Effects of chili consumption on postprandial glucose, insulin, and energy metabolism[1–3]

Kiran DK Ahuja, Iain K Robertson, Dominic P Geraghty, and Madeleine J Ball

ABSTRACT

Background: Animal and some human studies have indicated that the consumption of chili-containing meals increases energy expenditure and fat oxidation, which may help to reduce obesity and related disorders. Because habitual diets affect the activity and responsiveness of receptors involved in regulating and transporting nutrients, the effects of regular consumption of chili on metabolic responses to meals require investigation.

Objective: The objective was to investigate the metabolic effects of a chili-containing meal after the consumption of a bland diet and a chili-blend (30 g/d; 55% cayenne chili) supplemented diet.

Design: Thirty-six subjects with a mean (±SD) age of 46 ± 12 y and a body mass index (in kg/m^2) of 26.3 ± 4.6 participated in a randomized, crossover, intervention study with 2 dietary periods (chili and bland) of 4 wk each. The postprandial effects of a bland meal after a bland diet (BAB), a chili meal after a bland diet (CAB), and a chili meal after a chili-containing diet (CAC) were evaluated. Serum insulin, C-peptide, and glucose concentrations and energy expenditure (EE) were measured at fasting and up to 120 min postprandially.

Results: Significant heterogeneity was observed between the meals for the maximum increase in insulin and the incremental area under the curve (iAUC) for insulin ($P = 0.0002$); the highest concentrations were with the BAB meal and the lowest with the CAC meal. When separated at the median BMI (26.3), the subjects with a BMI ≥ 26.3 also showed heterogeneity in C-peptide, iAUC C-peptide, and net AUC EE ($P < 0.02$ for all); the highest values occurred after the BAB meal and the lowest after the CAC meal. Conversely, the C-peptide/insulin quotient (an indicator of hepatic insulin clearance) was highest after the CAC meal ($P = 0.002$).

Conclusion: Regular consumption of chili may attenuate postprandial hyperinsulinemia. *Am J Clin Nutr* 2006;84:63–9.

KEY WORDS Insulin resistance, chili, obesity, energy expenditure, postprandial effects

INTRODUCTION

Herbs and spices are natural food additives that contribute significantly to the taste and flavor of our food. In some cultures, these herbs and spices have also been used as medicines and preservatives. Research over the past 3 decades has indicated several potential beneficial health effects of spices (eg, turmeric, cinnamon, garlic, and chili), especially concerning lipid metabolism, diabetic control, digestive function, and antioxidative potential (1–9). Chili and its active principle capsaicin reduce adiposity in rats by enhancing energy and lipid metabolism, possibly by increasing catecholamine secretion from the adrenal medulla through the activation of the sympathetic nervous system (SNS) (10, 11).

Obesity generally occurs as a result of increased energy intake and decreased energy expenditure (EE) and is a major contributor to the development and progression of disorders such as insulin resistance, hyperinsulinemia, type 2 diabetes, and cardiovascular disease. Adding chili or red pepper to meals reduces the cumulative ad libitum energy and macronutrient intake in men and women (12–14). Acute (postprandial) studies with meals containing chili in lean young subjects have shown an increase in EE (15, 16), increased fat oxidation in women (16), or increased carbohydrate oxidation in men (17, 18). An increase in plasma epinephrine and norepinephrine after consumption of a single chili meal has also been reported (17). Conversely, a study comparing the effects of a chili-containing meal (3 mg capsaicin) with those of a bland meal in lean and obese women reported an increase in SNS activity (as measured by heart rate variability spectral analysis) and EE in lean but not in obese women (19).

The abovementioned acute studies investigated the effects of a single chili-containing meal in small groups ($n = 8–13$) of lean young subjects (mean age: 20–26 y) with a mean body mass index (BMI; in kg/m^2) of 22−24 after control for background diets for a maximum of 2 d (13, 15, 16). Because medium- and long-term habitual diets may affect the activity and responsiveness of receptors involved in the regulation and transportation of nutrients, we investigated the acute effects of chili meals with those of 2 different types of background diets, namely bland (spice-free) and chili-containing diets in a larger group of men and women with wider age (22–70 y) and weight (BMI: 18–35) ranges. The aim of this study was to examine the effects of a chili meal after 4 wk of a bland (spice-free) diet and of a chili meal after 4 wk of regular consumption of chili (chili diet) on plasma glucose, serum insulin, C-peptide, and EE and to compare the responses with those of a bland meal after a bland diet.

