Ginger consumption enhances the thermic effect of food and promotes feelings of satiety without affecting metabolic and hormonal parameters in overweight men: A pilot study

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Abstract

Objective—Evidence suggests that ginger consumption has anti-inflammatory, anti-hypertensive, glucose-sensitizing, and stimulatory effects on the gastrointestinal tract. This study assessed the effects of a hot ginger beverage on energy expenditure, feelings of appetite and satiety and metabolic risk factors in overweight men.

Methods—Ten men, age 39.1 ± 3.3 y and body mass index (BMI) 27.2 ± 0.3 kg/m^2, participated in this randomized crossover study. Resting state energy expenditure was measured using indirect calorimetry and for 6 h after consumption of a breakfast meal with or without 2 g ginger powder dissolved in a hot water beverage. Subjective feelings of satiety were assessed hourly using visual analog scales (VAS) and blood samples were taken fasted and for 3 h after breakfast consumption.

Results—There was no significant effect of ginger on total resting energy expenditure ($P = 0.43$) or respiratory quotient ($P = 0.41$). There was a significant effect of ginger on thermic effect of food (ginger vs control = 42.7 ± 21.4 kcal/d, $P = 0.049$) but the area under the curve was not different ($P = 0.43$). VAS ratings showed lower hunger ($P = 0.002$), lower prospective food intake ($P = 0.004$) and greater fullness ($P = 0.064$) with ginger consumption versus control. There were no effects of ginger on glucose, insulin, lipids, or inflammatory markers.

Conclusions—The results, showing enhanced thermogenesis and reduced feelings of hunger with ginger consumption, suggest a potential role of ginger in weight management. Additional studies are necessary to confirm these findings.

Keywords

spices; energy expenditure; appetite

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Introduction

Ginger has been used to treat metabolic ailments [1] in ancient civilizations [2]. Ginger has been purported to have anti-inflammatory, anti-hypertensive, and glucose-sensitizing effects as well as stimulatory effects on the gastrointestinal tract by increasing gastric secretions [3,4] and peristalsis [5]. Rats consuming a high fat diet with ginger for 6 wk had lower weight gain, glucose, insulin, total cholesterol, low-density lipoprotein cholesterol, triglycerides, free fatty acids and phospholipids compared to rats fed the high-fat diet without ginger [4]. Evidence from the animal literature supports the use of ginger as a functional dietary agent for weight management and prevention of metabolic disorders [4,6-10] but research in humans is limited.

Ginger may play a role in energy balance regulation by activating the transient-receptor potential vanilloid uncoupling pathway [3,11,12], implicated in the thermogenic effect of capsaicin [13]. However, consumption of a meal with ginger sauce did not increase post-prandial EE over a 2-h period to a greater extent than a similar meal without ginger [14]. On the other hand, ginger is proposed to modulate serotonin concentrations [7], which play a role in controlling appetite. Thus, there is evidence to suggest that ginger could be involved in weight management.

We hypothesized that ginger consumption would lead to a greater thermogenic response and satiety relative to a no ginger control condition in overweight men. In addition, we hypothesized that appetite-regulating hormones and inflammatory markers would differ between ginger and no ginger conditions.

Subjects and Methods

Men, age 19-50y, BMI 25-29.9kg/m^2 were recruited to participate in this study via online advertisements. Exclusion criteria included smoking, recent weight change (>10lbs in the previous 3mo), medications known to affect EE or gastrointestinal function, excessive caffeine use (>6 caffeinated beverages/d), chronic health conditions (e.g. hypertension, diabetes), or allergies to any of the food products or ingredients provided in this study. The study was approved by the St. Luke's/Roosevelt Hospital Institutional Review Board and all participants provided informed consent prior to randomization into the study.

This study employed a 2-arm, randomized, crossover design. Participants refrained from alcohol, spice and energy drink consumption for 2d prior to each test day. Additionally, they were asked not to perform any structured exercise and to consume the same evening meal the day before each test day.

