผัดไข่				
11. ข้าวผัดแหนม				
12. ข้าวผัดปลาทูนึ่ง				
13. ข้าวผัดน้ำพริกเผา				
14. ข้าวผัดน้ำพริกกะปิ				
15. ข้าวผัดกุนเชียง				
16. ข้าวคลุกกะปิ				

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รายการอาหาร	รับประทาน	ไม่รับประทาน	ไม่รู้จัก	อื่นๆ ระบุ.....
อาหารจานเดียว				
17. ข้าวต้มหมู				
18. มักกะโรนีขี้เมา				
19. ผัดหมี่ซั่ว				
20. สปาเกตตี้ราดซอส				
น้ำพริก				
1. น้ำพริกอ่อง				
2. น้ำพริกมะขามสดใส่หมูสับ				
3. น้ำพริกกะปิ				
4. น้ำพริกผัด				
อื่น ๆ				
1. สลัดผัด				
2. ลาบหมู				
3. ยำวุ้นเส้น				
4. หมูอบซอส				
5. ไข่ตุ๋น				
6. ฉู่ฉี่ปลาทู				
7. หมูสับนึ่งแครอท				

2. อาหารหรือส่วนประกอบใดในอาหารใดบ้างที่ท่านไม่ชอบหรือไม่รับประทานกรุณาระบุ .เช่น ผักชีฝรั่ง กะปิ

....

....

ขอขอบคุณในความร่วมมือ

สถาบันวิจัยโภชนาการ มหาวิทยาลัยมหิดล

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Appendix C

ตัวอย่างเมนูอาหารทุก 2 สัปดาห์ของอาสาสมัคร

สัปดาห์ที่ 1

	Energy (Kcal)	Energy distribution (%)			Dietary fiber (mg)	NSD		LSD	
		CHO	PROT	FAT		Na (mg)	K (mg)	Na (mg)	K (mg)
วันจันทร์									
เช้า แกงส้มผักรวม	78.68	36.56	47.60	15.84	3.11	1011.16	490.92	670.46	1012.86
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	411.18	63.81	24.26	8.94	3.61	1096.16	710.92	755.46	1232.86
กลางวัน เส้นเล็กผัดซีอิ๊ว	625.93	42.56	16.36	41.08	2.01	731.20	552.27	548.36	818.41
รวม	625.93	42.56	16.36	41.08	2.01	731.20	552.27	548.36	818.41
เย็น ผัดวุ้นเส้นกะหล่ำปลี	130.34	50.61	11.78	37.61	0.77	682.51	180.74	358.65	569.75
หมูผัดซีอิ๊ว	141.31	3.35	38.33	58.32	0.05	579.51	254.80	359.85	533.83
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	604.15	48.34	17.01	32.65	1.32	1347.02	655.54	803.50	1323.58
รวมทั้งวัน	1641.26	52.53	19.32	26.15	6.94	3174.38	1918.73	2107.32	3374.85

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สัปดาห์ที่ 1

		Energy	Energy distribution (%)			Dietary fiber	NSD		LSD	
			CHO	PROT	FAT		Na	K	Na	K
วันอังคาร										
เช้า	ข้าวผัดกุนเชียง	616.85	41.69	15.67	42.64	2.33	1495.42	527.88	816.72	1359.72
	รวม	616.85	41.69	15.67	42.64	2.33	1495.42	527.88	816.72	1359.72
กลางวัน	ต้มจับฉ่ายไก่	110.24	12.20	18.43	69.37	1.50	405.06	247.05	288.54	431.38
	ปลาหมึกผัดพริกสด	97.96	16.76	41.66	41.58	1.46	277.07	160.90	236.04	635.33
	ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
	รวม	540.70	40.01	20.33	37.66	3.46	767.13	627.95	609.58	1286.71
เย็น	มัสมั่นไก่	324.29	18.24	15.77	65.99	3.10	1100.69	369.91	706.18	889.08
	ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
	รวม	656.79	54.65	8.34	34.01	3.60	1185.69	589.91	791.18	1109.08
รวมทั้งวัน		**1814.34**	**45.17**	**15.56**	**37.27**	**9.39**	**3448.24**	**1745.74**	**2217.48**	**3755.51**

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สัปดาห์ที่ 1

	Energy (Kcal)	Energy distribution (%)			Dietary fiber (mg)	NSD		LSD	
		CHO	PROT	FAT		Na (mg)	K (mg)	Na (mg)	K (mg)
วันพุธ									
เช้า เส้นหมี่หมูตุ๋น	273.23	53.85	22.66	23.49	0.72	1187.33	285.80	470.45	743.74
รวม	273.23	53.85	22.66	23.49	0.72	1187.33	285.80	470.45	743.74
กลางวัน เส้นใหญ่ขี้เมา	638.37	51.55	13.33	35.12	2.94	1359.94	658.19	907.14	1233.22
รวม	638.37	51.55	13.33	35.12	2.94	1359.94	658.19	907.14	1233.22
เย็น ฉู่ฉี่ปลาทู	269.73	5.73	20.04	74.23	0.50	524.98	142.53	283.04	386.01
ผัดวุ้นเส้นใส่ไข่	251.92	59.00	12.01	52.40	0.94	149.91	181.79	97.41	159.68
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	854.15	51.93	10.99	42.89	1.94	759.89	544.32	465.45	765.69
รวมทั้งวัน	1765.75	52.24	13.79	37.45	5.60	3307.16	1488.31	1843.04	2742.65

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สัปดาห์ที่ 1

	Energy	Energy distribution (%)			Dietary fiber	NSD		LSD	
	(Kcal)	CHO	PROT	FAT	(mg)	Na (mg)	K (mg)	Na (mg)	K (mg)
วันพฤหัส									
เช้า ข้าวต้มหมูปรุงเครื่อง	276.83	59.27	17.42	23.31	1.53	1399.74	394.25	582.42	625.27
รวม	276.83	59.27	17.42	23.31	1.53	1399.74	394.25	582.42	625.27
กลางวัน ข้าวผัดน้ำพริกกะปิหมูสับ	565.44	53.80	10.99	35.12	1.36	918.43	441.40	791.71	669.52
รวม	565.44	53.80	10.99	35.12	1.36	918.43	441.40	791.71	669.52
เย็น แกงจืดเต้าหู้หมูแผ่น	204.18	16.04	32.19	51.77	2.01	1148.66	443.31	521.35	590.93
ยำวุ้นเส้น	278.12	42.62	26.80	30.57	0.38	568.40	298.80	420.56	564.94
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	1765.75	52.24	13.79	37.45	5.60	3307.16	1488.31	1843.04	2742.65
รวมทั้งวัน	**1657.07**	**52.56**	**17.66**	**28.56**	**5.78**	**4120.23**	**1797.76**	**2401.04**	**2670.66**

