

Refined grain consumption and the metabolic syndrome in urban Asian Indians (Chennai Urban Rural Epidemiology Study 57)

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Abstract

The objective of the study was to evaluate the association of refined grains consumption with insulin resistance and the metabolic syndrome in an urban south Indian population. The study population comprised 2042 individuals aged ≥ 20 years randomly selected from the Chennai Urban Rural Epidemiology Study (CURES), a cross-sectional study on a representative population of Chennai, southern India. The *metabolic syndrome* was defined according to modified Adult Treatment Panel III guidelines; and insulin resistance, by the homeostasis assessment model. The mean refined grain intake was 333 g/d (46.9% of total calories) in this population. After adjustment for age, sex, body mass index, metabolic equivalent, total energy intake, and other dietary factors, higher refined grain intake was significantly associated with higher waist circumference (8% higher for the highest vs the lowest quartile, P for trend $< .0001$), systolic blood pressure (2.9%, P for trend $< .0001$), diastolic blood pressure (1.7%, P for trend = .03), fasting blood glucose (7.9%, P for trend = .007), serum triglyceride (36.5%, P for trend $< .0001$), low high-density lipoprotein cholesterol (-10.1% , P for trend $< .0001$), and insulin resistance (13.6%, $P < .001$). Compared with participants in the bottom quartile, participants who were in the highest quartile of refined grain intake were significantly more likely to have the metabolic syndrome (odds ratio, 7.83; 95% confidence interval, 4.72–12.99). Higher intake of refined grains was associated with insulin resistance and the metabolic syndrome in this population of Asian Indians who habitually consume high-carbohydrate diets.

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1. Introduction

The term *metabolic syndrome* (MS) refers to the clustering of metabolic abnormalities including glucose intolerance, central obesity, dyslipidemia, and hypertension [1]. Individuals with MS are at increased risk of cardiovascular diseases (CVDs) [2] and have a 5-fold excess risk of developing type 2 diabetes mellitus [3]. Recent studies have shown that the prevalence of MS is higher among Asian Indians despite relatively lower levels of generalized obesity as measured by body mass index (BMI) [4]. The term *Asian*

Indian phenotype is used to describe the increased susceptibility of Asian Indians to diabetes and premature coronary artery diseases due to the increased insulin resistance and MS among this ethnic group [5,6]. Although genetic causes [7] and sedentary lifestyle [8] have been shown to contribute to the so-called Asian Indian phenotype, very little is known about the role of dietary factors.

Earlier studies from India have focused on the role of dietary fats in the etiology of chronic diseases [9,10]. The role of dietary carbohydrates from staple cereals is less well studied. As cereals continue to be the main staple [11] and provide the bulk (60%–70%) of total energy intake in Asian Indian diets, data on the health impact of cereals are of great significance. Cereal-based diets consumed in the past were rich not only in fiber but also in other micronutrients and have been associated with a lower risk of CVD and type 2 diabetes mellitus [12]. However, because of changing food processing technology and the modern milling process, the

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grains available today are more refined. Refining destroys the structure of the grain kernel and removes dietary fiber and other essential micronutrients in grains [13]. Intake of many refined grains including commonly used types of white rice can also induce high glycemic responses. Long-term consumption of refined grains has been associated with components of the MS and a higher risk of type 2 diabetes mellitus and CVD in some previous studies [14,15].

Although there are many studies from the West that have evaluated the role of grains intake in the development of metabolic risk factors [16–20], to our knowledge, there are none from an Asian Indian population that habitually consumes high-carbohydrate diets with rice as the staple. In this study, we examined the association between refined grains intake and several metabolic risk factors including waist circumference, blood pressure, fasting serum triglyceride, fasting plasma glucose, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), insulin resistance, and the MS in an urban south Indian population.

2. Material and methods

2.1. Sample selection

Participants were recruited from the Chennai Urban Rural Epidemiology Study (CURES) conducted on a representative population of Chennai city (formerly Madras) in southern India, with a population of about 5 million people. The methodology of the study has been published elsewhere [21], and our Web site <http://www.drmohansdiabetes.com/mdrf/CURES.pdf> provides details of the sampling frame. Briefly, Chennai is divided into 155 corporation wards, representing a socioeconomically diverse group. In phase I of CURES, 26 001 adults (aged ≥ 20 years) from 46 corporation wards were screened for diabetes by fasting capillary blood glucose using systematic random sampling technique. Phase 2 of CURES deals with studies on prevalence of microvascular and macrovascular complications of diabetes.

