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Rice and noodle consumption is associated with insulin resistance and hyperglycaemia in an Asian population

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Abstract

High consumption of refined grains, particularly white rice, has been reported to be associated with a higher risk of type 2 diabetes. Therefore, in the present study, we evaluated the association between rice and noodle consumption and markers of glucose homeostasis, inflammation and dyslipidaemia in an Asian population. We carried out a population-based cross-sectional study in 2728 Singaporean Chinese men and women aged between 24 and 92 years. Rice and noodle intake was assessed using a validated FFQ and studied in relation to glycaemic (fasting glucose, glycated Hb, homeostasis model assessment (HOMA) index for insulin resistance (HOMA-IR) and HOMA index for β-cell function (HOMA-β)), inflammatory (plasma adiponectin and C-reactive protein (CRP)) and lipid (fasting TAG and HDL-cholesterol (HDL-C)) markers. We used multiple linear regression analyses with adjustment for total energy intake and socio-demographic, anthropometric (BMI and waist:hip ratio) and lifestyle factors. Higher rice consumption was found to be associated with higher fasting glucose concentrations (0·81 % higher values per portion increment; 95 % CI 0·09, 1·54) and HOMA-IR (4·62 %; 95 % CI 1·29, 8·07). Higher noodle consumption was also found to be significantly associated with higher fasting glucose concentrations (1·67 %; 95 % CI 0·44, 2·92), HOMA-IR (6·17 %; 95 % CI 0·49, 12·16) and fasting TAG concentrations (9·17 %; 95 % CI 3·44, 15·22). No significant association was observed between rice and noodle consumption and adiponectin, CRP and HDL-C concentrations or HOMA-β in the fully adjusted model. These results suggest that high consumption of rice and noodles may contribute to hyperglycaemia through greater insulin resistance and that this relationship is independent of adiposity and systemic inflammation.

Key words: Carbohydrates: Glucose metabolism: Inflammation: Cholesterol: Adiponectin: C-reactive protein

More than half of all individuals with diabetes mellitus in the world reside in Asia, and a large increase in the number of diabetes cases has been predicted for this continent[1]. Grains in the form of rice and noodles are the primary carbohydrate source for most Asian populations, with rice providing as much as 60 % of total energy in Southeast Asia[2,3]. Rice and other grains are predominantly consumed as refined grains by most Asian populations[4,5]. Accounts as to when polished rice started being consumed in Asia are limited; in Japan, consumption of polished white rice began at the end of the seventeenth century and was initially limited to the urban upper class due to its high price[6]. Currently, mechanised steel roller mills and automated sifting devices are being used to efficiently refine grains, resulting in a huge loss of vitamins, minerals, essential fatty acids and phytochemicals[4,7,8].

Recently published data suggest that there is an association between high white rice consumption and a higher risk of type 2 diabetes in both Western[9] and Asian[10–12] populations. However, the biological mechanisms that may underlie this association are unclear. In addition, data on the association between consumption of noodles, another major source of refined grains in Asia, and risk of type 2 diabetes

Abbreviations: CRP, C-reactive protein; FPG, fasting plasma glucose; HDL-C, HDL-cholesterol; HOMA, homeostasis model assessment; HOMA-β, homeostasis model assessment of β-cell function; HOMA-IR, homeostasis model assessment of insulin resistance; SP2, Singapore Prospective Study Program-2.

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are sparse[11]. High consumption of white rice and noodles may lead to high dietary glycaemic index[13] and glycaemic load, which have been suggested to increase the risk of type 2 diabetes through excess postprandial variations in blood glucose and insulin concentrations[14–19]. Such variations in insulin concentrations may increase circulating NEFA concentrations[20] and reduce the number of insulin receptors, both of which can contribute to insulin resistance[21]. Furthermore, in contrast to whole grains, refined grains contain less amounts of fibre and phytochemicals that may lower the risk of type 2 diabetes[11,18,19,22–24]. Studies on the association between refined grain consumption and metabolic risk factors can provide insights into the potential mechanisms that may contribute to a higher risk of type 2 diabetes. However, few of these studies have been carried out in Asian populations in which rice and noodles are the main carbohydrate source[25–28]. Insulin resistance and impaired β-cell function are key contributors to the development of type 2 diabetes, and chronic inflammation and dyslipidaemia can contribute to these conditions[29,30]. To better understand the mechanisms by which rice or noodle intake may be involved in the pathogenesis of type 2 diabetes and its impact on the metabolic traits associated with type 2 diabetes, we evaluated the relationship between rice and noodle consumption and markers of insulin resistance, insulin secretion, inflammation and dyslipidaemia in a cross-sectional study in Singaporean Chinese.

Methods

Study population

The present study used cross-sectional data of the Singapore Prospective Study Program-2 (SP2) cohort. These data were collected from 2004 to 2007. SP2 was a follow-up study of the participants of four population-based studies carried out in Singapore during 1982–8: the Thyroid and Heart Study; the National Health Survey (1992); the National University of Singapore Heart Study; the National Health Survey (1998). These studies selected participants by stratified random sampling of individuals aged 18–69 years with oversampling of ethnic minority groups (i.e. Malays and Asian-Indians)[31]. The present study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human subjects were approved by the National University of Singapore and the Singapore General Hospital Institutional Review Boards. Written informed consent was obtained from all the participants.

For SP2, 10 747 study subjects qualified for participation, and 7744 completed interviewer-administered questionnaires on demographics, lifestyle and medical history. Of these participants, 5163 made a clinic visit during which blood was drawn and anthropometric parameters and blood pressure were measured. Details on participant recruitment and study methodology have been reported elsewhere[32]. As the type of refined grain foods consumed by Indians and Malays can be substantially different and may be less comprehensively represented in our questionnaire, we focused only on the Chinese subpopulation in the present study. Of the 5163 participants who visited the clinic, 3439 were Chinese. From this subpopulation, we excluded subjects with pre-existing CVD (n 109), diabetes diagnosis or known diabetes (n 232), current cancer (n 47) or a current pregnancy (n 2), as these conditions may affect diet or reporting of dietary intake potentially leading to reverse causation. From the remaining 3088 participants, we also excluded individuals who changed their diet in the month preceding the interview (n 203) and those with extremes of energy intake ($>29\,288$ or <2092 kJ (>7000 or <500 kcal)) and extreme energy intake based on the ratio of energy intake:energy expenditure (lowest or highest 2·5 percentage of the ratio, n 138)[33]. From the remaining cohort comprising 2758 participants, we further excluded individuals with missing covariates (n 30). As a result, 2728 persons remained for the analysis.

