Original Article

Diurnal differences in glycemic responses, insulin responses and cognition after rice-based meals

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Background and Objectives: The variation in glycemic responses to white rice caused by the circadian rhythm has been widely investigated but remain controversial. This study investigated diurnal differences in the effect of rice meals on glycemic responses, insulin responses, satiety, and acute cognitive function. **Methods and Study Design:** A total of 20 healthy participants in Group 1 and 14 in Group 2 were served identical servings of cooked white rice containing 50 g of available carbohydrates at 8:00 a.m. (rice at breakfast), 12:30 p.m. (rice at lunch), and 5:00 p.m. (rice at early supper) in a randomized order. Postprandial blood glucose, insulin, satiety, and cognitive performance tests were conducted for each test meal. **Results:** The rice at an early supper elicited significantly milder glycemic responses than did the rice at lunch and resulted in a lower insulin sensitivity than did rice at breakfast. No difference was observed among the test meals in terms of hunger and prospective food intake. Diurnal acute cognitive performance did not differ considerably among the meals. A correlation analysis indicated that low variability in glycemic responses was positively associated with superior cognitive performance. **Conclusions:** A high–glycemic index white rice supper at 5:00 p.m. may facilitate daily glycemic management.

Key Words: glycemic response, diurnal rhythm, white rice, cognitive function, satiety

INTRODUCTION

White rice is among the most common staple foods in East and Southeast Asian countries, and its health effects have been widely investigated yet remain controversial.[1,2] A study demonstrated that individuals who consumed white rice two to three times per week had a lower risk of diabetes than did those who rarely consumed white rice,[3] whereas other studies have observed a positive[4-6] or null[7,8] association between white rice intake and the subsequent incidence of type 2 diabetes. Nevertheless, strategies to control the glycemic response (GR) to polished rice for preventing and managing type 2 diabetes should be developed.

The circadian system regulates metabolism through daily 24-h cycles and plays a major role in regulating glucose, lipid, and energy metabolism.[9,10] Meals consumed in the morning (7:00 a.m.) elicit a milder GR than do those consumed in the afternoon (1:00 p.m.) and evening (7:00 p.m.).[11] A meta-analysis of feeding trials reported that unlike meals consumed early in the day, late-night eating negatively affected the GR, insulin response (IR), and glucose tolerance.[12] In addition, surveys have demonstrated that late-night suppers (8:00 p.m. and later)[13] and higher energy, protein, and fat intake at supper than at breakfast[14] increase the risk of hyperglycemia and type 2 diabetes. However, the diurnal pattern of rice in terms of the GR and IR is yet to be explored.

A moderate increase in the blood glucose concentration is associated with improved learning and memory, partly because of the increased passage of glucose to the brain,[15,16] and a stable glycemic state plays a key role in the prevention of cognitive dysfunction.[17,18] Individuals with long-term impaired blood sugar regulation have an increased risk of Alzheimer's disease and cognitive impairment.[19] However, whether this association is affected by the circadian rhythm is yet to be determined.

In most studies, supper has been scheduled late at night (7:00–8:00 p.m.),[20-23] and the GR to early white rice–based suppers (~5:00 p.m.) has rarely been reported. This study investigated the diurnal difference in the glycemic, insulin, and acute cognitive effects of rice meals. In addition, subjective appetite was evaluated to identify possible side effects. We assumed that (1) a white rice–based supper at 5:00 p.m. would not negatively affect the GR, IR, and subjective appetite and that (2) circadian rhythm would determine the effects of white rice–based meals on acute cognitive performance.

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Manuscript received 26 January 2022. Initial review completed 08 February 2022. Revision accepted 25 February 2022.
doi: 10.6133/apjcn.202203_31(1).0007

METHODS

Participant recruitment

Healthy university students aged 20–25 years with a BMI between 18.5 and 24 kg/m^2, a regular sleep–wake cycle, bedtime between 10:00 p.m. and 12:00 a.m., and a regular menstrual cycle (if female) were recruited. Those with a diagnosis of genetic or metabolic diseases (diabetes, impaired glucose tolerance, and hypertension); irregular sleep or eating schedules; dependency on alcohol, tobacco, or drugs; the use of medications or supplements known to affect sleep, the circadian rhythm, or metabolism; eating disorders (bulimia, anorexia nervosa, and binge eating); or participation in competitive or endurance sports were excluded. All eligible individuals who passed duplicated oral glucose tolerance tests provided written informed consent.

