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The relationship between dietary intake and other cardiovascular risk factors with blood pressure in individuals without a history of a cardiovascular event: Evidence based study with 5670 subjects

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Running title: The relationship between dietary intake and cardiovascular risk factors with blood pressure

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Abstract:

Background and aim: Raised blood pressure is a leading cause of morbidity and mortality worldwide; improved nutritional approaches to population-wide prevention are required.We aimed to investigate the relationship between dietary intake and other cardiovascular risk factors with blood pressure in individuals without a history of a cardiovascular event in an Iranian cohort.

Material and method: A cross-sectional study of 5670 healthy subjects [approximately 40 % (n=2179) males and 60 % (n=3491) females] was undertaken in a sample from northeastern Iran. Subjects were recruited from an urban population, using a stratified-cluster method and derived from the Mashhad Stroke Heart Atherosclerosis Disorder (MASHAD) study, Mashhad, Iran. The age of the subjects was between 35-64 years. None of the subjects had a past history of major disease.

Results: The mean ages for the male and female subgroups were 50.1± 8.1years and 48.2 ±7.8 y respectively. Not unexpectedly, subjects without hypertension (HTN) were younger than those with established HTN. Individuals with HTN were significantly more adipose than those without ($p<0.01$). We found no significant differences in crude or total energy adjusted intake of nutrients between the three groups ($p> 0.05$), except for crude and energy adjusted phosphorus intake ($p<0.05$) and crude intake of the cholesterol ($p<0.05$). There was a significant correlation between the dietary intake of total fatty acids, phosphorus and vitamin E with both systolic blood pressure (SBP) and diastolic blood pressure (DBP). PUFA (odds ratio [OR] [95% confidence interval (CI)], 1.56 [1.05-1.06]; $P < 0.01$), sodium (OR [95% CI], 1.00 [(1.00-1.01)]; $P < 0.01$) and phosphorus (OR [95% CI], 1.00 [(1.00-1.01)]; $P < 0.01$)were significant independent predictors of HTN after adjustment for energy intake.

Conclusion: In our representative population from North-Eastern Iran, it appears that in adults without a history of cardiovascular disease, crude or energy adjusted intake of phosphorus and total fatty acid intake were significant determinants of BP, however we found no association between sodium and potassium intake with BP.

Key words: Cardiovascular risk factors, Hypertension, dietary intake

Introduction:

Hypertension (HTN) is a prevalent public health concern and a major risk factor for coronary heart disease (CHD), stroke, and renal disease. HTN affects approximately one-quarter of the world's adult population and it has been estimated that its prevalence will increase to 29% by 2025 [1]. Early management of hypertension can lead to a substantial reduction in the risk of cardiovascular morbidity and mortality [2].

The World Health Organization (WHO) has estimated that a sub-optimal blood pressure (BP) (a systolic BP >115 mm Hg) is accountable for 62% of cerebrovascular disease and 49% of ischemic heart disease (IHD), and a major attributable risk factor for death [3]. In the developed world, the prevalence of HTN has been reduced by population screening that has raised awareness and led to improved management of the risk factors [4]. In contrast, the prevalence of HTN in many developing countries is increasing due to changes in lifestyle that are associated with economic growth and urbanization [5]. The current prevalence of HTN in developing countries is estimated to be 22.9% whilst that reported for developed countries is approximately 37.3% [2].

Epidemiological studies have shown that diet is an important determinant of many chronic diseases: these include several cancers [6, 7], cardiovascular disease [8,9] and metabolic conditions [10,11]. Moreover, lifestyle modification is a key element of hypertension prevention and treatment [12]. To date, lifestyle interventions for lowering BP have concentrated on dietary sodium reduction and weight loss or maintenance [13]; however, a number of studies have also reported that dietary calcium, potassium, and magnesium are associated with a lower BP [14].

The DASH (Dietary Approaches to Stop Hypertension) trial reported that a dietary intervention has similar BP lowering effects to a single drug therapy [15], and the DASH dietary pattern is consistent with the Dietary Guidelines for Americans [16] and is recommended by the American Heart Association [17].

The aim of this current study was to investigate the association between dietary factors (micro- and macronutrients), other cardiovascular risk factors and BP.

