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Hunger and satiety responses to diets enriched with cottonseed oil vs. olive oil

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ABSTRACT

Studies suggest that the type of dietary fat consumed habitually may modulate appetite and further influence weight management. The purpose of this study was to evaluate the impact of an 8-week diet intervention enriched with either cottonseed oil (CSO; polyunsaturated fat-rich) or olive oil (OO; monounsaturated fat-rich) on appetite responses in adults with high cholesterol. This was a parallel design, randomized partial outpatient feeding trial designed to provide approximately 60% of participants daily energy needs with ~30% of energy needs as CSO ($n = 21$, BMI 27.3 ± 0.92 kg/m^2, age 53 ± 2y) or OO ($n = 21$, BMI 27.6 ± 1.20 kg/m^2, age 54 ± 2y). A high saturated fat meal challenge was completed at pre- and post-intervention visits with 5 h postprandial blood draws and visual analog scales (VAS) for cholecystokinin (CCK), peptide YY (PYY), ghrelin, and subjective appetite, respectively. Participants also completed VAS questionnaires hourly and recorded dietary intake after leaving the lab for the remainder of the day. There was a greater increase in fasting CCK (CSO: 0.54 ± 0.03 to 0.56 ± 0.04; OO: 0.63 ± 0.07 to 0.60 ± 0.06 ng/ml $p = 0.05$), a greater suppression of postprandial ghrelin ($p < 0.01$), and a greater increase in postprandial VAS fullness ($p = 0.04$) in CSO compared to OO. Additionally, there was a greater decrease in self-reported energy intake in CSO compared to OO (CSO: 2464 ± 123 to 2115 ± 123; OO: 2263 ± 147 to 2,434 ± 184 kcal/d $p = 0.02$). Only postprandial VAS prospective consumption showed greater suppression ($p = 0.03$) in OO vs. CSO. Altogether, these data show that CSO has a greater effect on appetite suppression than OO diet enrichment and may be beneficial for weight maintenance, especially in a population at-risk for chronic disease. Registered at clinicaltrials.gov: NCT04397055

1. Introduction

Obesity is a growing problem on the global scale [1,2] and is a risk factor for other chronic diseases [3]. Therefore, it is essential to identify weight management practices to serve as primary prevention of weight gain. One effective method may be through enhancement of appetite regulation [4]. Accepted approaches to assessing appetite include physiologic (gastrointestinal hormones), subjective, or applied measurements. Ghrelin is the only known "hunger hormone" and is released from the stomach in low energy states to activate orexigenic neurons, stimulating hunger and promoting feeding behavior [5]. Released from the proximal and distal gut in response to a meal, cholecystokinin (CCK) and peptide YY (PYY) are satiety hormones that stimulate anorexigneic neurons and inhibit orexigenic neurons leading to satiation and satiety [6, 7]. Additionally, CCK is known to stimulate the release of some hormones including PYY and suppress the release of ghrelin [8]. Subjective feelings or ratings of appetite are commonly measured using visual analog scales (VAS) anchored to questions about hunger and fullness [9]. Finally, applied methods of measuring appetite most commonly involve assessments of energy intake (EI) and/or macronutrient intake [10].

While high-fat (HF) diets are typically associated with weight gain [11] due to its highly palatable nature [12], the fatty acid (FA) composition of the diet may differentially influence appetite responses, and potentially impact long-term weight management [13–15]. Some acute meal challenge studies measuring subjective and applied appetite found no differences based on FA composition [16–22], while other studies do report differences [23–26]. Our lab has previously shown that single meals rich in polyunsaturated fat (PUFA) elicit a stronger PYY response when compared to a monounsaturated fat (MUFA) rich meal [22, 26]. Additionally, a 7-day, PUFA-rich diet has been shown to increase fasting and postprandial PYY, and decrease fasting ghrelin [27].

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These limited data suggest PUFA may have a greater effect on physiologic measures of appetite than MUFA, but is insufficient to make strong, or longer-term, conclusions.

There are very few dietary intervention studies focusing on FA composition, specifically PUFA vs. MUFA, and appetite [27–29], and even fewer using a whole foods approach. Consumers recognize olive oil (OO) as a "heart healthy" MUFA-rich oil, primarily based on research on the Mediterranean Diet [30]. While not as familiar to consumers, cottonseed oil (CSO), which is rich in PUFAs, is present in the food supply and has been linked to improvements in lipid metabolism and chronic disease risk reduction [31–34]. Only one previous study has examined CSO vs. OO on appetite [35]. They demonstrated the ability of CSO-rich diets and meals to improve physiologic and subjective measures of appetite, even when compared to OO [35]. However, that trial only included healthy males and was a 5-day diet protocol. The purpose of this study was to examine the impact of an 8-week, HF diet enriched with CSO or OO on fasting and postprandial appetite measures in a parallel design in adults at-risk for chronic disease [36]. We evaluated subjective ratings of appetite, self-reported EI, and biological hunger and satiety hormones in response to a partial outpatient feeding protocol. We hypothesized that CSO would elicit more favorable changes in physiologic and subjective measures of appetite with no differences in EI compared to OO based on the previous short-term CSO trial [35].

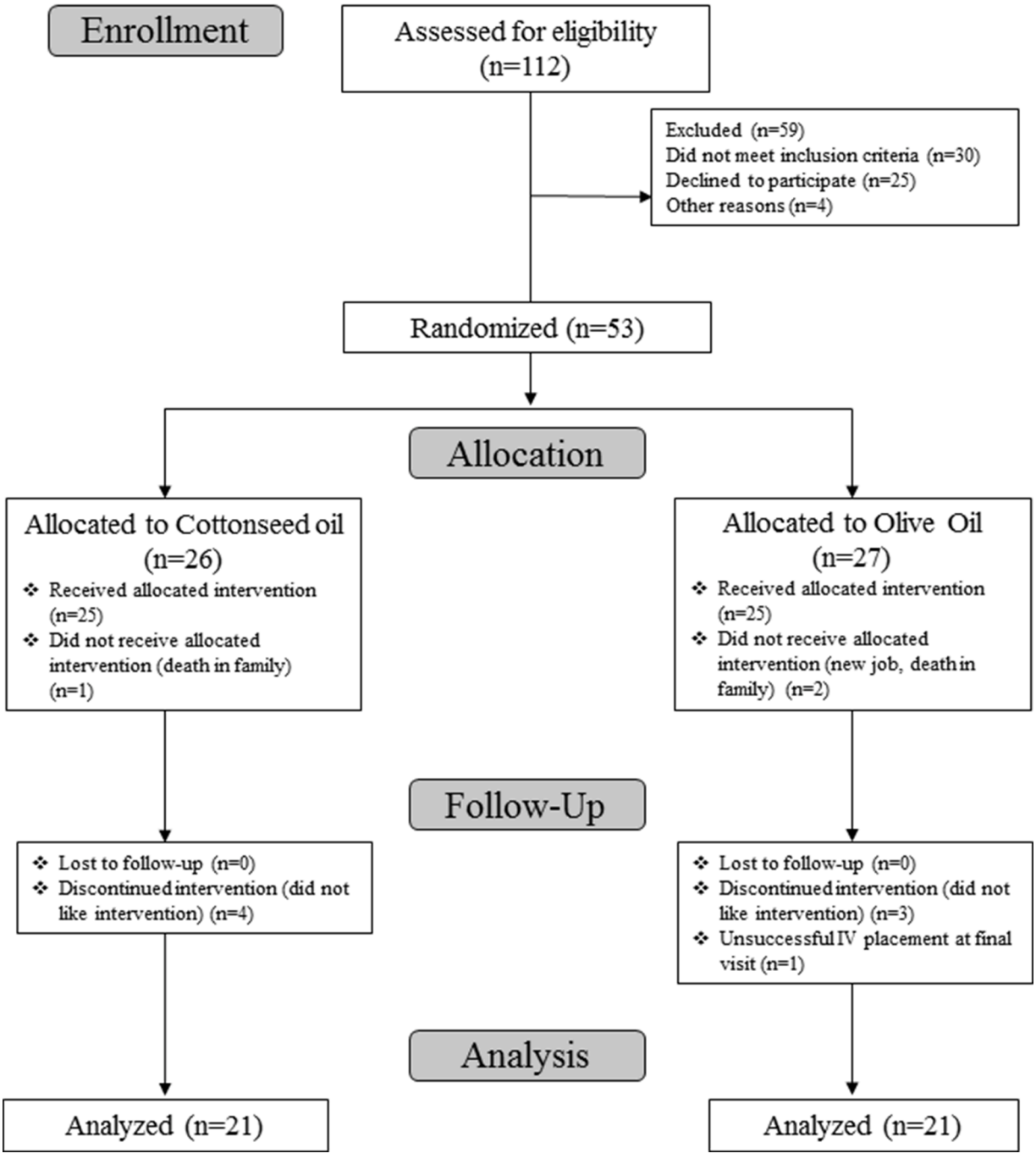


Fig. 1. CONSORT flow diagram selection of participants.