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สัปดาห์ที่ 1

	Energy	Energy distribution (%)			Dietary fiber	NSD		LSD	
	(Kcal)	CHO	PROT	FAT	(mg)	Na (mg)	K (mg)	Na (mg)	K (mg)
วันศุกร์									
เช้า ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
ผัดผักรวมมิตร	121.46	25.73	5.92	68.35	1.79	697.60	228.48	363.80	633.68
แกงจืดมะระหมูบด	83.50	20.06	45.72	34.22	1.87	478.76	306.71	328.35	545.68
รวม	1765.75	52.24	13.79	37.45	5.60	3307.16	1488.31	1843.04	2742.65
กลางวัน ผัดหมี่ซั่ว	702.86	54.10	13.06	32.84	2.99	672.99	494.91	525.06	761.05
รวม	702.86	54.10	13.06	32.84	2.99	672.99	494.91	525.06	761.05
เย็น แกงเขียวหวาน	262.61	10.95	15.02	74.04	2.55	841.57	227.91	674.64	664.44
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	656.79	54.65	8.34	34.01	3.60	1185.69	589.91	791.18	1109.08
รวมทั้งวัน	**1835.43**	**48.83**	**13.59**	**35.59**	**10.20**	**2860.92**	**1698.01**	**2061.85**	**3044.85**

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สัปดาห์ที่ 1

	Energy (Kcal)	Energy distribution (%)			Dietary fiber (mg)	NSD		LSD	
		CHO	PROT	FAT		Na (mg)	K (mg)	Na (mg)	K (mg)
วันเสาร์									
เช้า เส้นหมี่หมูน้ำ	405.58	31.74	20.75	47.51	0.90	1365.53	238.42	725.58	734.66
รวม	405.58	31.74	20.75	47.51	0.90	1365.53	238.42	725.58	734.66
กลางวัน ผัดเผ็ดหมูป่า	99.45	24.33	22.38	53.29	2.24	527.39	215.74	232.69	490.29
ปีกไก่หมักซอส	462.39	8.28	15.50	76.22	0.47	905.88	209.83	375.55	514.31
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	1765.75	52.24	13.79	37.45	5.60	3307.16	1488.31	1843.04	2742.65
เย็น ทอดมันปลา	192.28	5.49	24.62	69.89	1.21	693.09	213.77	288.31	416.89
ซี่โครงหมูต้มยำ	144.29	9.41	34.64	55.95	0.55	571.98	260.70	424.18	526.84
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	1765.75	52.24	13.79	37.45	5.60	3307.16	1488.31	1843.04	2742.65
รวมทั้งวัน	**1968.99**	**37.34**	**17.10**	**43.85**	**6.37**	**4233.87**	**1578.46**	**2216.31**	**3122.99**

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สัปดาห์ที่ 1

	Energy	Energy distribution (%)			Dietary fiber	NSD		LSD	
	(Kcal)	CHO	PROT	FAT	(mg)	Na (mg)	K (mg)	Na (mg)	K (mg)
วันอาทิตย์									
เช้า แกงคั่วหมูกะทิสด	337.43	11.00	29.08	59.92	0.78	1373.17	463.65	772.92	877.20
ผัดผักกาดขาว	61.40	21.79	10.38	67.83	1.52	338.89	232.25	171.99	434.85
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	1765.75	52.24	13.79	37.45	5.60	3307.16	1488.31	1843.04	2742.65
กลางวัน ข้าวผัดน้ำพริกเผา	640.37	49.41	9.78	40.81	3.80	542.70	533.42	334.30	753.38
รวม	640.37	49.41	9.78	40.81	3.80	542.70	533.42	334.30	753.38
เย็น หมูผัดพริกไทยดำ	191.96	14.61	47.64	37.75	1.78	607.46	491.24	345.36	750.43
น้ำพริกมะขามสด	116.77	36.96	25.50	37.55	0.29	804.68	155.75	551.69	326.23
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	1765.75	52.24	13.79	37.45	5.60	3307.16	1488.31	1843.04	2742.65
รวมทั้งวัน	**2012.93**	**50.82**	**17.74**	**35.42**	**9.17**	**3836.90**	**2316.31**	**2346.26**	**3582.09**

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สัปดาห์ที่ 2

	Energy	Energy distribution (%)			Dietary fiber	NSD		LSD	
	(Kcal)	CHO	PROT	FAT	(mg)	Na (mg)	K (mg)	Na (mg)	K (mg)
วันจันทร์									
เช้า ข้าวผัดแหนม	552.19	46.69	12.55	40.76	2.33	1198.42	442.78	676.02	1072.80
รวม	552.19	46.69	12.55	40.76	2.33	1198.42	442.78	676.02	1072.80
กลางวัน ราดหน้าหมูหมัก	588.36	52.79	13.51	33.70	2.48	1102.17	626.14	779.34	1079.42
รวม	588.36	52.79	13.51	33.70	2.48	1102.17	626.14	779.34	1079.42
เย็น แกงเลียง	173.72	27.71	37.90	34.39	2.58	830.91	534.54	542.16	524.62
ไก่ผัดพริกสด	172.74	42.00	23.33	56.24	1.02	325.63	227.99	245.37	372.47
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	678.96	53.59	20.71	30.89	4.10	1241.54	982.53	872.53	1117.09
รวมทั้งวัน	**1819.51**	**52.05**	**17.64**	**33.42**	**8.91**	**3542.13**	**2051.45**	**2327.89**	**3269.31**

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สัปดาห์ที่ 2

	Energy	Energy distribution (%)			Dietary fiber	NSD		LSD	
	(Kcal)	CHO	PROT	FAT	(mg)	Na (mg)	K (mg)	Na (mg)	K (mg)
วันอังคาร									
เช้า หมูอบซอส	131.70	22.16	44.97	32.87	2.90	598.84	547.96	309.16	870.66
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	464.20	56.61	22.94	17.45	3.40	341.92	767.96	394.16	545.33
กลางวัน น้ำพริกอ่อง, ไข่ต้ม	323.99	19.16	19.18	61.67	1.70	1263.66	394.80	579.09	439.82
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	656.79	54.65	8.34	34.01	3.60	1185.69	589.91	791.18	1109.08
เย็น แกงเขียวหวาน	262.61	10.95	15.02	74.04	2.55	841.57	227.91	674.64	664.44
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	656.79	54.65	8.34	34.01	3.60	1185.69	589.91	791.18	1109.08
รวมทั้งวัน	1715.80	60.66	7.39	28.36	8.65	2959.07	1830.67	1817.89	2634.92