In phase 3 of CURES, every 10th subject recruited in phase 1 ($n = 2600$) was invited to our center for detailed anthropometric measurements and biochemical tests. Of these, 2220 participated in the dietary assessment study, of whom participants with self-reported history of diabetes or CVD and those with unrealistic reported energy intakes (<500 or >4000 kcal/d) were excluded. Thus, a total of 2042 participants were included for the present analysis.

2.2. Dietary assessment

Dietary intakes were assessed using a previously validated and published interviewer-administered meal-based semiquantitative food frequency questionnaire (FFQ) containing 222 food items to estimate the usual food intake over the past year [22]. Interviews were conducted by nutritionists who were well trained in the methodology before any field work commenced.

Individuals were asked to estimate the usual frequency (number of times per day/week/month/year or never) and their usual serving size of the various food items in the FFQ. Common household measures such as household cups, bowls, ladles, spoons, wedges, and circles of different diameters and a visual atlas of different sizes of fruits (small, medium, large) were shown. Besides these questions, the participants were asked to specify the brand and type of cereals usually eaten. Refined grains include foods in which the bran and the germ layer are removed, with loss of dietary fiber, vitamins, and minerals, leaving the starchy endosperm; and these included mainly polished white rice, vermicelli, semolina, and white flour (refined breakfast cereals, cookies, biscuits, white bread, pastries). To avoid confounding by body size, physical activity, and metabolic efficiency and to reduce extraneous variation, refined grains intake was adjusted for total energy intake using the residual method [23].

Detailed description of this FFQ and the data on reproducibility and validity have been published elsewhere [22]. Validity of the FFQ has been documented by comparisons with six 24-hour recalls collected at 2-month intervals for a period of 1 year covering all seasons and included weekdays and weekends to capture variability. The energy-adjusted deattenuated Pearson correlation coefficient between the FFQ estimate and the average of six 24-hour recalls was 0.70 for refined cereals, 0.65 for pulses and legumes, 0.60 for roots and tubers, 0.69 for meat and poultry, 0.58 for fish and seafoods, 0.61 for dairy products, 0.71 for sugars, 0.28 for fruits and vegetables, and 0.61 for nuts and oil seeds.

2.3. Anthropometric measurements

Anthropometric measurements including weight, height, and waist measurements were measured by the trained research assistant using standardized techniques as described earlier [21]. The BMI was calculated using the formula weight (in kilograms)/height (in square meters). Blood pressure was recorded in the sitting position in the right arm to the nearest 2 mm Hg with a mercury sphygmomanometer (Diamond Deluxe BP apparatus, Pune, India). Two readings were taken 5 minutes apart, and the mean of the 2 was taken as the blood pressure. Demographic data, medical history, medications, family history of diabetes, smoking and alcohol consumption were also obtained. Details on physical activity were assessed using a previously validated physical activity questionnaire [7]. The protocol for the study was approved by the Institutional Ethics Committee of the Madras Diabetes Research Foundation, and informed consent was obtained from all study participants.

2.4. Laboratory measurements

A fasting blood sample was collected after an overnight fast of at least 10 hours for biochemical investigations, and serum was separated and stored at -70°C until the assays were performed. Fasting plasma glucose (glucose oxidase-

peroxidase method), serum cholesterol (cholesterol oxidase-peroxidase-aminopyrine), serum triglyceride (glycerol phosphate oxidase-peroxidase-aminopyrine method), and HDL-C (direct method polyethylene glycol-pretreated enzymes) were measured using the Hitachi 912 autoanalyzer (Hitachi, Mannheim, Germany). Low-density lipoprotein cholesterol level was calculated by using the Friedewald formula, as follows: LDL-C = total cholesterol – (HDL + triglyceride/5) (for subjects with triglyceride level <400 mg/dL). The intra- and interassay coefficients of variation for biochemical assays ranged from 3.1% to 7.6%. Serum insulin concentration was estimated using enzyme-linked immunoassay (Dako, Glostrup, Denmark). The intra- and the interassay coefficients of variation of insulin assay were 5.7% and 8.9%, respectively; and the lower detection limit was 0.5 μ IU/mL.