2. Materials and methods

2.1. Study design

This was a single-blind, randomized, parallel trial (clinicaltrials.gov: NCT04397055) involving an 8-week partial outpatient feeding intervention. Recruitment began in May 2018 and final testing took place in June 2021 when all primary and secondary outcomes were addressed. The protocol included a screening visit and two testing visits (pre- and post-intervention). Subjects were randomly assigned using balanced blocks to either an OO or CSO intervention arm as previous described [36]. This study was conducted according to the guidelines laid down by the Declaration of Helsinki and the Institutional Review Board for human subjects at the University of Georgia approved all procedures involving human subjects (STUDY00005869). Informed written consent was obtained from each participant prior to any testing procedures.

2.2. Participants

One hundred and twelve sedentary adults between the ages of 30 and 75y were assessed for eligibility (Fig. 1). Inclusion and exclusion criteria were measured at the screening visit and has been described previously [36]. Briefly, exclusion criteria included excessive alcohol use, tobacco use, exercise (>3 h/week), weight instability (gain or loss >5% of body weight in the past 3mo), lipid-lowering medications, diabetes medications, steroid/hormone therapies, and ADD/ADHD medications and chronic/metabolic diseases.

2.3. Protocol

2.3.1. Screening visit

Prior to arriving at the Human Nutrition Lab (HNL) on the morning of the screening visit, participants completed an 8–12 h overnight fast and 24 h abstaining from exercise and alcohol. Following anthropometric measures, a 30 min indirect calorimetry measurement (TrueOne 2400; Parvo Medics, Sandy, Utah, USA) was taken under standard conditions [37]. The Weir equation was applied to the final 20 min of respiratory gasses to calculate resting metabolic rate (RMR) [38]. Participants' RMRs were multiplied by 1.65, an average U.S. physical activity factor [39], to estimate total daily energy needs. This was used to calculate the test meal, as well as assignment to diet intervention energy tiers. Using a random number generator and stratified balanced blocks [36], a researcher who was not involved in the collection or analysis of these data assigned qualifying participants to either the OO or the CSO intervention groups.

2.3.2. Pre-intervention visit

Prior to the pre-intervention visit, participants completed a two-day food record consisting of one week day and one weekend day [40]. For this visit, participants arrived between 0700 and 0800 h to the HNL in the fasted state (8–12 h fast) after consuming a standardized dinner meal and snack (energy: 50% carbohydrate, 15% protein, 35% fat) the evening before the visit, which was provided by research personnel. Participants were instructed to consume enough of the standardized meal to be comfortably full. Height, weight, waist and hip circumference, blood pressure, and body composition were measured upon arrival. Body composition was measured by Bod Pod (Cosmed USA, Inc.). Next, participants completed a 100 mm visual analog scale (VAS) for hunger, fullness, prospective consumption, desire to eat to measure subjective appetite [41]. An intravenous catheter was placed in the antecubital vein and a fasting blood sample was collected. Saline was used to keep the IV-line patent throughout the protocol.

Once fasting measures were completed, participants consumed a high saturated fat (SFA) test meal which has been previously described [36]. Briefly, 35% of individual estimated daily energy needs were provided with 5%, 25%, and 70% of energy provided as protein, carbohydrate and fat, respectively. The FA breakdown was 47%, 16%, and 7% of total energy from SFA, MUFA, and PUFA, respectively. A high SFA meal was chosen to assess how changes in the fat composition of the chronic diet may influence appetite responses to an occasional "unhealthy" (high SFA) meal, which is commonly observed with weekend eating habits [42]. Four ounces of water was used to rinse the container and ingested to ensure the entire meal was consumed. A validated sensory questionnaire was administered to assess sensory modalities of the high SFA meal [43,44]. This questionnaire used a modified 9-point hedonic scale with '1' indicating 'dislike extremely' and '9' indicating 'like extremely'. Following the SFA-rich test meal, blood samples were collected, and VAS questionnaires were administered, intermittently for 5 h (time points: 30, 60, 90, 120, 150, 180, 240, 300 min). VAS measures taken at these time points were denoted as in lab (LAB) measures. Four ounces of water was provided hourly. After 5 h, the IV was removed and participants were instructed to eat a self-selected lunch within 1 h of leaving the lab and a self-selected dinner 4 h later. Participants were also provided with VAS questionnaires to complete once per hour for the next 7 h (starting at 360 min post SFA test meal). VAS measures taken at these time points were denoted as at-home (HOME) measures. In addition to our record of the SFA test meal, participants recorded all food and drink for the rest of the day on a food record. Participants were instructed to record everything consumed including condiments and beverages with attention to ingredients in recipes and brands of products. Estimated quantities were recorded in standard measures such as cups and tablespoons. Food records were analyzed using The Food Processor SQL software (version 10.12.0). The combined analysis of the SFA test meal and self-reported intake made up the total EI data.

2.3.3. Intervention protocol

The day following the pre-intervention visit, participants began the 8-week diet intervention. Participants were assigned to either CSO or OO intervention groups but were blinded to their assignment. The partial outpatient feeding of the intervention was designed to provide approximately 60% of energy needs (actual range was 56–87% of energy needs, with an average of 66±2% for CSO and 67±2% for OO) was utilized to allow for control of most of the diet, while allowing participants to maintain some of their usual diet. Participants were sent home with a one-week supply of daily meals and snacks that corresponded to their assigned group. They were informed of their estimated daily energy needs, how many kilocalories were being provided by the study foods, and how many kilocalories were left for them to fill with their own foods.

With the exception of the intervention oils (CSO or OO), the two diets were identical. All study foods were prepared by research personnel, and ingredients were weighted to the 0.01 g. A 7-day rotating menu was used to provide two meals plus snacks daily including breakfast and lunch/dinner entrée. This allowed for one self-selected meal daily. The meals provided included soups, sautéed vegetables with rice, turkey meatloaf with mashed potatoes and baked products. Breakfast shakes were provided 5 days per week as one of the two meals. Participants were instructed to prepare this breakfast shake by mixing proportioned shake mix with milk of choice and designated amount of the assigned intervention oil. The rest of the meals were pre-portioned by research personnel, packaged in microwave and freezer safe containers, and frozen. Participants were instructed on safe reheating practices.

Kilocalorie tiers, as described previously [36], were used to determine the quantity of food to provide participants. The intention of the tiers were to provide some (approximately 60%; actual range of 56–87%) of the estimated energy needs as foods from the study, leading to about 30% of their total energy needs being provided by the assigned oil (CSO or OO). Kilocalorie tier assignment was non-random and dependent on their estimated energy requirement, derived from their RMR at screening. Tier intervals are presented in Table 1. Fatty acid analyses of CSO and OO are presented in Supplemental Table 1.

During the intervention, participants completed weekly compliance

Table 1
Nutrient breakdown of provided intervention foods for each kilocalorie tier.

Intervention tiers (kcal)	Cottonseed oil				Olive oil			
	<1600	1600–2299	2300–3000	>3000	<1600	1600–2299	2300–3000	>3000
Energy (kcal)	1090	1402	1678	2107	1090	1402	1678	2107
Energy (KJ)	4561	5866	7021	8816	4561	5866	7021	8816
Energy from protein (%)	7.2	6.7	6.5	6.8	7.2	6.7	6.5	6.8
Protein (g)	19.1	23.0	26.8	35.2	19.1	23.0	26.8	35.2
Energy from carbohydrates (%)	36.9	36.3	36.3	39.0	36.9	36.3	36.3	39.0
Carbohydrates (g)	98.0	124.0	148.5	200.6	98.0	124.0	148.5	200.6
Fiber (g)	3.2	4.1	5.1	7.9	3.2	4.1	5.1	7.9
Sugar (g)	51.3	64.8	76.5	101.5	51.3	64.8	76.5	101.5
Energy from fat (%)	55.9	57.0	57.2	54.1	55.9	57.0	57.2	54.1
Total Fat (g)	65.5	85.9	103.1	122.7	65.5	85.9	103.1	122.7
Saturated fat (g)	15.3	20.0	24.0	28.7	12.3	16.0	19.3	23.2
Trans fat (g)	0.17	0.22	0.27	0.38	0.17	0.22	0.27	0.38
Monounsaturated fat (g)	13.3	17.4	20.8	24.7	44.8	58.8	70.6	83.5
Polyunsaturated fat (g)	36.8	48.4	58.1	68.8	8.3	10.9	13.0	15.5
Omega 3 fatty acid (g)	0.28	0.36	0.43	0.51	0.60	0.79	0.95	1.13
Omega 6 fatty acid(g)	36.6	48.0	57.6	68.3	7.8	10.1	12.1	14.4
Cholesterol (mg)	57.5	75.2	89.8	121.0	57.5	75.2	89.8	121.0
Total fat from intervention oil (%)	88.2	87.3	87.3	85.6	88.2	87.3	87.3	85.6
Fat from intervention oil (g)	57.8	75.0	90.0	105.0	57.8	75.0	90.0	105.0

Daily nutrients delivered through the provided study foods within each treatment and energy tier. Participants were assigned to a kilocalorie tier based on their estimated energy requirements from a resting metabolic rate measurement at the screening visit. Energy tiers are named for the range of total energy requirements of the participants that were assigned to that tier. Energy (kcal in the first row and (KJ) in the second row) is the amount of energy actually provided each day from the diet intervention foods. The only difference between treatments was from the different treatment oil used (cottonseed oil vs. olive oil). <1600 kcal = <6694 KJ; 1600–2299 kcal = 6694–9619 KJ; 2300–3000 kcal = 9623–12,552 KJ; >3000 kcal = >12,552 KJ.

checklists as a self-reported measure of compliance, bi-weekly two-day food records including a week day and a weekend day, and the sensory evaluation questionnaire the first time they ate any of the provided foods. Participants returned to the HNL on a weekly basis to pick up meals and turn in study materials.