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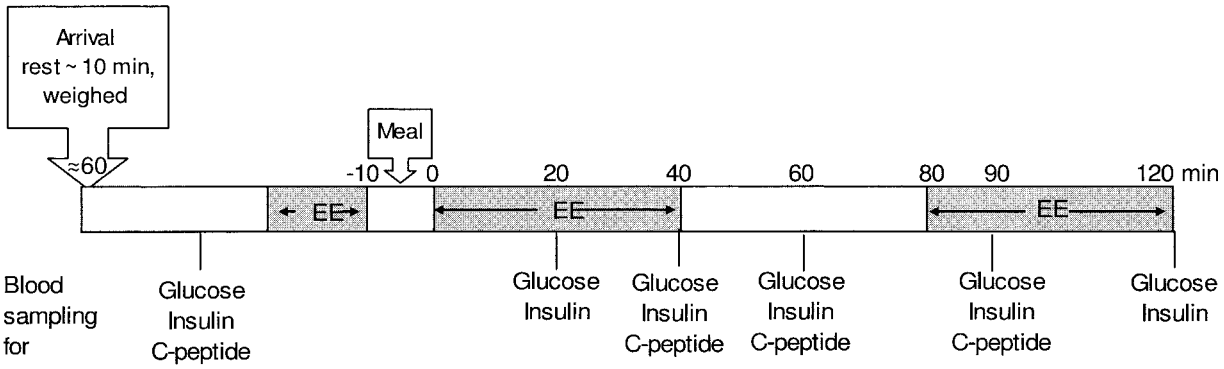

FIGURE 1. Test meal protocol.

SUBJECTS AND METHODS

Subjects

Thirty-six men and women aged 22–70 y were recruited through advertisements in local newspapers and university newsletters. The subjects were nonsmokers; had no known or self-reported history of diabetes or heart, renal, or hepatic disease; were not taking any prescription medications; and were not everyday consumers of chili. The study (H7437) was approved by the Northern Human Medical and Research Ethics Committee of Tasmania, Australia. All participants provided written informed consent.

Study design

Each participant completed two 4-wk dietary periods (bland and chili) in a randomized crossover design. A randomization sheet was obtained by using GRAPHPAD (Internet: http://www.graphpad.com/quickcalcs/RandMenu.cfm). Participants ate their normal diet without any spices for 4 wk (bland diet) and their normal diet plus 30 g/d of Freshly Chopped Chilli blend (MasterFoods, Wyong, Australia) minus other spices for another 4 wk. Thirty grams of this chili blend was chosen after 2 palatability test sessions, at which time 10 members of the staff (everyday users as well as occasional users) ingested different products and amounts of chili with bread. Thirty to thirty-five grams of chili was generally acceptable. The composition of this chili blend (as provided on the container) was 55% cayenne chili, water, sugar, salt, acetic acid, and xanthan. We were unable to analyze the capsaicin content of the chili blend, but the manufacturer (MasterFoods) reported to us (11 February 2003) that cayenne pepper contains 2000 ppm capsaicin. On the basis of this information, the capsaicin content of the product used in the present study was ≈33 mg/30 g chili blend (55% chili). This is comparable with the amount of capsaicin (30 mg) used in earlier studies that investigated the effects of meals containing chili on energy metabolism in lean young persons (16–18).

At the end of each dietary period, the subjects participated in acute meal tests: 2 at the end (≈days 22 and 29) of the bland diet and 1 at the end (day 29) of the chili dietary period. The meal on day 22 of the bland diet was a bland meal (BAB; bland meal after bland diet), that on day 29 of the bland diet was a meal containing chili (CAB; chili meal after bland diet), and that on day 29 of the chili diet was a meal containing chili (CAC; chili meal after chili diet).

Test day protocol

The protocol for postprandial testing is shown in **Figure 1**. The subjects were asked to fast for 10–12 h overnight and to refrain from alcohol, fried food, and any vigorous exercise for ≥24 h before the meal tests. On arrival at the university's clinical room, the participants were seated for 10 min. After body weight was measured, an intravenous cannula was inserted into a large antecubital vein for repeated blood sampling. A blood sample was collected for subsequent measurement of fasting plasma glucose, serum insulin, and C-peptide. After an additional 15 min of rest, fasting indirect calorimetry was performed while the subjects were seated in a semirecumbent position for 15 min. The test meal was then consumed over 10 min. No other food or drink was allowed after the meal, except for water (<250 mL). Blood sampling and indirect calorimetry were performed at regular intervals after the meal for 120 min. The subjects remained in a semirecumbent position for the duration of the postprandial meal test and were not allowed to sleep; they were allowed to read magazines and listen to the radio.