Ethics and design

This randomized controlled crossover study was approved by the Ethics Committee of China Agricultural University (ethics number CAUHR-2021011), registered on the Chinese Clinical Trial Registry (ChiCTR2100050541), and conducted in full compliance with the Helsinki Declaration.

Each participant was assigned to three test sessions, with each session being separated by at least 3 days in a randomized order. All test sessions lasted approximately 5 h and were identical in all respects except for food ingestion time. During the sessions, the participants were served meals with cooked white rice at 8:00 a.m. (rice at breakfast, RB), 12:30 p.m. (rice at lunch, RL) and 5:00 p.m. (rice at early supper, RES). The participants were required to arrive at the laboratory 30 min before meal time. They were instructed to schedule and record their daily diet during the test sessions. The trial consisted of two groups. In Group 1, the diurnal postprandial and subsequent-meal glycemic effects were assessed through continuous glucose monitoring along with subjective appetite and acute cognitive function. In Group 2, the diurnal glycemic and insulin effects were investigated through blood collection. The participants were required to follow an identical meal plan the day prior to and after the test sessions.

Test meals

The administered glucose solution contained 55.6 g of dextrose monohydrate powder diluted in 300 mL of water. The meal included cooked rice containing 50 g of available carbohydrates and 184.1 mL of water for weight balance. The rice was prepared in an electric pressure cooker (MY-HT5093, Midea, China). Each serving was prepared using 66.1 g of polished rice (Oryza sativa spp. Japonica, Heilongjiang, China) and 132.2 mL of water. The meals were cooked before each session, immediately served to the participants, and consumed within 15 min to prevent the retrogradation of starch.

Continuous glucose monitoring

Continuous glucose monitors (CGMs; Abbott, Shanghai, China) were inserted under the participants' skin 2 days before the first test and removed 24 h after the last test. The participants were instructed to use the sensor at least once every 8 h in accordance with the manufacturer's instructions. Sensor data were retrospectively stored every 15 min, and occasional missing values (<0.06% of all data) were imputed by averaging adjacent values.[24]

Subjective appetite and acute cognitive function

Subjective appetite, comprising satiety, fullness, hunger, desire to eat, and prospective food intake, was assessed using a visual analogue scale[25,26] before each test meal and at 15, 30, 45, 60, 90, 120, 150, 180, 240, and 270 min after meal ingestion (Figure 1). The Hopkins Verbal Learning Test (HVLT) was used to test short-term listening and memory,[27] the Map Test (MT) was used to test spatial memory,[28] and the visual recall test (VRT) was used to test memory[28] at 30 min before meals and at 60 and 210 min after meal ingestion.

Plasma chemistry

Blood for insulin analysis was obtained through finger pricking by using a sterile, single-use lancing device (Meisheng, China). The participants were encouraged to warm their hands in supplied water before finger pricking and to massage them from the bottom of the palm toward the fingertips to increase blood flow. Before each meal and 15, 30, 45, 60, 90, and 120 min after meal ingestion (Figure 1), 150 μL of capillary blood was collected in Microvette capillary blood collection tubes treated with dipotassium ethylenediamine tetraacetic acid (Lihui Inc., Jiangsu, China) and stored in crushed ice immediately thereafter. The last blood drop was retained for a glucose assay performed using a glucometer (LifeScan Inc., Milpitas, CA, USA). Within 30 min after blood collection, the Microvette tubes with the blood were centrifuged at 1000 × g for 15 min, and 50 μL of the supernatant plasma was pipetted into 0.5-mL Eppendorf tubes (Biosharp Inc., Anhui, China) and stored in a freezer at −80℃ until analysis. The insulin concentration was determined using an ELISA-based test kit (Dogesce Inc., Beijing, China) in accordance with the manufacturer's instructions.