2.3.4. Post-intervention visit

Participants returned to the HNL for their post-intervention visit after completing the 8-week partial outpatient feeding. All procedures from the pre-intervention visit were repeated at the post-intervention visit.

2.4. Plasma sample analyses

All blood samples were drawn into EDTA vacutainers (Becton, Dickinson and Company, Franklin Lakes, NJ) with 10 μl/ml whole blood each of 4-benzenesulfonyl fluoride hydrochloride (AEBSF) and Dipeptidyl Peptidase IV (DPP IV) inhibitor (Sigma Aldrich, St. Louis, MO, USA), immediately placed on ice, and then centrifuged for 15 min at 4 °C. The plasma was aliquoted and stored at −80 °C until analysis. Total PYY and active ghrelin were measured using radioimmunoassays (Millipore, Billerica, MA, USA), and total CCK was measured using an extraction free enzyme immunoassay (Phoenix Pharmaceuticals, Burlingame, CA, USA). Each participant's total number of samples were run within the same assay.

2.5. Statistical analysis

SAS version 9.2 (SAS Institute Inc., Cary, NC, USA) was used for statistical analyses. The Food Processor SQL software (version 10.12.0) was used to assess all food record data. All values are reported as mean ± SEM unless otherwise noted. Statistical significance was set at $p \leq 0.05$. A sample size of 40 (20 per group) was estimated using a Cohen's F of 0.233 to detect a difference in postprandial ghrelin based on the results from Polley et al. [35]. A second sample size calculation estimated a sample size of 24 (12 per group) using a Cohen's F of 0.312 based on the postprandial hunger VAS ratings presented also in Polley et al. [35]. Sample size was calculated using G*power 3.19.7 assuming at least 80% power and an α of 0.05. The decision to use per-protocol analyses was made a priori. A repeated measures (RM) linear mixed model for treatment and visit was used to determine between and within group differences for fasting hormone and VAS data, energy and macronutrient intake on test days anthropometric data, and sensory evaluation data of SFA test meals. Postprandial hormone and VAS data was assessed for between and within group differences using a RM linear mixed model for treatment, visit, and time point. All VAS data (incremental area under the curve (iAUC) and time course) was analyzed with all time points (0 – 720 min), as well as LAB (time points 0 – 300 min) and HOME (time points 360 – 720 min) subgroups separately using similar linear mixed models. In all linear mixed models, participants were modeled as random effects. When significance was found, post hoc analyses were done using Tukey's test. Finally, unpaired t-tests were used to assess differences between groups in self-reported compliance and sensory evaluation data of intervention foods.

3. Results

3.1. Participants

Fifty-three participants were allocated to an intervention group, but eleven did not start or complete the intervention and/or testing visits (Fig. 1). Thus, forty-two individuals (12 women and 9 men in CSO and 14 women and 7 men in OO) completed the study and were included in the final analysis. Participant anthropometrics at pre- and post-intervention visits are presented in Table 2. Weight (kg) and BMI (kg/m^2) increased in both groups but this increase was not different between groups. No other anthropometric characteristic changed during the intervention. Compliance of subjects averaged at 91 ± 2% and 92 ± 1% for CSO and OO groups, respectively, with no differences in compliance between groups.

3.2. Physiologic responses

Fasting plasma CCK, PYY, and ghrelin levels are presented in Table 3. There was a treatment by visit interaction ($p < 0.05$) with an increase in fasting CCK in CSO and a decrease in OO from pre- to post-intervention visit. There were no changes in fasting PYY or ghrelin. The time course of the meal responses and corresponding iAUC for CCK, PYY, and ghrelin

Table 2
Population characteristics.

	Cottonseed oil ($n = 21$)		Olive oil ($n = 21$)		p-values		
	Pre-intervention	Post-intervention	Pre-intervention	Post-intervention	Treatment	Visit	Interaction
Anthropometric Characteristics							
Age (y)	53±2	–	54±2	–	0.73	–	–
Height (cm)	169.0 ± 2.2	169.0 ± 2.2	168.0 ± 1.9	168.0 ± 1.9	0.72	0.66	0.56
Weight (kg) *	78.6 ± 3.6	79.6 ± 3.5	77.9 ± 3.6	78.9 ± 3.7	0.89	<0.001	0.79
Body Mass Index (kg/m^2) *	27.3 ± 0.9	27.7 ± 0.9	27.6 ± 1.2	27.9 ± 1.2	0.86	<0.001	0.74
Waist to Hip ratio	0.85 ± 0.02	0.85 ± 0.02	0.82 ± 0.02	0.82 ± 0.02	0.39	0.24	0.34
Body Fat (%)	31.0 ± 2.1	32.2 ± 1.9	32.9 ± 2.6	33.2 ± 2.6	0.65	0.17	0.43

Values are presented as mean ± SEM. Age was analyzed using a two-sample *t*-test. All other characteristics were analyzed using a linear mixed model for treatment and visit.

* indicates visit effect for increase from pre-intervention to post-intervention regardless of group assignment.

Table 3
Fasting hormones and subjective appetite.

	Cottonseed oil ($n = 21$)		Olive oil ($n = 21$)		p-values		
	Pre-intervention	Post-intervention	Pre-intervention	Post-intervention	Treatment	Visit	Interaction
CCK (ng/ml) *	0.54 ± 0.03	0.56 ± 0.04	0.63 ± 0.07	0.60 ± 0.06	0.40	0.59	0.05
PYY (pg/ml)	96.5 ± 7.05	93.6 ± 6.37	97.5 ± 9.78	97.7 ± 9.75	0.82	0.61	0.56
Ghrelin (pg/ml)	125 ± 14.3	128 ± 13.3	134 ± 14.6	131 ± 15.7	0.98	0.11	0.44
Hunger (mm)	46.6 ± 4.8	51.4 ± 7.1	48.1 ± 5.7	51.2 ± 5.7	0.93	0.24	0.81
Fullness (mm)	28.7 ± 5.4	21.5 ± 4.4	17.1 ± 4.4	16.3 ± 5.0	0.17	0.15	0.25
Prospective Consumption (mm)	49.3 ± 4.6	47.4 ± 4.9	50.8 ± 4.2	52.0 ± 4.4	0.59	0.92	0.59
Desire to Eat (mm)	51.8 ± 5.7	49.1 ± 5.5	52.6 ± 5.0	50.0 ± 5.7	0.89	0.43	0.99
Appetite Score (mm)	54.7 ± 4.3	56.6 ± 4.8	58.6 ± 4.0	59.2 ± 4.6	0.57	0.58	0.79

All values are presented as mean ± SEM. All fasting data was analyzed using linear mixed model for treatment and time.

* indicates a significant difference between groups (treatment by visit interaction at $p < 0.05$). Tukey's test reveals this difference was driven by slight increases in CCK in CSO and slight decreases in OO. CCK = Cholecystokinin, CSO = cottonseed oil, mm = millimeter, OO = Olive oil, PYY = Peptide YY.