Meals

All the meals consisted of bread, a beef patty (soy patty for vegetarians; $n = 7$), and a glucose drink. The CAB meal and the CAC meal also contained 30 g of the chili blend, which replaced some of the carbohydrate content of the glucose drink. The same commercial brands of bread, beef patty, and glucose drink were used for all 3 meals. The macronutrient composition of the chili blend (per 100g) was 84.61 kcal (354 kJ) energy, 1.7 g protein, 1.2 g fat (<0.1 g saturated fat), and 20.7g carbohydrate (14.7 g sugar); that of the whole meals was 467.3 kcal (1955 kJ) energy, 67% carbohydrate, 18% protein, 15% fat, and 2.4 g fiber (as analyzed by using FOODWORKS software; Xyris, Brisbane, Australia).

Indirect calorimetry

Indirect calorimetry was performed with the use of an open-circuit ventilated hood system (Deltatrac Metabolic Monitor; Datex Instrumentation Corp, Helsinki, Finland) 3 times (before the meal and 0–40 and 80–120 min after the meal), as outlined in Figure 1. Gas exchange rates were recorded at 1-min intervals. For baseline data, an equilibrium period of 5 min was allowed; data from the last 10 min were averaged for the calculation of EE (kcal/d) and respiratory quotient (ratio of carbon dioxide production to oxygen consumption).

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TABLE 1
Some metabolic variables at baseline and in the fasting state before the meals were eaten[1]

Variable	No. of subjects	Baseline	BAB	CAB	CAC
Weight (kg)	36	75.50 (70.02, 80.99)[2]	75.29 (69.85, 80.72)	75.26 (69.82, 80.71)	75.41 (69.93, 80.91)
BMI (kg/m^2)	36	26.38 (24.78, 27.97)	26.32 (24.69, 27.85)	26.30 (24.71, 27.87)	26.34 (24.75, 27.93)
Glucose (mg/dL)	36	88.55 (83.99, 93.10)	89.92 (85.98, 93.86)	88.71 (85.16, 92.29)	87.01 (82.34, 91.78)
Insulin (μIU/mL)	34	6.40 (4.89, 7.90)	6.56 (4.94, 8.17)	5.48 (4.19, 6.77)	6.04 (4.58, 7.51)
C-peptide (ng/mL)	29	ND	4.20 (3.40, 5.00)	3.79 (3.08, 4.50)	3.85 (3.03, 4.67)
EE (kcal/d)	36	ND	1502 (1393, 1612)	1530 (1424, 1634)	1491 (1389, 1592)

[1] BAB, bland meal after bland diet; CAB, chili meal after bland diet; CAC, chili meal after chili diet; ND, not determined. The BAB meal was eaten after 3 wk (day 22) of the bland diet, the CAB meal was eaten after 4 wk (day 29) of the bland diet, and the CAC meal was eaten after 4 wk (day 29) of the chili diet. The data were compared with repeated-measures ANOVA by using general linear modeling with bootstrap estimation of SEs followed by Holm's test to identify the heterogeneity of groups and by a partial Holm's test to identify significant intergroup differences adjusted for multiple comparisons.

[2] $\bar{x}$; 95% CI in parentheses (all such values).

Biochemical variables

Blood samples were collected into tubes with no anticoagulant (for the measurement of serum insulin and C-peptide) under fasting conditions and at 20, 40, 60, 90, and 120 min and were allowed to coagulate at room temperature. Samples with anticoagulant fluoride oxalate (for analysis of plasma glucose) were placed immediately on ice. Blood tubes were centrifuged at 1335 × *g* at 4 °C for 20 min. Serum and plasma were separated, portioned, and frozen at −80 °C until further analysis. Serum insulin and plasma glucose samples from each time point were analyzed. C-peptide was analyzed in fasting samples and in samples collected 40, 60, and 90 min postprandially.

