


BMJ Open Association of dietary patterns and practices on metabolic syndrome in adults with central obesity attending a mission hospital in Kenya: a cross-sectional study

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ABSTRACT

Objective Dietary patterns and practices can predispose or protect against metabolic syndrome (MetS) in humans. Despite the growing problem of MetS in adults, the underpinning dietary behaviour is poorly understood. We determined the dietary patterns and practices relevant to MetS in adults with central obesity attending a mission hospital in Kenya.

Study design Descriptive, cross-sectional.

Setting Outpatient clinic of a mission-based hospital in Nairobi.

Participants Adults (N=404) aged 18–64 years diagnosed with central obesity as per the International Diabetes Federation definition for MetS.

Primary outcomes Anthropometric measurements, clinical-biochemical markers and dietary components, quantity and frequency of food intake, as well as time-lapse between consumption of dinner and sleeping.

Results A high (87.2%) prevalence of MetS was observed for respondents who reported consumption of large amount of carbohydrates ($p<0.001$), proteins ($p<0.001$), processed/fast foods ($p<0.001$) and sugar ($p=0.009$). Frequent consumption of legumes ($p<0.001$), nuts ($p<0.001$), fruits ($p<0.001$) and vegetables ($p=0.021$) was linked to reduced MetS. Additionally, longer interval between eating dinner and going to bed was associated with reduced MetS.

Conclusion Regular consumption of fruits, vegetables, legumes and nuts, as well as observing sometime after eating dinner before sleeping, was the dietary pattern significantly associated with a lower risk of MetS. Whereas, consumption of a large quantity of carbohydrates, proteins, processed/fast foods and sugar is likely to predispose to MetS. The findings underscore the need to focus on specific dietary intake patterns including frequency, quantity, quality and variety for MetS prevention and management. The MetS-related interventions could be implemented during individual consultation, group and community health messaging sessions.

INTRODUCTION

A dietary pattern refers to the quantity, variety or combination of different foods in a diet and the frequency with which they

Strengths and limitations of this study

- This was the first study conducted among the informal settlements (slums) in Kenya that determined the association between dietary intake patterns and metabolic syndrome.
- The relatively large sample size increases the possibility of replication and generalisation of the findings.
- The use of widely recognised and validated dietary questionnaires is another strength of the study.
- The study was limited by its cross-sectional design.
- The self-reported dietary patterns and practices may suffer from information bias.

are habitually consumed. The importance of nutritious dietary patterns and practices on metabolic syndrome (MetS) cannot be overstated. Indeed, components of MetS namely: central obesity, raised blood glucose, elevated blood pressure (BP) and dyslipidaemia¹ are closely linked to dietary behaviour. Dietary patterns and practices have been implicated in the risk for MetS.^{2–3} Several important factors including genetic, unhealthy eating habits and urbanisation have been cited as risk factors for MetS,⁴ however, unhealthy dietary pattern plays a major role in the incidence of MetS. Unhealthy diet characterised by consumption of processed/fast foods has been reported to be associated with MetS and cardiovascular diseases (CVDs).^{3–5} Whereas, a healthy dietary pattern characterised by regular consumption of fruits and vegetables,⁶ legumes^{7–8} and nuts⁹ is strongly associated with a lower risk of MetS. Saturated fatty acids mainly from processed/fast foods as well as refined carbohydrates are the major dietary factors fully responsible for the occurrence of MetS.¹⁰ Lifestyle modification involving adjusting the type, quality and quantity of diet is attributed to reduction of the risk for

MetS.^{5 11} Of significance is the fact that MetS is linked to high propensity of escalation into non-communicable diseases (NCDs) notably hypertension and type 2 diabetes and the resultant morbidity and mortality.^{12 13} Indeed, individuals with MetS are more than seven times more likely to develop diabetes and twofold likely to develop and die from CVDs.^{11 13}

A growing epidemic of MetS has been observed globally with sub-Saharan and developing countries bearing the biggest burden. For example, according to the International Diabetes Federation (IDF), the global prevalence of MetS is approximately 25%.¹⁴ MetS just like the NCDs is disproportionately heavy in sub-Saharan Africa with a resultant burden on the health system and economy. For example, the prevalence of MetS has been found to be 35.1% in Nigeria,¹⁵ 35.9% in Ghana,¹⁶ 39.0% in Cameroon¹⁷ and 42.1% in Egypt.¹⁸ In Kenya, the prevalence of MetS has been reported at 25.6%.¹⁹

The growing burden of MetS in Africa is associated with nutritional transition^{14 20} characterised by intake of high energy-dense foods,²¹ high quantity,³ lack of variety and quality of foods.²² Kenya is experiencing a rapid epidemiological and nutritional transition accompanied by increased consumption of unhealthy dietary pattern characterised by high intakes of refined carbohydrates, processed/fast foods, sugar-sweetened beverages, and low fruits and vegetables.² Evidence by Kimani *et al*²⁰ showed that patients with hypertension who daily consumed vegetables and fruits had lower rates of obesity, hypertension and cholesterol levels, some of the components of MetS. Elsewhere, reports from one of Nairobi's slums showed a high prevalence of overweight and abdominal obesity related to consumption of low vegetables and fruits.²² The reports and other documented evidence underscore the importance of diet in relation to MetS and its related components providing a window of prevention through awareness creation using health facilities or community as avenues for the interventions.

Unhealthy dietary pattern characterised by consumption of high-calorie diet such as processed/fast foods that are high in fats and sugars promotes obesity compared with low-energy foods, for example fruits and vegetables.²³ The high-calorie diets are causally linked to insulin resistance, type 2 diabetes, dyslipidaemia and high BP—the main components of MetS.²⁴ Moreover, diet has been associated with risk for high BP and poor hypertension control.²⁰ Although some studies in Kenya have demonstrated the association between nutrition and MetS, they are limited in number and methodological rigour. For well-thought evidence guided dietary/nutritional-oriented public health interventions on MetS studies are required. We sought to determine dietary patterns/practices (frequency intake of fruits, vegetables, legumes, nuts, processed and/or fast foods, proportion of protein and carbohydrate, amount of salt and sugar, as well as time interval between taking dinner and sleeping) relevant to MetS among Kenyan adults with central obesity attending a mission hospital in Nairobi.

METHODS AND MATERIALS

The study methods and materials have been well elaborated in our published work.²⁵ This is part of the larger community-based lifestyle intervention study for managing MetS among adults in an ongoing project.

Study setting

The study was executed at St Mary's Mission Hospital—a faith-based health facility located in Langata constituency Nairobi County. The hospital provides affordable services to a large low-income-earning population from the neighbouring Kibera, Mukuru-Kwa-Njenga and Kuwinda slums. Specifically, Kibera is the largest and poorest slum in Africa, with individual resident's average monthly income of US\$39 per household.²⁶ The hospital has an inpatient bed capacity of 350 offering medical, surgical, maternity, paediatric, postnatal, newborn unit, operating theatre, inpatient gynaecology and physiotherapy services. Additionally, the facility has a 24-hour outpatient department that offers general outpatient care, maternal and child health, diabetic and hypertension, nutrition, dental, eye, pharmacy, laboratory and imaging services, as well as HIV/AIDS prevention treatment and care services. The hypertension–diabetic clinic operates on daily basis from Monday to Friday serving about 600 patients per month. The clinic is run by a team of professionals comprising of physicians, nurses, nutritionists, laboratory technicians, pharmacists and social workers.