On test day, participants arrived at the New York Obesity Nutrition Research Center metabolic chamber in the fasted state. Upon arrival, they rested for 30-min after which their RMR was measured by indirect calorimetry (Vmax29, Sensor Medics, Yorba Linda, CA). Participants were then given a standard breakfast (5 small corn muffins: 2197kJ [525kcal], 67.5g total carbohydrate, 25g fat, 7.5g protein, General Mills Sales, Inc., Minneapolis, MN; 4.23fl. oz. orange juice: 293kJ [70kcal], 17.0g total carbohydrate, Nestlé Juicy Juice™, Vevey, Switzerland; and 6oz. of hot water) to consume in 15 min. Dried ginger powder (2g, The McCormick Science Institute, McCormick & Company, Inc., Sparks, MD) was dissolved in hot water for the ginger test day. Hot water alone was given during the control day. Post-prandial EE measurements were obtained over a 40min period hourly for 6h. The calorimeter was calibrated hourly as specified by the manufacturer using standard gases. Data from the metabolic cart were used to calculate total post-prandial EE over the 6h measurement period and TEF (difference between post-prandial EE and RMR). Minute-by-minute data obtained during each 40min period were extrapolated to the full hour. Only data

points that showed greatest stability based on a calculated t-score, VO_2 flow rate $\pm 10\%$ and RQ $\pm 5\%$ were retained in the analyses. To obtain greatest period of stability, the first 10 min of each measurement period was discarded.

Immediately before and hourly after breakfast consumption, participants completed VAS to report their feelings of satiety and appetite [15]. Participants were asked: (1) how hungry do you feel? (2) how satisfied do you feel? (3) how full do you feel? (4) how much do you think you could eat? (5) how energetic do you feel? (6) how sluggish do you feel? (7) how alert do you feel? (8) how warm do you feel?

Sample Collection and Analysis

Blood samples were obtained in the fasted state immediately before and after breakfast consumption and at 30, 45, 60, 120, and 180 min relative to breakfast. At least 10 mL of blood was collected in EDTA-coated chilled tubes for hormone assays: leptin, adiponectin, glucagon-like peptide 1, ghrelin, and peptide YY. The tubes were pre-treated with addition of the protease inhibitor aprotinin (0.6 TIU/mL of blood) and DPP-IV inhibitor (10 μL/mL of blood) to prevent degradation of gut hormones (ghrelin, glucagon-like peptide 1 and peptide YY). Upon collection, blood was immediately placed on ice; plasma was separated within 60 min of collection. Samples to be analyzed for active ghrelin levels were acidified with 50 μL of 1N HCl and then frozen at −80 °C until assayed. Another 5 mL of blood was collected in EDTA-coated tubes for the measurement of other metabolic markers: glucose, insulin, TG, C-reactive protein, interleukin-6, tumor necrosis factor α. All hormone analyses were performed in the Hormone and Metabolite Core Laboratory of the New York Nutrition Obesity Research Center according to standard procedures. Glucose was measured using the glucose oxidase method (Analox Instruments, MA) and insulin, leptin, adiponectin, ghrelin, glucagon-like peptide 1 and peptide YY were measured by radioimmunoassay (Millipore, Billerica, MA). Inflammatory markers were analyzed in duplicate using commercially available ELISA kits (R&D Systems, Minneapolis, MN; Alpco diagnostics, Windham, NH) on Magellan spectrophotometric plate reader (Tecan, Durham, NC) at the appropriate wavelengths. TG was determined by spectrophotometry using an Ektachem DT 11 System (Johnson and Johnson Clinical Diagnostics, Rochester, NY) with appropriate reagents and standards (CliniqaRaichem, San Marco, CA).