Assessment of dietary intakes

A semi-quantitative 169-item FFQ developed by the Institute of Health, Singapore, was used to assess the dietary intake of participants during the month preceding the interview. Food items were selected if they were significant contributors to the intake of energy and selected nutrients in adult Singaporeans based on the National Nutrition Survey. The questionnaire has been validated previously[34]. The participants were asked to estimate the frequency of consumption of each food group based on a reference portion size and to indicate consumption on a per-d, per-week or per-month basis or as never/rarely. Daily energy and nutrient intakes were subsequently calculated by the Health Promotion Board based on an in-house database of energy and nutrient values of local foods.

Data on rice intake were obtained by asking the participants about the frequency of consumption of a standard bowl or portion of rice. Household measures such as bowls of different sizes or visual aids for various food groups and individual food items were used to help the participants estimate amounts consumed as fractions or multiples of the illustrated reference portions. In total, ten rice dishes and thirteen noodle dishes were included in the questionnaire. Options for the type of rice consumed included plain rice or plain rice porridge, flavoured rice (e.g. fried rice and chicken rice) and flavoured rice porridge. For noodle intake (mainly rice noodles, wheat noodles, bean noodles or pasta), the questionnaire assessed the consumption of different types of noodles in soup, dry noodles, fried noodles, noodles in lemak gravy (with coconut milk) and other noodles (including instant noodles). One portion of cooked white rice weighs approximately 200 g and one portion of cooked noodles weighs approximately 275 g.

Assessment of outcome variables

Fasting venous blood samples were sent to the National University Hospital Reference Laboratory for analysis on the day the blood was drawn for biochemical testing of fasting plasma glucose (FPG; ADVIA 2400, Siemens), glycated

Hb (Biorad Variant II analyser, Bio-Rad Laboratories), HDL-cholesterol (HDL-C) and TAG. Fasting serum samples were analysed for total cholesterol, TAG and HDL-C (Siemens Medical Solutions Diagnostics), high-sensitivity C-reactive protein (CRP, Roche Diagnostics), total adiponectin (Sekisui Medical Company Limited) and insulin (microparticle enzyme immunoassay, Abbott AXSYM, Abbott Laboratories). LDL-cholesterol concentrations were calculated using the Friedewald equation. The respective intra-batch and inter-batch CV for the variables were as follows: 0·93–1·15 and 0·56–0·65 % for TAG; 0–3·85 and 1·18–2·00 % for HDL-C; 1·27–3·40 and 2·50–6·60 % for glucose; 4·00–4·50 % for insulin; 0–2·00 and 0·85–1·54 % for glycated Hb, 0·60–1·30 and 2·30–3·10 % for CRP; 18·10 and 15·90 % for adiponectin. Homeostasis model assessment (HOMA) indices were used as measures of insulin resistance and β-cell function. The HOMA insulin resistance (HOMA-IR) index was computed as FPG (mmol/l) × fasting serum insulin (mU/l)/22·5, where for insulin 1 mU = 6·00 pmol. The HOMA-β-cell (HOMA-β) index was computed as 20 × fasting serum insulin (mU/l)/FPG (mmol/l) − 3·5[35].

Assessment of covariates

Height was measured using a wall-mounted measuring tape and weight using a digital scale. BMI was computed as weight (kg) divided by height (m^2). Data on alcohol and coffee intake were obtained using the FFQ and those on cigarette smoking status, education level and physical activity the main questionnaire. Total physical activity, expressed as metabolic equivalents of task-h/week, was assessed using a validated questionnaire based on activities in four domains (household, occupational, leisure-time and transport)[33]. Data on history of hypertension or dyslipidaemia were obtained from self-reports of physician diagnosis or use of medications to treat these conditions.

Statistical analyses

The participants were classified into quintiles of rice and noodle consumption, and we compared the characteristics of the participants across the quintiles using ANOVA (for continuous variables with a normal distribution), Kruskal–Wallis tests (for continuous variables with a non-normal distribution) or χ^2 tests (for categorical variables). Rice and noodle consumption is expressed as portions per 8368 kJ (2000 kcal). All the response variables were transformed using natural logarithms to achieve normality. Geometric means and 95 % CI were obtained by exponentiation of means and 95 % CI of values on the logarithmic scale. Sensitivity analyses were carried out, in which we truncated response variables that were more than 4 SD from the mean to reduce the potential impact of outliers. As this did not materially affect the results, we report results for data without truncation.

We used multiple linear regression analyses to study rice and noodle consumption in relation to biological risk factors. Sociodemographic factors and type 2 diabetes risk factors that were identified from the literature were adjusted for in the analyses as potential confounders. Variables were incorporated into four multivariable models as follows: (1) adjusted for age (years), sex and total energy intake (kJ/d); (2) further adjusted for physical activity (metabolic equivalents of task-h/week), alcohol intake (non-drinker, <1 serving/d, ≥1 serving/d), cigarette smoking status (non-smoker, ex-smoker, current smoker <10 cigarettes/d, current smoker ≥10 cigarettes/d), education level (primary/below, secondary, polytechnic/diploma, university), dyslipidaemia diagnosis or known dyslipidaemia (yes/no) and hypertension diagnosis or known hypertension (yes/no); (3) further adjusted for BMI (kg/m^2) and waist:hip ratio; (4) further adjusted for dietary confounders, specifically consumption of coffee (never/rarely, <1 cup daily, 1–2 cups daily, ≥3 cups daily), protein (% energy), wholemeal bread (0, 0·1 to <1, ≥1 serving daily), white bread (0, 0·1 to <1, ≥1 serving daily), fruits (servings per 8368 kJ (2000 kcal)), vegetables (servings per 8368 kJ (2000 kcal)), cholesterol (mg/8368 kJ (2000 kcal)), ratio of PUFA:SFA and rice (portions per 8368 kJ (2000 kcal)) or noodles (portions per 8368 kJ (2000 kcal)) when appropriate. Rice and noodle intakes were fitted as quintiles; bread and coffee intakes were fitted in the categorical scale, while the other dietary variables were modelled in the continuous scale. Analyses with HOMA-β as the outcome variable were further adjusted for HOMA-IR. Participants with diagnosed dyslipidaemia were excluded from the analyses with HDL-C or TAG as the outcome variable (remaining sample size *n* 1978). We also carried out analyses modelling rice and noodle intakes as continuous variables. The regression coefficients are expressed as percentage changes in the outcome variables for each portion increment in rice or noodle intake[36].