Study design, sampling methods and respondents

A cross-sectional study involving adults (N=404) aged 18–64 years. For inclusion into the study, we considered central obesity (waist circumference (WC) ≥ 94 cm for men and ≥ 80 cm for women) as the primary criteria for screening for the other MetS components as per the IDF guidelines.¹ We excluded pregnant and lactating women, individuals with contraindication for exercise due to serious diseases such as CVDs, cancer, mental illness and physical disability. However, some known patients with hypertension or diabetes were included based on the components of MetS. A systematic random sampling method was used to recruit the study respondents. During the study period, the clinic served for about 600 adults with hypertension and/or diabetes per month, which translated to 1800 patients in 3 months equivalent to the duration for completion of the data collection. Thus, the sampling interval was determined by dividing the target population (1800) in a 3-month period by the number of patients with hypertension–diabetes calculated to be screened for MetS (n=125) to get the sample interval of 14. Accordingly, every 14th client with hypertension/diabetes was included in the study after consenting until the desired sample size was achieved. An additional 279 participants who included those attending outpatients and their accompanying visitors were screened using systematic random sampling as they waited at the main laboratory waiting area of the hospital. During the same period, the hospital's main laboratory served for about

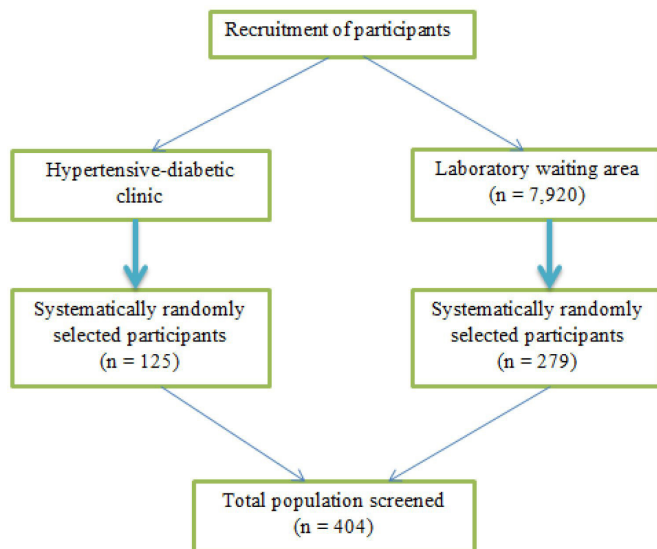


Figure 1 Flow chart showing recruitment of the participants.

2640 adult clients (aged 18–64) per month, equivalent to 7920 in a 3-month period, the duration required to complete the data collection. Then, the total population for 3 months (7920) was divided by the initially adjusted sample size (375) to get a sample interval of 21. Then every 21st participant was included in the screening until the desired sample size was achieved (figure 1).

Data collection tools and procedures

Data were collected using a researcher-assisted structured questionnaire adopted from the WHO STEPwise approach to NCD risk factor surveillance.²⁷ The questionnaire had four categories including sociodemographics, dietary intake patterns and practices, anthropometrics and biochemical markers. The data were collected by two trained research assistants with a bachelor's degree in nursing training background. The blood glucose and lipid levels were collected and analysed by two experienced laboratory technicians.

Assessment of dietary patterns/practices

A pre-intervention and post-intervention dietary intake patterns including quantity, variety and the frequency with which the respondents habitually consumed were assessed using 32 food frequency questionnaires adopted from the WHO STEPwise approach to NCD risk factor surveillance,²⁷ with some modifications to fit the Kenyan dietary context. The respondents were asked how frequently they consumed a particular food product, and the quantity usually they eat per food item by making comparisons with the specific reference portions. Frequency intake of fruits, vegetables, legumes, nuts, processed/fast foods, quantity or proportion of protein and carbohydrate, amount of salt and sugar, as well as time interval between taking dinner and sleeping, was assessed by asking the respondents how often a week they consumed these foods. The recommended frequency of legume and nut intake is four to five times

per week and therefore we grouped the responses as (often \geq four times/week, sometimes (two to three times/week and rarely (\leq one time/week). Whereas the recommended frequency of fruit and vegetable intake is four to five times a day, and thus we grouped as daily and not daily. Consumption of processed/fast foods was categorised as (always \geq 5 days/week, sometimes 2–4 days/week, rarely $<$ 2 days/week). Common household measuring equipment including measuring cups, spoons and plates were shown to assist the participants in the estimation of the amounts to avoid measurement bias. As part of the dietary pattern assessment, the dietary approach to stop hypertension (DASH) eating plan was used to estimate the quantity or proportion of protein (eg, meat, eggs, whole milk), carbohydrate (eg, ugali, bread, chapatti, rice, maize, potatoes, pasta) and vegetables/fruits as well as frequency of legume and nut consumption. According to the DASH diet, vegetables and/or fruits constitute one-half of a plate at each meal. One-quarter of the plate is filled with carbohydrates and the remaining one-quarter is filled with plant proteins like legumes, soy products, nuts and seed proteins to control high BP. Animal proteins in the diet should mainly compose of lean meats, low-fat dairy, eggs and fish. We measured the proportion of a meal by drawing a plate into four parts. Then we asked the respondents to estimate the proportion of vegetables and/or fruits, carbohydrate and proteins they usually fill their plate. The processed and/or fast foods such as chips, sandwiches, hamburgers, fried chicken, French fries, sausages, samosas, pizza, hot dogs, ice cream among others were captured using specific questions focused on such theme integrated in the questionnaire. Time interval between taking dinner and sleeping was assessed by asking respondents at what time do they usually eat their dinner. The responses were: before 20:00, between 20:00 and 21:00, between 21:00 and 22:00 and after 22:00. Then, they were asked the time they usually go to bed to sleep. The responses were: before 21:00, between 21:00 and 22:00, between 22:00 and 23:00, between 23:00 and midnight and after midnight.

Anthropometric variables

The anthropometric parameters included weight, height, WC and hip circumference (HC). These parameters were measured using standard measurement tools. The body weight was measured to the nearest 0.1 kg using a Sohenle mechanical weighing scale with the respondent in light clothing. The height (m) was measured using a portable stadiometer to the nearest 0.5 cm, with subjects standing upright on a flat surface without shoes, the back of the heels and the occiput on the equipment. The height and weight was used to calculate the body mass index that is the ratio of weight (kg) over height in m^2 . The WC was taken at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest to the nearest 0.1 cm using a procedure by WHO. The HC was taken at the greatest posterior protuberance of the buttocks to the nearest 0.1 cm using a flexible tape.²⁸

Clinical variables (BP and heart rate)

BP and heart rates were measured using OMRON automatic BP monitor (model: M3; HEM-141-E, serial no: 20170916247VG, Japan) after a rest period of 5–10 min in a sitting position. The BP was measured two times with a 5 min time interval with the mean of the two measurements being recorded. Elevated BP as a component of MetS was defined as $\geq 130/85$ mm Hg.¹ A systolic BP of 120–139 mm Hg and/or diastolic BP of 80–89 mm Hg were considered as prehypertension. Hypertension was defined as systolic BP ≥ 140 mm Hg and/or diastolic BP ≥ 90 mm Hg.²⁹ The heart rate was measured for 1 min as well by the BP monitor.

Biochemical variables

The blood samples for fasting blood glucose (FBG), lipids (triglycerides (TGs) and high-density lipoprotein cholesterol (HDL-C)) levels were drawn and analysed following an overnight fasting of 8–12 hours. FBG sample was obtained from the respondents' finger using HemoCue B-Glucose photometer (photometer, 1995). A sample of 3 mL of blood was obtained from the brachial vein following standard infection prevention procedures to determine TGs and HDL-C values. Each blood sample was labelled with the participants' number to avoid errors of recording. Raised FBG level was defined as FBG level ≥ 5.6 mmol/L.¹ Pre-diabetes and diabetes were defined as FBG of 5.6–6.9 mmol/L and ≥ 7 mmol/L, respectively.³⁰ Raised TG was defined as TGs level ≥ 1.7 mmol/L irrespective of gender. Whereas, low HDL-C level was defined as HDL-C < 1.03 mmol/L in men and < 1.29 mmol/L in women.¹

Validity and reliability of the study tools

The WHO STEPwise approach to NCD risk factor surveillance questionnaire²⁷ was used to collect the data. Additionally, the tools were reviewed for content validity by experts in the field of CVD and nutrition to ascertain relevance and completeness. The recommendations and suggestions were incorporated in the final questionnaire. To measure reliability of the questionnaire, a test re-test method was employed, whereby a repeat pre-test was carried out after 3 weeks, and Cohen's kappa statistic was used to measure the level of agreement of the two results. The result of the repeated questions had a kappa value of 0.91 therefore, the questionnaire was considered reliable.

Patient and public involvement

There were no patients and members of the public involved in developing the research design and questions. This research was solely done by researchers without involving patients or public in any part of the research work. However, this research involved human participants. The results will be disseminated to the public during health message sharing sessions in the study area as well as seminars/conferences.

Definition of MetS

MetS was defined using the IDF criteria¹ to include central obesity (WC of ≥ 94 cm for men and ≥ 80 cm for women) that was compulsory. The criteria also included other components namely: (1) raised TGs level ≥ 1.7 mmol/L (≥ 150 mg/dL) or history of specific treatment for the lipid abnormality; (2) reduced HDL-C < 1.03 mmol/L (< 40 mg/dL) in men and < 1.29 mmol/L (< 50 mg/dL) in women or history of specific treatment for the lipid abnormality; (3) elevated BP: systolic BP ≥ 130 mm Hg or diastolic BP ≥ 85 mm Hg or on treatment for previously diagnosed hypertension; (4) raised FBG level of ≥ 100 mg/dL (≥ 5.6 mmol/L) or previously diagnosed type 2 diabetes mellitus. The respondents had to display at least two of the four metabolic abnormalities in addition to the central obesity.