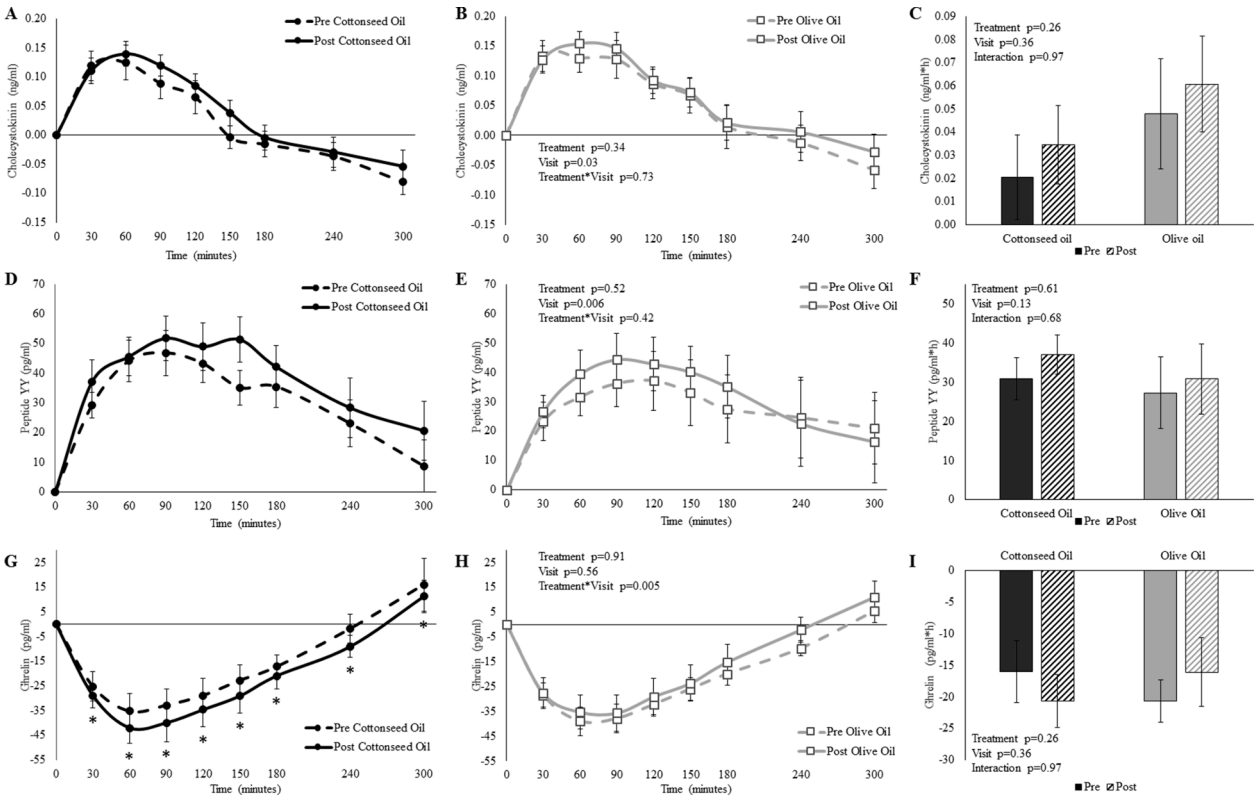


Fig. 2. Plasma postprandial change from baseline cholecystokinin (CCK) (A, B), peptide YY (PYY) (D, E), and ghrelin (G, H) at pre- and post-intervention visits. Data were analyzed using a linear mixed model for treatment, time, and visit. * indicates significant treatment by visit interaction. Plasma iAUC at pre- and post-intervention visit for CCK (C), PYY (F), and ghrelin (I) were analyzed using a linear mixed model for treatment and visit. No significant differences were found for iAUC data. Pre- pre-intervention visit, post- post-intervention visit, iAUC- incremental area under the curve.

are presented in Fig. 2. There was a visit effect ($p = 0.03$) for higher postprandial CCK (Fig. 2A, B) from pre- to post-intervention visit in both groups, and this increase was not different between groups. Similarly, the PYY meal response (Fig. 2D, E) was increased from pre- to post-intervention in both groups (visit effect $p = 0.006$), but this change was not different between groups. For ghrelin (Fig. 2G, H), there was a significant treatment by visit interaction ($p = 0.005$) which was driven by greater postprandial ghrelin suppression in CSO vs. OO, although post hoc analyses did not reach significance. No significant differences between groups were observed for iAUC measures (Fig. 2C, F, I).

3.3. Subjective ratings of appetite

There were no differences between groups for any fasting subjective measure of appetite (Table 3). The time course of meal responses and corresponding iAUC at pre-and post-intervention visits for VAS are presented in Figs. 3 and 4 and Table 4. For the appetite score time course data (Fig. 3A, B), there was a visit effect ($p = 0.004$) for an increase from pre- to post-intervention, but no difference between groups. When analyzed further, this 'all day' visit effect was driven by the LAB measures (visit effect $p < 0.001$) but not the HOME measures (visit effect $p = 0.44$).

Despite the time course composite appetite score (Fig. 3A, B) changing in similar ways in both groups, there were differences between groups for individual VAS questions. There was a treatment by visit interaction ($p = 0.04$) for fullness (Fig. 3C, D). Post hoc analyses revealed greater increases in fullness in the CSO group ($p = 0.02$) vs. the OO group ($p = 1.00$). After further analysis, the HOME (interaction $p = 0.04$) rather than the LAB measures (interaction $p = 0.46$) drove the fullness interaction. Conversely, prospective consumption ratings (Fig. 4A, B) ("how much do you think you could eat right now?") were lower at the post- vs. pre-intervention in both groups (visit effect $p = 0.003$), but there was a greater decrease in OO vs. CSO group (treatment by visit interaction $p = 0.03$; OO $p = 0.003$; CSO $p = 0.92$). The LAB measures were found to mirror the visit ($p < 0.001$) and interaction effects (interaction $p = 0.02$; OO $p < 0.001$; $p = 0.48$) but the HOME measures were not significant. Time course ratings of hunger (Fig. 4C, D) decreased regardless of group assignment (visit effect $p < 0.001$). After further analysis, this decrease was present for both LAB (visit effect $p < 0.001$) and HOME measures (visit effect $p = 0.009$). There were no differences between groups for desire to eat (Fig. 4E, F). Finally, for iAUC measures, appetite score and hunger showed visit effects for LAB (Table 4) but no other main or interaction effects for iAUC were observed.

3.4. Applied responses

Total energy and macronutrient intake from the pre- and post-intervention visit days based on the analysis of the food diaries and the provided SFA meal are presented in Fig. 5. There was a significant difference in total EI (treatment by visit interaction $p = 0.02$) driven by a trend for decrease in the CSO group ($p = 0.11$) compared to non-significant increase in the OO group ($p = 0.66$). This reduction in total EI in the CSO group and increase in the OO group was driven by corresponding changes in energy from protein (treatment by visit interaction $p < 0.001$; CSO $p = 0.015$; OO $p = 0.011$)) and carbohydrate (treatment by visit interaction $p = 0.002$; CSO $p = 0.02$; OO $p = 0.34$). There were no changes in EI from fat or alcohol.

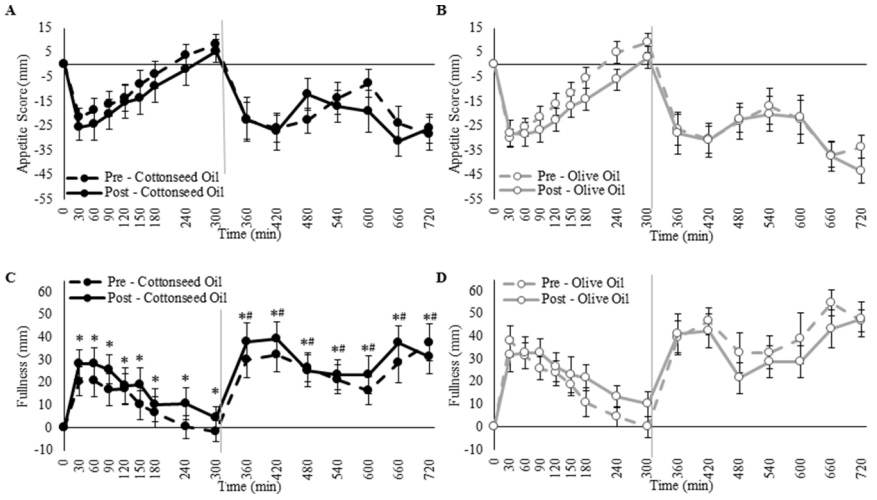
3.5. Sensory evaluation

Sensory evaluations of the SFA meal and the intervention foods are presented in Table 5. There were no significant differences within or between groups for any characteristic of the SFA test meal. Additionally, there were no differences between groups for overall acceptance of the CSO or OO-enriched foods provided during the intervention.

4. Discussion

For the first time, we have shown that an 8-week, CSO-enriched diet leads to better improvement in appetite control, compared to an OO-enriched diet in adults at risk for chronic disease. Specifically, CSO diet enrichment led to increases in fasting CCK, increased postprandial feelings of fullness, and suppression of postprandial ghrelin when compared to the OO-enriched diet. The only measure where OO was better than CSO was for greater improvements in postprandial desire to eat ratings. Similar between group improvements were observed in postprandial feelings of hunger, appetite score, CCK and PYY. Finally, and possibly most importantly, the aforementioned greater improvements in subjective and physiologic measures of appetite with CSO diet enrichment resulted in reductions in EI in the CSO, but not OO group.