Materials and Methods:

Subjects

The study was carried out on a sample of 5670 subjects recruited from an urban population, using a stratified-cluster method and derived from the Mashhad Stroke Heart Atherosclerosis Disorder (MASHAD) study, Mashhad, Iran (18). The age of the subjects was 35-64 years. None of the subjects had a past history of a cardiovascular event (unstable angina, MI and stroke), heart failure, peripheral vascular disease including transient ischemic attack or amaurosis fugax , or a history of any previous

cardiovascular interventions or surgery. Individuals with any major morbidity such as cancer, autoimmune, infectious and inflammatory diseases were also excluded. Each subject gave informed written consent to participate in the study, which was approved by the Mashhad University of Medical Science Ethics Committee.

Anthropometric Measurement

For all individuals, anthropometric parameters including weight, height, and waist circumference were measured using standard protocols. Height, body weight, and waist circumference were measured with subjects dressed in very light clothing after an overnight fast. Waist circumference was measured at the level of the umbilicus (at the level midway between the lower rib margin and the iliac crest). Body weight was measured with a standard scale to an accuracy of ±0.1 kg and height was measured to an accuracy of ±0.1 cm (a stadiometer was used for measuring height). Blood pressure (BP) was measured twice while patients were seated and rested for 15 min, using a standard mercury sphygmomanometer calibrated by the Iranian Institute of Standards and Industrial Research. The interval between each BP measurement was at least 30 min, and in this interval patients were at rest (not doing heavy activities or running) and the average of the two measurements was taken as the BP. Hypertension was diagnosed in individuals with systolic blood pressure at or above 140 mmHg and/or diastolic blood pressure at or above 90 mmHg, and in persons who were on anti-hypertension medications and pre hypertension were defined as systolic blood pressure at 130-139 and/or diastolic blood pressure at DBP:85-89 [19].Body mass index was calculated as weight (kg) divided by height squared (m^2).

Routine Biochemical Analysis

A full fasted lipid profile, comprising total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and LDL-C, was determined for each patient. Serum lipid and fasting blood glucose (FBG) concentrations were measured by enzymatic methods.

Dietary assessment

Dietary information was collected using a questionnaire for 24 h recall, administered by a trained dietary interviewer in a face-to-face interview, to recall and describe every item of food and beverage consumed over the previous 24 h period [20]. Individual nutritional intakes were assessed using Dietplan6 software (Forest field Software Ltd., UK). The dietary variables selected for the purpose of this study were crude, and total energy adjusted intake of micronutrients [20]. An adjustment was made for total energy intake through the residual method as an alternative to using nutrient densities to control for confounding by total energy intake and to remove extraneous variation due to total energy intake. Regression analyzes were used to compute residuals of nutrient intake by removing the

variation caused by total energy intake. In this procedure, the nutrient intakes of the individuals in a group are regressed on their total energy intakes. The residuals from the regression represent the differences between each individual's actual intake and the intake predicted by their total energy intake [21-23]. Total energy-adjusted nutrient intakes were calculated as the residuals from the regression model, with absolute nutrient intake as the dependent variable, and total energy intake as the independent variable [21].

Statistical analysis

SPSS software (version 11.5, Chicago, IL, USA) was used for statistical analysis. Kolomogrov-Smirnov test was used to evaluate the normality of data. Values were expressed as mean ±SD for normally distributed variables. Baseline demographic and clinical characteristics were compared between groups using independent samples *t*-test, chi-square and/or Fisher's exact test, as appropriate. We used multiple linear regression analysis to determine the association between the independent variable with clinical and biochemical factors. A p-value ≤0.05 was considered significant.

Results:

Of the 5670 participants, approximately 40 % (n=2179) males and 60 % (n=3491) females. The means (and SD) for age were 50.1± 8.1years for the male and 48.2 ±7.8 years for the female subgroups respectively.

Clinical, demographic and anthropometric data for subjects in different blood pressure categories

Table (1) shows the mean and standard deviation for clinical and biochemical parameters in the subgroups who were pre-hypertensive, hypertensive and normotensive. Age, weight, WC, FBG, total cholesterol, TGs, hs-CRP were significantly different between the groups ($p<0.01$); subjects without HTN were younger than those with established HTN ($p<0.0.5$). Furthermore subjects with HTN were significantly more adipose than those without ($p<0.0.5$).