Data processing and statistical analysis

The sample size was verified through calculations in PASS 13 Power Analysis and Sample Size software (NCSS, Kaysville, UT, USA) on the basis of a study in which a 36% reduction in the glycemic index (GI) was

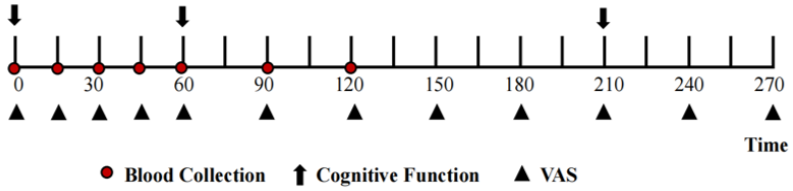

Figure 1. Study design diagram.

observed for whole grain oats and cooked rice compared with the glucose control.[29] If the standard deviation (SD) was assumed to be <15.80, the test would have 85% power to examine a difference (p<0.05) among 13 participants for a GI of 36.

Glycemic and insulin variability were evaluated in terms of the maximum amplitudes of glucose excursion (MAGE),[30] the incremental peak (ΔPeak) and low (ΔLow) of glucose and insulin concentrations, and the positive increments under the curve of GRs and IRs (iAUC).[31] To estimate insulin sensitivity (IS), the homeostatic model of β-cell function (HOMA-B) was calculated as follows: (20 × fasting insulin)/ (fasting glucose − 3.5).[32] IS indices (ISIs) were calculated as follows: 10,000/square root of (fasting glucose × fasting insulin × mean glucose × mean insulin).[33] The insulin secretion sensitivity index-2 (ISSI-2) was calculated as follows: (AUCins/AUCgluc) × ISI.[34]

The results are presented as means (standard errors [SEs]) unless otherwise noted. The Kolmogorov–Smirnov test was performed to check for normal distributions prior to analysis, and a natural logarithmic transformation was used when data were nonnormally distributed. Differences between treatments were identified through ANOVA with Duncan's multiple range test, and statistical significance was set at p<0.05. Correlations among the data were identified through a Pearson's correlation analysis. Statistical analysis was performed using SPSS (version 21.0, SPSS Inc. Chicago, IL, USA).

RESULTS

Participant characteristics

A total of 20 participants in Group 1 and 14 in Group 2 passed the screening and completed all the tests. No adverse events were reported during the test sessions, and all data were included in the analysis. Table 1 lists the participants' baseline characteristics.

GRs from CGMs

Figure 2 presents the GRs to the rice meals indicated by the CGMs. RES led to significantly lower glucose levels at 15, 30, 45, 60, 165, 180, 195, 225, 240, and 270 min than did RB. The GRs to RES were considerably lower than those to RL during the sessions. No significant difference between RB and RL was observed.

Figure 3 presents the GRs to the subsequent and routine meals in Group 1. The lunch after the test breakfast meal elicited significantly higher glycemic increments than did the routine lunch at 15 and 30 min. The blood glucose increments of the supper after the test lunch meal were higher than those of the routine supper at 45, 60, and 75 min. The breakfast after the test supper meal led to an advanced peak and higher glucose increments at 30 and 45 min and lower glucose increments from 90 to 150 min than did the routine breakfast.

GRs and IRs in blood tests

Figure 4 presents the GRs and IRs to the test meals in Group 2. RES elicited significantly lower glycemic in-

Table 1. Participant baseline characteristics

Characteristics	Group 1		Group 2	
	Mean	SD	Mean	SD
Number of participants (male/female)	20 (8/12)		14 (5/9)	
Age (year)	21.8	1.7	22.5	1.9
Body height (cm)	168.3	8.7	166.5	8.0
Body weight (kg)	61.5	10.4	57.4	7.5
BMI (kg/m^2)	21.6	2.2	20.7	2.1
Fat mass (%)	26.0	6.7	25.2	5.5
Basal metabolism rate (BMR) (kcal/day)	1354.2	198.1	1298.5	152.2