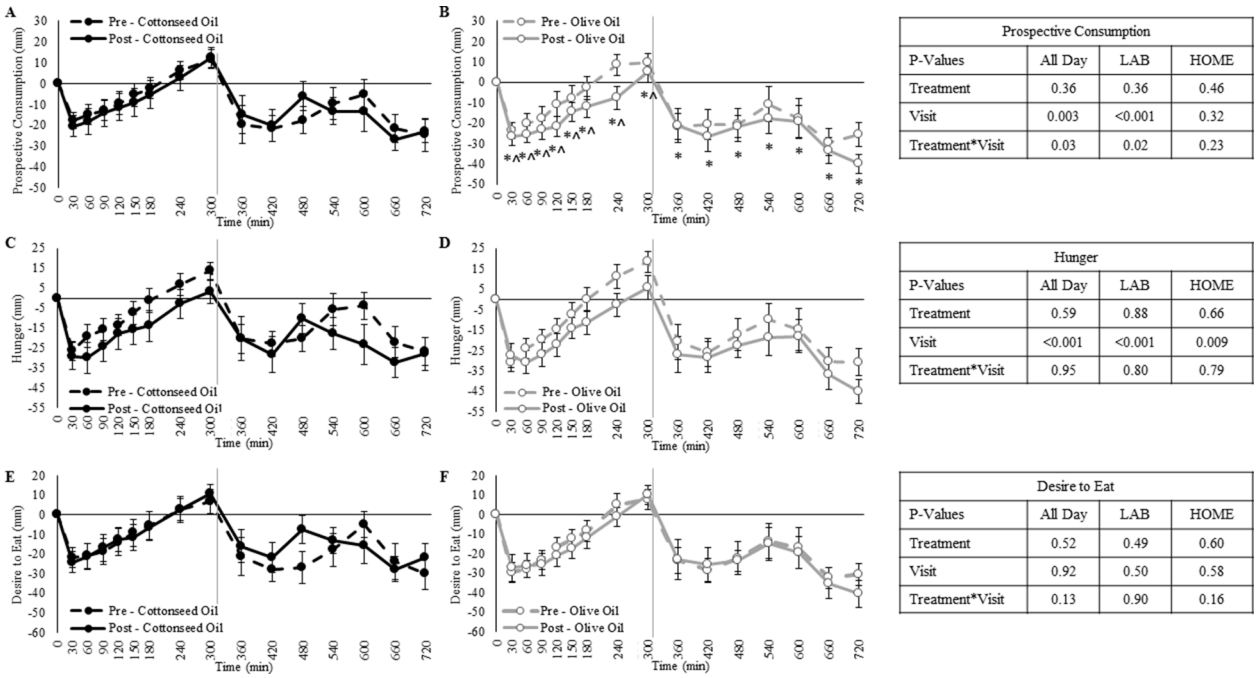
Of the appetite hormones measured, CCK and ghrelin appear to be consistently responsive to CSO consumption either in fasting or postprandial measures [35]. Our results demonstrated more favorable



Appetite Score			
P-Values	All Day	LAB	HOME
Treatment	0.37	0.45	0.41
Visit	0.004	<0.001	0.44
Treatment*Visit	0.61	0.45	0.84

Fullness			
P-Values	All Day	LAB	HOME
Treatment	0.14	0.26	0.15
Visit	0.04	<0.001	0.68
Treatment*Visit	0.04	0.46	0.04

Fig. 3. Postprandial change from baseline subjective VAS (visual analog scale) appetite score (A, B) and fullness ratings (C, D) at pre- and post-intervention visits. Data were analyzed using a linear mixed model for treatment, time, and visit. * indicates significant treatment by visit interaction for all day measures (time 0–720). # indicates significant treatment by visit interaction for HOME measures. Pre- pre-intervention visit, post- post-intervention visit, All Day- VAS measures taken across the whole visit day, LAB- VAS measures taken time 0–300, HOME- VAS measures take time 360–720.



Prospective Consumption			
P-Values	All Day	LAB	HOME
Treatment	0.36	0.36	0.46
Visit	0.003	<0.001	0.32
Treatment*Visit	0.03	0.02	0.23

Hunger			
P-Values	All Day	LAB	HOME
Treatment	0.59	0.88	0.66
Visit	<0.001	<0.001	0.009
Treatment*Visit	0.95	0.80	0.79

Desire to Eat			
P-Values	All Day	LAB	HOME
Treatment	0.52	0.49	0.60
Visit	0.92	0.50	0.58
Treatment*Visit	0.13	0.90	0.16

Fig. 4. Postprandial change from baseline subjective VAS (visual analog scale) ratings of prospective consumption (A,B), desire to eat (C, D), and appetite score (E, F) at pre- and post-intervention visits. Data were analyzed using a linear mixed model for treatment, time, and visit. * indicates significant treatment by visit interaction for all day measures (time 0–720). ^ indicates significant treatment by visit interaction for LAB measures. Pre- pre-intervention visit, post- post-intervention visit, All day- all VAS measures taken across the whole visit day, LAB- VAS measures taken time 0–300, HOME- VAS measures take time 360–720.

Table 4
Incremental area under the curve subjective appetite.

	Cottonseed oil ($n = 21$)		Olive oil ($n = 21$)		p-values		
	Pre-intervention	Post-intervention	Pre-intervention	Post-intervention	Treatment	Visit	Interaction
Hunger							
All day (mm/d*h)	−9.9 ± 3.8	−16.3 ± 5.8	−13.2 ± 4.7	−18.6 ± 4.8	0.65	0.06	0.87
LAB (mm/d*h) #	−5.6 ± 4.3	−13.7 ± 6.4	−6.3 ± 4.6	−14.9 ± 5.0	0.89	0.01	0.94
HOME (mm/d*h)	−13.8 ± 4.4	−18.9 ± 6.6	−16.9 ± 6.3	−23.1 ± 6.0	0.62	0.13	0.87
Fullness							
All day (mm/d*h)	17.1 ± 4.6	22.1 ± 4.8	28.2 ± 4.7	27.8 ± 4.7	0.16	0.40	0.33
LAB (mm/d*h)	9.4 ± 5.2	15.9 ± 5.8	17.3 ± 4.5	22.9 ± 5.5	0.27	0.06	0.89
HOME (mm/d*h)	22.9 ± 4.8	28.2 ± 4.9	37.3 ± 5.6	33.4 ± 5.0	0.12	0.84	0.19
Prospective Consumption							
All day (mm/d*h)	−9.8 ± 4.0	−10.2 ± 4.9	−13.3 ± 4.6	−17.4 ± 4.0	0.33	0.39	0.50
LAB (mm/d*h)	−4.0 ± 4.1	−6.6 ± 5.3	−6.1 ± 4.6	−14.3 ± 4.1	0.38	0.07	0.33
HOME (mm/d*h)	−14.3 ± 4.9	−12.4 ± 5.7	−17.5 ± 5.9	−21.0 ± 5.2	0.39	0.81	0.40
Desire to Eat							
All day (mm/d*h)	−13.6 ± 4.9	−10.9 ± 4.6	−16.9 ± 4.3	−17.0 ± 5.0	0.43	0.65	0.62
LAB (mm/d*h)	−8.1 ± 5.5	−8.0 ± 5.4	−10.9 ± 4.1	−13.8 ± 4.9	0.49	0.63	0.62
HOME (mm/d*h)	−18.4 ± 5.7	−13.5 ± 5.3	−20.5 ± 6.0	−21.6 ± 6.3	0.48	0.58	0.40
Appetite Score							
All day (mm/d*h)	−12.6 ± 3.4	−14.9 ± 4.6	−17.6 ± 3.8	−19.9 ± 4.1	0.33	0.26	0.99
LAB (mm/d*h) #	−6.8 ± 4.2	−11.1 ± 5.4	−9.9 ± 3.6	−16.2 ± 4.2	0.46	0.03	0.66
HOME (mm/d*h)	−17.3 ± 3.8	−18.2 ± 5.2	−23.1 ± 5.3	−24.8 ± 5.1	0.33	0.60	0.88

All values presented as mean±SEM. These data were analyzed using linear mixed models for treatment and visit.