Comparison of the crude and total energy adjusted intake of nutrients between the pre-hypertensive, hypertensive and normotensive subjects

Tables 2, shows the crude dietary intake and total energy adjusted intake of macro- and micronutrients. We found no differences in crude or total energy adjusted intake of nutrients between the three groups ($p> 0.05$) except for crude cholesterol and phosphorus intake and energy adjusted phosphorus intake($p< 0.05$).

Correlation between SBP and DBP with demographic, anthropometric, laboratory and nutritional data

To determine the relationship between systolic blood pressure (SBP), diastolic blood pressure (DBP) and other clinical and biochemical parameters, Pearson's correlation and Spearman's correlation coefficients were assessed. As can be seen from table (3), the correlation between SBP and DBP were significant for all parameters, except for HDL-C and hs-CRP respectively. Weaker negative associations were found between SBP and DBP with crude intake of fat ($r = -0.026$, $p<0.05$; $r=-0.021$, $p<0.05$), and saturated fatty acid ($r=0.027$, $p<0.05$; $r=0.036$, $p<0.05$). We found a significant correlation between both SBP and DBP with adjusted energy intake of the phosphorus, vitamin E and cholesterol ($p<0.05$) (Table 3).

Logistic Regression Analysis of Different Associated Factors with HTN

We show in table 4 the results of binary regression logistic analysis, which was used to determine the equation modeling the relationships between demographic, clinical, biochemical and dietary characteristics and HTN. Age, weight, WC and LDL-C were significantly related to the presence of HTN (Table 4; $p < 0.01$). Moreover, crude intake of fat, PUFA, and phosphorus were significantly associated with HTN ($p < 0.01$). However, only PUFA (odds ratio [OR] [95% confidence interval (CI)], 1.56 [1.05-1.06]; $P < 0.01$), sodium (OR [95% CI], 1.00 [(1.00-1.01)]; $P < 0.01$) and phosphorus (OR [95% CI], 1.00 [(1.00-1.01)]; $P < 0.01$) were significant independent predictors of HTN after adjustment for energy intake.

Table (5) shows the results of binary regression logistic analysis, which was used to determine the effect of each parameter in combination on the presence of MS, dyslipidaemia (Dys) and DM with HTN. In the first group the crude intake of total dietary fat, PUFA, and phosphorus were found to be significant predictors of HTN plus MS (Table 5; $p < 0.01$) moreover dietary calcium was a significant predictor for HTN plus dyslipidaemia. Trans fatty acid and calcium were significant ($p<0.05$) predictors of HTN plus obesity. As can be seen from table 6, when the total population was divided into quartiles for nutrient intake to remove the potential confounding for dietary change due to the diagnosis of hypertension, we can see the net effect of the dietary intake on hypertension. There were no significant differences between selected dietary intakes by quartile in the different category of the SBP, DBP, MS, dyslipidaemia and DM.

Discussion:

This study was undertaken primarily to determine the association between dietary micronutrients, macronutrients and cardiovascular risk factors for hypertension, including lipid profile, and demographic factors. This is the first population-based study to provide data on the relationship between hypertension and its associated determinants in this population.

We found that HTN was equally prevalent among those who were overweight and obese. Adiposity has previously been established as a risk factor for HTN, appearing to have an impact on HTN for a BMI as low as 23-25kg/m^2[24]. In a study based in Taiwan, using a logistic regression model, HTN was reported to be related to age and BMI [25].

The relationship between obesity and hypertension is well established both in adults and children [26,27]. The mechanism by which obesity causes hypertension remains an area of active research. Activation of the sympathetic nervous system (SNS) has been considered to have a crucial function in the pathogenesis of hypertension among obese individuals; however there is also evidence of high muscle SNS activity in obese subjects [28]. High-caloric intake increases norepinephrine turnover in peripheral tissues raises resting plasma norepinephrine concentrations and amplifies the rise of plasma norepinephrine in response to stimuli such as upright posture [29]. We found a significant correlation between both crude and energy adjusted intake of the total carbohydrates and fatty acids, this may partially be related to the effects of high dietary content of fat and carbohydrate on peripheral α- and β-adrenergic activity[30].