Data analyses

Statistical analyses were performed using the SPSS V.22. Descriptive data were analysed using proportions and summarised in frequency tables. The χ^2 test of independence and binary logistic regression were used to determine associations between categorical variables such as frequency of specific food consumption, prevalence of MetS and its related components. A multiple logistic regression model with backward conditional was carried out to determine the dietary-related variables independently contributed to the occurrence of MetS. Backward conditional method was specified with removal at $p < 0.05$ to determine the independent predictors of MetS as it removes the confounding variables until no further variables can be removed without a statistically insignificant loss of fit (last or reduced model). The fitness model was also performed to describe the variance and classification of MetS. A p value of less than 0.05 was considered to be significant.

RESULTS

Demographic characteristics of the respondents

Respondents totalling 404 were recruited with mean age of 42.5 ± 11.9 (mean \pm SD) years. Most (59.2%, $n=239$) of them were between 31 and 50 years. A high proportion of the respondents were married (76.0%, $n=307$), women (54.5%, $n=220$), Protestants (59.7%, $n=241$) and self-employed (52.2%, $n=211$). Respondents with secondary level education were 48.8% ($n=197$). Economically family monthly income was reported to be between US\$100 and US\$300 (45.5%; $n=184$) (table 1).

Relationship between the DASH eating plan and MetS-related components

The DASH diet was used to determine quantity or proportion of meals usually consumed by the respondents. According to the DASH diet, at each meal, vegetables and/or fruits constitute one-half of a plate, carbohydrates one-quarter of the plate and the remaining one-quarter of the plate to be filled with plant proteins like legumes,

Table 1 Demographic characteristics of the respondents

Characteristics	Number	%
Age (years)		
≤30	68	16.8
31–40	124	30.7
41–50	115	28.5
>50	97	24
Gender		
Male	184	45.5
Female	220	54.5
Marital status		
Married	307	76
Single	69	17.1
Divorced	6	1.5
Separated	11	2.7
Widowed	10	2.5
Cohabiting	1	0.2
Religion		
Protestant	241	59.7
Catholic	131	32.4
Muslim	32	7.9
Level of education		
No formal education	7	1.7
Primary	75	18.6
Secondary	197	48.8
College/university	125	30.9
Occupation		
Government employee	15	3.7
Non-government employee	111	27.5
Self-employed	211	52.2
Unemployed	67	16.6
Family monthly income (US\$)		
Less than 100	67	16.6
101–300	184	45.5
301–500	66	16.3
Over 500	49	12.1
No response	38	9.4
Total	404	100

soy products, nuts and seed proteins. Animal proteins in the diet should be limited to lean meats, low-fat dairy, eggs and fish. Most (77%; n=311) of the respondents reported consuming less than the recommended portion of a plate as vegetables and/or fruits. Further analysis revealed that those who consumed less than the recommended portion of a plate as vegetables and/or fruits were more likely to have MetS ($\chi^2=32.004$, $p<0.001$), elevated BP ($\chi^2=116.082$, $p<0.001$) and raised FBG level ($\chi^2=5.590$, $p=0.018$) compared with those who frequently took the

recommended portion as vegetables/fruits. Most, 70.8% (n=286) of the respondents, frequently consumed a large proportion (over one-quarter of a plate) of a carbohydrate diet as part of the main meals. Respondents who frequently consumed a large proportion of carbohydrate foods were more likely to have MetS ($\chi^2=33.866$, $p<0.001$) and elevated BP ($\chi^2=79.690$, $p<0.001$) compared with those who ate the recommended amounts ($\leq 25\%$ portion of a plate). With regards to protein consumption, slightly above a quarter (29%; n=117) of the respondents frequently consumed a large proportion (over one-quarter of a plate) of protein diet as part of the main meals. Respondents who frequently consumed large proportion of foods rich in proteins were more likely to have MetS ($\chi^2=13.122$, $p<0.001$), elevated BP ($\chi^2=33.342$, $p<0.001$) and raised FBG level ($\chi^2=6.393$, $p=0.011$) compared with those who ate the recommended amounts (less than or 25% portion of a plate) (table 2).

Frequency of fruit and vegetable intake in relation to MetS and related components

Of the respondents, some, 17.8% (n=72) and 41.1% (n=166), daily consumed fruits and vegetables, respectively. Further analysis showed a significant association between daily consumption of fruits and vegetables and MetS with some of its components. Respondents who did not consume fruits daily were more likely to have MetS ($\chi^2=14.276$, $p<0.001$), elevated BP ($\chi^2=13.505$, $p<0.001$) and raised FBG level ($\chi^2=9.301$, $p=0.002$) compared with those who frequently consumed fruits. Similarly, respondents who did not daily consume vegetables were more likely to have MetS ($\chi^2=5.313$, $p=0.021$) and elevated BP ($\chi^2=24.677$, $p<0.001$) compared with those who ate daily (table 3).

Association between legume and nut consumption and MetS-related components

According to the DASH eating plan, the recommended frequency of legume and nut consumption is four to five times per week. Less than a quarter (20.5%; n=83) of the respondents included legumes/pulses in their meals as recommended (four to five times in a week). Similarly, a small proportion (15.1%; n=61) of the respondents consumed nuts 4–5 days in a week as part of their meals. Further analysis with binary logistic regression showed a significant association between frequency of legume and nut consumption and MetS. Respondents who consumed legumes less than the recommended frequency were more likely to have MetS (OR=125.8; $p<0.001$), elevated BP (OR=2.3; $p=0.001$), low HDL-C (crude OR/COR=3.0; $p<0.001$), raised TGs (OR=4.1; $p<0.001$) and raised FBG level (adjusted OR=2.2; $p=0.037$) compared with those who ate them as recommended. Respondents who sometimes (two to three times/week) consumed nuts were more likely to have MetS (OR=105.6; $p<0.001$), elevated BP (COR=5.4; $p<0.001$), reduced HDL-C (OR=3.0; $p<0.001$), raised TGs (OR=6.2; $p<0.001$) and raised FBG level (OR=3.2; $p=0.011$) compared with those who

Table 2 Relationship between the DASH eating plan and metabolic syndrome (MetS)-related components

MetS and its components	Proportion of a plate filled with vegetables and/or fruits		Total	χ^2	Df	P value
	Less than half of a plate	Half and above of a plate				
MetS	N (%)	N (%)	N (%)	32.004	1	<0.001
Yes	287 (81.5)	65 (18.5)	352 (100)			
No	24 (46.2)	28 (53.8)	52 (100)			
Total	311 (77.0)	93 (23.0)	404 (100)			
Blood pressure (BP)				116.082	1	<0.001
Elevated BP	248 (93.2)	18 (6.8)	266 (100)			
Normal BP	63 (45.7)	75 (54.3)	138 (100)			
High-density lipoprotein cholesterol (HDL-C)				1.084	1	0.298
Reduced HDL-C	231 (75.7)	74 (24.3)	305 (100)			
Normal HDL-C	80 (80.8)	19 (19.2)	99 (100)			
Triglycerides (TGs)				1.729	1	0.189
Raised TGs	207 (79)	55 (21)	262 (100)			
Normal TGs	104 (73.2)	38 (26.8)	142 (100)			
Fasting blood glucose (FBG)				5.59	1	0.018
Raised FBG level	76 (86.4)	12 (13.6)	88 (100)			
Normal FBG level	235 (74.4)	81 (25.6)	316 (100)			
	Proportion of a plate filled with carbohydrates					
	≤one-quarter of a plate	>one-quarter of a plate				
MetS	N (%)	N (%)	N (%)	33.866	1	<0.001
Yes	85 (24.1)	267 (75.9)	352 (100)			
No	33 (63.5)	19 (36.5)	52 (100)			
Total	118 (29.2)	286 (70.8)	404 (100)			
BP				79.69	1	<0.001
Elevated BP	39 (14.7)	227 (85.3)	266 (100)			
Normal BP	79 (57.2)	59 (42.8)	138 (100)			
HDL-C				0.054	1	0.816
Reduced HDL-C	90 (29.5)	215 (70.5)	305 (100)			
Normal HDL-C	28 (28.3)	71 (71.7)	99 (100)			
TGs				2.974	1	0.085
Raised TGs	69 (26.3)	193 (73.7)	262 (100)			
Normal TGs	49 (34.5)	93 (65.5)	142 (100)			
FBG				1.554	1	0.213
Raised FBG level	21 (23.9)	67 (76.1)	88 (100)			
Normal FBG level	97 (30.7)	219 (69.3)	316 (100)			
Total	118 (29.2)	286 (70.8)	404 (100)			
	Proportion of a plate filled with protein					
	≤one-quarter of a plate	>one-quarter of a plate				
MetS	N (%)	N (%)	N (%)	13.122	1	<0.001
Yes	239 (67.9)	113 (32.1)	352 (100)			