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สัปดาห์ที่ 2

	Energy	Energy distribution (%)			Dietary fiber	NSD		LSD	
	(Kcal)	CHO	PROT	FAT	(mg)	Na (mg)	K (mg)	Na (mg)	K (mg)
วันพุธ									
เช้า เส้นหมี่หมูตุ๋น	273.23	53.85	22.66	23.49	0.72	1187.33	285.80	470.45	743.74
รวม	273.23	53.85	22.66	23.49	0.72	1187.33	285.80	470.45	743.74
กลางวัน เส้นใหญ่ขี้เมา	638.37	51.55	13.33	35.12	2.94	1359.94	658.19	907.14	1233.22
รวม	638.37	51.55	13.33	35.12	2.94	1359.94	658.19	907.14	1233.22
เย็น ฉู่ฉี่ปลาทู	269.73	5.73	20.04	74.23	0.50	524.98	142.53	283.04	386.01
ผัดวุ้นเส้นใส่ไข่	251.92	59.00	12.01	52.40	0.94	149.91	181.79	97.41	159.68
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	854.15	51.93	10.99	42.89	1.94	759.89	544.32	465.45	765.69
รวมทั้งวัน	1765.75	52.24	13.79	37.45	5.60	3307.16	1488.31	1843.04	2742.65

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สัปดาห์ที่ 2

	Energy	Energy distribution (%)			Dietary fiber	NSD		LSD	
	(Kcal)	CHO	PROT	FAT	(mg)	Na (mg)	K (mg)	Na (mg)	K (mg)
วันพฤหัส									
เช้า ผัดเปรี้ยวหวาน	192.59	20.50	29.06	50.44	1.50	478.71	356.23	323.03	607.08
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	525.09	55.78	14.99	26.24	2.00	563.71	576.23	408.03	827.08
กลางวัน ราดหน้าพริกเผา	659.36	51.05	12.20	36.75	4.90	1618.09	901.72	1301.30	1472.02
รวม	659.36	51.05	12.20	36.75	4.90	1618.09	901.72	1301.30	1472.02
เย็น น้ำพริกกะปิ+ปลาทูทอด	165.12	23.3/8	37.06	39.56	0.92	724.49	258.36	640.01	410.44
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	497.62	91.06	18.99	20.80	1.42	809.49	478.36	725.01	630.44
รวมทั้งวัน	1682.07	63.42	16.03	26.16	8.32	2991.29	1956.31	2434.34	2929.54

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สัปดาห์ที่ 2

	Energy	Energy distribution (%)			Dietary fiber	NSD		LSD	
	(Kcal)	CHO	PROT	FAT	(mg)	Na (mg)	K (mg)	Na (mg)	K (mg)
วันศุกร์									
เช้า แกงจืดตำลึงหมูสับ	149.51	11.19	37.42	51.39	1.84	903.34	360.31	610.00	745.91
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	482.01	51.13	19.17	26.71	2.34	988.34	580.31	695.00	965.91
กลางวัน ข้าวคลุกกะปิใหม่	728.51	48.63	13.75	37.61	1.67	1642.21	660.61	1067.76	1339.42
รวม	728.51	48.63	13.75	37.61	1.67	1642.21	660.61	1067.76	1339.42
เย็น ทอดมันปลา	192.28	5.49	24.62	69.89	1.21	693.09	213.77	288.31	416.89
ผัดผักกะหล่ำดอก	97.34	41.83	13.83	44.33	2.76	479.88	365.56	229.53	669.51
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	622.12	46.13	13.12	38.75	4.47	1257.97	799.33	602.84	1306.40
รวมทั้งวัน	**1832.64**	**48.21**	**15.24**	**34.55**	**8.48**	**3888.52**	**2040.25**	**2365.60**	**3611.73**

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สัปดาห์ที่ 2

	Energy (Kcal)	Energy distribution (%)			Dietary fiber (mg)	NSD		LSD	
		CHO	PROT	FAT		Na (mg)	K (mg)	Na (mg)	K (mg)
วันเสาร์									
เช้า พะโล้	278.41	18.29	24.44	57.27	0.41	1377.73	134.29	592.90	551.99
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	610.91	54.68	12.68	29.65	0.91	1462.73	354.29	677.90	771.99
กลางวัน ข้าวผัดปลาทูนึ่ง	574.01	47.83	17.30	34.88	2.73	1541.55	865.49	1044.10	1436.42
รวม	574.01	47.83	17.30	34.88	2.73	1541.55	865.49	1044.10	1436.42
เย็น ผัดผักคะน้า	107.62	16.13	19.30	64.57	2.19	381.95	324.13	215.05	526.73
กระเพราไก่	212.54	12.58	28.58	58.84	1.99	711.26	416.30	542.34	720.46
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	652.66	39.92	16.26	41.81	4.68	1178.21	960.43	842.39	1467.19
รวมทั้งวัน	**1837.58**	**46.16**	**15.24**	**36.60**	**8.32**	**4182.49**	**2180.21**	**2564.39**	**3675.60**

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สัปดาห์ที่ 2

	Energy (Kcal)	Energy distribution (%)			Dietary fiber (mg)	NSD		LSD	
		CHO	PROT	FAT		Na (mg)	K (mg)	Na (mg)	K (mg)
วันอาทิตย์									
เช้า เกาเหลาลูกชิ้น	176.31	20.82	40.82	38.36	2.91	1062.45	499.06	590.10	837.37
ข้าวสวย 180 กรัม	239.40	91.06	0.91	2.03	0.36	61.20	158.40	61.20	158.40
รวม	415.71	55.94	20.87	20.20	3.27	1123.65	657.46	651.30	995.77
กลางวัน ลาบหมูผัก	231.19	9.87	49.72	40.41	1.21	733.64	606.61	564.72	910.77
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	563.69	50.47	25.32	21.22	1.71	818.64	826.61	649.72	1130.77
เย็น ต้มข่าไก่	270.02	13.54	19.86	66.61	0.91	1055.39	296.11	578.96	583.86
ปลาทูทอด 50 กรัม	118.00	0.53	45.41	54.06	0.00	66.00	166.50	66.00	166.50
ข้าวสวย 250 กรัม	332.50	91.06	0.91	2.03	0.50	85.00	220.00	85.00	220.00
รวม	720.52	35.04	22.06	40.90	1.41	1206.39	682.61	729.96	970.36
รวมทั้งวัน	**1699.92**	**41.21**	**23.36**	**33.03**	**3.12**	**3148.68**	**2166.68**	**2030.98**	**3096.90**