Secondary analyses were carried out to evaluate whether associations were similar for men and women and for overweight (BMI ≥23 kg/m^2) and non-overweight (BMI <23 kg/m^2) individuals using a commonly accepted cut-off value for overweight in Asians[37]. A multiplicative interaction term with sex or overweight as a dichotomous variable and rice or noodle intake as a continuous variable was included in the multivariable models. We observed no significant interaction by sex and therefore present the results for men and women together. All the data were analysed using the Statistical Analysis System version 9.2 software (SAS Institute, Inc.). Two-sided *P* values <0·05 were considered statistically significant.

Results

The median intake of rice was 1·57 portions/d (1·75 portions/8368 kJ (2000 kcal)) and the median intake of noodles was 0·57 portions/d (0·63 daily portions/8368 kJ (2000 kcal)). Rice and noodle consumption explained 64·6 % of starch intake in the study population independent of total energy intake. The characteristics of the study population according to rice and noodle consumption are given in Table 1. The study participants were aged between 24·6 and 91·8 years (mean 48·7 (SD 11·5) years). Participants with higher rice intake tended to be older and male, have a low education level, smoke, be less physically active and have hypertension diagnosis or

Table 1. Characteristics of the study participants according to quintiles of rice and noodle consumption

(Mean values and standard deviations; median values, interquartile ranges and percentages)

	Rice						Noodles					
	Quintile 1 (low)		Quintile 3 (medium)		Quintile 5 (high)		Quintile 1 (low)		Quintile 3 (medium)		Quintile 5 (high)	
	n 546		*n* 545		*n* 545		*n* 546		*n* 545		*n* 545	
Characteristics	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Daily intake												
Median	0·98		1·75		2·79		0·22		0·63		1·21	
Interquartile range	0·77–1·10		1·67–1·84		2·56–3·27		0·12–0·29		0·58–0·68		1·08–1·42	
Age (years)	45·67	10·44	47·91	10·89	52·81	11·89	52·89	11·91	47·58	11·28	47·12	10·75
BMI (kg/m^2)	22·65	3·58	23·07	3·86	22·54	3·55	22·56	3·46	22·47	3·58	22·98	3·56
WHR												
Men												
Median	0·89		0·88		0·89		0·88		0·88		0·88	
Interquartile range	0·85–0·92		0·84–0·92		0·84–0·92		0·84–0·92		0·84–0·91		0·85–0·92	
Women												
Median	0·79		0·80		0·80		0·81		0·79		0·80	
Interquartile range	0·76–0·84		0·77–0·84		0·77–0·84		0·77–0·86		0·75–0·83		0·76–0·84	
Sex (%)												
Men	34·07		49·72		54·13		46·15		44·95		48·62	
Education (%)												
Primary or below	14·84		24·59		38·72		32·60		21·83		23·49	
Secondary	37·18		38·17		34·50		32·97		36·70		43·12	
Polytechnic or diploma	22·34		16·15		12·84		15·75		19·45		17·61	
University	25·64		21·10		13·94		18·68		22·02		15·78	
Alcohol intake (%)	50·55		42·02		44·40		41·39		49·54		46·97	
Coffee consumption (%)												
Never/rarely	30·59		27·71		28·99		26·56		28·44		28·26	
<1 cup/d	11·17		11·19		9·17		11·36		12·48		9·36	
1–2 cups/d	47·80		54·68		54·50		53·85		51·74		54·13	
≥3 cups/d	10·44		6·42		7·34		8·24		7·34		8·26	
Cigarette smoking status (%)												
Non-smoker	85·53		82·20		76·15		84·07		83·12		77·61	
Ex-smoker	7·69		8·81		8·26		7·69		7·34		8·26	
Current, <10 cigarettes/d	2·38		2·39		4·22		2·38		3·67		4·04	
Current, ≥10 cigarettes/d	4·40		6·61		11·38		5·86		5·87		10·09	
Physical activity (MET-h/week)												
Median	22·08		18·00		17·50		23·80		17·50		17·50	
Interquartile range	10·00–53·50		7·00–36·35		7·00–45·00		8·63–51·00		7·00–38·50		7·00–45·75	
Diagnosed dyslipidaemia (%)	24·73		28·44		29·36		31·68		25·69		28·81	
Diagnosed hypertension (%)	17·03		16·88		20·55		18·86		16·15		17·80	
Energy intake (kcal/d)												
Median	2164		1806		1610		1686		1846		1987	
Interquartile range	1621–2665		1419–2472		1313–1902		1357–2148		1502–2328		1523–2432	
Energy intake (kJ/d)												
Median	9054		7556		6736		7054		7724		8314	
Interquartile range	6782–11150		5937–10343		5494–7958		5678–8987		6284–9740		6372–10175	
Carbohydrate (% energy)	52·22	6·13	55·95	5·14	61·25	5·77	57·62	7·36	56·30	6·10	55·88	5·37
Protein (% energy)	15·68	2·22	14·94	1·68	14·13	1·77	14·69	2·13	14·77	1·79	15·21	1·77