Continued

Table 2 Continued

MetS and its components	Proportion of a plate filled with vegetables and/or fruits		Total	χ^2	Df	P value
	Less than half of a plate	Half and above of a plate				
No	48 (92.3)	4 (7.7)	52 (100)			
Total	287 (71.0)	117 (29.0)	404 (100)			
BP				33.342	1	<0.001
Elevated BP	164 (61.7)	102 (38.3)	266 (100)			
Normal BP	123 (89.1)	15 (10.9)	138 (100)			
HDL-C				0.464	1	0.496
Reduced HDL-C	214 (70.2)	91 (29.8)	305 (100)			
Normal HDL-C	73 (73.7)	26 (26.3)	99 (100)			
TGs				0.238	1	0.626
Raised TGs	184 (70.2)	78 (29.8)	262 (100)			
Normal TGs	103 (72.5)	39 (27.5)	142 (100)			
FBG				6.393	1	0.011
Raised FBG level	53 (60.2)	35 (39.8)	88 (100)			
Normal FBG level	234 (74.1)	82 (25.9)	316 (100)			
Total	287 (71)	117 (29)	404 (100)			

DASH, dietary approach to stop hypertension.

included nuts as recommended (four to five times/week) (table 4).

Consumption of processed and/or fast foods relative to MetS and related components

Slightly over one-third (36.4%; n=147) of the respondents always consumed processed and/or fast foods, of which, 96.6% presenting with MetS. Further analysis showed a significant association between frequency of eating processed/fast foods and MetS. Respondents who always consumed processed/fast foods were more likely to have MetS ($\chi^2=66.34$; $p<0.001$) compared with those who rarely ate those types of food. Moreover, respondents who sometimes ate processed/fast foods were more likely to have elevated BP (OR=6.3; $p<0.001$), low HDL-C (OR=2.5; $p=0.002$), raised TGs (OR=2.6; $p<0.001$) and raised FBG level (OR=5.8; $p<0.001$) compared with those who rarely ate these foods (table 5).

Consumption of sugar and salt in relation to MetS-related components

Most (62.6%; n=253) of the respondents consumed more than the recommended (more than five teaspoons) amount of sugar in a day. With regards to salt intake, the majority, 57.9% (n=234) of the respondents added salt to their meal right before they ate or as they were eating it. Further analysis revealed respondents who consumed more than the recommended amount of sugar were more likely ($\chi^2=6.917$, $p=0.009$) to have MetS. However, raised FBG level was significantly ($\chi^2=25.099$, $p<0.001$) related to consumption of less than five teaspoons of sugar per

day. Respondents who added salt to foods right before they ate were more likely ($\chi^2=21.718$, $p<0.001$) to have elevated BP compared with those who did not add salt after the food has been cooked. However, respondents who did not add salt after the food has been cooked were more likely to have both raised TGs ($\chi^2=9.697$, $p=0.002$) and FBG ($\chi^2=10.028$, $p=0.002$) levels compared with those who added salt while they were eating their meals (table 6).

Time interval after consumption of dinner and sleeping in relation to MetS-related components

Less than a quarter (19.6%; n=79) of the respondents observed more than a 2-hour interval between taking dinner and sleeping. Analysis with binary logistic regression revealed a significant association between the time interval of taking dinner and sleeping with MetS. Respondents who observed more than a 2-hour interval between eating dinner and sleeping were 20% ($p<0.001$), 40% ($p=0.001$), 50% ($p=0.027$) and 50% ($p=0.011$) less likely to have MetS, elevated BP, reduced HDL-C and raised TGs, respectively, compared with those who observed less than a 1-hour time interval between eating dinner and sleeping (table 7).

Multivariable analysis for dietary risk factors of MetS

Binary logistic regression analysis was performed to model MetS (presence or absence) as a dependent variable and the independent variables that revealed significant association at $p<0.05$ during the bivariate analysis.

Table 3 Frequency of fruit and vegetable intake in relation to metabolic syndrome (MetS) and related components

MetS and its components	Frequency of fruit intake			χ^2	Df	P value
	Daily	Not daily	Total			
MetS	N (%)	N (%)	N (%)	14.276	1	<0.001
Yes	53 (15.1)	299 (84.9)	352 (100)			
No	19 (36.5)	33 (63.5)	52 (100)			
Total	72 (17.8)	332 (82.2)	404 (100)			
Blood pressure (BP)				13.505	1	<0.001
Elevated BP	34 (12.8)	232 (87.2)	266 (100)			
Normal BP	38 (27.5)	100 (72.5)	138 (100)			
High-density lipoprotein (HDL)				1.029	1	0.31
Reduced HDL	51 (16.7)	254 (83.3)	305 (100)			
Normal HDL	21 (21.2)	78 (78.8)	99 (100)			
Triglycerides (TGs)				2.403	1	0.121
Raised TGs	41 (15.6)	221 (84.4)	262 (100)			
Normal TGs	31 (21.8)	111 (78.2)	142 (100)			
Fasting blood glucose (FBG)				9.301	1	0.002
Raised FBG level	6 (6.8)	82 (93.2)	88 (100)			
Normal FBG level	66 (20.9)	250 (79.1)	316 (100)			
Total	72 (17.8)	332 (82.2)	404 (100)			
	Frequency of vegetable intake					
	Daily	Not daily				
MetS	N (%)	N (%)	N (%)	5.313	1	0.021
Yes	137 (38.9)	215 (61.1)	352 (87.1)			
No	29 (55.8)	23 (44.2)	52 (12.9)			
Total	166 (41.1)	238 (58.9)	404 (100)			
BP				24.677	1	<0.001
Elevated BP	86 (32.3)	180 (67.7)	266 (65.8)			
Normal BP	80 (58)	58 (42)	138 (34.2)			
HDL				0.748	1	0.387
Reduced HDL	129 (42.3)	176 (57.7)	305 (75.5)			
Normal HDL	37 (37.4)	62 (62.6)	99 (24.5)			
TGs				0.019	1	0.89
Raised TGs	107 (40.8)	155 (59.2)	262 (64.9)			
Normal TGs	59 (41.5)	83 (58.5)	142 (35.1)			
FBG				0.204	1	0.652
Raised FBG level	38 (43.2)	50 (56.8)	88 (100.0)			
Normal FBG level	128 (40.5)	188 (59.5)	316 (100.0)			
Total	166 (41.1)	238 (58.9)	404 (100.0)			

Accordingly, the logistic model included the following factors: proportion of a plate filled with vegetables and/or fruits; frequency of processed/fast food, fruit, vegetable, legume and nut intake; sugar consumption status; and time interval between taking dinner and sleep. Backward conditional method was specified with removal at $p < 0.05$ to determine the independent predictors of MetS as it removes the confounding variables until no further variables can be removed without a statistically

insignificant loss of fit (last or reduced model). After considering all, frequency of processed/fast food, legume and nut intake was independently associated with MetS. The fitness model according to Hosmer and Lemeshow Test was 0.248, which indicates the model fits. Respondents who often consumed processed/fast foods were three times (95% CI: 1.48–7.29, $p = 0.003$) more likely to develop MetS compared with those who rarely consumed legumes. Respondents who rarely and

Table 4 Association between legume and nut consumption and metabolic syndrome-related components