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Appendix D

The amount of sodium and potassium contents of low sodium and normal sodium condiments products*

Kind	Sodium content (mg/ 100 g)	Potassium content (mg/ 100 g)
Salt		
Normal	39173	109
Low sodium	11790 (decreased 69.9 %)	1910 (increased 94.3 %)
Fish sauce		
Normal	7237	738
Low sodium	5125 (decreased 41.2 %)	5276 (increased 86.0 %)
Soy sauce		
Normal	6405	581
Low sodium	3750 (decreased 41.5 %)	4300 (increased 86.5 %)
Flavored sauce		
Normal	8890	536
Low sodium	3680 (decreased 58.6 %)	6034 (increased 90.7 %)
Osyter sauce		
Normal	3065	201
Low sodium	1395 (decreased 54.5 %)	2226 (increased 91.0 %)
Chilli sauce		
Normal	1557	149
Low sodium	836 (decreased 46.3 %)	1208 (increased 87.7 %)
Tomato sauce		
Normal	939	272
Low sodium	271 (decreased 71.1 %)	1081 (increased 74.8 %)

* In parenthesis means percent change of sodium or potassium base on normal condiments.

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Appendix E

คำแนะนำในการเก็บอาหารที่บริโภคจริง 24 ชั่วโมง (Duplicate meals)

1. เก็บอาหารที่ท่านบริโภคจริงทุกอย่าง ทุกมื้อ เช่น เมื่อท่านรับประทานข้าวผัด 1 จาน เติมพริกน้ำปลา 1 ช้อนชา, มะนาว1/4 ลูก ท่านก็ต้องนำข้าวผัด 1 จานที่เติมพริกน้ำปลาและมะนาวเท่ากันกับที่ท่านรับประทานเก็บใส่ภาชนะแล้วนำมา ทำเช่นนี้ทั้ง 3 มื้อ
2. กรณีที่ท่านรับประทานอาหารเหลือกรุณาช่วยเก็บแยกจากข้อ1. มาให้ด้วย
3. กรณีที่ท่านรับประทานอาหาร, ขนม, ขนมขบเคี้ยวอื่นๆนอกจากอาหารมื้อหลัก กรุณาเก็บปริมาณและจำนวนที่เท่ากันให้ด้วยโดยทำเช่นเดียวกับอาหารหลักในข้อ 1 เช่น ท่านรับประทานข้าวเหนียวถั่วดำ 1 ถ้วยท่านก็ต้องซื้อข้าวเหนียวถั่วดำอีก 1 ถ้วยใส่ถุงนำมาให้ด้วย และกรณีที่รับประทานไม่หมดให้ปฏิบัติตามข้อ 2

คำแนะนำในการเก็บปัสสาวะ 24 ชั่วโมง

การตรวจปัสสาวะจะได้ผลแน่นอนเมื่อใช้ปัสสาวะที่เก็บไว้ครบ 24 ชั่วโมงตามวิธีต่อไปนี้

1. เริ่มต้นการเก็บปัสสาวะ 24 ชั่วโมงโดยตื่นนอนตอนเช้า*ถ่ายปัสสาวะทิ้งไป* จดเวลาไว้ข้างขวด (เช่น 6:00น.)
2. ปัสสาวะที่ถ่ายครั้งต่อๆไปตลอดวันรวมทั้งปัสสาวะในตอนกลางคืนด้วยให้ถ่ายลงภาชนะที่สะอาดแล้วเทเก็บไว้ในขวดที่จัดไว้ให้ทุกครั้ง
3. เช้าวันรุ่งขึ้นในเวลาเดียวกันกับเวลาที่จดไว้ในข้อ 1. (เช่น 06:00 น.) ให้ถ่ายปัสสาวะเก็บลงในขวดเป็นครั้งสุดท้าย

ขอขอบคุณในความร่วมมือ สถาบันวิจัยโภชนาการ มหาวิทยาลัยมหิดล

กรณีที่ท่านมีปัญหาหรือข้อสงสัยกรุณาติดต่อ วีรวรรณ ลิ้มมานนท์ 0-1329-9219

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Appendix F

แบบทดสอบระดับความเครียดโครงการวิจัยการศึกษาประสิทธิผลของการจำกัดปริมาณการบริโภคโซเดียมโดยใช้เครื่องปรุงรสที่มีโซเดียมต่ำแทนเครื่องปรุงรสปกติต่อความดันโลหิตในผู้ที่มีความดันโลหิตสูง

ชื่อ-สกุล................................วันที่.........ครั้งที่ประเมิน....รหัส.........

คำชี้แจง ในระยะเวลา 2 เดือนที่ผ่านมานี้ ท่านมีอาการ พฤติกรรม หรือความรู้สึกต่อไปนี้มากน้อยเพียงใด โปรดทำเครื่องหมายลงในช่องที่แสดงระดับอาการที่เกิดขึ้นกับตัวท่าน ตามความเป็นจริงมากที่สุด

อาการ พฤติกรรม หรือความรู้สึก	ไม่เคยเลย	เป็นครั้งคราว	เป็นบ่อย ๆ	เป็นประจำ
1. นอนไม่หลับเพราะคิดมากหรือกังวลใจ				
2. รู้สึกหงุดหงิด รำคาญใจ				
3. ทำอะไรไม่ได้เลย เพราะประสาทตึงเครียด				
4. มีความวุ่นวายใจ				
5. ไม่อยากพบปะผู้คน				
6. ปวดหัวข้างเดียว หรือปวดบริเวณขมับทั้ง 2 ข้าง				
7. รู้สึกไม่มีความสุขและเศร้าหมอง				
8. รู้สึกหมดหวังในชีวิต				
9. รู้สึกว่าชีวิตตนเองไม่มีคุณค่า				
10. กระวนกระวายอยู่ตลอดเวลา				
11. รู้สึกว่าตนเองไม่มีสมาธิ				
12. รู้สึกเพลียจนไม่มีแรงจะทำอะไร				
13. รู้สึกเหนื่อยหน่ายไม่อยากทำอะไร				
14. มีอาการหัวใจเต้นแรง				