It is apparent from the table 3 that there is a relationship between crude and adjusted intake of Ca and hypertension. In the meta-analysis of Griffith et al. [31], analysis of sixty-six randomized trials showed a distinct beneficial effect of adequate dietary Ca intake on blood pressure. There were significant reductions in systolic blood pressure of 1·3 and 4·3 mmHg in the general population and in hypertensive subjects respectively. Moreover, calcium supplementation was associated with reductions in systolic and diastolic blood pressure of 0·2 and 1·5 mmHg respectively. They have also pointed out that groups at high risk for hypertension, such as African-Americans, salt-sensitive individuals and pregnant women, were particularly sensitive to the effect of increased Ca intake.

We found a significant inverse relationship between dietary phosphorus intake with BP. This possible association has not been reported previously. One animal study found a significant BP-lowering effect of dietary phosphorus in spontaneously hypertensive and normotensive rats [32]. A direct association was reported from the National Health and Nutrition Examination Survey I, based on a single 24-hour recall, [33,34] whereas the Honolulu Heart Study (also with a single 24-hour recall) reported 3/2 mm Hg lower systolic/diastolic pressures for the top compared with bottom quartile (2.2 SDs) of phosphorus intake (milligrams per day), with adjustment for age and body mass index [35]. An increase in dietary phosphorus from an estimated 940 to 1481 mg (at 2100 kcal) was a component of the Dietary Approaches to Stop Hypertension combination diet [36,37]; it also involved increases in dietary calcium, magnesium, and other micronutrients [38]. The critical importance of phosphorus in

cellular structure and function could have profound effects on BP regulation through its role in plasma membrane structure (phospholipids), energy production and storage (adenosine triphosphate, creatine phosphate, and other phosphorylated compounds), enzyme activation, cellular messengers such as G-proteins, and acid-base regulation [39] Phosphorus is also intimately involved in calcium regulation [40]; calcium has a membrane stabilizing effect [41] with a key role in vascular smooth muscle function [42].

Further statistical tests revealed a significant correlation between energy intake adjusted and crude intake of vitamin E with systolic and diastolic BP. Some antioxidant vitamins have previously been identified as having a role in blood pressure regulation. The role of stress and cellular oxidative damage through the formation of free radicals is well recognized as contributing to several chronic conditions. Free radicals have an effect on LDL oxidation and hence on endothelial function and atherogenesis, which are associated with hypertension. Also, the most important endothelium-dependent vasodilator, NO, is susceptible to oxidative damage and is protected by mechanisms inhibiting the formation of free radicals. Vitamin E is the antioxidant vitamin in the membrane regions of the cell and, hence, indirectly affects blood pressure regulation. Other mechanisms have also been suggested in this protective action. Vitamin E is an endogenous antioxidant: it breaks the chain of lipid peroxidation in cell membranes, prevents the formation of lipid hydroperoxides [43] and also stabilizes the membrane [44]. Thus, vitamin E improves cellular free radical defence potential and seems to have a beneficial effect on glucose transport and insulin sensitivity [45,46]. Wen et al. they found that erythrocyte tocopherol levels, which reflect the content of vitamin E in membranes, were also significantly lower in hypertensive than in healthy normotensive subjects; however, plasma levels of vitamin E were comparable in both hypertensive and normotensive subjects. These results suggest that hypertensive patients might have reduced tissue levels of the antioxidant vitamins and, hence, increased lipid peroxidation [47]. The role of vitamin E as an antioxidant is well recognized. Vitamin E supplements appear to decrease blood pressure in a variety of hypertensive conditions. Several studies indicate an association between dietary intake or plasma concentrations of the antioxidants vitamin E, vitamin C and -carotene, and hypertension [48]. Suboptimal levels of vitamin C, vitamin E and carotene have been reported as additive risk factors for cardiovascular diseases.

It is possible that in the present study there may have been a modification in the dietary intake of the subjects by previous medical, or public health recommendations. Furthermore the cross-sectional nature of the present study should be considered when interpreting our findings. The responses were based on self-recall and recall bias is a problem with any self-report survey. Although the 24-hour recall methodology has been largely used in cross-sectional surveys, they could be considered limited by their lack of quantitative accuracy. Scoliosis et al have suggested that, 'Frequent under-reporters' have a greater BMI, social desirability score, body dissatisfaction score and lower income. These four variables seemed to be able to discriminate individuals who are more prone to systematic under reporting [49]. Underreporting of energy intake may be a problem when obese subjects are under

investigation [50]. Another limitation is that that subjects below 35 years and over 65 years were not included because of financial constraints.