Frequency of legume intake	Metabolic syndrome		Total	COR (95% CI)	P value
	Yes	No			
Always	40 (48.2)	43 (51.8)	83 (100)	1	
Sometimes	78 (91.8)	7 (8.2)	85 (100)	125.8 (29.3–539.9)	<0.001
Rarely	234 (99.2)	2 (0.8)	236 (100)	10.5 (2.1–51.6)	0.004
Total	352 (87.1)	52 (12.9)	404 (100)		
Frequency of legume intake	Blood pressure (BP)		Total	COR (95% CI)	P value
	Elevated BP	Normal BP			
Often	43 (51.8)	40 (48.2)	83 (100)	1	
Sometimes	54 (63.5)	31 (36.5)	85 (100)	2.3 (1.4–3.9)	0.001
Rarely	169 (71.6)	67 (28.4)	236 (100)	1.4 (0.9–2.4)	0.167
Total	266 (65.8)	138 (34.2)	404 (100)		
Frequency of legume intake	High-density lipoprotein (HDL)		Total	COR (95% CI)	P value
	Low HDL	Normal HDL			
Often	48 (57.8)	35 (42.2)	83 (100)	1	
Sometimes	67 (78.8)	18 (21.2)	85 (100)	3.0 (1.8–5.2)	<0.001
Rarely	190 (80.5)	46 (19.5)	236 (100)	1.1 (0.6–2.0)	0.739
Total	305 (75.5)	99 (24.5)	404 (100)		
Frequency of legume intake	Triglycerides (TGs)		Total	COR (95% CI)	P value
	Raised TGs	Normal TGs			
Often	33 (39.8)	50 (60.2)	83 (100)	1	
Sometimes	57 (67.1)	28 (32.9)	85 (100)	4.1 (2.4–6.9)	<0.000
Rarely	172 (72.9)	64 (27.1)	236 (100)	1.3 (0.8–2.3)	0.309
Total	262 (64.9)	142 (35.1)	404 (100)		
Frequency of legume intake	Fasting blood glucose (FBG)		Total	COR (95% CI)	P value
	Raised FBG level	Normal FBG level			
Often	10 (12)	73 (88)	83 (100)	1	
Sometimes	24 (28.2)	61 (71.8)	85 (100)	2.2 (1.0–4.5)	0.037
Rarely	54 (22.9)	182 (77.1)	236 (100)	0.8 (0.4–1.3)	0.325
Total	88 (21.8)	316 (78.2)	404 (100)		
Frequency of nut intake	Metabolic syndrome		Total	COR (95% CI)	P value
	Yes	No			
Often	19 (31.1)	42 (68.9)	61 (100)	1	
Sometimes	94 (94.9)	5 (5.1)	99 (100)	105.6 (37.4–298.4)	<0.001
Rarely	239 (98)	5 (2)	244 (100)	2.5 (0.7–9.0)	0.147
Total	352 (87.1)	52 (12.9)	404 (100)		
Frequency of nut intake	BP		Total	COR (95% CI)	P value
	Elevated BP	Normal BP			
Often	22 (36.1)	39 (63.9)	61 (100)	1	
Sometimes	60 (60.6)	39 (39.4)	99 (100)	5.4 (3.0–9.9)	<0.001
Rarely	184 (75.4)	60 (24.6)	244 (100)	2.0 (1.2–3.3)	0.007
Total	266 (65.8)	138 (34.2)	404 (100)		
Frequency of nut intake	HDL		Total	COR (95% CI)	P value
	Reduced HDL	Normal HDL			
Often	35 (57.4)	26 (42.6)	61 (100)	1	
Sometimes	74 (74.7)	25 (25.3)	99 (100)	3.0 (1.7–5.5)	<0.001
Rarely	196 (80.3)	48 (19.7)	244 (100)	1.4 (0.8–2.4)	0.254
Total	305 (75.5)	99 (24.5)	404 (100)		

Continued

Table 4 Continued

Frequency of legume intake	Metabolic syndrome		Total	COR (95% CI)	P value
	Yes	No			
Frequency of nut intake	TGs				
	Raised TGs	Normal TGs			
Often	17 (27.9)	44 (72.1)	61 (100)	1	
Sometimes	73 (73.7)	26 (26.3)	99 (100)	6.2 (3.3–11.5)	<0.001
Rarely	172 (70.5)	72 (29.5)	244 (100)	0.9 (0.5–1.4)	0.547
Total	262 (64.9)	142 (35.1)	404 (100)		
Frequency of nut intake	FBG				
	Raised FBG level	Normal FBG level			
Often	6 (9.8)	55 (90.2)	61 (100)	1	
Sometimes	19 (19.2)	80 (80.8)	99 (100)	3.2 (1.3–7.8)	0.011
Rarely	63 (25.8)	181 (74.2)	244 (100)	1.5 (0.8–2.6)	0.194
Total	88 (21.8)	316 (78.2)	404 (100)		

COR, crude OR.

Table 5 Relationship between consumption of processed/fast foods and metabolic syndrome-related components

Frequency of eating processed/ fast foods	Metabolic syndrome		Total	χ^2	Df	P value
	Yes	No				
Rarely	90 (70.3)	38 (29.7)	128 (100)	66.34	2	<0.001
Sometimes	120 (93.0)	9 (7.0)	129 (100)			
Always	142 (96.6)	5 (3.4)	147 (100)			
Total	352 (87.1)	52 (12.9)	404 (100)			
	Blood pressure (BP)			COR (95% CI)		P value
	Elevated BP	Normal BP				
Rarely	65 (49.2)	67 (50.8)	132 (100)	1		
Sometimes	79 (60.8)	51 (39.2)	130 (100)	6.288 (3.51–11.27)		<0.001
Always	122 (85.9)	20 (14.1)	142 (100)	3.938 (2.18–7.10)		<0.001
Total	266 (65.8)	138 (34.2)	404 (100)			
	High-density lipoprotein (HDL)			COR (95% CI)		P value
	Low HDL	Normal HDL				
Rarely	88 (66.7)	44 (33.3)	132 (100)	1		
Sometimes	99 (76.2)	31 (23.8)	130 (100)	2.458 (1.39–4.34)		0.002
Always	118 (83.1)	24 (16.9)	142 (100)	1.540 (0.85–2.79)		0.156
Total	305 (75.5)	99 (24.5)	404 (100)			
	Triglycerides (TGs)			COR (95% CI)		P value
	Raised TGs	Normal TGs				
Rarely	67 (50.8)	65 (49.2)	132 (100)	1		
Sometimes	92 (70.8)	38 (29.2)	130 (100)	2.562 (1.55–4.23)		<0.001
Always	103 (72.5)	39 (27.5)	142 (100)	1.091 (0.64–1.85)		0.747
Total	262 (64.9)	142 (35.1)	404 (100)			
	Fasting blood glucose (FBG)			COR (95% CI)		P value
	Raised FBG	Normal FBG level				
Rarely	13 (9.8)	119 (90.2)	132 (100)	1		
Sometimes	20 (15.4)	110 (84.6)	130 (100)	5.787 (2.98–11.25)		<0.001
Always	55 (38.7)	87 (61.3)	142 (100)	3.477 (1.94–6.24)		<0.001
Total	88 (21.8)	316 (78.2)	404 (100)			

COR, crude OR.

Table 6 Consumption of sugar and salt in relation to metabolic syndrome (MetS)-related components

MetS and its components	Sugar consumption status		Total	χ^2	Df	P value
	≤5 tsp per day	>5 tsp per day				
				6.917	1	0.009
MetS	N (%)	N (%)	N (%)			
Yes	123 (34.9)	229 (65.1)	352 (100)			
No	28 (53.8)	24 (46.2)	52 (100)			
Total	151 (37.4)	253 (62.6)	404(100)			
Blood pressure (BP)				0.016	1	0.9
Elevated BP	100 (37.6)	166 (62.4)	266 (100)			
Normal BP	51 (37)	87 (63)	138 (100)			
High-density lipoprotein (HDL)				1.428	1	0.232
Reduced HDL	109 (35.7)	196 (64.3)	305 (100)			
Normal HDL	42 (42.4)	57 (57.6)	99 (100)			
Triglycerides (TGs)				0.438	1	0.508
Raised TGs	101 (38.5)	161 (61.5)	262 (100)			
Normal TGs	50 (35.2)	92 (64.8)	142 (100)			
Fasting blood glucose (FBG)				25.099	1	<0.001
Raised FBG level	53 (60.2)	35 (39.8)	88 (100)			
Normal FBG level	98 (31)	218 (69)	316 (100)			
Total	151 (37.4)	253 (62.6)	404 (100)			
	Adds salt to meals at the table		Total			
	Yes	No				
MetS	N (%)	N (%)	N (%)	0.113	1	0.736
Yes	205 (58.2)	147 (41.8)	352 (100)			
No	29 (55.8)	23 (44.2)	52 (100)			
Total	234 (57.9)	170 (42.1)	404 (100)			
BP				21.718	1	<0.001
Elevated BP	176 (66.2)	90 (33.8)	266 (100)			
Normal BP	58 (42)	80 (58)	138 (100)			
HDL				0.006	1	0.936
Reduced HDL	177 (58)	128 (42)	305 (100)			
Normal HDL	57 (57.6)	42 (42.4)	99 (100)			
TGs				9.697	1	0.002
Raised TGs	137 (52.3)	125 (47.7)	262 (100)			
Normal TGs	97 (68.3)	45 (31.7)	142 (100)			
FBG				10.028	1	0.002
Raised FBG level	38 (43.2)	50 (56.8)	88 (100)			
Normal FBG level	196 (62)	120 (38)	316 (100)			
Total	234 (57.9)	170 (42.1)	404 (100)			

sometimes consumed legumes were six (95% CI: 3.27–12.52, $p < 0.001$) and four (95% CI: 1.24–10.83, $p = 0.022$) times, respectively, more likely to develop MetS compared with those who always consumed legumes. With regards to nut intake, respondents who rarely consumed nuts were seven (95% CI: 3.68–13.62, $p < 0.001$) and four (95% CI: 1.36–9.54, $p = 0.011$) times, respectively, more likely to develop MetS compared with those who always consumed nuts (table 8).