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Table 1. *Continued*

	Rice						Noodles					
	Quintile 1 (low)		Quintile 3 (medium)		Quintile 5 (high)		Quintile 1 (low)		Quintile 3 (medium)		Quintile 5 (high)	
	n 546		*n* 545		*n* 545		*n* 546		*n* 545		*n* 545	
Characteristics	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Fat (% energy)	31·81	5·51	28·82	4·51	24·33	4·76	27·42	6·11	28·57	5·23	28·53	4·84
Starch (% energy)	29·65	6·84	35·81	5·12	44·02	6·72	34·93	9·69	35·63	6·63	38·57	6·07
Mono- and disaccharides (% energy)	22·31	7·24	19·90	5·79	17·02	5·39	22·41	7·33	20·42	5·73	17·13	5·24
PUFA:SFA ratio												
Median	0·48		0·51		0·50		0·57		0·51		0·44	
Interquartile range	0·37–0·74		0·37–0·73		0·38–0·78		0·39–0·87		0·37–0·73		0·36–0·62	
Fibre (g/8368 kJ (2000 kcal))	21·41	5·72	20·83	4·39	21·21	4·66	22·11	5·50	20·86	4·48	20·14	4·16
Cholesterol (mg/4184 kJ (1000 kcal))	125·97	42·66	124·03	39·90	109·17	43·06	106·46	40·61	123·64	36·14	126·79	42·00
Rice*	0·90	0·27	1·75	0·10	3·00	0·62	2·16	0·98	1·82	0·66	1·54	0·62
Noodles*	0·83	0·54	0·69	0·38	0·52	0·33	0·20	0·11	0·63	0·06	1·32	0·37
Wholemeal bread (portions/d)	0·44	0·82	0·32	0·77	0·23	0·62	0·51	1·01	0·31	0·65	0·17	0·44
White bread (portions/d)												
Median	0·57		0·70		0·43		0·64		0·60		0·43	
Interquartile range	0·10–1·18		0·61–1·29		0·07–1·00		0·07–1·78		0·14–1·27		0·07–1·00	
Fruits*												
Median	1·00		1·00		1·00		1·00		1·00		1·00	
Interquartile range	1·00–2·00		1·00–2·00		1·00–2·00		1·00–2·00		1·00–2·00		1·00–2·00	
Vegetables*												
Median	1·95		1·57		1·40		1·62		1·48		1·54	
Interquartile range	1·24–3·05		1·09–2·35		0·94–1·97		1·04–2·56		1·07–2·23		1·10–2·30	

WHR, waist:hip ratio; MET-h/week, metabolic equivalents of task-h/week.
* Expressed in portions per 8368 kJ (2000 kcal).

known hypertension. Higher rice consumption was associated with higher intakes of carbohydrate and starch, but with lower intakes of protein, fat, mono- and disaccharides and noodles. Participants with higher noodle intake tended to be younger and be less physically active. Higher noodle consumption was associated with higher intakes of protein and starch, but with lower intakes of carbohydrates, mono- and disaccharides, and fibre and a lower ratio of PUFA:SFA.

Table 2 summarises the concentrations of metabolic markers according to quintiles of rice consumption. Higher rice consumption was not significantly associated with fasting glucose concentrations and HOMA-IR in the basic model adjusted for age, sex and energy intake. However, after further adjustment for potential confounders, particularly BMI and protein and noodle intake, higher rice consumption was strongly associated with higher fasting glucose concentrations and HOMA-IR. Rice consumption was not significantly associated with glycated Hb, CRP, adiponectin, HDL-C, and fasting TAG concentrations and HOMA-β.

Higher noodle consumption was associated with higher fasting glucose, glycated Hb, CRP, and fasting TAG concentrations and HOMA-IR, but with lower adiponectin and HDL-C concentrations in the basic model (Table 3). After further adjustment for additional potential confounders, noodle consumption remained significantly directly associated with fasting glucose and TAG concentrations and HOMA-IR. Associations between noodle consumption and other biomarkers were not statistically significant in the fully adjusted model.

In secondary analyses, we examined the odds of having hyperglycaemia using the American Diabetes Association FPG cut-off ≥5·6 mmol/l[38] across the tertiles of rice and noodle consumption (Supplementary Table 1, available online). In the fully adjusted model, we observed higher odds of having high FPG concentrations with higher noodle consumption (OR per portion increment: 1·55; 95 % CI 1·09, 2·20; *P* trend=0·01), but not with higher rice consumption (0·99; 95 % CI 0·79, 1·22; *P* trend=0·89).

We also examined the association between total refined grain consumption (the sum of rice and noodle intakes) and metabolic risk factors that were significantly associated with either rice or noodle consumption. Each portion increment in total refined grain consumption was associated with higher FPG concentrations (percentage change 0·92; 95 % CI 0·22, 1·62; *P* trend=0·01), higher fasting TAG concentrations (3·42; 95 % CI 0·33, 6·60; *P* trend=0·03) and a higher insulin resistance index (4·96; 95 % CI 1·75, 8·27; *P* trend=0·002).

It has been suggested that the association between refined grain consumption and markers of hyperglycaemia may be more pronounced among overweight individuals[39,40]. Therefore, we evaluated the possible interaction of rice and noodle consumption with overweight status (BMI ≥23 kg/m^2) in relation to metabolic risk factors for which we observed significant associations in the main analysis (Supplementary Tables 2 and 3, available online). The direct association between noodle consumption and HOMA-IR was stronger in the overweight group (change per portion 13·15 %, 95 % CI 3·02, 24·29) than in the leaner group (1·49 %, 95 % CI −5·02, 8·45; *P* interaction=0·001). However, the direct association between rice consumption and HOMA-IR tended to be weaker in the overweight participants (1·88 %, 95 % CI −3·69, 7·78) than in the leaner participants (6·87 %, 95 % CI 2·80, 11·10, *P* interaction=0·33).

Discussion

In the present population-based study of 2728 Singaporean Chinese adult men and women, high consumption of rice and noodles was found to be associated with higher fasting glucose concentrations and a higher HOMA index for insulin resistance. In contrast, rice and noodle consumption was found to be not associated with adiponectin or CRP concentrations after adjustment for potential confounders. The present results thus suggest that high consumption of refined grains may contribute to hyperglycaemia through greater insulin resistance, rather than through increased systemic inflammation.

The results obtained for rice consumption in the present study are consistent with the findings of several other studies carried out in Asian populations in which white rice intake is high. Higher consumption of rice was found to be significantly associated with a higher risk of type 2 diabetes in a cohort of older Shanghai women (relative risk 1·78; 95 % CI 1·48, 2·15 for ≥300 *v.* <200 g/d)[10]. In a Japanese cohort, higher rice consumption was also reported to be associated with a higher risk of type 2 diabetes in women (relative risk 1·65; 95 % CI 1·06, 2·57 for ≥420 *v.* <200 g/d), but not in men[11]. In a cross-sectional study carried out in India, high refined grain consumption (of which white rice comprised 75·8 %) was found to be associated with a higher prevalence of newly diagnosed type 2 diabetes[27]. In the same study, higher refined grain consumption was found to be associated with significantly 7·9 % higher fasting glucose concentrations, 13·6 % higher HOMA-IR values, 36·5 % higher serum TAG concentrations and 10·1 % lower HDL-C concentrations for the highest (median 449 g/d) *v.* the lowest (median 218 g/d) quartile[18]. In a cross-sectional study in Japanese female farmers, white rice was found to be the major contributor to the dietary glycaemic load, which is associated with higher fasting TAG and glucose concentrations and lower HDL-C concentrations[41].