In the present survey the differences in mean intake levels of nutrients between under reporters and those who give valid records reduced by energy adjustment through the residual model. Our study includes a large sample of adults to examine our hypothesis. Future longitudinal studies are needed to establish the potential effect and causality.

Conclusion:

In our represented population from North-Eastern Iran, in individuals without a history of cardiovascular disease, crude and energy adjusted dietary phosphorus and fatty acids intakes were correlated with BP.

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Declaration of Interest: The authors have no conflict of interests to declare.

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Tables

Table 1 ***Clinical and Biochemical features' in blood pressure categories.***

Variables	Normal	Pre- HTN	HTN	p-value
Age(years)	47.5±7.1	51±7.6 a	52±7.7 a	<0.01
Weight(kg)	70±12	74±11 a	75±12 a	<0.01
BMI(kg/m^2)	27±4.3	28±4.2	29±4.5 a	0.32
WC (cm)	93±11	97±10.6 a	99±11 b	<0.01
FBG (mg/dl)	88±3.2	94±3.8 a	96±3.9 b	<0.01
HDL_C (mg/dl)	42±9.5	41±8.7	42±9.4	0.13
LDL_C (mg/dl)	119±31	122±3	122±34	0.11
TC (mg/dl)	189±36	195±36 a	197±36 a	<0.01
TGs (mg/dl)	121(86-174)	135(98-182) a	140(99-189) b*	<0.01
hsCRP	3.7±1.1	4.2±1.4 a	4.9±1.5 a*	<0.01
Current Smoking (%)	22%	21%	19.7%	<0.01
DM (%)	6.3%	9.2%	10.8%	<0.01
MS (%)	8.2%	30.9%	31.3%	<0.01
Dys (%)	25%	67.1%	69.1%	<0.01

Values expressed as mean ± SD for normally distributed data, and median and interquartile range for non-normally distributed data. ANOVA One-way analysis of variance and Tokay test were used for comparison between groups.

Compared to healthy subjects: a<0.05, b<0.01.

Compared to Pre-HTN subjects: *<0.05.

TC = Total cholesterol, HDL-C = High density lipoprotein cholesterol, LDL-C = Low density lipoprotein cholesterol, TGs = Triglycerides, BMI = Body mass index, DM = Diabetes Mellitus, WC=Waist Circumference, FBG=Fasting Blood Glucose, MS, Metabolic syndrome; Dys, Dyslipidemia; DM, Diabetes Mellitus;

Table 2 Nutritional Intake Characteristics of the subject *in blood pressure categories*

Variable		Groups		
		Normal	**Pre-HTN**	**HTN**
Crude daily Intake	**Energy intake (kcal)**	**1753(1326-2209)**	**1751(1366-2189)**	**1729(1353-2171)**
	Carbohydrate (g)	**232(175-300)**	**234(180-301)**	**232(176-297)**
	Fat (g)	**66(48-87)**	**65(47-85)**	**64(47-84)**
	Fiber (g)	**15.7(10.5-22.7)**	**16(10-23.8)**	**15.7(10.6-22.9)**
	MUFA (g)	**17.6(12.9-24.3)**	**17(12-23.1)**	**17.5(12.5-23.4)**
	Saturated-Fat (g)	**17.4(12.6-23.8)**	**17(12.5-23)**	**16.9(12.2-22.9)**
	PUFA (g)	**22.2(15.9-32)**	**22(15-31.9)**	**21.8(15.4-31)**
	Trans-Fat (g)	**0.8(0.4-1.2)**	**0.8(0.4-1.2)**	**0.7(0.4-1.2)**
	Cholesterol (mg)	**184(104-317)**	**176(102-290)**	**169(97-297)***
	Sodium (mg)	**1801(1159-2943)**	**1809(1207-2902)**	**1817(1157-2922)**
	Calcium (mg)	**794(549-1063)**	**806(590-1080)**	**816(567-1106)**
	Phosphorus (mg)	**1222(935-1588)**	**1257(952-1641)**	**1265(966-1639)***
Total energy adjusted intake	**Carbohydrate (g)**	**234(205-267)**	**237(208-266)**	**238(208-266)**
	Fat (g)	**67.1(55.4-78.7)**	**65.2(53.9-77.3)**	**65.6(55.0-77.1)**
	MUFA (g)	**18.1(14.3-21.7)**	**17.9(14.4-21.4)**	**17.9(14.6-21.1)**
	Saturated-Fat (g)	**17.5(14.1-21.8)**	**17.0(13.7-21.3)**	**16.9(13.7-21.0)**
	PUFA (g)	**22.9(16.5-28.8)**	**22.1(15.7-28.5)**	**22.3(15.9-28.4)**
	Trans-Fat (g)	**0.7(0.4-1.1)**	**0.7(0.4-1.0)**	**0.7(0.4-1.0)**
	Cholesterol (mg)	**178(116-300)**	**171.2(107.5-288.7)**	**169.8(110.1-286.4)**
	Sodium (mg)	**1951(995-2858)**	**2017(1053-2867)**	**2057(1106-2965)**