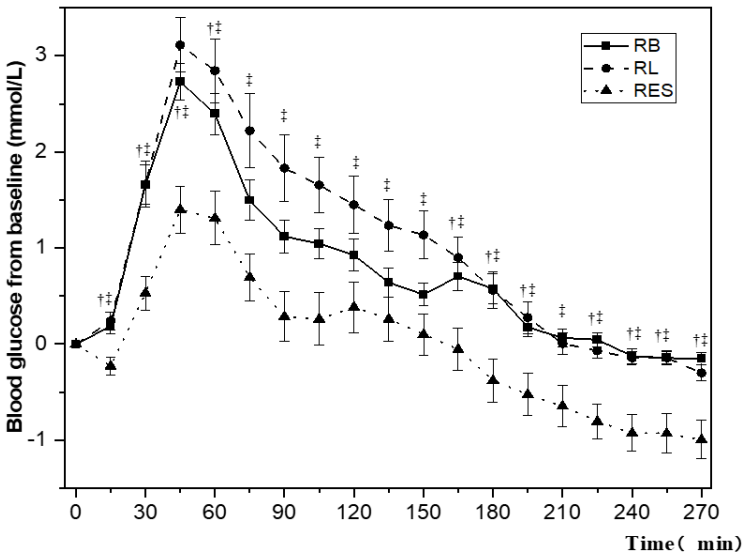

Figure 2. GRs to rice meals (n=20). RB: rice ingested at breakfast (8:00 a.m.); RL: rice ingested at lunch (12:30 p.m.); RES: rice ingested at early supper (5:00 p.m.). †Differences between RB and RES, ‡differences between RL and RES (p<0.05). Values are presented as means, with SE represented by vertical bars.

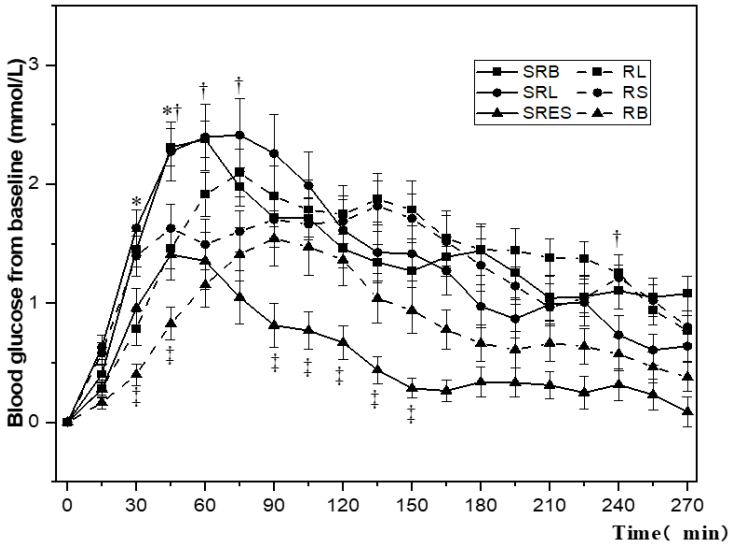

Figure 3. GRs to subsequent and routine meals (n=20). SRB: subsequent meal of rice ingested at breakfast; SRL: subsequent meal of rice ingested at lunch; SRES: subsequent meal of rice ingested at supper; RL: routine lunch; RS: routine supper; RB: routine breakfast. Values are presented as means, with SEs represented by vertical bars. *Differences between SRB and RL, †differences between SRL and RD, ‡differences between SRES and RB (p<0.05).