2.5. Ascertainment of outcome measures

Hypercholesterolemia (serum cholesterol ≥ 5.2 mmol/L [≥ 200 mg/dL]), hypertriglyceridemia (serum triglycerides ≥ 1.7 mmol/L [≥ 150 mg/dL]), low HDL-C (male participants, HDL-C <1.04 mmol/L [<40 mg/dL]; female participants, HDL cholesterol <1.3 mmol/L [<50 mg/dL]), *abnormal glucose metabolism* (as defined by a fasting glucose ≥ 6.1 mmol/L [≥ 110 mg/dL]), and high blood pressure (systolic blood pressure ≥ 130 mm Hg or diastolic

blood pressure ≥ 85 mm Hg) were diagnosed based on the Adult Treatment Panel III guidelines [24]; and *abdominal obesity* (defined as waist circumference ≥ 90 cm for male participants and ≥ 80 cm for female participants), according to modified Asia Pacific World Health Organization guidelines [25]. Metabolic syndrome was diagnosed based on modified Adult Treatment Panel III guidelines if any 3 of the above abnormalities were present. Insulin resistance was calculated using the homeostasis model assessment (HOMA-IR) using the following formula: fasting insulin (in micro-international units per milliliter) \times fasting plasma glucose (in millimoles per liter)/22.5 [26].

2.6. Statistical analysis

Statistical analyses were performed with SAS software (version 9.1; SAS Institute, Cary, NC). In separate models, first-order interaction between sex and refined grain intake was entered to determine whether association was similar between men and women. There was no significant interaction by sex on the association of refined grains and individual metabolic risk factors, and we therefore present results for men and women combined. Mean values of MS risk factors were examined across quartiles of refined grain intake. Values were expressed as the mean \pm SD or percentages. One-way analysis of variance (for continuous

Table 1

Baseline characteristics by quartiles of refined grain intake in 2042 participants of the CURES study

Characteristics	Quartiles of energy-adjusted refined grains intake ^a				P value
	1 (low)	2	3	4 (high)	
n	510	511	510	511	
Median refined grain intake (g/d)	218.1	298.9	364.9	448.8	–
Mean age (y)	40.7 \pm 14.4	38.9 \pm 12.0	39.4 \pm 12.1	40.3 \pm 11.3	.94
Men (%)	49.0	38.0	43.1	42.2	.005
Weight (kg)	57.7 \pm 12.2	58.9 \pm 12.5	61.7 \pm 12.9	63.1 \pm 12.1	.001
BMI, kg/m ²	22.8 \pm 4.4	23.0 \pm 4.3	24.5 \pm 4.7	25.2 \pm 4.4	<.0001
Current smoker, %	18	14.7	16.6	17.1	.543
Alcohol consumers, %	33.9	33.2	37.1	44.1	.001
Physical inactivity, %	68	71.9	72.2	74.6	.03
<i>Dietary variables (daily intake)</i>					
Total energy intake (kcal)	2519 \pm 611	2307 \pm 554	2548 \pm 513	2567 \pm 574	<.0001
Protein (energy %)	12.5 \pm 1.8	12.0 \pm 1.5	11.7 \pm 1.5	11.5 \pm 1.6	<.0001
Total fat (energy %)	27.1 \pm 4.9	24.5 \pm 3.3	22.4 \pm 3.4	21.1 \pm 5.4	<.0001
Carbohydrates (energy %)	59.5 \pm 6.7	63.2 \pm 8.7	65.5 \pm 4.7	67.1 \pm 6.7	<.0001
Dietary fiber g/1000 kcal	14.8 \pm 4.7	13.7 \pm 3.4	13.3 \pm 3.27	12.3 \pm 3.8	<.0001
Pulses and legumes (g)	57.9 \pm 24.5	52.1 \pm 18.2	51.6 \pm 17.6	48.2 \pm 18.4	<.0001
Meat and poultry (g)	51.7 \pm 40.0	45.2 \pm 35.9	51.4 \pm 45.6	53.1 \pm 50.3	.267
Dairy products (g)	488.4 \pm 275.0	373.7 \pm 197.1	382.6 \pm 225.4	335.6 \pm 184.2	<.0001
Visible fats and oils (g)	37.5 \pm 13.1	33.2 \pm 11.71	33.3 \pm 11.5	30.9 \pm 10.3	.001
Sugars (g)	30.1 \pm 24.6	22.6 \pm 17.8	21.9 \pm 16.7	17.9 \pm 14.5	<.0001
Fruits and vegetables (g)	281.1 \pm 136.9	234.9 \pm 105.1	228.4 \pm 107.7	211.1 \pm 100.47	<.0001
Fish and seafoods (g)	27.9 \pm 27.5	21.5 \pm 18.2	25.7 \pm 24.4	23.8 \pm 22.3	.0013
Roots (g)	31.5 \pm 28.0	30.4 \pm 29.5	29.9 \pm 27.6	32.3 \pm 32.4	.6844
Tubers (g)	144.0 \pm 55.4	132.9 \pm 49.1	139.6 \pm 57.5	136.4 \pm 51.1	.019
Nuts and oil seeds (g)	25.7 \pm 18.0	21.9 \pm 12.6	22.2 \pm 12.1	22.0 \pm 12.7	.001