	Fiber(g)	**15.7(11.1-21.9)**	**16.3(11.6-22.2)**	**16.2(11.5-22.1)**
	Calcium(mg)	**778(586-1003)**	**807(620.1-1024)**	**812(624.0-1040)**
	Phosphorus (mg)	**1230(1048-1436)**	**1281(1082-1468.7)**	**1286(1086-1480) ***

Data were expressed as median (IQ). MUFA, mono unsaturated fatty acid; PUFA, poly unsaturated fatty acid. ANOVA One-way analysis of variance test was used. *p < 0.05

Table 3 Correlation (r) between Systolic and Diastolic blood pressure with Clinical and Biochemical factors as well as crude a energy adjusted nutrient intake.

Variable		Systolic Blood Pressure	Diastolic Blood Pressure
Age(years)		0.3*	0.22*
Weight(kg)		0.18*	0.22*
BMI(kg/m^2)		0.21*	0.21*
WC(cm)		0.25*	0.24*
FBG(mg/dl)		0.13*	0.65*
HDL_C(mg/dl)		-0.14	-0.09
LDL_C(mg/dl)		0.1*	0.1*
TC(mg/dl)		0.1*	0.1*
TGs(mg/dl)		0.1*	0.1*
hsCRP		0.19	0.02
Crude Intake	Energy intake (kcal)	0.03	0.03
	Carbohydrate (g)	0.01	0.011*
	Fat (g)	-0.026*	-0.021*
	Fiber (g)	0.012	0.001
	MUFA (g)	-0.016	0.027*
	Saturated-Fat (g)	-0.027*	-0.036*
	PUFA (g)	-0.01	-0.026*
	Trans-Fat (g)	-0.026*	-0.032*
	Cholesterol (mg)	-0.01	-0.028*
	Sodium (mg)	-0.04*	0.01
	Calcium(mg)	0.05*	0.04*
	Phosphorus (mg)	0.04*	0.03*
	Carbohydrate (g)	0.03*	0.02*

Total energy adjusted	**Fat (g)**	**0.04✸**	**-0.06✸**
	Fiber (g)	**0.01**	**0.01**
	MUFA (g)	**-0.032✸**	**0.051✸**
	Saturated-Fat (g)	**-0.05✸**	**-0.05✸**
	PUFA (g)	**0.026✸**	**0.042✸**
	Trans-Fat (g)	**-0.038✸**	**0.042✸**
	Cholesterol (mg)	**0.022**	**-0.032✸**
	Sodium (mg)	**-0.036✸**	**0.01**
	Calcium(mg)	**0.01✸**	**0.046✸**
	Phosphorus (mg)	**0.032✸**	**0.044✸**
	Vit E	**-0.028✸**	**-0.04✸**

Correlations were assessed using Pearson correlation coefficients. For non-normally distributed data such as triglycerides Pearson correlations were used.

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

TC = Total cholesterol, HDL-C = High density lipoprotein cholesterol, LDL-C = Low density lipoprotein cholesterol, TGs = Triglycerides, BMI = Body mass index, WC=Waist Circumference , ,FBG=Fasting Blood Glucose.

Table 4 The risk of hypertension in relation to demographic, clinical, biochemical and dietary characteristics.