# indicates a significant visit effect $p < 0.05$. Regardless of group, "LAB" hunger and appetite score were reduced at post-intervention vs. pre-intervention. All day = measures taken time points 0–720 min, LAB = VAS measures taken time points 0–300 min, HOME = VAS measures taken time points 360 – 720 min, VAS = visual analog scale, mm = millimeters.

fasting CCK responses with CSO compared to OO diets; however, the previous CSO v. OO study by Polley et al. found CSO meals to elicit greater postprandial CCK responses [35]. Additionally, postprandial ghrelin is consistently suppressed whether CSO is consumed daily (and not part of the test meal), or as part of the test meal [35]. While Polley et al. did not find changes in ghrelin responses from pre- to 5-day post-diet intervention, they were able to show that consuming CSO meals suppressed ghrelin more than OO meals [35]. It is possible that the longer intervention and lack of CSO and OO in test meals used in the present study is responsible for the slight differences in hormone responses between trials. Of note, there is no established reference range for changes in these hormones that would be considered clinically relevant for appetite suppression. The work of others has demonstrated that increases in CCK and decreases in ghrelin are associated with subjective feelings of appetite and decreased energy intake [45, 46]. However, these examples observed more robust changes in those hormones than presented here. Importantly, though, others have observed changes in these hormones postprandially in similar magnitude as what

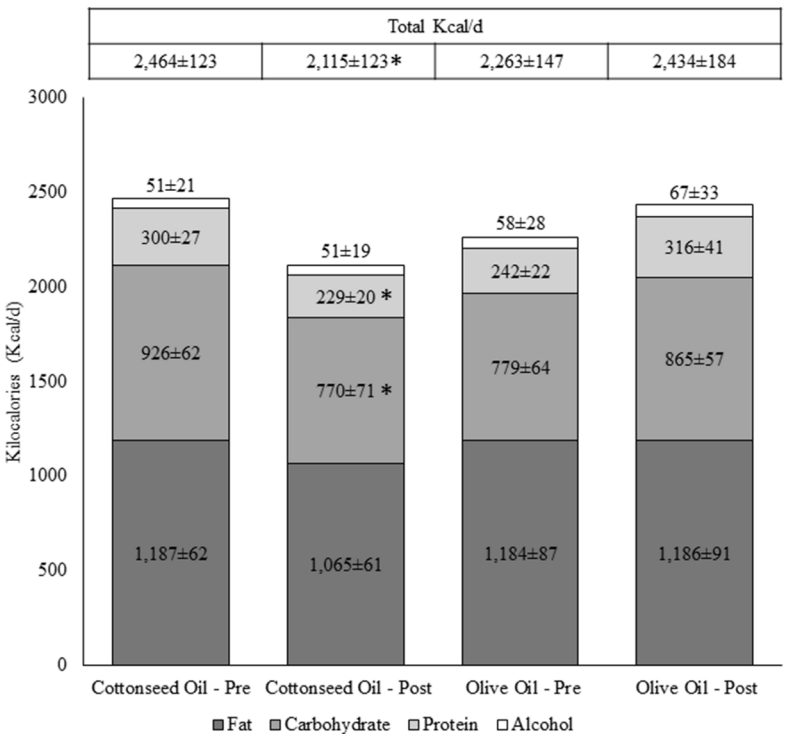


Fig. 5. Total and macronutrient intake consumed on the days of the pre- vs. post-intervention visits. Data were analyzed using a linear mixed model for treatment and visit and are expressed in kcals (kilocalories). * indicates significant treatment by visit interaction. Pre- pre-intervention visit, post- post-intervention visit.
Kilojoule (KJ) equivalents of energy intake are as follows.
CSO pre: total EI 10,309±515 KJ/d; alcohol 213±87 KJ/d; protein 1256±112 KJ/d; carbohydrate 3874±261 KJ/d; fat 4964±258 KJ/d.
CSO post: total EI 8849±515 KJ/d, alcohol 212 ±81 KJ/d; protein 958±82 KJ/d; protein 3223 ±297 KJ/d; fat 4456±256 KJ/d.
OO pre: total EI 9468±615; alcohol 241±118 KJ/d; protein 1013±92 KJ/d; carbohydrate 3259±267 KJ/d; fat 4956±365 KJ/d.
OO post: total EI 1184±770 KJ/d; alcohol 280 ±138 KJ/d; protein 1322±172 KJ/d; carbohydrate 3620±240 KJ/d; fat 4962±382 KJ/d.

Table 5
Sensory evaluation.

	Cottonseed oil (n = 21)		Olive oil (n = 21)		p-values		
	Pre-intervention	Post-intervention	Pre-intervention	Post-intervention	Treatment	Visit	Interaction
Sensory evaluation of SFA meal							
Appearance	5.5 ± 0.4	5.7 ± 0.4	5.2 ± 0.4	5.6 ± 0.4	0.71	0.36	0.68
Taste/Flavor	6.8 ± 0.3	6.6 ± 0.4	6.2 ± 0.4	6.0 ± 0.5	0.23	0.33	1.00
Texture/Consistency	5.9 ± 0.4	5.8 ± 0.4	5.6 ± 0.4	5.7 ± 0.4	0.60	0.77	0.77
Aroma/Smell	6.4 ± 0.4	6.4 ± 0.4	6.0 ± 0.4	5.6 ± 0.4	0.20	0.56	0.44
Overall Acceptability	6.9 ± 0.3	6.5 ± 0.3	6.0 ± 0.4	6.0 ± 0.4	0.10	0.29	0.29
Sensory evaluation of intervention food							
Appearance	6.6 ± 0.2	–	6.9 ± 0.3	–	0.50	–	–
Taste/Flavor	7.0 ± 0.2	–	7.4 ± 0.2	–	0.14	–	–
Texture/Consistency	6.7 ± 0.2	–	7.1 ± 0.2	–	0.16	–	–
Aroma/Smell	6.8 ± 0.2	–	7.2 ± 0.2	–	0.23	–	–
Overall Acceptability	7.0 ± 0.2	–	7.2 ± 0.2	–	0.32	–	–

Values presented as mean ± SEM. Ratings are based on a 9-point hedonic liking scale with 1 indicating "extremely dislike", 5 indicating "neither like or dislike", and 9 indicating "extremely like". Sensory evaluation of SFA meal was analyzed using a linear mixed model for treatment and visit while sensory evaluation of intervention food was analyzed using a two-sample t-test. There were no differences within or between groups for sensory evaluation of the SFA meal or the intervention foods. SFA = saturated fatty acid.

was observed in the current study but without reductions in energy intake [47–49]. Furthermore, increases in fasting CCK are not commonly observed in response to diet interventions; yet, small differences in basal CCK (<5 pmol/L) (similar to our observed changes) are suggested to be a driver of reduced appetite and energy intake associated with old age [50]. Taken together, this highlights that small changes in fasting or postprandial appetite hormones are difficult to interpret at this time but may still be physiologically relevant. The clinical relevance of our results may lie more in the concurrence of improvements in physiologic and subjective measures of appetite than either hormone change independently, along with the subsequent changes in self-reported energy intake.

For VAS, the changes in fullness we observed in the CSO group were similar to 4 weeks of a PUFA supplement study that resulted in improvements in fullness ratings and a reduction in EI compared to a control diet [51]. Conversely, Polley et al. [35] reported decreased hunger ratings with CSO, but similar improvements in fullness for CSO and OO. Our study design was different from these two studies, and most other FA composition and appetite literature, in that we included "HOME" measures of VAS. While this gives us a more complete picture of subjective appetite ratings across the entire day, it is difficult to compare our results to previous studies. Some of our results were driven by the HOME measures, demonstrating the importance for obtaining this data outside of laboratory visits.

While our measure of EI is self-reported and should be interpreted with some caution, it is not common to find differences in EI based on FA composition acutely or with diet interventions [16–21,25,26,35]. This highlights the importance of our data showing suppressed EI with CSO vs. OO diet enrichment. It also suggests there may be additional characteristic besides the FA composition of CSO that is suppressing appetite, or the FA composition of CSO is uniquely able to suppress EI compared to other oils. Unlike most other PUFA-rich oils, CSO also contains a relatively high amount of SFA [52]. Since SFAs have been shown to have a greater effect on postprandial appetite hormones and VAS in some

studies [13,23,26,53], it is possible that the combination of high PUFA and moderately high SFA of CSO has a more potent effect on appetite regulation.

While CSO was more favorable than OO in many appetite-related outcomes, we also observed several visit effects in both subjective and physiologic measures that could have been a result of multiple factors. First, there was a 1 kg average weight gain across the intervention that was the same in both groups. Weight gain has known implications on appetite and may explain some of the improvements observed in both groups from pre- to post-intervention. However, the fact that this weight gain was consistent between groups preserves the validity of the between group differences we have observed. Second, most studies that are looking at the impact of FA composition on appetite regulation are incorporating HF meals and/or diets [22,26–28, 54]. It is possible that simply being on a HF diet could have driven some of our visit effects. Lastly, differences in the rate of absorption of FA have been hypothesized to drive differences in appetite hormone responses observed in acute meal challenge studies [55]. Saturated FAs are absorbed more slowly than unsaturated FAs suggesting that having the same high SFA meal challenge could have driven some of the visit effects and overrode the daily diet FA composition differences. However, our use of an SFA-rich meal was intentional. Since it has already been shown that acute CSO consumption more favorably alters appetite hormone levels compared to OO [35], we sought to determine whether daily CSO consumption could be protective, from an appetite standpoint, against an occasional unhealthy (high SFA) meal.