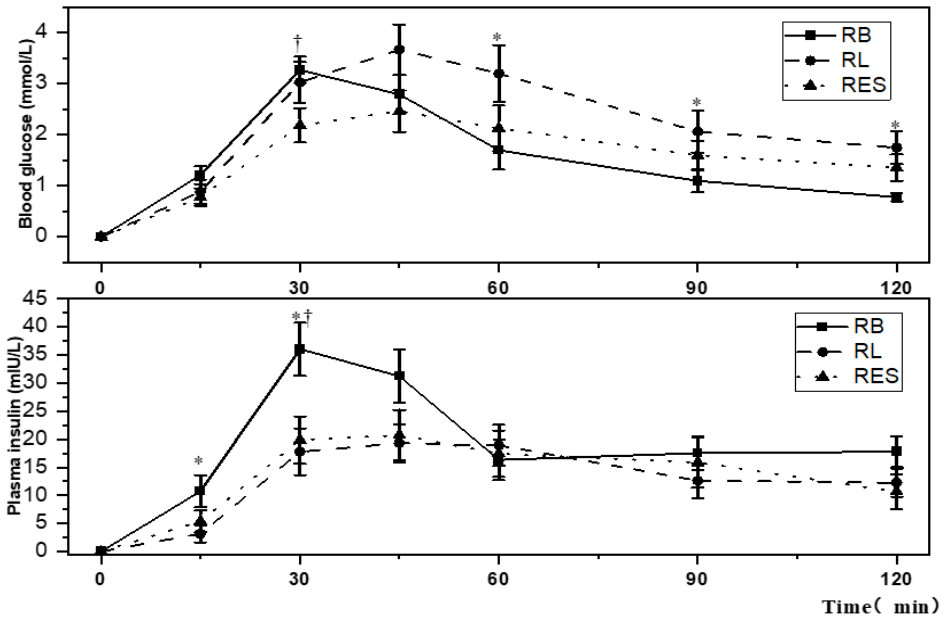

Figure 4. Changes in blood glucose and plasma insulin from baseline for test foods (n=14). RB: rice ingested at breakfast (8:00 a.m.); RL: rice ingested at lunch (12:30 p.m.); RES: rice ingested at supper (5:00 p.m.). Values are presented as means, with SEs represented by vertical bars. *Differences between RB and RL, †differences between RB and RES (p<0.05).

crements than did RB at 30 min, and RB led to lower glucose increments at 60, 90, and 120 min than did RL. The insulin increments of RB were higher than those of RL and RES at 30 min and higher than those of RL at 15 min. No difference in insulin increments between RL and RES was observed.

Table 2 displays the glycemic and insulin variability indices for the test meals. The glycemic peak value, MAGE, and SD of RES were significantly lower than those of RL. RB resulted in higher insulin peak values than did RL and RES. RES increased insulin sensitivity more than did RB in terms of ISI.

Subjective appetite

The RES satiety increments at 90 and 120 min were significantly lower than those of RB and those from 90 to 270 min in relation to those of RL (Figure 5). The fullness of RL was higher than that of RES at 180, 240, and 270 min. The desire to eat for RB was lower than that for RL at 45, 60, and 90 min and lower than that for RES at 120 min. No difference among the test meals in terms of hunger and prospective food intake was observed.

Acute cognitive function

Table 3 presents the VRT results before and after lunch. Significantly more correct numbers in the VRT were observed at −30 min than at 60 min. No significant difference in the VRT results was observed between the breakfast and supper tests or in the results of the MT.

Table 4 presents the results of HVLT before and after

Table 2. Glycemic and insulin variability indices for test meals (mean values and SEs, n=14)

	RB		RL		RES	
	Mean	SE	Mean	SE	Mean	SE
Blood glucose						
ΔPeak (mmol/L)	3.7†‡	0.3	4.2†	0.4	2.8‡	0.4
ΔLow (mmol/L)	-0.1†	0.0	-0.1†	0.1	-0.0†	0.0
MAGE (mmol/L)	3.8†‡	0.3	4.4†	0.3	2.9‡	0.4
SD	1.4†‡	0.1	1.6†	0.1	1.1‡	0.1
$iAUC_{0\text{-}120}$ (mmol/L·min)	192.6†	18.9	277.8†	36.5	197.6†	30.2
Plasma insulin						
ΔPeak (mIU/L)	44.0†	4.1	27.5‡	3.8	29.0‡	4.7
$iAUC_{0\text{-}120}$ (mIU/L·min)	2330.3†	222.6	1598.3†	243.8	1724.2†	345.5
Insulin sensitivity						
HOMA-B	86.8†	6.3	81.1†	6.3	80.8†	5.9
ISI	20.5‡	1.5	23.5†‡	3.0	31.6†	5.3
ISSI-2	267.5†	35.0	203.1†	72.3	315.8†	91.5

RB: rice ingested at breakfast (8:00 a.m.); RL: rice ingested at lunch (12:30 p.m.); RES: rice ingested at supper (5:00 p.m.).
Values are the mean glycemic characteristics of the test meals with their SE.
†‡Significant differences among test meals ($p<0.05$).

Table 3. VRT results before and after lunch (mean values and SE, n=20)

Time (min)	Correct number in VRT (n)
-30	13.21±0.91†
60	9.16±0.81‡
210	12.32±1.06†‡

VRT: Visual Recall Test.
Values are the mean test results with SEs.
†‡Significant differences among time points ($p<0.05$).

each meal. The FT error at −30 min was higher than that at 60 and 210 min for lunch, and the TT error at −30 min was higher than that at 210 min. No differences among the tests at individual time points were observed, but omissions showed a decreasing trend.