Refined grain includes white rice, semolina, vermicelli, and white flour. Mean \pm SD (all such variables). *Physical inactivity* was defined as no exercise and being sedentary (watching television, slow walking).

^a Adjusted for total energy intake using the residual method.

variables) and the χ^2 test (for proportions) were used to test differences across quartiles of refined grain intakes.

To evaluate the relationship of refined grain intake with HOMA-IR and components of the MS, we used generalized linear model with least square means with adjustment for age (5-year category), sex (male/female), smoking (current, past, and never smokers; smokers—smoked at least 1 cigarette per day for >6 months), alcohol (current, past, and never consumers; consumers—having ever consumed spirits, wine, or beer for >6 months), BMI (quintiles), 24-hour metabolic equivalent (METs), total energy (in kilocalories), legumes (in grams per day), visible fats and oils (in grams per day), dairy products (in grams per day), sugars (in grams per day), fruits and vegetables (in grams per day), tubers (in grams per day), and nuts and oil seeds (in grams per day). Relative differences in adjusted mean variables between refined grains in quartile 1 and 4 were calculated. We also examined the association between the refined grain con-

sumption and the MS using logistic regression analysis and calculated the odds ratio and their 95% confidence interval (CI), with individuals in the lowest quartile category of refined grains as the reference category. To assess trends across quartile categories, we assigned median intake of each quartile category to individuals with intakes in the category and then included this quartile median variable as continuous factor in regression models. The *P* for trend was the resulting *P* value for the associated logistic regression coefficient. All tests of significance were 2-tailed, and *P* values less than .05 were considered statistically significant.

3. Results

Baseline characteristics of the study population by quartiles of refined grains are shown in Table 1. The study included 2042 participants, with an average age of 39.9 (SD,

Table 2
Multivariate adjusted mean (95% CI) HOMA-IR and metabolic risk factors by quartiles of refined cereal intake in 2042 participants of the CURES study