DISCUSSION

Our findings underscore the relationship between dietary patterns/practices and MetS in adults with

central obesity. In sum, we report that MetS is linked with frequent consumption of: large proportion of carbohydrates, proteins, processed/fast foods, large amounts of sugars, less than 50% portion of a plate filled as vegetables and/or fruits, and adding salt to food. However, adequate and/or frequent consumption of: fruits, vegetables, legumes and nuts were linked with protection against MetS. Additionally, those who observed more than a 2-hour interval between eating dinner and sleeping were less likely to have MetS. Although these findings show the importance of diet practices/patterns in the development and sustainability of MetS—a well-documented narrative, such link has also been locally reported with

Table 7 Relationship between time interval of taking dinner and sleeping relative to metabolic syndrome-related components

Time interval between taking dinner and sleep	Metabolic syndrome		Total	COR (95% CI)	P value
	Yes	No			
Less than 1 hour	97 (90.7)	10 (9.3)	107 (100)	1	
1–2 hours	201 (92.2)	17 (7.8)	218 (100)	0.2 (0.1–0.5)	<0.001
More than 2 hours	54 (68.4)	25 (31.6)	79 (100)	0.2 (0.1–0.4)	<0.001
Total	352 (87.1)	52 (12.9)	404 (100)		
Blood pressure (BP)					
	Elevated BP	Normal BP			
Less than 1 hour	69 (64.5)	38 (35.5)	107 (100)	1	
1–2 hours	157 (72)	61 (28)	218 (100)	0.6 (0.3–1.0)	0.059
More than 2 hours	40 (50.6)	39 (49.4)	79 (100)	0.4 (0.2–0.7)	0.001
Total	266 (65.8)	138 (34.2)	404 (100)		
High-density lipoprotein (HDL)					
	Reduced HDL	Normal HDL			
Less than 1 hour	92 (86)	15 (14)	107 (100)	1	
1–2 hours	164 (75.2)	54 (24.8)	218 (100)	0.3 (0.1–0.5)	<0.001
More than 2 hours	49 (62)	30 (38)	79 (100)	0.5 (0.3–0.9)	0.027
Total	305 (75.5)	99 (24.5)	404 (100)		
Triglycerides (TGs)					
	Raised TGs	Normal TGs			
Less than 1 hour	62 (57.9)	45 (42.1)	107 (100)	1	
1–2 hours	156 (71.6)	62 (28.4)	218 (100)	0.9 (0.5–1.6)	0.76
More than 2 hours	44 (55.7)	35 (44.3)	79 (100)	0.5 (0.3–0.9)	0.011
Total	262 (64.9)	142 (35.1)	404 (100)		
Fasting blood glucose (FBG)					
	Raised FBG level	Normal FBG level			
Less than 1 hour	21 (19.6)	86 (80.4)	107 (100)	1	
1–2 hours	50 (22.9)	168 (77.1)	218 (100)	1.1 (0.5–2.3)	0.752
More than 2 hours	17 (21.5)	62 (78.5)	79 (100)	0.9 (0.5–1.7)	0.796
Total	88 (21.8)	316 (78.2)	404 (100)		

COR, crude OR.

intake of refined carbohydrate, processed/fast foods, sugar-sweetened beverages, low fruits and vegetables^{20 22} as the main associated risks.

Consumption of large amounts of carbohydrates and proteins, as well as small proportion of vegetables and/or fruits, was linked to higher prevalence of MetS, elevated BP and raised FBG level. The aforementioned does not meet the DASH diet criteria. The DASH eating plan has been shown to be beneficial on MetS.^{9 31} However, excess consumption of carbohydrates³ and specifically animal-based protein foods³² increases the risks for MetS. Importantly, consumption of unhealthy diet characterised by a high-calorie content is known risk factor for obesity, a principal element for various metabolic-clinical abnormalities such as dyslipidaemia, high BP, insulin resistance features of MetS.²¹ The cardiometabolic protective effects of fruits and vegetables can be attributed to their richness in vitamins, minerals, phytochemicals, fibres, potassium, magnesium and antioxidants. Fruits and vegetables are rich in phytochemicals and flavonoids which have been reported to support cardioprotective properties.³³ Both

fruits and vegetables are also rich in soluble dietary fibres that may decrease the intestinal absorption for cholesterol and bile salts, thus controlling their levels.³⁴ Consumption of high-fibre diets such as fruits and vegetables slows absorption of foods in the gut, resulting in a regulated release of insulin from the pancreas, thus maintaining normal glucose level. Furthermore, vegetables and fruits are rich in potassium, an important cofactor for BP regulation. Mechanistically, when serum potassium level is low, sodium and water retention increases resulting in high BP.^{35 36}

We show that the prevalence of MetS and related components was inversely associated with legume consumption. The findings are consistent with reported beneficial effect of legumes on MetS,^{7 8} TGs and BP.³⁷ The cardiometabolic protective effect of dietary legumes is attributed to the high content of viscous soluble fibres which contributes to slow absorption of carbohydrates, cholesterol and bile salts in the intestine resulting in improved blood sugar control³⁸ and blood lipid levels.³⁴ Similarly, regular consumption of nuts is associated with lower risk of MetS

Table 8 Multivariable analysis for dietary risk factors of metabolic syndrome

Variable	AOR	95% CI		P value
		Lower	Upper	
Full/first model				
Proportion of plate filled with vegetables and/or fruits				
Less than half of a plate	2.15	0.63	7.29	0.221
Half and above of a plate	Ref			
Frequency of processed/fast food intake				
Always	3.192	1.246	6.903	0.004
Sometimes	2.132	0.775	5.648	0.109
Rarely	Ref			
Frequency of fruit intake				
Not daily	2.015	0.526	7.716	0.306
Daily	Ref			
Frequency of vegetable intake				
Not daily	1.048	0.293	3.744	0.942
Daily	Ref			
Frequency of legume intake				
Rarely	6.19	2.624	11.725	<0.001
Sometimes	3.702	1.096	10.248	0.039
Often	Ref			
Frequency of nut intake				
Rarely	6.667	3.78	12.811	<0.001
Sometimes	4.718	1.934	9.18	0.012
Always	Ref			
Sugar consumption status				
>5 tsp per day	1.505	0.426	5.315	0.525
≤5 tsp per day	Ref			
Time interval between taking dinner and sleep				
Less than 1 hour	4.21	0.819	21.644	0.085
1–2 hours	3.824	0.908	16.11	0.061
More than 2 hours	Ref			
Reduced/last model				
Frequency of processed/fast food intake				
Always	3.286	1.482	7.289	0.003
Sometimes	3.358	0.864	13.053	0.08
Rarely	Ref			
Frequency of legume intake				
Rarely	6.395	3.267	12.519	<0.001
Sometimes	4.28	1.235	10.832	0.022
Often	Ref			
Frequency of nut intake				
Rarely	7.081	3.68	13.622	<0.001
Sometimes	4.03	1.359	9.538	0.011
Often	Ref			

AOR, adjusted OR.

and related components. The beneficial effect of regular consumption of nuts on MetS,⁹ central obesity, type 2 diabetes, BP and TGs³⁹ is well established. Nuts may exert protective effect on MetS through several mechanisms. First, they are rich in both macronutrients and micronutrients including unsaturated fatty acids, fibre, non-sodium minerals, tocopherols and bioactive phytochemicals such as polyphenols and phytosterols.⁴⁰ These biomolecules have cardioprotective effect via improving inflammation, oxidative stress and endothelial function. The mechanisms can improve insulin secretion and sensitivity and thus reduce the risk of type 2 diabetes, dyslipidaemia, central obesity and hypertension.⁴¹ Furthermore, dietary fibres from nuts have cholesterol³⁸ and blood glucose³⁴ reducing effects. Nuts have magnesium that can reduce peripheral inflammation improving insulin resistance as well as stimulating production of vasodilators specifically nitric oxide and prostacyclins,⁴² hence controlling both blood glucose and BP.