Correlation analysis

The correlation analysis indicated that the glycemic MAGE had a significant and positive correlation with the number of errors in three HVLTs and with the number of category C errors at −30 min in the MT (Table 5). The glycemic peak value and $iAUC_{0\text{-}270}$ were positively correlated with the number of category B errors at 60 min in the MT. A negative correlation between $iAUC_{0\text{-}270}$ and the correct number at 60 min in the VRT was observed.

DISCUSSION

The early (5:00 p.m.) supper elicited a milder GR than did the lunch (12:30 p.m.) and reduced IS more than did the breakfast (8:00 a.m.) with the same amount of white rice containing 50 g of available carbohydrates. The satiety responses and diurnal acute cognitive performance did not differ considerably among meals. However, the results suggested that stable GRs were positively associated with superior cognitive performance.

The CGM and capillary blood tests consistently indicated that RES produced lower ΔPeak (−1.9 mmol/L for CGM and −1.4 mmol/L for plasma blood) and MAGE (−1.2 mmol/L for CGM and −1.5 mmol/L for plasma blood) values than did RL. In addition, RES led to a lower insulin ΔPeak and higher IS than did RL.

The results suggested that if served early, a high-carbohydrate supper may not necessarily lead to a considerable surge in blood glucose or low insulin sensitivity, as observed in other studies.[12] The inconsistency between the results of this study and those of others can be explained by several factors.

First, the prescribed supper times in other studies have been 7:00 p.m.,11 7:30 p.m.,[21] 8:00 p.m.,[22] and 10:00 p.m.,[23] whereas it was 5:00 p.m. in this study. One study demonstrated that the GRs to supper at 6:00 p.m. were significantly lower than those to supper at 10:00 p.m.; thus, earlier supper time may lead to more stable GRs.[23]

Second, unlike meals in others studies, which consisted of multiple foods, the meals in this study consisted of rice and were thus low protein, low fat, and high carbohydrate. One study reported that consuming mostly protein during the day and mostly carbohydrates at night for 8 weeks led to a nonsignificant decrease in fasting blood glucose in individuals with overweight and obesity.[35] An epidemiological study reported that consuming more total energy, total fat, and protein but not carbohydrate at supper than at breakfast increased the risk of diabetes, cardiovascular disease, and all-cause mortality.[36] Studies have attributed the hypoglycemic effect of high-GI suppers to an increase in insulin levels.[37] However, in this study, the postprandial insulin level of RES was lower, and the IS was higher than that of RB.

Third, this study limited the window of mealtime to a 9-h period. An early time-restricted feeding pattern might have contributed to stable GRs at night.[38] Because most adults work from 9:00 a.m. to 5:00 p.m., the only practical window for mealtime would be breakfast at 8:00 a.m. to supper at 5:00 a.m. In addition, dinner service at uni-