Serum insulin and C-peptide were measured by radioimmunoassay with the use of commercially available kits (Diagnostic Systems Laboratories Inc, Webster, TX) and a 1261 multigamma counter (LKB, Wallace Oy, Turku, Finland) plus RIACALC software (version 3). Plasma glucose was analyzed on a DataPro clinical analyzer (Thermo Electron Corporation,) with the use of Thermotrace reagents (Thermo Electron Corporation, Melbourne, Australia). Samples for each person were assayed in the same batch. The intraassay CV for plasma glucose was 2% and for serum insulin and C-peptide was <6%.

Calculations and statistics

Sample size calculations indicated that 32–34 subjects needed to complete the study to detect a 5% difference in insulin concentrations [logarithm of area under the curve AUC)] with 90% power based on a repeated-measures design and an SD to difference ratio of 0.094 measured in the first 10 subjects. The AUC was calculated for all variables measured by using the trapezoidal method (GRAPHPAD PRISM, version 4.00; GraphPad Software, San Diego CA). Incremental AUC (iAUC; the AUC above baseline) and net AUC (iAUC minus AUC below baseline) for glucose, insulin, C-peptide, and EE were calculated. The insulin and glucose quotient (iAUC insulin/iAUC glucose) was used as an indicator of insulin sensitivity. Although C-peptide and insulin are secreted in equimolar amounts, some insulin (but not C-peptide) is quickly cleared by the liver. Therefore, the C-peptide concentration was considered an indicator of insulin secretion and the quotient for iAUC C-peptide/iAUC insulin as an indicator of hepatic insulin clearance.

Repeated-measures analysis of variance by general linear modeling with bootstrap estimation of SEs was used to test for any differences between the metabolic responses with different meals (STATA, version 8.2, StataCorp LP, College Station, TX). Postestimation Holm test analysis was then used to adjust *P* values for multiple comparisons (20). All analysis was carried out after adjustment for order and period effects of the meals. Data in the tables are shown as mean AUCs (95% CIs). Prism 4 GRAPHPAD was used to plot the data for the figures. Because not all analyses were available for every participant, the number of participants for each variable tested is shown in graphs and tables.

RESULTS

Thirty-six participants (22 women and 14 men) completed the study. There was no significant difference in BMI ($P = 0.29$) on the meal test days (**Table 1**). Similarly, no significant differences were observed for fasting plasma glucose ($P = 0.42$), serum insulin ($P = 0.42$), serum C-peptide, and EE ($P = 0.30$) before the consumption of the 3 meals (as shown in Table 1 and as time zero in **Figure 2**, **Figure 3**, and **Figure 4**).

Although the iAUC for plasma glucose was 10% lower after consumption of the CAB and the CAC meals than after that of the BAB meal (Figure 2), no statistically significant differences between the 3 meals were shown ($P = 0.065$). There was no difference in the peak glucose response or time response on the 3 meals (**Table 2**). Maximum increases in insulin concentration and iAUC for serum insulin (Figure 3) were heterogeneous ($P = 0.0008$) after the 3 meals: these measures were 36% and 32% lower, respectively, after consumption of the CAC meal than after that of the BAB meal ($P < 0.001$ for both). The maximum increases in insulin concentration and iAUC for serum insulin after the CAB meal were in between but closer to those with the CAC meal than with the BAB meal. The maximum increases in insulin concentration and iAUC for serum insulin after the CAB meal were 15% ($P = 0.074$) and 17% ($P = 0.072$) lower, respectively, than after the BAB meal. The maximum increases in insulin concentration and iAUC for serum insulin after the CAC meal were 24% ($P = 0.24$) and 18% ($P = 0.52$) lower, respectively, than after the CAB meal. The maximum increases in C-peptide concentration and iAUC for C-peptide were not significantly different between the 3 meals (Table 2). There was no significant difference ($P = 0.1$) in EE (Figure 4) or in the respiratory quotient between the 3 meals (Table 2).

We hypothesized that there might be a difference in the measured variables between those with different BMIs. To confirm this hypothesis, general linear modeling and postestimation heterogeneity testing between meals was conducted for the measured variables after the whole group was separated into 2 groups