Variables	Quartile of refined grain intake				<i>P</i> trend ^a	% Difference between Q1 and Q4
	1 (low)	2	3	4 (high)		
n	510	511	510	511	—	
Median refined-grain intake (g/d)	218.1	298.9	364.9	448.8		
HOMA-IR, μ IU/mL						
Unadjusted	1.9 (1.7–2.0)	1.9 (1.7–2.0)	2.2 (2.1–2.4)	2.5 (2.3–2.7)	.001	24.0
Multivariable ^b	1.9 (1.8–2.2)	1.9 (1.8–2.2)	2.2 (1.9–2.3)	2.2 (1.9–2.4)	.001	13.6
Weight, kg						
Unadjusted	57.7 (56.7–58.8)	58.9 (57.6–59.7)	61.7 (60.6–62.8)	63.1 (62.0–64.2)	.001	9.4
Multivariable ^b	56.1 (55.0–57.2)	58.6 (57.5–59.7)	61.2 (60.1–62.3)	62.9 (61.6–64.1)	.001	12.1
Waist circumference, cm						
Unadjusted	80.9 (79.9–81.9)	81.8 (80.7–82.8)	86.0 (85.0–87.1)	87.3 (86.3–88.3)	.001	7.3
Multivariable ^b	80.5 (79.4–81.6)	82.5 (81.5–83.6)	85.8 (84.7–86.9)	87.5 (86.3–88.8)	.001	8.0
Systolic BP, mm Hg						
Unadjusted	116.3 (114.6–117.9)	117.5 (115.9–119.1)	120.3 (118.6–121.9)	122.9 (121.3–124.6)	.001	5.4
Multivariable ^b	117.1 (115.2–118.9)	118.2 (116.5–119.9)	118.8 (117.1–120.6)	120.6 (118.5–122.6)	.001	2.9
Diastolic BP, mm Hg						
Unadjusted	72.6 (71.6–73.5)	72.9 (71.9–73.9)	74.3 (73.3–75.3)	75.6 (74.6–76.6)	.001	3.9
Multivariable ^b	73.0 (71.9–74.2)	73.3 (72.2–74.3)	74.1 (72.9–75.2)	74.3 (73.0–75.6)	.029	1.7
HDL-C, mg/dL						
Unadjusted	44.3 (43.4–45.2)	43.4 (42.5–44.3)	41.9 (41.0–42.8)	40.6 (39.7–41.5)	.001	–9.1
Multivariable ^b	44.8 (43.8–45.8)	42.8 (41.8–43.7)	42.2 (41.2–43.1)	40.7 (39.6–41.9)	.001	–10.1
Triglyceride, mg/dL						
Unadjusted	101.7 (94.8–108.7)	119.9 (113.0–126.9)	129.4 (122.5–136.4)	149.7 (142.7–156.7)	.001	32.1
Multivariable ^b	96.8 (88.4–104.6)	122.8 (115.1–130.4)	130.9 (122.9–138.8)	152.4 (143.3–161.5)	.001	36.5
LDL-C, mg/dL						
Unadjusted	110.3 (107.5–113.2)	109.6 (106.8–112.5)	110.5 (107.6–113.3)	112.9 (110.1–115.8)	.947	2.4
Multivariable ^b	112.1 (108.6–115.7)	111.2 (107.9–114.6)	110.6 (107.4–114.3)	114.6 (110.7–118.6)	.137	2.2
Fasting blood glucose, mg/dL						
Unadjusted	88.5 (86.2–90.8)	92.8 (90.5–95.1)	92.7 (90.4–95.0)	98.2 (95.8–100.5)	.001	9.9
Multivariable ^b	87.9 (84.9–90.8)	92.9 (90.2–95.6)	90.8 (87.9–93.7)	95.4 (92.2–98.7)	.007	7.9

Numbers in parentheses are 95% CIs. BP indicates blood pressure.

^a *P* for trend calculated by using the median of each category of intake as a continuous variable in the regression model.

^b Adjusted for age (5-year category), sex (male/female), smoking (current, past and never smokers; smokers—smoked at least 1 cigarette per day for >6 months), alcohol (current, past, and never consumers; consumers—having ever consumed spirits, wine, or beer for >6 months), BMI (quintiles), METs, total energy (in kilocalories), legumes (in grams per day), visible fats and oils (in grams per day), dairy products (in grams per day), sugars (in grams per day), fruits and vegetables (in grams per day), tubers (in grams per day), fish and seafoods (in grams per day), and nuts and oil seeds (in grams per day) (Bonferroni correction).

12.6) years and a median intake of refined grain of 333 g/d. Compared with participants in the lowest quartile, those in the highest quartile of refined grain intake had a higher BMI, were more likely to consume alcohol, and were less likely to engage in physical activity. There was no significant difference in the age and smoking behavior across quartiles of refined grain intake. Those who tended to eat more refined grains also tended to eat less protein and dietary fat. Similarly, participants with higher intake of refined grains consumed less legumes, fruits and vegetables, tubers, visible fats and oils, sugars, dairy products, fish and seafoods, and nuts and oil seeds; but no significant difference was observed in the intake of meat and poultry and roots. Almost half (46.9%) of the daily calories in our population was derived from refined grains, which include polished rice, refined wheat flour, and semolina, of which rice was a major contributor (75.8%; mean intake, 253.4 g/d).