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อาการ พฤติกรรม หรือความรู้สึก	ไม่เคยเลย	เป็นครั้งคราว	เป็นบ่อย ๆ	เป็นประจำ
15. เสียงสั่น ปากสั่น หรือมือสั่นเวลาไม่พอใจ				
16. รู้สึกกลัวผิดพลาดในการทำสิ่งต่าง ๆ				
17. ปวดหรือเกร็งกล้ามเนื้อบริเวณท้ายทอย หลัง หรือไหล่				
18. ตื่นเต้นง่ายกับเหตุการณ์ที่ไม่คุ้นเคย				
19. มึนงงหรือเวียนศีรษะ				
20. ความสุขทางเพศลดลง				

แบบประเมินความเครียดนี้ สร้างโดย กรมสุขภาพจิต กระทรวงสาธารณสุข

เกณฑ์การให้คะแนน

รวมคะแนนไม่เกิน 60 คะแนน โดยจำนวนคำถาม 20 ข้อ ตอบว่า

ไม่เคยเลย =0 คะแนน

เป็นครั้งคราว = 1 คะแนน

ไม่เคยเลย = 2 คะแนน

ไม่เคยเลย = 3 คะแนน

ผลการประเมินและวิเคราะห์ความเครียด

ระดับคะแนน 0-5

ท่านมีความเครียดอยู่ในระดับต่ำกว่าเกณฑ์ปกติ ความเครียดในระดับต่ำมากเช่นนี้ อาจมีความหมายว่า

- ท่านตอบไม่ตรงตามความเป็นจริง หรือ
- ท่านอาจเข้าใจคำถาคลาดเคลื่อนไป
- ท่านอาจเป็นคนที่ขาดแรงจูงใจ มีความเฉื่อยชา
- ชีวิตประจำวันซ้ำซากจำเจ น่าเบื่อ ปราศจากความตื่นเต้น

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ระดับคะแนน 6-17

ท่านมีความเครียดอยู่ในเกณฑ์ปกติ สามารถจัดการกับความเครียดที่เกิดขึ้นในชีวิตประจำวัน และสามารถปรับตัวกับสถานการณ์ต่าง ๆ ได้อย่างเหมาะสม รู้สึกพึงพอใจเกี่ยวกับตนเองและสิ่งแวดล้อมเป็นอย่างมาก

ความเครียดในระดับนี้ถือว่ามีประโยชน์ในการดำเนินชีวิตประจำวันเป็นแรงจูงใจที่นำไปสู่ความสำเร็จในชีวิตได้

ระดับคะแนน 18-25

ท่านมีความเครียดอยู่ในระดับสูงกว่าปกติเล็กน้อย ซึ่งถือว่าเป็นความเครียดที่พบได้ในชีวิตประจำวัน อาจไม่รู้ตัวว่ามีความเครียดหรืออาจรู้สึกได้จากการเปลี่ยนแปลงของร่างกาย อารมณ์ ความรู้สึกและพฤติกรรมบ้างเล็กน้อย แต่ไม่ชัดเจนและยังพอทนได้ อาจต้องใช้เวลาในการปรับตัว แต่ในที่สุดก็สามารถจัดการกับความเครียดได้ และความเครียดระดับนี้ไม่เป็นผลเสียต่อการดำเนินชีวิต

ในกรณีนี้ท่านสามารถผ่อนคลายความเครียดด้วยการหากิจกรรมที่เพิ่มพลัง เช่น การออกกำลังกาย เล่นกีฬา ทำสิ่งที่สนุกสนานเพลิดเพลิน เช่น ดูหนัง ฟังเพลง อ่านหนังสือ หรือทำงานอดิเรกต่าง ๆ อย่าลืมพูดคุยกับผู้ที่ไว้วางใจ พิจารณาและลงมือแก้ไขปัญหาตามลำดับความสำคัญอย่างรอบคอบและมีสติ

ระดับคะแนน 26-29

คุณมีความเครียดอยู่ในระดับสูงกว่าปกติปานกลาง ขณะนี้ท่านเริ่มมีความตึงเครียดในระดับค่อนข้างสูงและได้รับความเดือดร้อนเป็นอย่างมากจากปัญหาทางอารมณ์ที่เกิดจากปัญหาความขัดแย้งและวิกฤตการณ์ในชีวิต เป็นสัญญาณเตือนขั้นต้นว่าท่านกำลังเผชิญกับภาวะวิกฤตและความขัดแย้ง ซึ่งท่านจัดการแก้ไขด้วยความยากลำบาก ลักษณะดังกล่าวจะเพิ่มความรุนแรงซึ่งมีผลกระทบต่อการทำงาน จำเป็นต้องหาวิธีแก้ไขข้อขัดแย้งต่าง ๆ ให้ลดน้อยลงหรือหมดไปด้วยวิธีการอย่างใดอย่างหนึ่ง

สิ่งแรกที่ต้องรีบจัดการคือ คุณต้องมีวิธีคลายเครียดที่ดี และสม่ำเสมอทุกวัน วันละ 1-2 ครั้ง ๆ ละ 10 นาที โดยนั่งในท่าที่สบาย หายใจลึก ๆ ให้หน้าท้องขยาย หายใจออกช้า ๆ นับ 1-10 ไปด้วย ท่านจะใช้วิธีนั่งสมาธิหรือสวดมนต์ก็ได้

ท่านควรแก้ไขปัญหาให้ดีขึ้น โดยค้นหาสาเหตุของปัญหาที่ทำให้เกิดความขัดแย้ง หาวิธีแก้ไขปัญหาหลาย ๆ วิธี พร้อมทั้งพิจารณาผลดี ผลเสียของแต่ละวิธี เลือกวิธีที่เหมาะสมกับ

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สภาวะของตนเองมากที่สุด ทั้งนี้ต้องไม่สร้างปัญหาให้เพิ่มขึ้นหรือทำให้ผู้อื่นเดือดร้อน วางแผนแก้ไขปัญหาเป็นลำดับขั้นตอนและลงมือแก้ปัญหา

ระดับคะแนน 30 ขึ้นไป

คุณมีความเครียดอยู่ในระดับสูงกว่าปกติมาก กำลังตกอยู่ในภาวะตึงเครียด หรือกำลังเผชิญกับวิกฤตการณ์ในชีวิตอย่างรุนแรง เช่น การเจ็บป่วยที่รุนแรง เรื้อรัง ความพิการ การสูญเสีย ปัญหาความรุนแรงในครอบครัว ปัญหาเศรษฐกิจ ซึ่งส่งผลต่อสุขภาพกายและสุขภาพจิตอย่างชัดเจน ทำให้ชีวิตไม่มีความสุข ความคิดฟุ้งซ่าน ตัดสินใจผิดพลาด ขาดความยับยั้งชั่งใจ อาจเกิดอุบัติเหตุได้ง่าย บางครั้งอาจมีพฤติกรรมก้าวร้าวรุนแรง เช่น เอะอะ โวยวาย ขว้างปาข้าวของ