Statistical analyses

We had 10 subjects with seven time points and two treatments each. We used Cohen's statistics for performing effect size/power analysis. (Cohen's f^2 is a standard effect size measure for regression based linear model analysis [16]; it is defined as the ratio of squared multiple correlation and its difference from one. By statistical convention Cohen's $f^2 = 0.02$, 0.15 and 0.25 are taken as small, medium and large effect sizes [16]). For these data we had 38% power to detect a small effect size (Cohen's f2=0.02), 99% power to detect a medium effect size (Cohen's f2=0.15) and almost 100% power to detect a large effect size (Cohen's f2=0.25).

Mixed model analysis of variance and Wilcoxon paired t-test were used to assess the effects of treatment (ginger or no ginger), time and treatment x time interaction on all variables. EE variable models were adjusted for age, race, and body weight. VAS models were adjusted for body weight alone. Area under the curve (AUC) was calculated for VAS ratings using the trapezoidal method and compared between treatments using the Wilcoxon paired t-test.

For each metabolite, blood samples were analyzed in duplicate and averaged for statistical analysis. Also, the AUC was calculated via the trapezoidal method to determine total treatment effect over the 3 h post-prandial period for each metabolite. Regression analysis

was conducted on metabolite concentrations and AUC, with treatment type (ginger vs no ginger), age, weight, time (concentrations only), and race used as independent variables. A separate analysis was also performed to examine the treatment by time interaction.

After analysis of blood samples, one patient was found to show signs of diabetes. At the time of screening, the patient reported no health issues, and no preliminary blood tests were conducted during the screening, so his condition went unreported until the samples were analyzed. Statistical analyses for hormones and metabolites were performed with and without this person. Results were not different when the analyses were performed without this person and, unless otherwise noted, we report here the results on the complete sample population.

Results

Of the 17 men screened for the study, 3 did not qualify and 4 dropped out after randomization due to claustrophobia (n = 1) and non-compliance with the protocol (n = 3), leaving 10 completers (age 39.1±3.3y, BMI 27.2±0.3kg/m^2).

There was a significant effect of treatment (ginger vs no ginger = 42.7±21.4kcal/d, P=0.049) but no time or treatment x time interaction on TEF (Figure 1). There was no difference in AUC for TEF between conditions (P=0.43). There was no significant effect of treatment or treatment x time interaction on post-prandial EE (P=0.425) and RQ (P=0.409). There was no treatment x time interaction for any of the VAS questions. AUC for VAS ratings was significantly lower for questions related to hunger (P=0.002) and prospective food intake (P=0.004) after ginger consumption compared to control (Figure 2). Additionally, there was a trend for greater fullness (P=0.064) after ginger consumption.

There was no significant ginger x time interaction (P = 0.84) or ginger effect (P = 0.92) on glucose concentration (Table 1). Similarly, for insulin, there was no significant ginger x time interaction (P = 0.82) or ginger effect (P = 0.76). Similar results were obtained for TG for ginger x time interaction (P = 0.96) and ginger effect (P = 0.72). When we examined the AUC over the 3 h post-breakfast, we found no difference in glucose, insulin, or TG concentrations between the ginger and no ginger conditions (all P > 0.8).

There was no significant ginger x time interaction (P = 0.71) or ginger effect (P = 0.45) on CRP concentrations, though average CRP levels were 16.3 ± 4.1% higher in the control phase than the ginger phase. Similarly, there was no significant ginger x time interaction (P = 0.71) or ginger effect (P = 0.17) on IL-6 concentrations, though average IL-6 levels were 16.4 ± 3.5% higher in the ginger phase than the control phase. Likewise, there was no significant ginger x time interaction (P = 0.91) or ginger effect (P = 0.49) on TNF-α concentrations. When we examined the AUC over the 3 h post-breakfast, we found no difference in CRP, IL-6, or TNF-α concentrations between the ginger and no ginger conditions (all P > 0.5).