As regards to frequent consumption of processed/fast foods, they were directly linked to MetS (elevated BP, low HDL-C, raised TGs and FBG levels). These findings are consistent with reports showing frequently consumption of processed/fast foods increases chances of having MetS.^{3,5} Processed/fast foods are high in refined carbohydrates, cholesterol, salt, processed sugars—MetS-friendly food but poor in whole grains, fruits and vegetables.⁴³ Additionally, excessive sugar consumption (more than five teaspoons in a day) is a risk factor for MetS. Intake of below 25 g (five teaspoons) per person is recommended by the WHO to prevent NCDs notably hypertension and diabetes.⁴⁴ Added sugars and/or sugar-sweetened beverages are linked with central obesity,^{44,45} dyslipidaemia,⁴⁶ insulin resistance,⁴⁷ type 2 diabetes,^{47,48} high BP⁴⁹ and MetS.^{45,50} Surprisingly, we found consumption of less sugar per day to be associated with raised FBG levels. This finding could have been attributed to the known patients with diabetes recruited into the study who were more likely to have been taking less or no sugar as per the recommendation compared with non-diabetics.

Adding salt to foods after the food has been cooked was shown to be associated with elevated BP. The relationship between salt consumption and high BP is well documented.^{32,51} Dietary guideline by WHO recommends daily salt intake of less than 5 g (one teaspoon) per person to help prevent high BP, reduce risk of heart disease and stroke in adults.⁵² Of particular interest in our study finding is that low-salt consumption was significantly associated with both raised TGs and FBG levels. Indeed, reducing or restriction salt intake has a beneficial effect on lowering BP. However, an unwanted side effect that we also show is consuming less salt has increased risk of elevated levels of blood cholesterol.⁵³ The mechanisms associated with low salt intake and hyperlipidaemia can be explained by the fact that limited sodium intake reduces body water content and in an attempt to revert the low plasma volume, epinephrine, renin and angiotensin increase. These hormones inhibit insulin action,

causing insulin resistance⁵⁴ and consequently high insulin level in the blood inhibits lipid metabolism and increases blood cholesterol.⁵⁵ With regards to the raised FBG level in relation to low salt intake, there is so far no explanation to this association. However, it could be due to mechanisms associated with the aforementioned hormones.⁵⁵

A finding of interest is the short time interval between taking dinner and going to bed and likelihood of MetS. This is related to the fact that quantity of food consumption is directly associated with shunting of blood into the mesenteric system resulting into early sleepiness. This finding is consistent with reports that eating too close to bedtime is a risk factor for obesity,^{56–58} dyslipidaemia,⁵⁸ MetS and hyperglycaemia,^{59,60} diabetes and cardiovascular morbidity.⁶¹ Indeed, eating an early dinner allows the body time to burn off those unwanted calories before going to sleep⁶² and thus reduces the risks of CVDs. Whether, the likelihood of sleepiness relative to the quantity and quality of food was not a subject of our investigation. However, reports show carbohydrate-rich food is associated with sleepiness due to their possibility of increasing plasma concentration of tryptophan—a precursor for serotonin and sleep-inducing agent.⁶³

Strengths and limitations of this study

This was the first study conducted among the informal settlements (slums) in Kenya that determined the association between dietary intake patterns and MetS. The relatively large sample size increases the possibility of replication and generalisation of the findings. The use of widely recognised and validated dietary questionnaires is another strength of the study. The findings reinforce the importance of dietary consideration in public health interventions addressing MetS and related cardiovascular problems. The study was limited by its cross-sectional design. The self-reported dietary patterns and practices may suffer from information bias. The current study was conducted in Nairobi, the capital city of Kenya, where consumption of processed and/or fast foods as well as sweetened beverages is common, thus, generalisability to the rural areas in the country may not be possible.

In conclusion, regular consumption of fruits, vegetables, legumes and nuts, as well as observing sometime after eating dinner before sleeping, was the dietary pattern significantly associated with reduced risk of MetS. However, consumption of a large quantity of carbohydrates, proteins, processed/fast foods and sugar is likely to predispose to MetS. The findings underscore the need to focus on specific dietary intake patterns including frequency, quantity, quality and variety for MetS prevention and management. The MetS-related interventions could be implemented during individual consultation, group and community health messaging sessions.

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Patient consent for publication Obtained.

Ethics approval Ethical approval to conduct this study was obtained from Kenyatta National Hospital-University of Nairobi Ethical Review Committee (KNH-UoN ERC) (approval number P430.07/2017). The institutional permission was granted by the administration of the St Mary's Mission Hospital. Consent was obtained from the study participants prior to data collection after an explanation on study aim and objectives. The participants were assured of confidentiality, privacy, anonymity and non-coercive nature of the study.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request. The dataset analysed during the current study is available from the corresponding author on a reasonable request.