ความเครียดในระดับนี้ถือว่ามีความรุนแรงมาก หากปล่อยไว้โดยไม่ดำเนินการแก้ไขอย่างเหมาะสมและถูกวิธี อาจนำไปสู่ความเจ็บป่วยทางจิตที่รุนแรง ซึ่งส่งผลเสียต่อตนเองและบุคคลใกล้ชิดต่อไปได้ ในระดับนี้ท่านต้องไปปรึกษาหรือใช้บริการปรึกษาปัญหาสุขภาพจิตทางโทรศัพท์ ซึ่งจะช่วยให้ท่านมองเห็นปัญหาและแนวทางแก้ไขที่ชัดเจนและเหมาะสมต่อไป

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Appendix G

Raw data of subjects

Name	Sex	Age	BW	HT	BMI	Baseline SBP	Baseline DBP
GS	F	47	59.20	156.00	24.30	140.67	96.66
TJ	F	47	51.50	150.00	22.89	144.67	86.66
PW	F	55	56.50	157.00	22.89	133.33	86.66
WS	M	38	53.40	155.00	22.03	144.67	106.00
KS	F	42	67.80	159.00	26.71	146.67	90.00
SJ	M	41	84.60	177.00	26.78	137.33	90.66
AK	F	44	57.90	174.00	19.05	140.00	100.00
YY	F	49	59.20	155.00	24.34	144.00	84.66
SG	F	52	54.50	156.00	22.47	144.66	91.00
SK	M	25	87.10	165.00	28.66	167.33	96.66
PP	M	42	61.50	166.00	26.11	175.00	117.00
NN	F	36	48.70	155.00	20.26	175.00	115.00
LT	F	33	64.70	155.00	26.90	149.00	99.00
PC	M	41	76.60	166.00	27.72	142.00	95.33
SM	M	47	72.20	167.00	25.10	145.33	91.33
KR	M	49	64.50	158.00	30.44	150.00	98.33
SS	M	56	64.90	170.00	21.15	149.33	100.00
WG	M	55	77.40	159.00	25.71	133.33	93.33

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Systolic blood pressure of subjects throughout the study.

Name	Run-in	beginning	Low period				washout	beginning	Control period			
			wk1	wk2	wk3	wk4			wk1	wk2	wk3	wk4
GS	124.00	124.00	115.00	124.00	114.67	108.00	126.00	120.00	121.33	114.00	118.67	118.00
TJ	135.00	138.00	132.00	127.33	128.67	129.33	138.00	138.00	132.67	133.00	124.67	126.00
PW	130.00	124.00	113.50	115.33	112.67	112.67	126.00	120.00	116.00	121.00	118.67	123.33
WS	134.00	136.00	140.00	138.00	126.00	127.33	148.00	140.00	144.00	146.00	132.00	143.33
KS	145.00	148.00	136.67	146.00	138.00	134.67	148.00	134.00	141.00	141.33	153.33	140.67
SJ	121.00	110.00	116.67	114.00	113.33	121.33	110.00	136.00	137.33	122.67	110.00	127.33
AK	136.00	110.00	115.00	112.33	109.33	117.33	121.33	110.00	118.67	110.67	110.00	112.67
YY	132.33	128.00	130.67	132.00	124.00	126.00	132.00	130.00	127.00	131.33	131.33	120.00
SG	141.33	148.00	137.00	140.67	134.00	120.00	138.67	138.00	136.67	139.33	133.33	135.33
SK	167.33	160.00	155.33	153.33	152.00	149.33	156.00	160.00	160.00	156.00	162.67	162.00
PP	178.00	180.00	170.67	162.67	167.33	176.67	180.00	180.00	180.67	188.00	183.33	178.67
NN	160.00	160.00	154.00	160.00	148.00	150.67	163.33	174.00	151.33	154.67	164.00	162.67
LT	131.00	134.00	114.67	118.00	120.00	118.00	122.00	110.00	140.33	133.33	128.00	126.67
PC	140.00	124.00	136.00	120.00	124.00	125.00	129.33	130.00	124.67	132.00	126.00	131.33
SM	159.33	150.00	144.67	134.00	138.00	135.00	149.33	144.00	146.00	137.00	154.00	150.67
KR	154.67	158.00	145.33	153.00	156.00	152.00	154.67	150.00	158.67	154.67	147.33	145.00
SS	154.67	150.00	155.33	150.00	154.00	143.33	152.67	150.00	158.33	160.00	151.33	151.00
WG	130.00	130.00	122.67	137.00	121.00	130.00	127.33	122.00	132.00	125.33	135.33	123.50

* beginning means the first day values of each intervention diet used to test the carry over effect

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Diastolic blood pressure of subjects throughout the study.

Name	Run-in	beginning	Low period				washout	beginning	Control period			
			wk1	wk2	wk3	wk4			wk1	wk2	wk3	wk4
GS	78.00	92.00	85.00	73.00	80.00	81.33	80.00	90.00	88.00	85.00	81.33	80.67
TJ	81.00	96.00	84.00	84.00	86.67	84.00	90.00	82.00	88.67	85.00	86.67	81.33
PW	82.00	90.00	72.00	74.00	73.33	79.33	80.00	72.00	77.33	82.00	80.67	76.67
WS	91.00	98.00	101.33	89.00	94.00	84.67	98.00	102.00	110.00	101.33	104.00	97.33
KS	85.00	98.00	89.33	104.00	91.33	92.67	98.00	80.00	93.00	92.00	99.33	86.00
SJ	85.00	84.00	80.67	77.00	82.00	80.67	84.00	94.00	92.67	87.33	90.00	80.67
AK	87.33	70.00	81.00	70.67	76.67	79.33	79.33	70.00	77.33	74.67	73.33	75.33
YY	77.33	80.00	86.67	78.00	73.33	84.00	83.33	70.00	84.00	74.00	78.00	73.33
SG	92.00	100.00	91.00	91.33	90.00	82.67	90.00	94.00	91.33	87.33	84.00	86.67
SK	106.67	110.00	103.33	99.33	94.00	94.67	96.00	108.00	110.00	100.00	104.00	113.33
PP	119.00	120.00	110.00	106.67	114.00	113.33	130.00	120.00	110.67	120.00	119.33	116.00
NN	105.00	92.00	93.33	114.00	99.33	96.00	111.33	102.00	100.00	101.33	106.00	106.67
LT	85.00	82.00	74.00	80.67	80.00	79.33	78.00	80.00	90.00	94.00	83.33	86.00
PC	92.00	90.00	87.33	84.00	85.33	85.00	90.00	92.00	86.67	81.00	79.00	89.33
SM	102.67	100.00	98.00	87.33	94.00	90.00	99.33	90.00	102.67	91.00	96.00	98.67
KR	95.33	110.00	93.33	95.00	100.00	97.33	101.33	90.00	94.67	96.67	98.67	94.00
SS	96.00	100.00	98.00	89.00	101.00	96.00	100.67	90.00	102.67	100.67	97.33	91.00
WG	85.00	92.00	82.67	91.00	84.00	96.00	88.67	86.00	87.33	83.33	86.00	87.00

* beginning means the first day values of each intervention diet used to test the carry over effect

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Energy intake of subjects throughout the study.