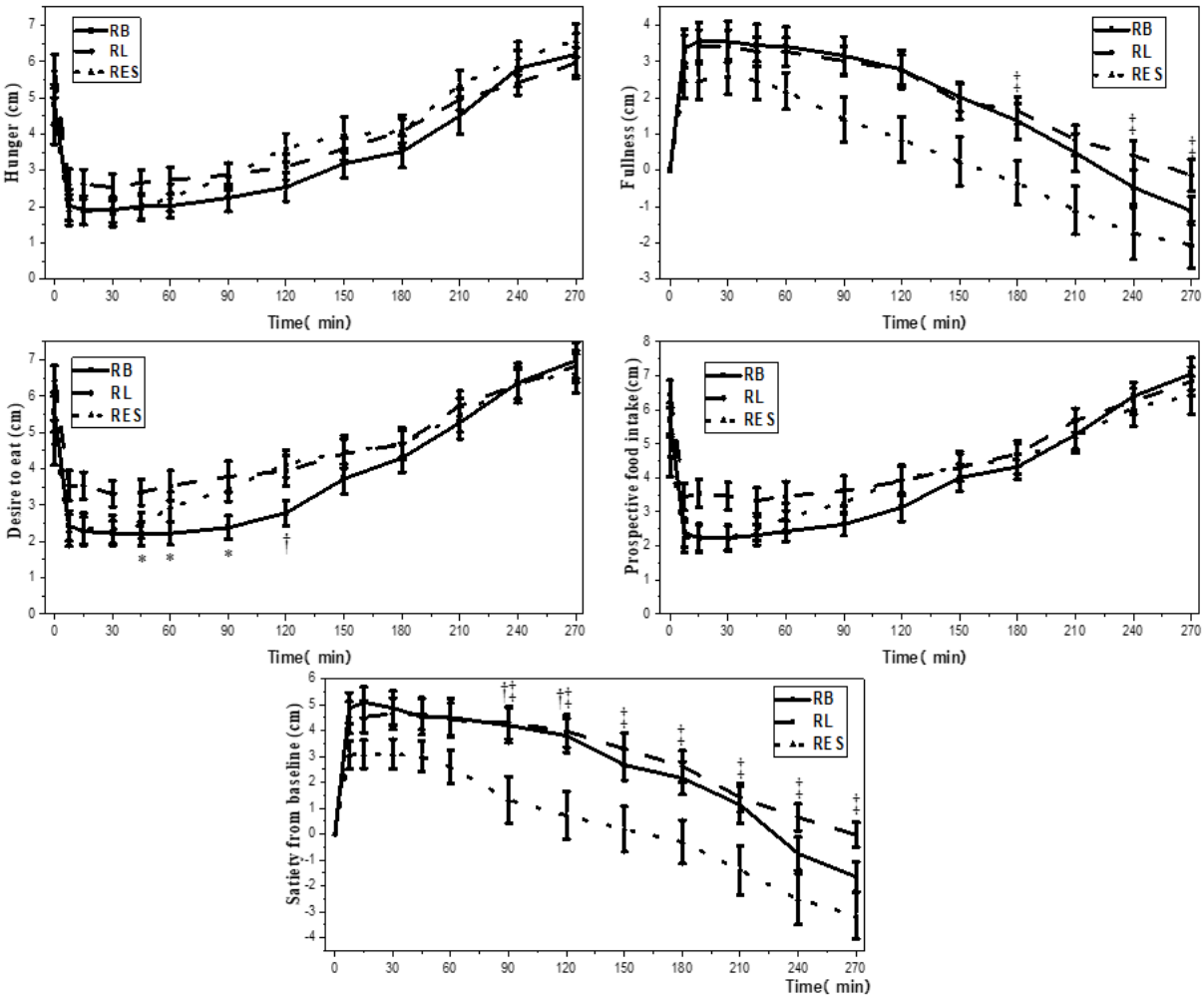

Figure 5. Changes in satiety from baseline for test foods assessed using a visual analogue scale (n=20). RB: rice ingested at breakfast (8:00 a.m.); RL: rice ingested at lunch (12:30 p.m.); RES: rice ingested at supper (5:00 p.m.). *Differences between RB and RL, †differences between RB and RES, ‡differences between RL and RES (p<0.05).

versity cafeterias and restaurants usually begins at 5:00 p.m.

When comparing GRs to the subsequent and routine meals, we observed that GRs to the supper after the test lunch were more severe than those to the routine suppers, whereas GRs to the breakfast after the test supper did not differ considerably from those to the routine breakfast. This result raised questions regarding the second-meal effect of high-GR meals[39] because the test meal at lunch led to the most severe GRs. Strong GRs after lunch might explain sleepiness after lunch; one study revealed that those without diabetes who nap during the day have high HbA1c levels and strong IRs.[40] The possible effects of lunchtime carbohydrate intake on postprandial GRs and after-meal drowsiness must be investigated.

Studies have demonstrated that early suppers do not significantly increase hunger scores.[41] In this study, the correlation analysis indicated a null association between GRs and satiety indicators. Although the GRs of RES were significantly lower, the three test meals resulted in similar levels of self-reported desire to eat. Therefore, a stable postprandial blood glucose pattern may help prevent hunger at night.[42]

No significant diurnal difference in acute cognitive function was observed, but the correlation analysis revealed that severe postprandial glycemic fluctuations were correlated with decreased short-term listening, spatial memory, and graphical memory. Studies have indicated that the key factor determining the effect of glucose on cognitive ability is not the concentration of glucose but the glycemic pattern after the release of glucose.[43,44] Variability in glycemic levels (rather than the absolute concentration of blood glucose) is crucial to the regulation of cognitive function.[45]

To the best of our knowledge, this is the first study to investigate diurnal differences in the effects of white rice meals on GRs, IRs, and acute cognitive function. Because white rice is a global staple food, the GRs, proper consumption time, and cognitive effects of white rice should be investigated. This study revealed that scheduling suppers at 5:00 p.m. and limiting mealtimes to a 9-h window could provide glycemic solutions for high-GI food.