Few studies on noodle consumption in relation to metabolic risk factors or risk of type 2 diabetes have been carried out. In a study in Hong Kong Chinese, higher consumption of rice, noodles and pasta was found to be associated with a significantly higher prevalence of newly diagnosed type 2 diabetes. However, specific results for noodles were not reported[25]. In a Japanese cohort, consumption of noodles was found to be not associated with the risk of type 2 diabetes[11]. It is possible that differences in the type of noodles consumed (e.g. wholegrain buckwheat noodles are commonly consumed in Japan but not in Singapore) are responsible for these differences in association. In the present study, we observed stronger associations between noodle consumption and metabolic risk markers than between rice consumption and the markers. In Singapore, both rice and wheat-based noodles typically

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Table 2. Metabolic risk factors according to quintiles of rice consumption

(Geometric means and 95 % confidence intervals; median values and interquartile ranges)

	Quintile 1 (*n* 546)		Quintile 2 (*n* 546)		Quintile 3 (*n* 545)		Quintile 4 (*n* 546)		Quintile 5 (*n* 545)		Percentage change per portion*		
Characteristics	Geometric mean	95 % CI	Geometric mean	95 % CI	Geometric mean	95 % CI	Geometric mean	95 % CI	Geometric mean	95 % CI	Geometric mean	95 % CI	*P* for trend†
Daily portion intake													
Median	0·98		1·40		1·75		2·15		2·79		–		–
Interquartile range	0·77–1·10		1·32–1·50		1·67–1·84		2·04–2·27		2·56–3·27		–		
FPG (mmol/l)													
Model 1‡	4·71	4·66, 4·77	4·75	4·70, 4·80	4·80	4·75, 4·85	4·83	4·78, 4·88	4·78	4·73, 4·83	0·22	−0·44, 0·89	0·51
Model 2§	4·76	4·67, 4·84	4·80	4·72, 4·88	4·84	4·76, 4·93	4·88	4·79, 4·96	4·83	4·74, 4·91	0·22	−0·45, 0·90	0·52
Model 3‖	4·73	4·65, 4·81	4·78	4·71, 4·86	4·81	4·73, 4·89	4·85	4·77, 4·92	4·82	4·74, 4·90	0·41	−0·23, 1·07	0·21
Model 4¶	4·70	4·61, 4·78	4·76	4·68, 4·84	4·80	4·71, 4·88	4·84	4·75, 4·92	4·83	4·74, 4·92	0·81	0·09, 1·54	0·03
HbA1c (%)													
Model 1	5·68	5·63, 5·73	5·69	5·65, 5·74	5·75	5·70, 5·80	5·72	5·67, 5·77	5·72	5·67, 5·77	0·18	−0·34, 0·71	0·49
Model 2	5·73	5·65, 5·81	5·75	5·67, 5·82	5·80	5·72, 5·88	5·76	5·68, 5·84	5·76	5·69, 5·84	0·05	−0·47, 0·58	0·84
Model 3	5·71	5·64, 5·79	5·73	5·66, 5·81	5·77	5·70, 5·85	5·74	5·66, 5·81	5·76	5·68, 5·84	0·19	−0·33, 0·71	0·48
Model 4	5·69	5·61, 5·78	5·71	5·64, 5·79	5·76	5·68, 5·84	5·73	5·65, 5·81	5·76	5·67, 5·84	0·29	−0·29, 0·86	0·33
HOMA-IR													
Model 1	1·19	1·12, 1·26	1·13	1·07, 1·20	1·26	1·20, 1·34	1·25	1·19, 1·33	1·23	1·16, 1·30	1·79	−1·70, 5·39	0·32
Model 2	1·26	1·15, 1·37	1·22	1·11, 1·33	1·33	1·22, 1·46	1·34	1·23, 1·46	1·30	1·19, 1·42	1·79	−1·66, 5·36	0·31
Model 3	1·19	1·10, 1·28	1·18	1·09, 1·27	1·25	1·16, 1·34	1·25	1·16, 1·35	1·29	1·20, 1·39	3·78	0·81, 6·85	0·01
Model 4	1·16	1·08, 1·26	1·16	1·08, 1·26	1·24	1·14, 1·34	1·25	1·16, 1·36	1·29	1·19, 1·40	4·62	1·29, 8·07	0·01
HOMA-β													
Model 1	101·95	97·51, 106·59	96·44	92·36, 100·70	96·49	92·45, 100·71	92·31	88·43, 96·36	97·44	93·24, 101·84	−0·07	−2·70, 2·62	0·96
Model 2	101·60	94·76, 108·94	96·05	89·72, 102·83	96·14	89·66, 103·09	92·07	85·98, 98·58	96·58	90·04, 103·59	−0·34	−3·00, 2·40	0·81
Model 3	101·80	94·94, 109·17	96·18	89·83, 102·97	96·37	89·87, 103·34	92·30	86·20, 98·83	96·38	89·86, 103·38	−0·52	−3·17, 2·22	0·71
Model 4	105·00	97·57, 113·00	98·62	91·80, 105·95	98·11	91·10, 105·66	93·44	86·92, 100·46	95·91	88·84, 103·55	−2·09	−4·99, 0·90	0·17
Adiponectin (μg/ml)													
Model 1	3·37	3·22, 3·52	3·45	3·30, 3·60	3·22	3·08, 3·36	3·36	3·22, 3·51	3·48	3·33, 3·64	2·07	−0·60, 4·82	0·13
Model 2	3·22	3·00, 3·45	3·28	3·06, 3·50	3·06	2·86, 3·28	3·19	2·98, 3·41	3·30	3·08, 3·53	1·62	−1·06, 4·38	0·24
Model 3	3·32	3·12, 3·54	3·33	3·13, 3·55	3·17	2·97, 3·38	3·30	3·10, 3·51	3·32	3·12, 3·55	0·64	−1·83, 3·18	0·62
Model 4	3·36	3·14, 3·60	3·36	3·15, 3·59	3·20	2·99, 3·43	3·32	3·11, 3·55	3·34	3·12, 3·59	0·56	−2·20, 3·39	0·7
CRP (mg/l)													
Model 1	0·87	0·79, 0·96	0·88	0·80, 0·96	0·96	0·87, 1·06	0·95	0·86, 1·04	0·92	0·84, 1·02	2·65	−3·19, 8·84	0·38
Model 2	0·88	0·76, 1·02	0·90	0·78, 1·04	0·95	0·82, 1·11	0·95	0·82, 1·10	0·89	0·77, 1·04	0·40	−5·32, 6·45	0·89
Model 3	0·83	0·72, 0·95	0·87	0·76, 1·00	0·88	0·77, 1·01	0·88	0·77, 1·01	0·89	0·78, 1·03	2·89	−2·47, 8·54	0·3
Model 4	0·83	0·72, 0·96	0·85	0·74, 0·98	0·86	0·74, 0·99	0·86	0·75, 0·99	0·87	0·74, 1·01	1·30	−4·54, 7·50	0·67
HDL-cholesterol (mmol/l)**													
Model 1	1·45	1·42, 1·49	1·45	1·42, 1·48	1·42	1·39, 1·45	1·42	1·39, 1·46	1·45	1·42, 1·49	−0·17	−1·55, 1·22	0·81
Model 2	1·47	1·42, 1·52	1·46	1·41, 1·51	1·44	1·39, 1·49	1·44	1·39, 1·49	1·48	1·43, 1·53	0·22	−1·17, 1·63	0·76
Model 3	1·48	1·44, 1·53	1·47	1·42, 1·52	1·46	1·41, 1·51	1·46	1·41, 1·51	1·48	1·43, 1·53	−0·24	−1·55, 1·08	0·72
Model 4	1·49	1·44, 1·54	1·47	1·42, 1·52	1·46	1·41, 1·52	1·46	1·41, 1·52	1·48	1·43, 1·54	−0·44	−1·89, 1·03	0·55
Fasting TAG (mmol/l)**													
Model 1	1·00	0·95, 1·05	1·01	0·96, 1·06	1·11	1·05, 1·16	1·06	1·01, 1·12	1·04	0·99, 1·09	1·94	−1·10, 5·08	0·21
Model 2	1·16	1·07, 1·25	1·16	1·08,1·25	1·28	1·18, 1·38	1·21	1·13, 1·31	1·18	1·09, 1·27	0·89	−2·13, 4·01	0·57
Model 3	1·12	1·05, 1·21	1·15	1·07, 1·23	1·24	1·15, 1·34	1·18	1·10, 1·26	1·18	1·09, 1·26	1·97	−0·89, 4·91	0·18
Model 4	1·11	1·03, 1·19	1·13	1·05, 1·22	1·22	1·13, 1·32	1·16	1·08, 1·25	1·16	1·07, 1·26	2·08	−1·10, 5·36	0·2