Name	Run-in		Low period								Control period							
			wk1		wk2		wk3		wk4		wk1		wk2		wk3		wk4	
	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD
GS	2088	146	2070	146	2079	82	2126	113	1912	691	2005	66	2132	179	2072	148	2086	92
TJ	1848	146	2097	173	2130	141	2082	99	1836	738	2431	354	1986	231	2131	171	2004	213
PW	2340	142	2310	247	2855	384	2739	418	2157	787	2592	266	2404	240	2199	400	2123	232
WS	1210	109	1948	464	1782	384	1982	421	1718	550	1295	137	1686	424	1894	345	1792	446
KS	2418	160	2217	299	2159	418	2202	342	2207	678	2372	189	2653	308	2112	367	2469	317
SJ	2656	429	2767	200	2892	180	2710	197	2722	189	2412	485	2906	236	2772	198	2904	200
AK	1842	267	1908	315	1819	238	1644	188	1840	296	1461	204	1900	253	1720	87	1940	165
YY	1746	174	1597	107	1735	162	1597	107	1699	159	1597	107	1735	162	1597	107	1741	176
SG	1542	276	1575	354	1334	242	1579	203	1534	175	1494	146	1306	149	1450	129	1262	90
SK	1525	176	1504	167	1527	161	1504	167	1558	762	1504	167	1527	161	1504	167	1499	155
PP	1423	99	1992	236	2035	154	1992	236	2001	138	1992	236	2035	154	1992	236	1965	964
NN	1139	119	1164	108	1127	113	1164	108	1122	122	1164	108	1127	113	1164	108	1146	121
LT	1370	108	1434	62	1346	117	1434	62	1343	121	1434	62	1346	117	1434	62	1327	115
PC	2419	253	2087	150	2312	179	2087	150	2341	184	2220	269	2470	223	2097	158	2300	887
SM	1988	237	2082	126	2095	107	2005	113	2238	1100	2212	183	2384	123	2361	224	2373	778
KR	1978	131	1900	172	2099	200	2061	156	1984	755	2059	299	2031	97	2031	108	1923	205
SS	1542	124	1538	90	1503	115	1532	88	1527	587	1486	101	1463	72	1462	105	1548	758
WG	1756	188	1895	351	1653	209	1601	125	1669	669	1597	134	1534	161	1521	175	1583	184

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Sodium intake of subjects throughout the study.

Name	Run-in		Low period								Control period							
			wk1		wk2		wk3		wk4		wk1		wk2		wk3		wk4	
	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD
GS	4076	539	2513	282	2423	252	2619	356	2479	216	3894	545	3882	461	3870	435	3751	368
TJ	3755	598	2418	337	2352	180	2501	398	2457	174	3641	323	3811	516	3905	476	3731	565
PW	4485	516	3053	611	3024	365	3164	379	2768	339	4575	682	4501	465	4295	614	4089	826
WS	2900	704	2525	784	2068	335	2403	629	2009	393	2884	333	3517	844	3361	1056	3280	757
KS	4022	376	2683	579	2419	363	2286	281	2182	353	3774	541	3999	565	3606	600	3631	452
SJ	4612	887	2908	304	2206	443	2858	278	2267	571	4026	1162	3555	703	4435	391	3479	724
AK	3448	452	2305	508	2935	867	2500	243	2362	1034	3280	598	3946	699	3764	1551	3363	429
YY	3986	362	2577	318	2501	126	2600	542	2718	407	4909	1631	4737	1159	4875	1446	4435	1766
SG	3613	551	2093	630	2293	683	2016	367	2366	930	3168	433	2999	432	2860	309	2963	197
SK	3951	360	2735	273	2710	241	2801	185	2759	280	3859	638	4179	473	3983	389	3997	1633
PP	3443	509	3613	1088	2892	747	3101	658	2763	1146	4254	1039	4967	859	4585	1278	3699	239
NN	2862	586	1986	434	1790	479	2006	258	1822	744	2590	1194	2682	382	2915	397	2557	533
LT	3158	428	2069	401	2014	211	2029	318	2323	366	3164	504	3171	372	3091	483	3140	1231
PC	4802	627	3026	560	3044	524	2891	573	3401	532	5042	813	4649	960	4442	529	4217	1795
SM	3752	565	2589	221	2386	368	2462	398	2351	250	4304	353	3975	596	4132	542	3867	1444
KR	3788	505	2352	289	2188	285	2182	184	2202	875	3378	326	3506	317	3484	330	3502	191
SS	3818	501	2658	256	2841	292	2678	289	2439	960	4096	440	4055	274	4191	427	3952	432
WG	3444	351	2141	164	2170	316	1947	146	2212	313	3148	275	3275	381	3102	275	3375	450

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Potassium intake of subjects throughout the study.