Because this study was conducted as an acute trial in healthy young volunteers, the applicability of the results to those with impaired glucose tolerance, diabetes, and other health conditions must be determined. The long-term effects and underlying mechanisms should be investigated through longer interventions and analyses of hormones such as glucagon-like peptide-1 and gastric inhibitory polypeptide.

Table 4. Results of HVLT before and after breakfast, lunch and dinner (mean values and SE, n=20).

Time (min)	FT error (n)	FT omission (n)	ST error (n)	ST omission (n)	TT error (n)	TT omission (n)
Breakfast						
-30	0.37±0.14†,††	4.42±0.40†,§	0.21±0.10†,††	1.79±0.44†,¶	0.11±0.07†,††	0.37±0.16†,††
60	0.37±0.16†,††	5.47±0.28†,§	0.23±0.13†,††	2.58±0.48†,¶	0.21±0.10†,††	1.00±0.28†,††
210	0.21±0.10†,‡‡	5.26±0.33†,§	0.21±0.10†,‡‡	1.95±0.36†,¶	0.16±0.09†,‡‡	0.95±0.31†,††
Lunch						
-30	0.47±0.12†,††	5.42±0.54†,§	0.37±0.11†,††	2.32±0.47†,¶	0.32±0.11†,††	1.21±0.38†,††
60	0.05±0.05†,††	5.05±0.46†,§	0.11±0.07†,††	2.21±0.52†,¶	0.16±0.09†,††	1.32±0.39†,¶
210	0.16±0.12‡,‡‡	5.68±0.32†,§	0.32±0.13†,‡‡	2.58±0.38†,¶	0.26±0.15†,‡‡	1.21±0.42†,††
Supper						
-30	0.21±0.10†,‡‡	6.26±0.23†,§	0.26±0.10†,‡‡	2.79±0.35†,¶	0.42±0.16†,‡‡	1.42±0.25†,††
60	0.21±0.10†,††	4.84±0.44‡,§	0.21±0.12†,††	1.68±0.47†,¶	0.21±0.12†,‡,††	1.11±0.43†,¶,††
210	0.16±0.09†,‡‡	6.05±0.35†,§	0.11±0.07†,‡‡	2.74±0.41†,¶	0.05±0.05†,‡‡	1.58±0.41†,††

FT error: the error number in the first test; FT omission: the omission number in the first test; ST error: the error number in the second test; ST omission: the omission number in the second test; TT error: the error number in the third test; ST omission: the omission number in the third test.
Values are the mean test results with SE.
†,‡ Significant differences among time points (p<0.05).
§,¶,††,‡‡ Significant differences among the tests at the same time point (p<0.05).

Table 5. Correlation between postprandial glucose and satiety in supper.

Glycemic characterastics	-30min FT error in HVLT (n)	210min ST error in HVLT (n)	210min TT error in HVLT (n)	-30minC-error in MT (n)	60minB-error in MT (n)	60min correct number in VRT (n)
ΔPeak (mmol/L)	0.935	0.935	0.945	0.915	1.000*	-0.993
MAGE (mmol/L)	1.000*	1.000*	1.000**	0.998*	0.948	-0.974
$iAUC_{0\text{-}270}$ (mmol·min/L)	0.958	0.958	0.966	0.941	0.999*	-0.999*

FT error: the error number in the first test; ST error: the error number in the second test; TT error: the error number in the third test; C-error: area name wrongly recalled; B-error: correct name placed in the wrong area.
$^{*}p$<0.05, ** p<0.01.

Conclusion

Our study revealed that consuming white rice containing 50.0 g of available carbohydrates at 5:00 p.m. for supper resulted in stable GRs, increased insulin sensitivity, and similar satiety level, subsequent-meal GRs, and cognitive performance. High–glycemic index white rice suppers at 5:00 p.m. may facilitate daily glycemic management.

ACKNOWLEDGEMENTS

We sincerely thank all participants for their time and cooperation.

AUTHOR DISCLOSURES

The authors declare no conflicts of interest and that this research received no external funding.

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