FPG, fasting plasma glucose; HbA1c, glycated Hb; HOMA-IR, homeostasis model assessment of insulin resistance; HOMA-β, homeostasis model assessment of β-cell function; CRP, C-reactive protein.

* Percentage changes (and 95 % CI) in plasma concentrations of metabolic risk factors for each portion increment of rice, approximately 200 g of cooked rice.

† From multiple linear regression models for the relationship between rice consumption and log-transformed markers.

‡ Adjusted for age, sex and energy intake. HOMA-β models were further adjusted for HOMA-IR.

§ Further adjusted for physical activity, alcohol intake, smoking status, education level, diagnosed dyslipidaemia and diagnosed hypertension.

‖ Further adjusted for BMI and waist:hip ratio.

¶ Further adjusted for coffee consumption and intake of protein, noodles, wholemeal bread, white bread, fruits, vegetables, cholesterol and ratio of PUFA:SFA.

** Participants with diagnosed dyslipidaemia were excluded from HDL-cholesterol and TAG analyses (remaining sample size *n* 1978, whereby n_1 411, n_2 395, n_3 390, n_4 397 and n_5 385 for quintiles 1–5, respectively).

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Table 3. Metabolic risk factors according to quintiles of noodle consumption
(Geometric means and 95 % confidence intervals; median values and interquartile ranges)