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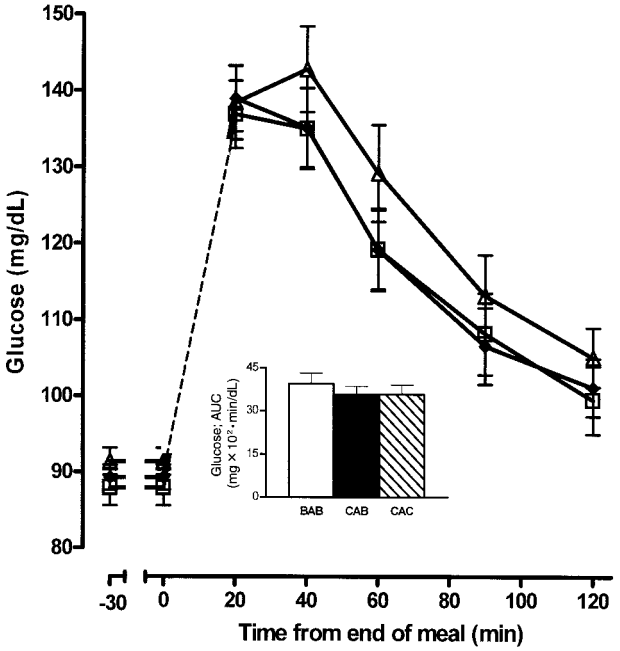

FIGURE 2. Mean (±SEM) glucose concentrations at fasting and up to 2 h after the consumption of the bland meal after the bland diet (BAB; △), the chili meal after the bland diet (CAB; ♦), and the chili meal after the chili diet (CAC; □). Baseline values (0 min) were obtained 30 min before the meal was eaten. Inset: incremental area under the curve (iAUC) for plasma glucose. $n = 32$. Overall $P = 0.065$ (repeated-measures ANOVA by using general linear modeling; P values for multiple comparisons were adjusted by using Holm's test).

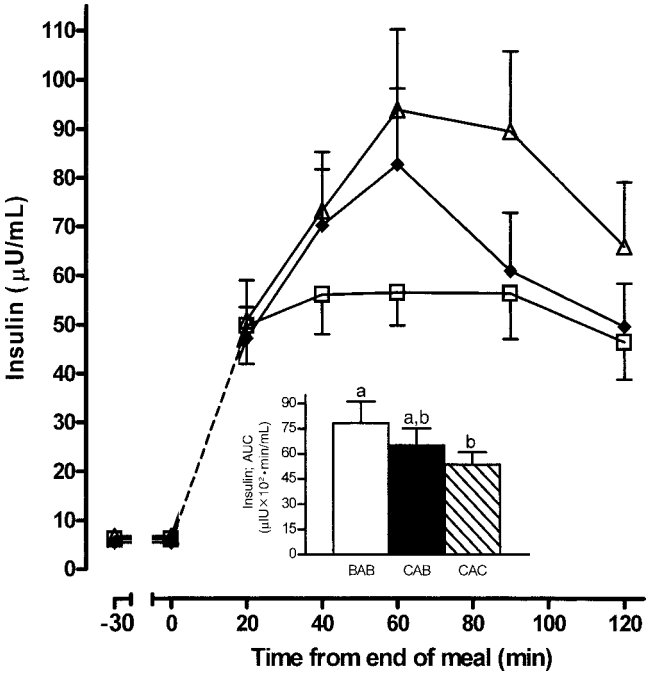

FIGURE 3. Mean (±SEM) insulin concentrations at fasting and up to 2 h after the consumption of the bland meal after the bland diet (BAB; △), the chili meal after the bland diet (CAB; ♦), and the chili meal after the chili diet (CAC; □). Baseline values (0 min) were obtained 30 min before the meal was eaten. Inset: incremental area under the curve (iAUC) for serum insulin. $n = 32$. Overall $P = 0.0008$ (repeated-measures ANOVA by using general linear modeling; P values for multiple comparisons were adjusted by using Holm's test, which indicated heterogeneity between the 3 meals). Bars with different lowercase letters are significantly different, $P < 0.05$.

at the median BMI value of 26.3. The analysis showed that the significant heterogeneity observed (for maximum insulin and insulin) between the 3 meals in the whole group came predominantly from the subjects with a BMI ≥ 26.3 (**Table 3**). In addition to these results, subjects with a BMI ≥ 26.3 showed significant heterogeneity ($P < 0.02$ for all) in maximum C-peptide, iAUC C-peptide, iAUC C-peptide/insulin quotient, and net AUC EE. Postestimation analysis showed a higher C-peptide/insulin quotient, and a lower net EE after the CAB meal ($P < 0.045$ for both) and the CAC meal ($P < 0.003$ for both) than after the BAB meal. The maximal change in C-peptide was smaller with the CAC meal than with the CAB and the BAB meals ($P < 0.02$ for both), and iAUC C-peptide was lower after the CAC meal than after the BAB meal ($P = 0.015$).