The implications of reduced EI during an afternoon and evening of self-selected meals becomes more apparent in the context of weight management. Traditional weight loss recommendations are to reduce intake by 500 – 1000 kcal/day (2092–4184 KJ/day) to observe 1–2 lbs. of weight loss per week [56,57]. While this is an oversimplification of the complicated physiologic mechanisms controlling weight loss [58, 59], it is of note that our observed reduction of 349 kcal (1460 KJ) corresponds to 69.7% of this 500 kcal/day (2092 KJ/day) deficit recommendation. This was achieved on a day when the breakfast test meal was a fixed percentage of energy needs thus magnifying the 349 kcal (1460 KJ) reduction for the remaining meals of the day. Furthermore, it is well known that appetite increases proportional to weight loss [60] making adherence to weight loss and weight maintenance programs difficult. Therefore, the reduction in EI with CSO diet enrichment, along with the improvements in physiologic and subjective appetite measures, demonstrates the potential usefulness of CSO in curbing appetite, supporting weight loss, and encouraging weight maintenance. This requires additional research with CSO incorporation as part of a weight loss or weight maintenance protocol,

This study is not without limitations. A completely controlled feeding trial may have been better to ensure weight maintenance, but our partial outpatient feeding trial shows that even with some variation in food choice we are still able to observe improvements between the two intervention groups. Along those lines, the use of kilocalorie tiers limited the precision of the proportion of energy we provided but were employed for practicality of batch cooking of the intervention. Importantly the percent energy provided by study foods was not different between groups, therefore preserving comparability between groups. Secondly, we measured total PYY (PYY $_{1–36}$ and PYY $_{3–36}$) and CCK (CCK 33) rather than active forms. The biologically active forms of both of these peptides are associated with appetite control [61,62], and by measuring the total peptide we may be missing changes in the concentration of the active forms specifically. However, the measurement of these total forms has been shown to track similarly with their active counterparts in postprandial responses [63,64]. Another limitation is our use of self-reported compliance and intake. Self-reported measures are known to introduce under- and over-reporting [65,66] which may have influenced our results. Furthermore, the use of a relatively high amount of oil may have limited the application of our results to people following lower fat diets. While HF diets are gaining popularity (e.g. ketogenic diet), further research is needed to determine the effects of lower doses of CSO. Additionally, the use of an average physical activity factor for estimated energy expenditures of all participants may have limited our ability to apply individual activity levels to our estimates, or to account for limitations in physical activity due to COVID-19 restrictions. However, we do believe this average activity factor still appropriately described our generally sedentary population. Finally, this study was not designed to evaluate the effect of sex, race, or BMI; thus, future work is required to elucidate these interactions.

In conclusion, this is the first study to find improvements in appetite control with CSO diet enrichment in adults at risk for cardiovascular disease. Fasting CCK and subjective feelings of fullness were higher, and postprandial ghrelin and EI were suppressed, with CSO diet enrichment compared to OO diet enrichment. The OO diet enrichment did improve subjective desire to eat but there were no other measures where OO enrichment was more favorable. Improvements in postprandial CCK, PYY, subjective ratings of hunger and appetite score were not different between groups and may be attributable to the HF diets or the slight energy surplus (based on 1 kg weight gain over 8 weeks) rather than characteristics of the individual oils. Future research should focus on the potential use of CSO in the context of weight management including diets with lower doses of CSO.

Ethical statement

We are writing to confirm that we received Institutional Review Board (IRB) ethics approval from the University of Georgia (STUDY00005869) to conduct the research that is presented in our manuscript titled “Hunger and satiety responses to diets enriched with cottonseed oil vs. olive oil”. All participants provided informed written consent prior to testing, and this information is provided within the manuscript.

Data code and availability

Available upon request.

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CRediT authorship contribution statement

M.Catherine Prater: Investigation, Data curation, Formal analysis, Visualization, Writing – original draft. **Alexis R. Scheurell:** Methodology, Investigation, Data curation, Writing – review & editing. **Chad M. Paton:** Conceptualization, Funding acquisition, Methodology, Supervision, Resources, Writing – review & editing. **Jamie A. Cooper:** Conceptualization, Funding acquisition, Methodology, Supervision, Resources, Project administration, Writing – review & editing.

Declaration of Competing Interest

None

Data availability

Data will be made available on request.

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None

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.physbeh.2022.114041.