Variables		Odds Ratio (95% CI)
		HTN
Age(year)		(1.05-1.07) **
Weight(Kg)		(1.02-1.03) **
BMI(kg/m^2)		(0.96-1.01)
WC(cm)		(1.01-1.03) **
FBG(mg/dl)		(1.01-1.03)**
LDL_C(mg/dl)		(0.99-1.01) **
TC(mg/dl)		(1.01-1.02)
TGs(mg/dl)		(0.99-1.02)
Crude Intake	Carbohydrate (g)	(0.98-1.01)
	Fat (g)	(0.96-0.99) **
	Saturated-Fat (g)	(0.94-1.03)
	MUFA (g)	(0.97-1.04)
	PUFA (g)	(1.05-1.06) **
	Trans-Fat (g)	(0.87-1.2)
	Cholesterol (mg)	(0.99-1.01)
	Sodium (mg)	(0.99-1.00)
	Potassium	(0.98-1.02)
	Calcium(mg)	(0.99-1.01)
	Phosphorus (mg)	(1.00-1.01) **
Total energy adjusted	Carbohydrate (g)	(0.97-1.07)
	Fat (g)	(0.63-1.08)
	Saturated-Fat (g)	(0.78-1.25)
	MUFA (g)	(0.74-1.03)
	PUFA (g)	(1.05-1.06) **

Trans-Fat (g)	**(0.87-1.2)**
Sodium (mg)	**(1.00-1.01) ****
Potassium	**(1.00-1.01)**
Calcium(mg)	**(0.09-1.02)**
Phosphorus (mg)	**(1.00-1.01) ****

Adjusted odds ratios with 95% confidence intervals (95% CI) obtained from multiple logistic regressions. Models were adjusted by logistic regression analysis for the association with increased HTN among our subjects. BMI, body mass ; HDL-C, high density lipoprotein cholesterol; FBS, fasting blood sugar; PUFA, poly unsaturated fatty acid; TG, triglyceride; MUFA, mono unsaturated fatty acid; **p < 0.01.

Table 5 Regression logistic analysis for HTN, MS, DM, Dys, Obese subject with Clinical and Biochemical factors as well as cr and energy adjusted nutrient intake.

Variable		Odds Ratio (95% CI)			
		HTN+MS	HTN+ Dys	HTN+DM	HTN+ Obese
Crude Intake	Carbohydrate (g)	(0.98-1.00)	(0.98-1.00)	(0.96-1.01)	(0.99-1.00)
	Fat (g)	(0.96-0.9)*	(0.76-1.02)	(0.92-1.03)	(0.97-1.01)
	Saturated-Fat (g)	(0.96 -1.02)	(0.67-1.04)	(0.92-1.03)	(0.92-1.00)
	MUFA (g)	(0.99-1.05)	(0.63-1.05)	(0.89-1.07)	(0.88-1.05)
	PUFA (g)	(1.02-1.04) *	(0.73-1.02)	(0.86-1.09)	(0.99-1.03)
	Trans-Fat (g)	(0.97-1.3)	(0.98-1.2)	(0.99-1.8)	(1.01 -1.1) *
	Cholesterol (mg)	(0.99-1.00)	(0.98-1.00)	(0.96-1.00)	(0.98-1.00)
	Fiber(g)	(0.98-1.03)	(0.76-1.01)	(0.97-1.10)	(0.97-1.03)
	Sodium (mg)	(0.98-1.00)	(0.99-1.00)	(1.00-1.02)	(0.75-1.05)
	Calcium(mg)	(0.97-1.00)	(1.00-1.02) *	(1.00-1.06)	(1.00-1.02) *
	Phosphorus (mg)	(1.00-1.01) *	(1.00-1.01)	(1.00-1.03)	(1.00-1.01)
Total energy adjusted	Carbohydrate (g)	(0.99-1.08)	(0.99-1.07)	(0.97-0.99)	(0.99-1.01)
	Fat (g)	(0.96-1.07)	(0.97-1.03)	(0.98-0.99)	(0.98-1.01)
	Saturated-Fat (g)	(0.97-1.03)	(0.96-1.04)	(0.86-1.1)	(0.94-1.03)
	MUFA (g)	(0.98-1.04)	(0.95-1.04)	(0.88-1.04)	(0.95-1.01)
	PUFA (g)	(1.04-1.05) *	(0.96-1.02)	(0.98-1.1)	(0.94-1.03)
	Trans-Fat (g)	(0.88-1.3)	(0.72-1.2)	(0.67-1.5)	(0.84-1.3)
	Cholesterol (mg)	(0.99-1.00)	(0.99-1.00)	(0.99-1.00)	(0.99-1.01)
	Sodium (mg)	(0.98-1.01)	(1.00-1.01)	(1.00-1.01)	(1.00-1.001)
	Calcium(mg)	(0.88-1.04)	(1.03-1.06)	(1.00-1.05)	(1.00-1.02)
	Phosphorus (mg)	(1.00-1.01)	(1.01-1.04)	(1.00-1.02)	(1.00-1.02)