There was no significant ginger x time interaction (P = 0.59) or ginger effect (P = 0.86) on leptin concentrations. AUC analysis over the 3 h post-breakfast was not significantly different between the ginger and no ginger conditions (P = 0.998). Total ghrelin levels tended to be higher in the ginger phase than in the control phase (P = 0.059). When the data were analyzed without the diabetic patient, the difference in total ghrelin levels between phases for the 9 remaining patients became statistically significant (P = 0.018). However, AUC analysis (P = 0.55) and interaction analysis (P = 0.67) showed no effect of ginger or ginger x time interaction on total ghrelin. On the other hand, no significant difference was found for active ghrelin levels between phases (P = 0.45). Similar results were observed with the AUC analysis (P = 0.87) and interaction analysis (P = 0.19). There was no

significant ginger x time interaction (P = 0.86) or ginger effect (P = 0.998) on adiponectin concentrations. AUC analysis over the 3 h post-breakfast was similar to leptin result (P = 0.998).

There was no significant ginger x time interaction (P = 0.49) or ginger effect (P = 0.88) on PYY concentrations. Similarly, there was no significant ginger x time interaction (P = 0.21) or ginger effect (P = 0.42) on GLP-1 concentrations. When we examined the AUC over the 3 h post-breakfast, we found no difference in PYY and GLP-1 concentrations between the ginger and no ginger conditions (all P > 0.75).

Discussion

This study provides preliminary evidence for the thermogenic and satiety-inducing effects of ginger in humans. Ginger powder dissolved in hot water increased the TEF of a standardized breakfast meal by 43kcal relative to hot water. Interestingly, this was accompanied by enhanced satiety and reduced hunger, suggesting that ginger consumption may also play a role in modulating appetite. However, contrary to our hypothesis, levels of inflammatory markers were not improved with ginger consumption.

Our results differ from those of Henry and Piggott [14] likely due to the different method of ginger administration (sauce from fresh ginger vs. dried ginger powder). Only dried ginger is known to contain shogaols, the dehydration product of gingerols, which increase ginger's pungency [17,18]. Our study used a well-characterized dried ginger powder.

Others have also found increased thermogenesis with various spices [19]. Consumption of a chili and mustard sauce was found to raise metabolic rate to a greater extent, 25% more, than a control meal devoid of spices. Our results are also supported by *in vitro* and *in vivo* rodent studies [20,21]. Ginger activates transient-receptor potential vanilloid 1 channels that give rise to currents that produce the characteristic burning sensation felt upon consumption of pungent spices [11,12]. Such a warming sensation seems to be integral in the sympathetic nervous system-associated transduction pathway by which pungent compounds enhance thermogenesis [13].

Our study found no significant effect of ginger on RQ. Based on previous reports [13], we expected ginger consumption to lower RQ, indicating greater fat oxidation, relative to control. It is possible that a habituation period, different meal formulation, or longer term consumption of ginger may be needed to increase fat oxidation. However, whether the effect of ginger consumption on metabolic rate and fat oxidation is sustained or attenuated with repeated exposure and long-term consumption is unknown.

Total ghrelin levels were significantly higher after ginger consumption compared to the no ginger condition, with the biggest difference occurring at 45 min after consumption. This suggests that ginger is exerting acute effects on serum ghrelin levels, as opposed to daily ghrelin cycles corresponding to expected meal times [22]. The increase in ghrelin post-prandially was opposite to our expectation that ginger consumption would decrease appetite, in line with our VAS data. However, a study in rats showed greater food consumption after 5 d of consuming ginger-containing chow compared to prior to the initiation of ginger consumption [23]. Another study examined rats that were fed a ginger drink appetizer, containing 12% ginger juice, for 10 d with their normal chow meal. Both body weights and food consumption increased significantly in the ginger group relative to controls at the end of the 10-d trial period, and leptin levels were lower [24]. Unfortunately, no previous study has examined the effects of ginger on food intake in humans, and future studies should measure food intake at an *ad libitum* meal following a ginger drink preload. The apparent disagreement between our self-reported measures of appetite and satiety and hormone data

serves to illustrate the complexity of the satiety response and suggests that the correlation between psychological and physiological indications of satiety may not be so straightforward.