DISCUSSION

The results of this study suggest that the amount of insulin required to control for the postprandial increase in glucose is reduced with a CAC meal. Furthermore, these results are more definitive with increasing BMI. In subjects with a BMI ≥ 26.3, chili meals also possibly result in lower C-peptide and insulin secretion and higher hepatic clearance of insulin (evidenced by a higher C-peptide/insulin quotient), and the effect is larger if chili is eaten regularly. These results may have implications in the control of postprandial hyperinsulinemia. In addition to high fasting blood glucose and insulin concentrations, postprandial hyperglycemia and hyperinsulinemia are also independent risk factors for atherosclerosis (21–23). Because humans spend much of their time in the postprandial state, controlling for food-induced hyperglycemia and hyperinsulinemia could be more important than controlling for fasting or the postabsorptive state.

Hyperinsulinemia, a byproduct of obesity and insulin resistance, results from increased insulin secretion (24–26) and decreased hepatic insulin clearance (26–28) with increasing BMI. In the present investigation, chili appeared to diminish the adverse effects related to obesity. The difference in maximum insulin and AUC insulin between subjects with a BMI < 26.3 and those with a BMI ≥ 26.3 was reduced from 119% and 96% with the BAB meal to 38% and 39% with the CAC meal, respectively. Similarly, the difference for C-peptide/insulin quotient changed from 40% to 22% from the BAB to the CAC meal. Although it is not possible to validate the mechanism of action of chili from this study, it is probable that chili affects the hepatic insulin receptors, either by increasing their number or affinity or both to result in increased insulin clearance and, hence, lower serum insulin concentrations. Additional research is warranted to elucidate this finding.

It has been hypothesized that insulin resistance and hyperinsulinemia are the compensatory mechanisms that limit additional weight gain, reestablish energy balance, and stabilize body weight in obese people (29, 30). Insulin secretion, SNS activity, and thermogenesis increase after food intake. Additionally, insulin increases SNS activity and, hence, thermogenesis, which may help prevent additional weight gain. The lower EE in subjects with a BMI ≥ 26 after the chili meals may have been the consequence of the reduced and improved profile of postprandial

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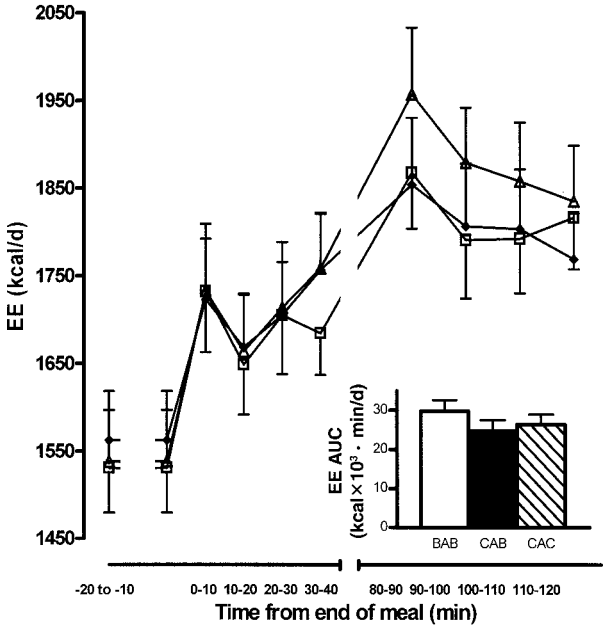

FIGURE 4. Mean (±SEM) energy expenditure (EE) at fasting and up to 2 h after consumption of the bland meal after a bland diet (BAB; △), the chili meal after the bland diet (CAB; ♦), and the chili meal after the chili diet (CAC; □). Inset: net area under the curve (AUC) for EE. $n = 30$. Overall $P = 0.1$ (repeated-measures ANOVA by using general linear modeling; P values for multiple comparisons were adjusted by using Holm's test).

insulin. Anecdotal evidence and the findings from studies conducted in lean, young persons suggest that meals containing chili increase EE (15, 16) and may be helpful in reducing body weight. However, the results from a study by Matsumoto et al (19) and the present study suggest a different action of chili on thermogenesis in adults with a BMI greater than that considered to be desirable, ie, 25.