		✸			

Adjusted odds ratios with 95% confidence intervals (95% CI) obtained from multiple logistic regressions. PUFA, poly unsaturated fatty acid; MUFA, mono unsaturated fatty acid; MS, Metabolic syndrome; Dys, Dyslipidemia; DM, Diabetes Mellitus; ✸p < 0.01.

Table 6 Mean SBP and DBP also prevalence of MS, Dys, DM in quartile for nutrient intake.

Variables	MUFA intake(g)			
	Q1	Q2	Q3	Q4
SBP	120.0±18.2	121±18.4	120±18.7	120±17.8
DBP	80±11.7	80±11.2	80±11.3	80±11.7
MS (%)	32.6	32.7	31.5	29.9
Dys(%)	28	28.7	28.7	26.5
DM(%)	6.6	7.5	8.1	9.8
	PUFA Intake(g)			
	Q1	Q2	Q3	Q4
SBP	123±18	122±18	123±18	121±15
DBP	80±11	79±11	79±11	79±10
MS (%)	32.7	31.1	31.0	31.5
Dys(%)	28.5	28	28	25.1
DM(%)	7.3	6.5	7.6	10.6
	Energy intake (kcal)			
	Q1	Q2	Q3	Q4
SBP	122±19	123±18	123±17	122±17
DBP	79±11	79±11	79±11	79±11
MS (%)	33.3	32.1	26.9	29.3
Dys(%)	29.3	28	31.7	25.6
DM(%)	6.1	7.7	8.7	9.3
	Saturated-Fat (g)			
	Q1	Q2	Q3	Q4
SBP	123±19	122±18	123±18	121±17
DBP	80±11	79±11	79±11	79±11

MS (%)	**32.5**	**34.3**	**31.1**	**28.3**
Dys(%)	**28.3**	**28**	**26.5**	**27.1**
DM(%)	**6.8**	**7.7**	**8.2**	**9.4**
	Trans-Fat (g)			
	Q1	**Q2**	**Q3**	**Q4**
SBP	**123±18**	**122±18**	**122±18**	**122±18**
DBP	**80±10**	**79±11**	**79±11**	**79±11**
MS (%)	**32.7**	**31.3**	**33**	**29.6**
Dys(%)	**27.8**	**28.8**	**28.7**	**27.9**
DM(%)	**7.6**	**7.5**	**7.6**	**9.1**
	Cholesterol (mg)			
	Q1	**Q2**	**Q3**	**Q4**
SBP	**122±18**	**122±18**	**122±17**	**122±17**
DBP	**80±11**	**78±11**	**79±10**	**79±18**
MS (%)	**34**	**31.5**	**30.4**	**30.3**
Dys(%)	**28.7**	**26.7**	**27.7**	**27**
DM(%)	**6.1**	**8.3**	**8.2**	**9.5**
Sodium (mg)				
SBP	**121±16**	**122±17**	**122±22**	**121±18**
DBP	**79±11**	**78±12**	**80±10**	**79±16**
MS (%)	**32**	**31**	**32.4**	**31.5**
Dys(%)	**29.7**	**27.7**	**26.3**	**25.4**
DM(%)	**9.3**	**5.3**	**7.2**	**6.1**
Phosphorus (mg)				
SBP	**120±13**	**122±13**	**123±26**	**120±15**
DBP	**80±13**	**79±10**	**82±13**	**79±18**
MS (%)	**30**	**32.9**	**32.7**	**32.5**

Dys(%)	**29.1**	**26.8**	**26.4**	**26.1**
DM(%)	**9.1**	**7.6**	**8.2**	**7.5**