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REFERENCES

- Alberti KGMM, Eckel RH, Grundy SM, *et al.* Harmonizing the metabolic syndrome: a joint interim statement of the International diabetes Federation Task force on epidemiology and prevention; National heart, lung, and blood Institute; American heart association; world heart Federation; international atherosclerosis Society; and international association for the study of obesity. *Circulation* 2009;120:1640–5.
- Saklayen MG. The global epidemic of the metabolic syndrome. *Curr Hypertens Rep* 2018;20:12.
- Suliga E, Kozielec D, Ciesla E, *et al.* Dietary patterns in relation to metabolic syndrome among adults in Poland: a cross-sectional study. *Nutrients* 2017;9:1366.
- Lind PM, Riserus U, Salihovic S, *et al.* An environmental wide association study (EWAS) approach to the metabolic syndrome. *Environ Int* 2013;55:1–8.
- Rodríguez-Monforte M, Sánchez E, Barrio F, *et al.* Metabolic syndrome and dietary patterns: a systematic review and meta-analysis of observational studies. *Eur J Nutr* 2017;56:925–47.
- Li X-T, Liao W, Yu H-J, *et al.* Combined effects of fruit and vegetables intake and physical activity on the risk of metabolic syndrome among Chinese adults. *PLoS One* 2017;12:e0188533.
- Hosseinpour-Niazi S, Mirmiran P, Mirzaei S, *et al.* Cereal, fruit and vegetable fibre intake and the risk of the metabolic syndrome: a prospective study in the Tehran lipid and glucose study. *J Hum Nutr Diet* 2015;28:236–45.
- Sala-Vila A, Estruch R, Ros E. New insights into the role of nutrition in CVD prevention. *Curr Cardiol Rep* 2015;17:26.
- Babio N, Toledo E, Estruch R, *et al.* Mediterranean diets and metabolic syndrome status in the PREDIMED randomized trial. *CMAJ* 2014;186:E649–57.
- Czekajło A, Róžańska D, Zatońska K, Zanska DR, *et al.* Association between dietary patterns and metabolic syndrome in the selected population of Polish adults—results of the pure Poland study. *Eur J Public Health* 2019;29:335–40.
- Kaur J. A comprehensive review on metabolic syndrome. *Cardiol Res Pract* 2014.
- Suzuki T, Hirata K, Elkind MSV, *et al.* Metabolic syndrome, endothelial dysfunction, and risk of cardiovascular events: the Northern Manhattan study (NOMAS). *Am Heart J* 2008;156:405–10.
- Ford ES, Li C, Sattar N, *et al.* Metabolic syndrome and incident diabetes: current state of the evidence. *Diabetes Care* 2008;31:1898–904.
- O'Neill S, O'Driscoll L. Metabolic syndrome: a closer look at the growing epidemic and its associated pathologies. *Obes Rev* 2015;16:1–12.
- Sabir AA, Jimoh A, Iwuala SO, *et al.* Metabolic syndrome in urban city of north-western Nigeria: prevalence and determinants. *Pan Afr Med J* 2016;23:19.
- Gyakobo M, Amoah AG, Martey-Marbell D-A, *et al.* Prevalence of the metabolic syndrome in a rural population in Ghana. *BMC Endocr Disord* 2012;12:25.
- Dandji MBS, Zambou FN, Dangang DSB, *et al.* Prevalence of metabolic syndrome in adult men of the Dschang health district in Western-Cameroon. *World J Nutr Health* 2018;6:1–10.
- H Maklady FA, Kamal HM, El-Eraky AZ, *et al.* Prevalence of metabolic syndrome among adults in Suez canal area. *Egyptian Heart J* 2014;66:15.
- Omuse G, Maina D, Hoffman M, *et al.* Metabolic syndrome and its predictors in an urban population in Kenya: a cross sectional study. *BMC Endocr Disord* 2017;17:37.
- Samuel K, Mirie W, Chege M, *et al.* Association of lifestyle modification and pharmacological adherence on blood pressure control among patients with hypertension at Kenyatta national Hospital, Kenya: a cross-sectional study. *BMJ Open* 2029;2:e023995.
- Popkin BM, Adair LS, Ng SW. Global nutrition transition and the pandemic of obesity in developing countries. *Nutr Rev* 2012;70:3–21.
- Hulzebosch A, van de Vijver S, Oti SO, *et al.* Profile of people with hypertension in Nairobi's slums: a descriptive study. *Global Health* 2015;11:26.
- Babio N, Bulló M, Basora J, *et al.* Adherence to the Mediterranean diet and risk of metabolic syndrome and its components. *Nutr Metab Cardiovasc Dis* 2009;19:563–70.
- DiNicolantonio JJ, Lucan SC, O'Keefe JH. The evidence for saturated fat and for sugar related to coronary heart disease. *Prog Cardiovasc Dis* 2016;58:464–72.
- Okube OT, Kimani ST, Mirie W. Gender differences in the pattern of socio-demographics relevant to metabolic syndrome among Kenyan adults with central obesity at a mission hospital in Nairobi, Kenya. *High Blood Press Cardiovasc Prev* 2020;27:61–82.
- Desgroppes A, Taupin S. Kibera: the biggest slum in Africa. *Les Cahiers de l'Afrique de l'Est* 2011;44:23–34.
- World Health Organization. *The WHO step-wise approach to non-communicable disease risk factor surveillance*, 2017.
- World Health Organization. *Western Pacific region, International association for the study of obesity, International obesity Task force: the Asia-Pacific perspective: redefining obesity and its treatment*. Sydney: Health Communications Australia, 2000.
- Chobanian AV, Bakris GL, Black HR, *et al.* The seventh report of the joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. *JAMA* 2003;289:2560–72.
- World Health Organization. *International diabetes Federation: definition and diagnosis of diabetes mellitus and intermediate hyperglycemia: report of a WHO/IDF consultation*, 2006.
- Saneei P, Fallahi E, Barak F, *et al.* Adherence to the DASH diet and prevalence of the metabolic syndrome among Iranian women. *Eur J Nutr* 2015;54:421–8.
- Cheng M, Wang H, Wang Z, *et al.* Relationship between dietary factors and the number of altered metabolic syndrome components in Chinese adults: a cross-sectional study using data from the China health and nutrition survey. *BMJ Open* 2017;7:e014911.
- Steemburgo T, Dall'Alba V, Almeida JC, *et al.* Intake of soluble fibers has a protective role for the presence of metabolic syndrome in patients with type 2 diabetes. *Eur J Clin Nutr* 2009;63:127–33.
- Visioli F. Nutritional support in the pharmacological treatment of metabolic syndrome. *Eur J Pharmacol* 2011;668:S43–9.
- Rheinschild E. Fruit and veggies rich in potassium may be key to lowering blood pressure, 2017. Available: <https://news.usc.edu/119637/how-to-lower-your-blood-pressure-eat-more-fruit-and-veggies>
- Savica V, Bellinghieri G, Kopple JD. The effect of nutrition on blood pressure. *Annu Rev Nutr* 2010;30:365–401.
- Bazzano LA, Thompson AM, Tees MT, *et al.* Non-soy legume consumption lowers cholesterol levels: a meta-analysis of randomized controlled trials. *Nutr Metab Cardiovasc Dis* 2011;21:94–103.
- Bouchenak M, Lamri-Senhadj M. Nutritional quality of legumes, and their role in cardiometabolic risk prevention: a review. *J Med Food* 2013;16:185–98.
- Blanco Mejia S, Kendall CWC, Viguioliou E, *et al.* Effect of tree nuts on metabolic syndrome criteria: a systematic review and meta-

- analysis of randomised controlled trials. *BMJ Open* 2014;4:e004660.
- 40 Ros E, Hu FB. Consumption of plant seeds and cardiovascular health: epidemiological and clinical trial evidence. *Circulation* 2013;128:553–65.
- 41 Ros E. Health benefits of nut consumption. *Nutrients* 2010;2:652–82.
- 42 Barbagallo M, Dominguez LJ, Galioto A, *et al.* Role of magnesium in insulin action, diabetes and cardio-metabolic syndrome X. *Mol Aspects Med* 2003;24:39–52.
- 43 Paniagua JA, Pérez-Martínez P, Gjelstad IMF, *et al.* A low-fat high-carbohydrate diet supplemented with long-chain n-3 PUFA reduces the risk of the metabolic syndrome. *Atherosclerosis* 2011;218:443–50.
- 44 World Health Organization. *Guideline: sugars intake for adult and children*. Geneva: World Health Organization, 2015.
- 45 Bray GA, Popkin BM. Dietary sugar and body weight: have we reached a crisis in the epidemic of obesity and diabetes?: health be damned! pour on the sugar. *Diabetes Care* 2014;37:950–6.
- 46 Welsh JA, Sharma A, Cunningham SA, *et al.* Consumption of added sugars and indicators of cardiovascular disease risk among US adolescents. *Circulation* 2011;123:249–57.
- 47 Stanhope KL. Sugar consumption, metabolic disease and obesity: the state of the controversy. *Crit Rev Clin Lab Sci* 2016;53:52–67.
- 48 DiNicolantonio JJ, O'Keefe JH, Lucan SC. Added fructose: a principal driver of type 2 diabetes mellitus and its consequences. *Mayo Clin Proc* 2015;90:372–81.
- 49 DiNicolantonio JJ, Lucan SC. The wrong white crystals: not salt but sugar as aetiological in hypertension and cardiometabolic disease. *Open Heart* 2014;1:e000167.
- 50 Denova-Gutiérrez E, Talavera JO, Huitrón-Bravo G, *et al.* Sweetened beverage consumption and increased risk of metabolic syndrome in Mexican adults. *Public Health Nutr* 2010;13:835–42.
- 51 Oh YS, Appel LJ, Galis ZS, *et al.* National heart, lung, and blood Institute Working Group report on salt in human health and sickness: building on the current scientific evidence. *Hypertension* 2016;68:281–8.
- 52 World Health Organization. *Guideline: sodium intake for adults and children*. Geneva: World Health Organization, 2012.
- 53 Graudal NA, Hubeck-Graudal T, Jurgens G. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride. *Cochrane Database Syst Rev* 2017;4:CD004022.
- 54 Graudal NA, Hubeck-Graudal T, Jurgens G. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride. *Cochrane Database Syst Rev* 2011;11:CD004022.
- 55 Soleimani M. Insulin resistance and hypertension: new insights. *Kidney Int* 2015;87:497–9.
- 56 Bechtold DA, Loudon ASI. Hypothalamic clocks and rhythms in feeding behaviour. *Trends Neurosci* 2013;36:74–82.
- 57 Drapeau V, Gallant AR. Homeostatic and circadian control of food intake: clinical strategies to prevent overconsumption. *Curr Obes Rep* 2013;2:93–103.
- 58 Yoshida J, Eguchi E, Nagaoka K, *et al.* Association of night eating habits with metabolic syndrome and its components: a longitudinal study. *BMC Public Health* 2018;18:1366.
- 59 Soga Y, Shirai C, Ijichi A. [Association between daily lifestyle and the risk of metabolic syndrome among young adults in Japan. An analysis of Kobe city young adult health examination data]. *Nihon Koshu Eisei Zasshi* 2013;60:98–106.
- 60 Nakajima K, Suwa K. Association of hyperglycemia in a general Japanese population with late-night-dinner eating alone, but not breakfast skipping alone. *J Diabetes Metab Disord* 2015;14:16.
- 61 Akerstedt T, Wright KP. Sleep loss and fatigue in shift work and shift work disorder. *Sleep Med Clin* 2009;4:257–71.
- 62 Baron KG, Reid KJ, Kern AS, *et al.* Role of sleep timing in caloric intake and BMI. *Obesity* 2011;19:1374–81.
- 63 Afaghi A, O'Connor H, Chow CM. High-glycemic-index carbohydrate meals shorten sleep onset. *Am J Clin Nutr* 2007;85:426–30.