Ginger intake had no significant effect on inflammatory markers measured in this study. However, our participants exhibited a wide range of serum metabolic risk factors, which may have reduced our ability to detect ginger effects in a small sample size. A recent study showed that rats consuming dissolved ginger powder displayed dose-dependent anti-inflammatory effects as compared to controls in the induced acute and chronic inflammatory model, verifying the expectation that ginger has anti-inflammatory effects and suggesting the role of future research in elucidating this finding [25]. In a human study, Skulas-Ray et al. examined the effects of a spice blend containing 1.51 g of ginger (10.8% of the total spice content by weight) on metabolic risk factors in overweight men [26]. The authors found a reduction in insulin and TG for the spice phase compared to the control phase. They also found a CRP response of an immediate increase up to 30 min post-prandially followed by a decrease below the control levels thereafter. Although our data were not significant, CRP values in our study were lower overall with ginger consumption as compared to no ginger condition, but as we did not analyze CRP 60 min post-consumption, we could not detect a similar peak in our data. The use of a combination of spices in the study by Skulas-Ray et al. [26] may explain the divergent results and the results obtained by this group are promising for this field of study and demonstrate the importance of future studies on the role of spices on human metabolism.

A potential limitation of this study is the mode of administration. Consumption of a high-fat beverage with the ginger may enhance solubility and micelle formation, further enhancing ginger's gastrointestinal stimulation [18]. A fatty beverage could have made the beverage less pungent/more palatable than hot water. Several participants reported that the ginger beverage was unusually strong and pungent. No other side effects from the ginger beverage were noted. Another limitation of our study was the absence of blinding. However, treatment order was assigned randomly and participants were not told of the order of beverage consumption a priori. Also, taste-blinding by using capsule administration would have limited the full physiological effect of ginger on transient-receptor potential vanilloid 1 receptor activation via lingual stimulation.

We cannot generalize our results to women. However, having both sexes within a small sample size would have increased the variability of our data. Future studies with larger sample size could clarify the thermogenic-enhancing effects of ginger found in this study. A future study in this direction might benefit from our work to determine the sample size needed to ensure enough power. Based on the observed measures, the effect size for a t-test is Cohen's $d = 0.224$ and for an ANOVA Cohen's effect size statistic is 0.124. With these effect sizes we would need 159 and 256 subjects to ensure 80% power in paired t-test and ANOVA respectively.

Although satiety and fullness were greater with ginger compared to control, we have no objective measure of food intake. The literature has established a satiety-inducing role for capsaicin [13,27] as well as suggestions of a satiety-inducing role for the serotonin pathway which is activated by ginger consumption [7].

From a public health perspective, due to the increase in TEF observed in this study, the use of ginger may have relevance for weight management. Moreover, effects may be increased with daily consumption. Nonetheless, the present study has provided intriguing preliminary evidence that 2g of powdered ginger dissolved in hot water can induce a small but significant increase in TEF in healthy overweight men and influence feelings of satiety

without any adverse side effects. Larger studies that include women are necessary to confirm our findings and determine the role of ginger on metabolic risk profile.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Abbreviations

EE	energy expenditure
PYY	peptide YY
RMR	resting metabolic rate
RQ	respiratory quotient
TEF	thermic effect of food
TG	triglycerides
VAS	visual analog scale