	Quintile 1 (*n* 546)		Quintile 2 (*n* 546)		Quintile 3 (*n* 545)		Quintile 4 (*n* 546)		Quintile 5 (*n* 545)		Percentage change per portion*		
Characteristics	Geometric mean	95 % CI	Geometric mean	95 % CI	Geometric mean	95 % CI	Geometric mean	95 % CI	Geometric mean	95 % CI	Geometric mean	95 % CI	*P* for trend†
Daily portion intake													
Median	0·22		0·45		0·63		0·84		1·21		–		–
Interquartile range	0·12–0·29		0·40–0·49		0·58–0·68		0·78–0·91		1·08–1·42		–		
FPG (mmol/l)													
Model 1‡	4·70	4·65, 4·75	4·79	4·74, 4·84	4·75	4·70, 4·80	4·79	4·74, 4·84	4·85	4·79, 4·90	2·09	0·93, 3·27	<0·001
Model 2§	4·75	4·66, 4·83	4·83	4·75, 4·92	4·79	4·71, 4·87	4·82	4·74, 4·91	4·88	4·80, 4·96	1·86	0·70, 3·05	0·002
Model 3‖	4·74	4·66, 4·82	4·81	4·73, 4·89	4·78	4·70, 4·86	4·79	4·71, 4·87	4·85	4·78, 4·93	1·50	0·38, 2·64	0·01
Model 4¶	4·73	4·65, 4·81	4·79	4·71, 4·88	4·77	4·68, 4·85	4·78	4·69, 4·86	4·85	4·76, 4·94	1·67	0·44, 2·92	0·01
HbA1c (%)													
Model 1	5·69	5·64, 5·73	5·70	5·65, 5·74	5·68	5·64, 5·73	5·73	5·68, 5·78	5·77	5·72, 5·82	1·43	0·52, 2·34	0·002
Model 2	5·73	5·65, 5·81	5·74	5·66, 5·82	5·73	5·65, 5·81	5·77	5·70, 5·85	5·80	5·72, 5·88	1·16	0·25, 2·08	0·01
Model 3	5·73	5·65, 5·80	5·72	5·64, 5·79	5·72	5·65, 5·80	5·75	5·67, 5·82	5·78	5·70, 5·85	0·95	0·07, 1·85	0·04
Model 4	5·73	5·65, 5·81	5·71	5·63, 5·79	5·72	5·64, 5·79	5·74	5·66, 5·82	5·77	5·69, 5·85	0·85	−0·12, 1·84	0·09
HOMA-IR													
Model 1	1·17	1·11, 1·24	1·17	1·10, 1·23	1·16	1·10, 1·23	1·27	1·20, 1·34	1·30	1·23, 1·37	10·64	4·19, 17·50	0·001
Model 2	1·25	1·14, 1·37	1·24	1·14, 1·35	1·23	1·12, 1·34	1·33	1·22, 1·45	1·36	1·25, 1·48	8·64	2·40, 15·27	0·01
Model 3	1·23	1·14, 1·32	1·17	1·09, 1·27	1·21	1·12, 1·30	1·23	1·15, 1·33	1·28	1·19, 1·38	4·74	−0·38, 10·12	0·07
Model 4	1·22	1·13, 1·32	1·16	1·08, 1·26	1·20	1·11, 1·30	1·23	1·13, 1·33	1·29	1·19, 1·40	6·17	0·49, 12·16	0·03
HOMA-β													
Model 1	102·52	98·16, 107·06	92·96	89·07, 97·02	97·14	93·07, 101·40	97·88	93·77, 102·17	94·01	90·05, 98·13	−4·17	−8·49, 0·35	0·07
Model 2	102·24	95·29, 109·70	92·71	86·51, 99·36	97·08	90·65, 103·97	97·67	91·18, 104·62	93·93	87·81, 100·49	−3·97	−8·34, 0·61	0·09
Model 3	102·19	95·24, 109·65	92·94	86·73, 99·61	97·10	90·67, 103·99	97·85	91·34, 104·83	94·02	87·89, 100·57	−3·89	−8·26, 0·69	0·1
Model 4	103·21	95·90, 111·08	94·60	87·96, 101·74	98·74	91·84, 106·16	99·24	92·25, 106·77	95·18	88·35, 102·53	−4·16	−8·92, 0·85	0·1
Adiponectin (μg/ml)													
Model 1	3·50	3·35, 3·65	3·40	3·25, 3·54	3·38	3·24, 3·53	3·26	3·12, 3·40	3·35	3·20, 3·49	−5·03	−9·29, −0·58	0·03
Model 2	3·31	3·09, 3·54	3·23	3·02, 3·46	3·23	3·01, 3·45	3·13	2·92, 3·35	3·21	3·00, 3·43	−4·18	−8·49, 0·32	0·07
Model 3	3·35	3·14, 3·57	3·32	3·12, 3·54	3·26	3·06, 3·47	3·25	3·05, 3·46	3·31	3·11, 3·52	−2·25	−6·34, 2·02	0·3
Model 4	3·36	3·14, 3·60	3·34	3·12, 3·57	3·28	3·07, 3·51	3·28	3·06, 3·50	3·34	3·11, 3·58	−2·27	−6·76, 2·43	0·34
CRP (mg/l)													
Model 1	0·87	0·79, 0·96	0·84	0·76, 0·92	0·93	0·85, 1·02	0·98	0·90, 1·08	0·96	0·87, 1·06	16·75	5·54, 29·16	0·003
Model 2	0·88	0·76, 1·02	0·85	0·73, 0·98	0·93	0·80, 1·08	0·97	0·84, 1·12	0·93	0·81, 1·08	12·24	1·51, 24·10	0·02
Model 3	0·87	0·76, 1·00	0·80	0·70, 0·92	0·91	0·80, 1·05	0·90	0·78, 1·03	0·87	0·76, 1·00	6·94	−2·44, 17·23	0·15
Model 4	0·87	0·97, 1·01	0·78	0·68, 0·90	0·89	0·77, 1·03	0·87	0·76, 1·01	0·86	0·74, 1·00	7·59	−2·71, 18·97	0·15
HDL-cholesterol (mmol/l)**													
Model 1	1·48	1·45, 1·52	1·43	1·40, 1·46	1·47	1·43, 1·50	1·42	1·39, 1·45	1·40	1·37, 1·44	−3·69	−6·00, −1·32	0·002
Model 2	1·50	1·45, 1·56	1·44	1·40, 1·50	1·48	1·43, 1·54	1·44	1·39, 1·49	1·43	1·38, 1·48	−3·31	−5·63, −0·94	0·01
Model 3	1·50	1·45, 1·55	1·46	1·41, 1·51	1·49	1·44, 1·54	1·46	1·42, 1·51	1·45	1·40, 1·49	−2·28	−4·50, −0·01	0·05
Model 4	1·51	1·45, 1·56	1·46	1·41, 1·51	1·49	1·44, 1·54	1·47	1·42, 1·52	1·45	1·40, 1·50	−2·24	−4·65, 0·24	0·08
Fasting TAG (mmol/l)**													
Model 1	0·99	0·94, 1·04	1·03	0·98, 1·08	0·99	0·95, 1·04	1·10	1·05, 1·15	1·11	1·06, 1·17	11·62	5·86, 17·69	<0·001
Model 2	1·14	1·05, 1·23	1·18	1·10, 1·27	1·13	1·05, 1·22	1·25	1·16, 1·34	1·25	1·16, 1·35	9·30	3·68, 15·22	0·001
Model 3	1·13	1·05, 1·22	1·15	1·07, 1·24	1·13	1·05, 1·21	1·21	1·12, 1·29	1·21	1·13, 1·30	6·67	1·52, 12·08	0·01
Model 4	1·11	1·03, 1·20	1·14	1·05, 1·22	1·11	1·03, 1·20	1·20	1·11, 1·29	1·22	1·13, 1·32	9·17	3·44, 15·22	0·001

FPG, fasting plasma glucose; HbA1c, glycated Hb; HOMA-IR, homeostasis model assessment of insulin resistance; HOMA-β, homeostasis model assessment of β-cell function; CRP, C-reactive protein.
* Percentage changes (and 95 % CI) in plasma concentrations of metabolic risk factors for each portion increment of noodles, approximately 275 g of cooked noodles.
† From multiple linear regression models for the relationship between noodle consumption and log-transformed markers.
‡ Adjusted for age, sex and energy intake. HOMA-β models were further adjusted for HOMA-IR.
§ Further adjusted for physical activity, alcohol intake, smoking status, education level, diagnosed dyslipidaemia and diagnosed hypertension.
‖ Further adjusted for BMI and waist:hip ratio.
¶ Further adjusted for coffee consumption and intake of protein, rice, wholemeal bread, white bread, fruits, vegetables, cholesterol and ratio of PUFA:SFA.
** Participants with diagnosed dyslipidaemia were excluded from HDL-cholesterol and TAG analyses (remaining sample size *n* 1978, whereby n_1 373, n_2 407, n_3 405, n_4 405 and n_5 388 for quintiles 1–5, respectively).