Name	Run-in		Low period wk1		Low period wk2		Low period wk3		Low period wk4		Control period wk1		Control period wk2		Control period wk3		Control period wk4	
	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD
GS	1784	315	3442	399	3348	431	3455	513	3468	336	1987	220	1831	243	2084	192	1850	285
TJ	1784	316	3774	371	4004	479	4003	533	4011	622	2153	498	2103	453	2688	399	2127	406
PW	2410	514	3765	692	4048	843	3809	418	3456	397	2329	316	2012	290	2280	399	2131	365
WS	1575	321	3684	921	3011	579	3600	769	2962	597	1293	219	1674	414	2138	392	1589	371
KS	3449	399	4187	925	4103	670	3422	519	4147	716	3366	296	3325	512	2898	480	2491	501
SJ	2763	226	4697	493	3520	780	4570	568	3564	954	2576	520	2134	529	2791	298	2195	525
AK	2081	145	3071	235	3243	616	3222	603	3045	707	1582	276	2034	290	1767	240	2035	271
YY	2210	328	3450	525	3785	294	3684	585	3943	643	1870	269	2226	368	1866	306	2178	877
SG	1710	199	2977	809	2918	752	2968	552	2916	1129	1682	291	1547	296	1614	307	1400	197
SK	1886	204	3573	430	3669	572	3646	369	3687	406	1819	383	1897	196	1831	323	1817	702
PP	1534	165	5033	998	3963	757	4464	906	3753	536	2242	232	2279	354	2276	297	2126	363
NN	1479	312	2720	577	2656	550	2730	396	2653	1033	1343	630	1417	303	1493	277	1321	198
LT	1491	107	3060	367	2644	330	3015	618	2877	408	1707	334	1528	124	1700	332	1542	597
PC	2848	555	4225	472	4464	834	4076	364	4945	395	2529	469	2762	616	2214	301	2530	996
SM	2626	407	3848	337	3584	596	3544	425	3825	591	3237	503	3163	493	3606	523	3200	943
KR	2013	303	3577	238	3188	505	3109	457	3015	1258	1790	188	1706	176	1671	202	1902	157
SS	2087	232	3998	364	4113	538	3865	663	3589	1465	2213	560	2146	161	2204	328	2016	376
WG	2595	855	3337	473	2914	230	2906	322	2700	1370	1762	144	1777	207	1635	194	1735	216

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Sodium, potassium intake and 24-hr urinary sodium, potassium excretion of subjects at intervention period.

	NSD						LSD					
	Week 2		**Week 4**		**Average 4 week**		**Week 2**		**Week 4**		**Average 4 week**	
	intake	**24-hr urinary**	**intake**	**24-hr urinary**	**intake**	**24-hr urinary**	**intake**	**24-hr urinary**	**intake**	**24-hr urinary**	**intake**	**24-hr urinary**
Na	3850.9 (727.6)	2486.4 (856.6)	3548.7 (628.8)	2675.4 (1083.7)	3494.0 (690.4)	2580.9 (967.5)	2617.8 (639.8)	2093.6 (746.1)	2597.7 (548.6)	1984.2 (583.8)	2441.9 (517.0)	2038.9 (662.5)
K	2112.9 (584.1)	1133.8 (671.4)	2069.8 (567.7)	1226.0 (622.3)	2091.4 (568.1)	1179.9 (637.9)	3409.2 (549.5)	1904.3 (1003.2)	3481.1 (625.3)	1698.7 (765.7)	3445.1 (581.3)	1801.5 (885.7)

Values are means (SD)

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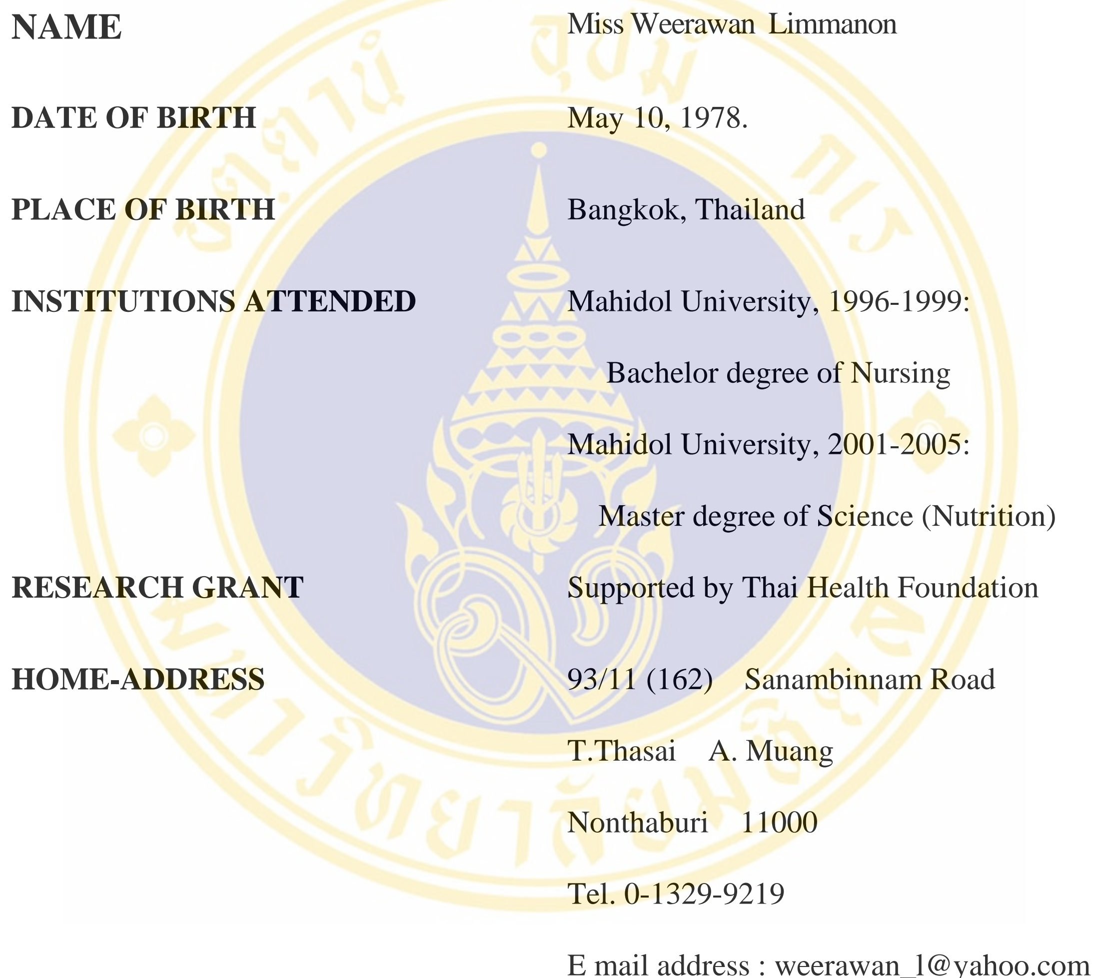
BIOGRAPHY



NAME	Miss Weerawan Limmanon
DATE OF BIRTH	May 10, 1978.
PLACE OF BIRTH	Bangkok, Thailand
INSTITUTIONS ATTENDED	Mahidol University, 1996-1999:
	Bachelor degree of Nursing
	Mahidol University, 2001-2005:
	Master degree of Science (Nutrition)
RESEARCH GRANT	Supported by Thai Health Foundation
HOME-ADDRESS	93/11 (162) Sanambinnam Road
	T.Thasai A. Muang
	Nonthaburi 11000
	Tel. 0-1329-9219
	E mail address : weerawan_l@yahoo.com

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