Table 2 presents the various metabolic abnormalities according to quartiles of refined grain intake. In the unadjusted analyses, subjects in the highest quartile of refined grain intake had significantly higher HOMA-IR values (+13.6%) than those in the lowest quartile. This association was weakened by adjustment for other major risk factors including BMI, age, sex, smoking, alcohol, METS, total energy intake, and other dietary factors but remained substantial and significant (mean HOMA-IR; first Q, 1.9 [1.8–2.2] vs fourth Q, 2.2 [1.9–2.4]; P for trend < .001). The associations did not substantially change after further adjustment for dietary fiber.

After multivariate adjustment for potential confounders, higher refined grain intake was also significantly associated

with higher body weight (+12.1% for highest vs lowest quartile), waist circumference (+8%), serum fasting plasma glucose (+7.9%), triglyceride levels (+36.5%), systolic blood pressure (+2.9%), and diastolic blood pressure (+1.7%) and lower HDL-C levels (–10.1%) (Table 2). However, there was no association with LDL-C levels. Results were similar whether or not dietary fiber was included in the model. The observed associations of refined grain intake with HOMA-IR, fasting glucose, blood pressure, triglycerides, and HDL-C were not materially different when waist circumference, instead of BMI, was included in the multivariable models.

The prevalence of the MS was 36.5% (P < .001) in the highest as compared with the lowest quartile (13.9%) of refined grain intake. There was a significant trend for a higher prevalence of the MS with higher intakes of refined grains, even after controlling for possible confounders. The multivariate adjusted odds ratio and CI for MS was 7.83 (95% CI, 4.72, 12.99; P for trend < .0001) for the highest as compared with the lowest quartile of refined grain consumption (Fig. 1).

4. Discussion

We observed a significant association between higher intake of refined grains and greater insulin resistance and prevalence of the MS independent of other lifestyle confounders. It was also interesting to note that MS and BMI were highest in the group that ate the least amount of sugar and fat, indicating that low sugar and fat intake is not always correlated with improved health conditions. Few studies have assessed the role of refined grain intake in relation to metabolic abnormalities such as insulin resistance and the MS [16–20]. Diets high in refined carbohydrates intake may lead to hypertension, dyslipidemia, and insulin resistance [27].

In the present study, the prevalence of the MS was higher in those with highest refined grain; and this is consistent with the other cross-sectional studies conducted among US and Iranian population that showed higher prevalence of MS in those with higher intakes of refined grains [16–18]. In contrast, the Framingham Cohort Study and the Bogalusa Heart Study [19,20] reported no such association. The average consumption of refined grains was much higher in our population (333 g/d) as compared with consumption reported for Western (117 g/d) [16] or Iranian (201 g/d) populations [17]. Consumption of refined grains, in the setting of lower intake of whole grains, fruits and vegetables, legumes, and dairy products and increased sugar intake, has been shown to be associated with metabolic risk factors in several prospective studies [9,15,28,29]. However, in the present study, adjusting for other food groups did not explain the association between refined grains and metabolic risk factors. The protective effect of whole grain intake in this study could not be