References

[1] C.M. Hales, et al., Prevalence of obesity and severe obesity among adults: United States, 2017–2018, NCHS Data Brief (360) (2020). Hyattsville, MD: National Center for Health Statistics.
[2] World Health Organization, Obesity and Overweight [Fact Sheet], 2021.
[3] Mayo Clinic, Obesity, Symptoms & Causes. 2021 May 24, 2022.]Available from, https://www.mayoclinic.org/diseases-conditions/obesity/symptoms-causes/syc-20375742.
[4] J.C. Halford, J.A. Harrold, Satiety-enhancing products for appetite control: science and regulation of functional foods for weight management, in: Proceedings of the Nutrition Society 71, 2012, pp. 350–362.
[5] K. Howick, et al., From belly to brain: targeting the ghrelin receptor in appetite and food intake regulation, Int. J. Mol. Sci. 18 (2) (2017) 273.
[6] H. Ueno, et al., The role of PYY in feeding regulation, Regul. Pept. 145 (1–3) (2008) 12–16.
[7] G.J. Dockray, Cholecystokinin, Curr. Opin. Endocrinol., Diabetes Obes. 19 (1) (2012) 8–12.
[8] L. Degen, et al., Effect of CCK-1 receptor blockade on ghrelin and PYY secretion in men, Am. J. Physiol.-Regul. Integr. Comp. Physiol. 292 (4) (2007) R1391–R1399.
[9] B.A. Parker, et al., Relation between food intake and visual analogue scale ratings of appetite and other sensations in healthy older and young subjects, Eur. J. Clin. Nutr. 58 (2) (2004) 212–218.
[10] J. Blundell, et al., Appetite control: methodological aspects of the evaluation of foods, Obes. Rev. 11 (3) (2010) 251–270.
[11] L. Lissner, B.L. Heitmann, Dietary fat and obesity: evidence from epidemiology, Eur. J. Clin. Nutr. 49 (2) (1995) 79–90.
[12] J.M. de Castro, et al., Palatability and intake relationships in free-living humans: characterization and independence of influence in North Americans, Physiol. Behav. 70 (3–4) (2000) 343–350.
[13] S. Kaviani, J. Cooper, Appetite responses to high-fat meals or diets of varying fatty acid composition: a comprehensive review, Eur. J. Clin. Nutr. 71 (10) (2017) 1154–1165.
[14] S. Nymo, et al., Timeline of changes in appetite during weight loss with a ketogenic diet, Int. J. Obes. 41 (8) (2017) 1224–1231.
[15] D. Jakubowicz, et al., Meal timing and composition influence ghrelin levels, appetite scores and weight loss maintenance in overweight and obese adults, Steroids 77 (4) (2012) 323–331.
[16] P. Casas-Agustench, et al., Acute effects of three high-fat meals with different fat saturations on energy expenditure, substrate oxidation and satiety, Clin. Nutr. 28 (1) (2009) 39–45.
[17] C.M. Strik, et al., No evidence of differential effects of SFA, MUFA or PUFA on post-ingestive satiety and energy intake: a randomised trial of fatty acid saturation, Nutr. J. 9 (2010) 24.
[18] A. Flint, et al., Effects of different dietary fat types on postprandial appetite and energy expenditure, Obes. Res. 11 (12) (2003) 1449–1455.
[19] A.J. Graybeal, S. Meena, J.L. Willis, Manipulation of fatty acid composition in a high-fat meal does not result in differential alterations in appetite or food intake in normal weight females: a single-blind randomized crossover study, Appetite 160 (2021), 105085.
[20] R.C. Alfenas, R.D. Mattes, Effect of fat sources on satiety, Obes. Res. 11 (2) (2003) 183–187.
[21] C.G. MacIntosh, S.H. Holt, J.C. Brand-Miller, The degree of fat saturation does not alter glycemic, insulinemic or satiety responses to a starchy staple in healthy men, J. Nutr. 133 (8) (2003) 2577–2580.
[22] J.L. Stevenson, H.C. Clevenger, J.A. Cooper, Hunger and satiety responses to high-fat meals of varying fatty acid composition in women with obesity, Obesity 23 (10) (2015) 1980–1986.
[23] C.L. Lawton, et al., The degree of saturation of fatty acids influences post-ingestive satiety, Br. J. Nutr. 83 (5) (2000) 473–482.
[24] F.X. Valente, et al., Effects of coconut oil consumption on energy metabolism, cardiometabolic risk markers, and appetitive responses in women with excess body fat, Eur. J. Nutr. 57 (4) (2018) 1627–1637.
[25] J. Maljaars, et al., Effect of fat saturation on satiety, hormone release, and food intake, Am. J. Clin. Nutr. 89 (4) (2009) 1019–1024.
[26] A. Kozimor, H. Chang, J.A. Cooper, Effects of dietary fatty acid composition from a high fat meal on satiety, Appetite 69 (2013) 39–45.
[27] J.L. Stevenson, C.M. Paton, J.A. Cooper, Hunger and satiety responses to high-fat meals after a high-polyunsaturated fat diet: a randomized trial, Nutrition 41 (2017) 14–23.
[28] M.M. Kamphuis, M.S. Westerterp-Plantenga, W.H. Saris, Fat-specific satiety in humans for fat high in linoleic acid vs fat high in oleic acid, Eur. J. Clin. Nutr. 55 (6) (2001) 499–508.
[29] F.E. Lithander, et al., No evidence of an effect of alterations in dietary fatty acids on fasting adiponectin over 3 weeks, Obesity (Silver Spring) 16 (3) (2008) 592–599.
[30] M. Pichierri, et al., Health claims' text clarity, perceived healthiness of extra-virgin olive oil, and arousal: an experiment using facereader, Trends Food Sci. Technol. 116 (2021) 1186–1194.
[31] F. Gomez-Delgado, et al., Apolipoprotein E genetic variants interact with Mediterranean diet to modulate postprandial hypertriglyceridemia in coronary heart disease patients: CORDIOPREV study, Eur. J. Clin. Invest. 49 (8) (2019) e13146.
[32] B. Gomez-Marin, et al., Long-term consumption of a Mediterranean diet improves postprandial lipemia in patients with type 2 diabetes: the cordioprev randomized trial, Am. J. Clin. Nutr. 108 (5) (2018) 963–970.
[33] C.M. Paton, et al., Dihydrosterculic acid from cottonseed oil suppresses desaturase activity and improves liver metabolomic profiles of high-fat-fed mice, Nutr. Res. 45 (2017) 52–62.
[34] K.E. Davis, C. Prasad, V. Imrhan, Consumption of a diet rich in cottonseed oil (CSO) lowers total and LDL cholesterol in normo-cholesterolemic subjects, Nutrients 4 (7) (2012) 602–610.
[35] K.R. Polley, et al., Appetite responses to high-fat diets rich in mono-unsaturated versus poly-unsaturated fats, Appetite 134 (2019) 172–181.
[36] M.C. Prater, et al., Comparison of blood lipid responses to diets enriched with cottonseed oil vs. olive oil in adults with high cholesterol in a randomized trial, J. Nutr. (2022).
[37] J.A. Cooper, et al., Assessing validity and reliability of resting metabolic rate in six gas analysis systems, J. Am. Diet Assoc. 109 (1) (2009) 128–132.
[38] J.d.V. Weir, New methods for calculating metabolic rate with special reference to protein metabolism, J. Physiol. 109 (1–2) (1949) 1.
[39] J.R. Lupton, et al., Dietary Reference Intakes For energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids, 5, National Academy Press, Washington, DC, USA, 2002, pp. 589–768.
[40] M. Gersovitz, J.P. Madden, H. Smiciklas-Wright, Validity of the 24-hr. dietary recall and seven-day record for group comparisons, J. Am. Diet Assoc. 73 (1) (1978) 48–55.
[41] T.V. Kral, L.S. Roe, B.J. Rolls, Combined effects of energy density and portion size on energy intake in women, Am. J. Clin. Nutr. 79 (6) (2004) 962–968.
[42] R. An, Weekend-weekday differences in diet among US adults, 2003–2012, Ann. Epidemiol. 26 (1) (2016) 57–65.
[43] M.D. BUHALY, P.L. BORDI, Development and sensory evaluation of a high-protein, vitamin-fortified fruit roll-up for children with cystic fibrosis, Foodservice Res. Int. 14 (4) (2004) 243–256.
[44] D.R. Peryam, F.J. Pilgrim, Hedonic scale method of measuring food preferences, Food Technol. (1957).
[45] B. Burton-Freeman, P.A. Davis, B.O. Schneeman, Plasma cholecystokinin is associated with subjective measures of satiety in women, Am. J. Clin. Nutr. 76 (3) (2002) 659–667.
[46] C. Gibbons, et al., Comparison of postprandial profiles of ghrelin, active GLP-1, and total PYY to meals varying in fat and carbohydrate and their association with hunger and the phases of satiety, J. Clin. Endocrinol. Metab. 98 (5) (2013) E847–E855.
[47] N. Godwin, et al., Mixed nuts may promote satiety while maintaining stable blood glucose and insulin in healthy, obese, and overweight adults in a two-arm randomized controlled trial, J. Med. Food 22 (4) (2019) 427–432.
[48] W.J. Pasman, et al., The effect of Korean pine nut oil on in vitro CCK release, on appetite sensations and on gut hormones in post-menopausal overweight women, Lipids Health Dis. 7 (1) (2008) 1–10.
[49] L.L. Guarneiri, C.M. Paton, J.A. Cooper, Appetite responses to pecan-enriched diets, Appetite 173 (2022), 106003.
[50] C.G. MacIntosh, et al., Effect of exogenous cholecystokinin (CCK)-8 on food intake and plasma CCK, leptin, and insulin concentrations in older and young adults: evidence for increased CCK activity as a cause of the anorexia of aging, J. Clin. Endocrinol. Metabolism 86 (12) (2001) 5830–5837.
[51] L. Payahoo, et al., Effects of n-3 polyunsaturated fatty acid supplementation on serum leptin levels, appetite sensations, and intake of energy and macronutrients in obese people: a randomized clinical trial, J. Diet Suppl. 15 (5) (2018) 596–605.
[52] Dowd, M.K., et al., Fatty acid profiles of cottonseed genotypes from the national cotton variety trials. 2010.
[53] K.L. Feltrin, et al., Comparative effects of intraduodenal infusions of lauric and oleic acids on antropyloroduodenal motility, plasma cholecystokinin and peptide YY, appetite, and energy intake in healthy men, Am. J. Clin. Nutr. 87 (5) (2008) 1181–1187.
[54] J.A. Cooper, et al., Impact of exercise and dietary fatty acid composition from a high-fat diet on markers of hunger and satiety, Appetite 56 (1) (2011) 171–178.
[55] K.G. Jackson, et al., Olive oil increases the number of triacylglycerol-rich chylomicron particles compared with other oils: an effect retained when a second standard meal is fed, Am. J. Clin. Nutr. 76 (5) (2002) 942–949.
[56] F.X. Pi-Sunyer, Obesity and hypertension, Obes. Manag. 5 (2) (2009) 57–61.
[57] D.M. Thomas, et al., Can a weight loss of one pound a week be achieved with a 3500-kcal deficit? Commentary on a commonly accepted rule, Int. J. Obes. 37 (12) (2013) 1611–1613.

[58] K.D. Hall, et al., Energy balance and its components: implications for body weight regulation, Am. J. Clin. Nutr. 95 (4) (2012) 989–994.
[59] K.D. Hall, et al., The energy balance model of obesity: beyond calories in, calories out, Am. J. Clin. Nutr. 115 (5) (2022) 1243–1254.
[60] D. Polidori, et al., How strongly does appetite counter weight loss? Quantification of the feedback control of human energy intake, Obesity (Silver Spring) 24 (11) (2016) 2289–2295.
[61] N. Muurahainen, et al., Effects of cholecystokinin-octapeptide (CCK-8) on food intake and gastric emptying in man, Physiol. Behav. 44 (4–5) (1988) 645–649.
[62] B. Sloth, et al., Effect of subcutaneous injections of PYY1-36 and PYY3-36 on appetite, ad libitum energy intake, and plasma free fatty acid concentration in obese males, Am. J. Physiol.-Endocrinol. Metabolism 293 (2) (2007) E604–E609.
[63] P. Pfluger, et al., Effect of human body weight changes on circulating levels of peptide YY and peptide YY3–36, J. Clin. Endocrinol. Metabolism 92 (2) (2007) 583–588.
[64] R.A. Liddle, et al., Cholecystokinin bioactivity in human plasma. Molecular forms, responses to feeding, and relationship to gallbladder contraction, J. Clin. Invest. 75 (4) (1985) 1144–1152.
[65] L.S. Freedman, et al., Pooled results from 5 validation studies of dietary self-report instruments using recovery biomarkers for energy and protein intake, Am. J. Epidemiol. 180 (2) (2014) 172–188.
[66] H. Wehling, J. Lusher, People with a body mass index⩾ 30 under-report their dietary intake: a systematic review, J Health Psychol 24 (14) (2019) 2042–2059.