The amount of food eaten, ingestion time, the physical state of the meal (solid or liquid), and the protein, fat, carbohydrate, and fiber contents may affect the postprandial glycemic and insulin responses (31, 32), but these variables were constant in the 3 meals used in this study. The meal ingestion time ranged from 4.30 to 10 min between persons, but was similar for each person for the 3 meals. Postprandial glucose and insulin responses are inversely related to gastric emptying time (33, 34). Although the available data are equivocal, some research suggests that chili delays gastric emptying and quickens the whole-gut transit time in healthy human volunteers (35, 36). It is possible that the lower postprandial glycemic response after the chili meals may have been due to reduced absorption and slower gastric emptying. Some people avoid consuming chili for fear of unwanted gastric hypermotility. However, in our study, all subjects except one reported a marked reduction or disappearance of any symptoms within 7–10 d of eating chili.

Different methods for calculating AUC (eg, iAUC, net AUC, and AUC from minimum value in the postprandial state) have been used in the literature, and the results differ for these analytic methods (37). We analyzed the data as both iAUC and net AUC for glucose and EE. Although no difference was observed for EE, the CIs for the mean difference in glucose between the CAB and the BAB meal were larger for net AUC (-12.96 to 27 mg $\times$ 10^2 · min/dL) than for iAUC (-11.56 to -1.03 mg $\times$ 10^2 · min/dL), which resulted in different P values ($P = 0.12$ for net AUC and $P = 0.04$ for iAUC). Insulin and C-peptide concentrations were above baseline for the entire 2 h after the meal.

TABLE 2
Comparison of maximal increase (Δ) from baseline and incremental area under the curve (iAUC) for some metabolic variables in response to the 3 meals[1]

	No. of subjects	Mean (95% CI)	P^2
ΔGlucose (mg/dL)			0.46
BAB	33	59.86 (50.99, 68.73)	
CAB	33	57.03 (49.80, 64.26)	
CAC	32	56.80 (49.90, 63.70)	
ΔInsulin (μIU/mL)			0.0002[3]
BAB	33	108.80 (72.90, 144.70)	
CAB	33	92.06 (61.53, 122.58)	
CAC	33	69.82 (51.46, 88.18)[4]	
ΔC-peptide (ng/mL)			0.24
BAB	29	12.55 (11.66, 13.44)	
CAB	29	12.26 (11.34, 13.18)	
CAC	29	11.97 (11.09, 12.84)	
iAUC insulin/iAUC glucose ($\times 10^{-5}$)			0.17
BAB	33	27.84 (19.13, 36.55)	
CAB	33	29.04 (15.61, 42.47)	
CAC	32	20.87 (15.04, 26.60)	
iAUC C-peptide (ng × 10 · min/mL)			0.32
BAB	29	77.65 (71.17, 84.12)	
CAB	29	75.50 (62.88, 82.72)	
CAC	29	75.19 (68.47, 81.91)	
iAUC C-peptide/iAUC insulin			0.08
BAB	29	12.44 (9.39, 15.49)	
CAB	29	13.91 (10.22, 17.61)	
CAC	29	14.13 (11.34, 16.92)	
iAUC RQ			0.69
BAB	30	4.30 (1.08, 7.52)	
CAB	30	4.41 (1.36, 7.45)	
CAC	30	6.02 (3.16, 8.87)	

[1] BAB, bland meal after bland diet; CAB, chili meal after bland diet; CAC, chili meal after chili diet; RQ, respiratory quotient. The data were compared with repeated-measures ANOVA by using general linear modeling with bootstrap estimation of SEs followed by Holm's test to identify the heterogeneity of groups and by a partial Holm's test to identify significant intergroup differences adjusted for multiple comparisons.
[2] Adjusted for by using Holm's test.
[3] Significant heterogeneity between the 3 meals.
[4] Significantly different from the BAB meal, $P < 0.05$.

Similar fasting concentrations of the measured variables before the meals (especially CAB and CAC) also indicate that there was no differential effect of 4 wk of chili and bland diets on these variables. It may be possible that the amount of capsaicin used was too small to show any significant change. Anecdotal information from participants suggests that regular ingestion of higher amounts of chili would not be possible over a long period. Similar to these findings, Lejeune et al (38) reported no difference in serum glucose, insulin, or EE between subjects who consumed 135 mg capsaicin/d for 9 wk (taken as supplements) and those who consumed placebo. It is probably relevant that factors such as body weight and the macronutrient composition of the diet, which can influence fasting glucose and insulin concentrations, were designed to be constant in the present investigation.