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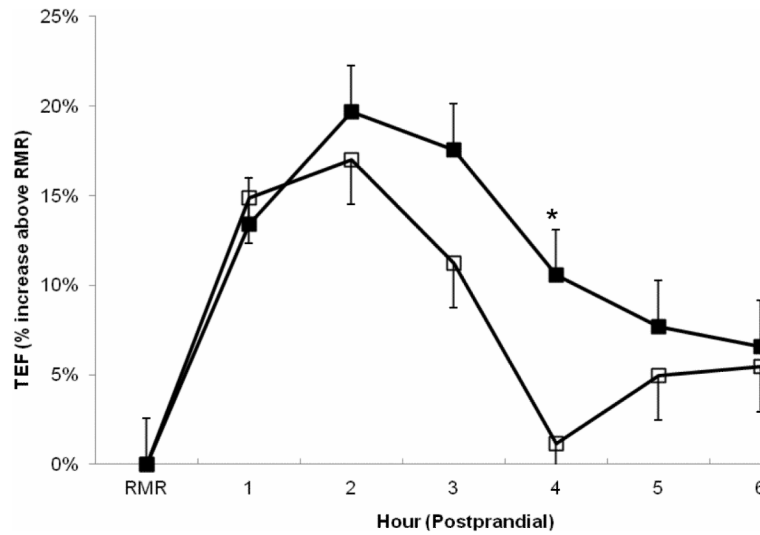
FIGURE 1.
TEF at baseline and hourly for 6 h after consumption of a breakfast with (black squares) or without (white squares) ginger in overweight men. There was a significant treatment effect (P = 0.049) with a TEF increase of 42.7 kcal/d from ginger treatment but no significant treatment x time interaction. * Significantly different between ginger and no ginger, P < 0.01. Data are means ± SEM adjusted for age, race and body weight.
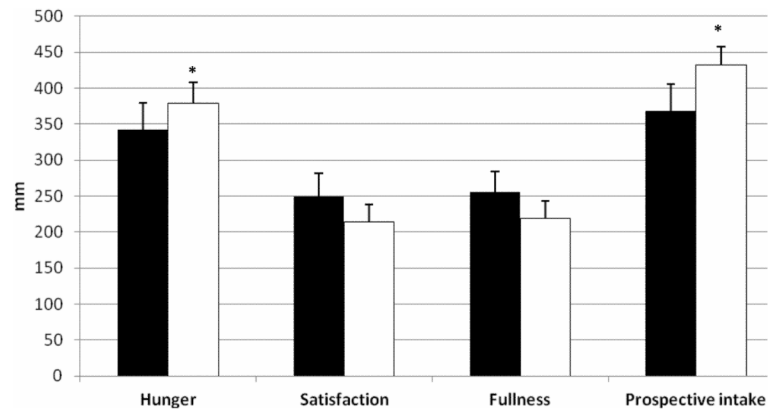
FIGURE 2.
Area under curve for visual analog scales assessing hunger, satisfaction, fullness, and prospective food intake after a breakfast with (black bars) or without (white bars) ginger. Data are means ± SEM, n = 10. *Significantly different from no ginger, P < 0.01.

Table 1

Overall metabolic profile before and after consumption of a breakfast with or without 2 g of powdered ginger.

Metabolite	Control	Ginger	P-value (main treatment effect)
Glucose, mg/dL	113.25 ± 2.19	112.90 ± 1.80	0.92
Triglycerides, mg/dL	104.75 ± 2.52	100.79 ± 2.46	0.72
C-reactive protein, ng/mL	1107.54 ± 8.99	952.61 ± 5.11	0.45
Tumor necrosis factor-α, pg/mL	1.29 ± 0.02	1.36 ± 0.01	0.49
Interleukin-6, pg/mL	0.766 ± 0.01	0.892 ± 0.01	0.17
Insulin, mg/dL	48.86 ± 3.81	46.02 ± 3.48	0.76
Leptin, mg/dL	6.95 ± 0.02	7.02 ± 0.03	0.86
Peptide YY, mg/dL[1]	111.05 ± 0.67	110.60 ± 0.61	0.88
Adiponectin, ng/mL	6032.33 ± 15.41	6030.70 ± 25.58	0.998
Glucagon-like peptide-1, mg/dL	37.99 ± 0.62	39.74 ± 0.68	0.42
Total ghrelin, mg/dL	695.94 ± 7.48	745.32 ± 8.87	0.059[2]
Active ghrelin, mg/dL	74.48 ± 1.83	79.65 ± 2.21	0.45

Data are metabolite concentrations throughout the measurement period, from fasting baseline through 3 h post-breakfast consumption, means ± SEM, n = 10.

[1] Data from n = 9.

[2] P-value became 0.018 after excluding one participant who had diabetes.