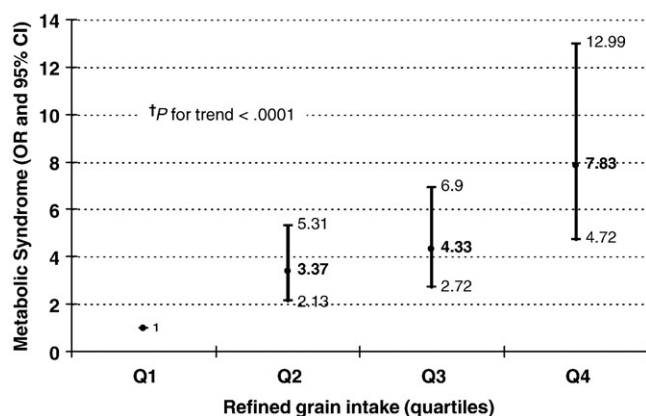


Fig. 1. Multivariate adjusted odds ratio and 95% CI of MS according to quartiles of refined grain among CURES participants. *Odds ratios were adjusted for age (5-year category), sex (male/female), smoking (current, past, and never smokers), alcohol (current, past, and never consumers), BMI (quintiles), METS, total energy (in kilocalories), legumes (in grams per day), visible fats and oils (in grams per day), dairy products (in grams per day), sugars (in grams per day), fruits and vegetables (in grams per day), tubers (in grams per day), fish and seafoods (in grams per day), and nuts and oil seeds (in grams per day). † P for trend calculated by using the median of each category of intake as a continuous variable in the regression model. The reference category was the lowest quartile.

evaluated because only 20% of study participants consumed whole grains, with a mean intake of 53 g/d.

Diets with high intake of carbohydrates induce hypertriglyceridemia mainly by enhancing hepatic synthesis of very low-density lipoprotein [30] and possibly by reducing lipoprotein lipase activity [31]. We observed that high-carbohydrate south Indian diets predominantly derived from refined grains (64% of total carbohydrates) were associated with higher triglycerides and lower HDL-C; and this probably contributes to the so-called Asian Indian phenotype characterized by higher glucose intolerance, dyslipidemia, central obesity, and MS [5]. We have also shown in our earlier study using dual-energy x-ray absorptiometry and computed tomographic scan that central obesity is common in Asian Indians and is predominantly due to visceral fat [32]. Randomized and metabolic studies have shown that white rice and refined wheat flour products increase fasting triglyceride levels and reduce HDL-C concentrations [33,34]. Our current results are in agreement with our previous finding that a higher dietary glycemic load intake was associated with lower HDL-C levels [35].

Consistent with another cross-sectional study conducted among older adults [16], we also found that higher refined grains intake was associated with higher fasting glucose concentration. However, this association was not observed in the Framingham Cohort Study [20]. The Dietary Approaches to Stop Hypertension diet that emphasized whole grain consumption more than refined grains and sugar-sweetened beverages among other dietary changes showed a significant decrease in both systolic and diastolic blood pressure [36]. In the present study, higher refined grain intake was significantly associated with systolic, but not diastolic, blood pressure.

Since 1980, the percentage of carbohydrate intake in Indian diets has remained relatively constant. However, the prevalence of diabetes and CVD has increased from 8% (1980) to 16% (2006) [37] in this urban population. This probably reflects changes in lifestyle including nutrition-related changes. Highly refined foods such as refined cereals and sugar-based products now comprise 70% of the all the products available in the Indian market. This could possibly be one of the factors contributing to the high prevalence of MS, diabetes, and CVD in this population. However, because refined grains were also associated with less protein, dietary fat, and sugar intake, it is more prudent to advise people on adopting an overall healthy diet approach by encouraging increased protein and dietary fiber intake and healthier fat options in addition to increasing lower glycemic carbohydrates and fruits and vegetable intake rather than focusing on decreasing refined grain intake alone.

This study had several limitations. First, being a cross-sectional study, it does not allow us to infer causation or definitely establish the direction of effects. Second, we adjusted for various potential confounders; but as in any observational study, residual confounding by unknown or imperfectly measured factors cannot be excluded. Third,

some measurements error is inevitable in the assessment of dietary intakes. However, our validation study indicated that the assessment of refined grains using a detailed interviewer-administered FFQ was reasonably accurate. Remaining measurement error would be expected to weaken rather than strengthen the observed association.

This study also has several strengths including the relatively large sample size, the detailed information on diet, and the large variation in refined grain intakes. We included a representative population of Chennai; and hence, the results can be extrapolated to urban India. The present data are particularly important given the paucity of available information on refined grains and the MS in populations with high white rice intakes.

In conclusion, refined grain intake was directly associated with insulin resistance and the MS in this south Indian population. This study further warrants an intervention trial replacing refined grains with whole grains to reduce the development of the MS in south Asian populations with high white rice intakes.

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