To our knowledge, the present study was the first to carefully control for background diets and to investigate the effects of a chili meal on different metabolic variables. The study was conducted in an age group of people (mean age: 46 y; BMI = 26.3)

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TABLE 3
Comparison of maximal increase (Δ) and incremental area under the curve (iAUC) for some metabolic variables in response to the 3 meals in different BMI (in kg/m^2) groups (separated at the median BMI of 26.3)[1]

	Mean (95% CI)	
	BMI < 26.3 (n = 12–18)	BMI ≥ 26.3 (n = 15–18)
ΔInsulin (μIU/mL)		
P	0.7	< 0.0001[2]
BAB	65.80 (27.93, 103.69)	144.62 (89.13, 100.12)
CAB	66.43 (29.07, 103.79)	113.41 (65.87, 160.95)[3]
CAC	57.90 (30.85, 84.94)	79.76 (53.19, 106.32)[3]
ΔC-peptide (ng/mL)		
P	0.3	0.007[2]
BAB	12.47 (11.17, 13.76)	12.61 (11.27, 13.94)
CAB	12.24 (10.97, 12.51)	12.27 (10.87, 12.69)
CAC	12.52 (11.50, 13.54)	11.58 (10.21, 12.96)[3,4]
iAUC insulin (μIU × 10^2 · min/mL)		
P	0.7	0.0001[2]
BAB	52.03 (23.29, 80.76)	101.71 (60.08, 143.34)
CAB	50.17 (24.93, 75.42)	78.12 (44.98, 111.27)[3]
CAC	44.02 (23.62, 64.41)	61.07 (38.27, 83.86)[3]
iAUC C-peptide (ng × 10 · min/mL)		
P	0.4	0.02[2]
BAB	76.94 (67.45, 86.43)	78.15 (68.49, 87.81)
CAB	77.33 (70.48, 84.18)	74.21 (62.19, 86.24)
CAC	78.33 (72.76, 83.90)	72.98 (61.70, 84.25)[3,4]
iAUC C-peptide/iAUC insulin		
P	0.7	0.002[2]
BAB	16.22 (11.07, 21.37)	9.777 (6.18, 12.36)
CAB	16.07 (10.25, 21.89)	12.40 (7.22, 17.58)[3]
CAC	16.18 (11.17, 21.19)	12.68 (9.19, 16.17)[3]
Net AUC EE (kcal × 10^3 · min/d)		
P	0.9	0.003[2]
BAB	26.71 (15.79, 37.64)	34.48 (25.56, 43.37)
CAB	22.48 (11.61, 33.34)	26.34 (20.30, 32.37)[3]
CAC	25.01 (10.70, 33.30)	25.90 (16.89, 34.90)[3]

[1] BAB, bland meal after bland diet; CAB, chili meal after bland diet; CAC, chili meal after chili diet. The data were compared with repeated-measures ANOVA by using general linear modeling with bootstrap estimation of SEs followed by Holm's test to identify the heterogeneity of groups and a partial Holm's test to identify significant intergroup differences adjusted for multiple comparisons. The *P* values were adjusted for by using Holm's test.
[2] Significant heterogeneity between the 3 meals.
[3] Significantly different from the BAB meal, $P < 0.05$.
[4] Significantly different from the CAB meal, $P < 0.05$.

in whom the risk of insulin resistance, cardiovascular disease, and type 2 diabetes is high. Also, a palatable chili flavoring was used rather than pure capsaicin—the active ingredient. In addition to containing capsaicin, chilies are a rich source of antioxidants, including vitamin C and carotenoids such as β-carotene and lutein, which could also be important in reducing the risk of atherosclerosis. Because of the dramatic increase in the number of persons with insulin resistance and hyperinsulinemia, a range of measures are being investigated or used to control or reverse this abnormality. These measures include the use of soluble fiber–rich guar and pectin or high fiber and foods with a low glycemic index, including whole-grain cereals, which will improve postprandial glucose and insulin profiles. Our study indicates that the habitual consumption of chili may also be useful in ameliorating meal-induced hyperinsulinemia. Additional research is needed to confirm this finding in persons at higher risk of hyperinsulinemia and to determine whether an improved insulin response might, however, induce negative effects on thermogenesis in overweight and obese persons.

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MJB, DPG, and IKR designed the study, discussed the data, and corrected the manuscript. IKR provided statistical support. KDKA planned the meals, recruited the subjects, conducted the study, performed the biochemical and statistical analyses, and wrote the manuscript. None of the authors had a conflict of interest.

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