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prepared from refined grains are commonly consumed. Also, noodle-based dishes are often prepared with lard and typically contain high amounts of Na and cholesterol. It is possible that addition of these ingredients to noodles during their preparation exacerbates the adverse effects of refined grain on metabolic outcomes. In a cohort of older Singaporean-Chinese, noodle consumption was found to be part of a dietary pattern comprising other less healthy food choices including red meats and deep-fried foods, which is associated with an increased risk of type 2 diabetes[42]. In the present study, noodle consumption was found to be correlated with a higher intake of cholesterol, a lower intake of total fibre and vegetables and a lower PUFA:SFA ratio, suggesting that noodle consumption may serve as a marker of unhealthy dietary choices in this population. In the present study, rice consumption was found to be associated with a lower consumption of vegetables, but not with the consumption of other unfavourable food items. However, associations of both rice and noodle consumption with metabolic risk factors remained after adjustment for dietary risk factors.

It has been postulated that the adverse metabolic effects of high carbohydrate intakes on glucose and lipid metabolism are stronger in insulin-resistant individuals than in insulin-sensitive individuals[41] and may thus be stronger in overweight individuals than in lean individuals[39]. In Asian populations, however, both stronger[10] and weaker[11,43] associations for glycaemic index, glycaemic load and high rice consumption have been observed for overweight individuals than for leaner individuals. In the present study, the association between rice consumption and HOMA-IR tended to be more pronounced among non-overweight participants, whereas the association between noodle consumption and HOMA-IR was more pronounced among overweight participants. Taken together, results regarding possible effect modification of the association between refined grain consumption and metabolic health outcomes by overweight status have not been consistent.

The specific biological mechanisms by which increased consumption of refined grains such as white rice and refined grain noodles may contribute to insulin resistance remain to be elucidated. Metabolic studies have shown that both the glycaemic index of foods and the amount of carbohydrates consumed contribute to the glycaemic response to a meal[40]. The glycaemic load reflects both the quality and quantity of carbohydrate intake. Commonly consumed rice varieties in this population such as Jasmine rice and glutinous rice typically have high glycaemic index values[44]. Data on the glycaemic index values of Asian types of noodles are limited. Published values are usually in the intermediate range (40–66 units, using glucose as a standard), although some types of noodles such as fresh wheat noodles have a high glycaemic index of 82[44,45]. Given the high carbohydrate content of both rice and noodles, their consumption can contribute to a higher dietary glycaemic load, which has been reported to be associated with a higher risk of type 2 diabetes in several[10,14–16] but not in all[23] studies. Consumption of foods with a high glycaemic load leads to high postprandial glucose and insulin concentrations. The resulting peak in postprandial glucose concentrations is typically followed by a rapid decline in glucose concentrations, which may trigger the secretion of counter-regulatory hormones and reduce the suppression of circulating NEFA concentrations. Chronic elevations in postprandial concentrations of insulin, counter-regulatory hormones and NEFA may contribute to insulin resistance[20].

Another possible explanation for the association between rice and noodle consumption and hyperglycaemia lies in the processing of the refined grains. This process removes most of the bran and some of the germ, resulting in the loss of various components that may be beneficial for glucose metabolism such as minerals, lignans and phenolic compounds[7]. Although we controlled for other dietary risk factors in the multivariable models in the present study, the possibility remains that high rice and noodle consumption contributes to metabolic disturbances by replacing foods that have a beneficial effect on glucose metabolism.

The strengths of the present study include the population-based selection of participants, the reasonably large sample size, and the detailed information on dietary intakes and potential confounders based on validated questionnaires. However, the study also has several limitations. First, the cross-sectional nature of the study did not allow us to determine the direction of effects. However, we excluded individuals with known diabetes or CVD. It seems unlikely that the remaining participants were aware of their glycaemic blood markers and that the concentrations of these biomarkers affected food choices. Second, measurement error in the assessment of dietary intakes was inevitable. However, this is unlikely to explain the observed associations in the present study, as this would have weakened rather than strengthened the associations. We did not distinguish between whole-grain noodles and brown rice in our questionnaire. However, in the Singaporean population, as in most Asian populations[4,5], consumption of brown rice and whole-grain noodles was rare at the time of the study. Our findings for rice consumption and metabolic risk factors can, therefore, be assumed to reflect associations for high white rice and refined grain noodle consumption. Finally, although we considered a wide range of potential confounders, residual confounding by unmeasured or imperfectly measured confounders may still have affected the results of the study.

In several cohort studies, high white rice consumption has been reported to be associated with a higher risk of type 2 diabetes. The results of the present study suggest that this association may be mediated by detrimental effects of high rice consumption on insulin sensitivity, rather than by the effects on systemic inflammation. We observed that high consumption of noodles may be at least as detrimental for metabolic risk factors as high consumption of white rice. Previous studies in Asian population have mainly focused on white rice, but attention on noodles, which are frequently consumed by many East Asian and South East Asian populations, is also warranted. If these detrimental effects of high white rice and noodle consumption on glucose metabolism are confirmed in further epidemiological studies and randomised trials, it could have major implications for Asian populations that have very high refined grain intakes and a rapidly increasing burden of type 2 diabetes[1].

Supplementary material

To view supplementary material for this article, please visit http://dx.doi.org/10.1017/S0007114513003486

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The authors' contributions are as follows: Y. L. M. Z. had the primary responsibility for writing the manuscript; S. A. R. contributed to the analytical design and revised and edited the manuscript; P. L. O. and H. Z. analysed the data and edited the manuscript; J. L. and E. S. T. collected the data; R. M. V. D. developed the analytical plan, interpreted the results, contributed to manuscript writing, reviewed the manuscript, and directed the study. All the authors read and approved the final version of the manuscript.

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