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ABSTRACT

Background: Individuals with impaired fat taste (FT) sensitivity have reduced satiety responses after consuming fatty foods, leading to increased dietary fat intake. Habitual consumption of dietary fat may modulate sensitivity to FT, with high consumption decreasing sensitivity [increasing fatty acid taste threshold (FATT)] and low consumption increasing sensitivity (decreasing FATT). However, some individuals may be less susceptible to diet-mediated changes in FATT due to variations in gene expression.

Objective: The objective of this study was to determine the effect of an 8-wk low-fat or high-fat diet on FATT while maintaining baseline weight (<2.0 kg variation) to assess heritability and to explore the effect of genetics on diet-mediated changes in FATT.

Design: A co-twin randomized controlled trial including 44 pairs (mean ± SD age: 43.7 ± 15.4 y; 34 monozygotic, 10 dizygotic; 33 women, 10 men, 1 gender-discordant) was conducted. Twins within a pair were randomly allocated to an 8-wk low-fat (<20% of energy from fat) or high-fat (>35% of energy from fat) diet. FATT was assessed by a 3-alternate forced choice methodology and transformed to an ordinal scale (FT rank) at baseline and at 4 and 8 wk. Linear mixed models were fit to assess diet effect on FT rank and diet effect modification due to zygosity. A variance components model was fit to calculate baseline heritability.

Results: There was a significant time × diet interaction for FT rank after the 8-wk trial ($P < 0.001$), with the same conclusions for the subset of participants maintaining baseline weight (low-fat; $n = 32$; high-fat: $n = 35$). There was no evidence of zygosity effect modification (interaction of time × diet × zygosity: $P = 0.892$). Heritability of baseline FT rank was 8%.

Conclusions: There appears to be little to no genetic contribution on heritability of FATT or diet-mediated changes to FATT. Rather, environment, specifically dietary fat intake, is the main influencer of FT sensitivity, regardless of body weight. This trial was registered with the Australian New Zealand Clinical Trials Registry at http://www.anzctr.org.au/ as ACTRN12613000466741. *Am J Clin Nutr* 2018;107:683–694.

Keywords: fat taste, fat intake, weight, randomized controlled trial, co-twin, zygosity, heritability

INTRODUCTION

Individuals with impaired oral fatty acid sensitivity [fat taste (FT)] may be more likely to consume greater amounts of fatty foods, mainly from foods high in saturated and monounsaturated fat (1–4). Impaired sensitivity to FT is paralleled by impaired satiety responses to fatty acids in the gastrointestinal tract, which could lead to increased consumption of dietary fat (5–7). In addition, impaired sensitivity to FT has been shown to be associated with increased liking, preference, and choice for fatty foods (5, 8).

Habitual fat intake is responsible for variation in FT, with increasing dietary fat causing increased fatty acid taste threshold (FATT). Although cross-sectional studies have shown positive associations between fat intake and FATT (1, 7, 9, 10), intervention studies have shown that modifications to long-term dietary fat intake mediate change in FATT. Two human 4-wk (11) and 6-wk (12) dietary fat crossover intervention studies reported decreased FATT after low-fat (LF) dietary intake (11, 12) and increased FATT after high-fat (HF) dietary intake, albeit only in lean individuals (11). However, in both studies, participants who consumed the LF diet lost significant weight over the

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Supplemental Figure 1 and Supplemental Table 1 are available from the "Supplementary data" link in the online posting of the article and from the same link in the online table of contents at https://academic.oup.com/ajcn/.

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Abbreviations used: AMDR, Acceptable Macronutrient Distribution Range; DZ, dizygotic; FATT, fatty acid taste threshold; FT, fat taste; HF, high-fat; ICC, intraclass correlation coefficient; LF, low-fat; LMS, labeled magnitude scale; MZ, monozygotic; SNP, single-nucleotide polymorphism; TG, triglyceride; TRA, Twins Research Australia; 3-AFC, 3-alternate forced choice; Δ, week 8 − baseline.

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intervention period (11, 12), and participants who consumed the HF diet gained significant weight (11). The authors could not rule out the possibility that at least part of the reported effect was due to weight differences, especially considering that in one of the studies the effect was only evident in lean individuals. However, body weight does not seem to be associated with FATT, because a recent meta-analysis of 7 cross-sectional studies found no relation between FT sensitivity and obesity (13).

Two variants of the *CD36* gene have been associated with FT sensitivity. The A allele of single-nucleotide polymorphism (SNP) rs1761667 for *CD36* was associated with reduced FT sensitivity and increased creaminess perception and liking of added fats in foods compared with the G allele in multiple populations (14–18). In addition, the T allele of SNP rs1527483 for *CD36* was associated with increased perception of fat content compared with the C allele in an African American population (14) but not in a white population (15). However, it is not known whether SNPs of these genes regulate changes in FT sensitivity after dietary intervention. Genetic components of a phenotype can be assessed by comparing monozygotic (MZ) and dizygotic (DZ) twin pairs under the assumption that MZ pairs share 100% of their genes and DZ pairs share, on average, 50% of their genes (19, 20).

We present here a study with a co-twin design that was controlled for age and the genetic and common environmental factors shared by a pair. This randomized controlled trial aimed to assess the effect of LF (<20% of energy from fat) or HF (>35% of energy from fat) dietary intakes on FATT while recommending that participants maintain stable weight over 8 wk. It also aimed to assess the heritability of FATT at baseline and to explore the effect of genetics on diet-mediated FATT changes by assessing whether zygosity modifies the diet effect. Secondary aims were to assess the effect of LF or HF dietary intake on triglyceride (TG) perception, liking of fatty foods, and intensity ratings on 5 prototypical tastants.

METHODS

Participants

Twins Research Australia (TRA) invited via mailings 1881 twin pairs (3762 individuals) from the Melbourne metropolitan area who had not participated in a TRA study in the past 18 mo. Reminders were sent to 3430 individuals who did not respond within 3 mo via a combination of mailings, phone calls, and e-mails. Twins were eligible to participate in the study if they were aged between 18 and 69 y, were able to attend 3 laboratory sessions in Burwood, Victoria, Australia, and were willing to alter their diet for a period of 8 wk. Both MZ and DZ twin pairs were invited to participate. Individuals were excluded from recruitment if they had any dairy allergies and intolerances, illnesses preventing them from eating foods included in the study, or if they were pregnant or lactating. Due to the nature of the twin study design, if 1 individual from a twin pair was excluded or withdrew from the study, their co-twin was also excluded. In 66 pairs, both twins expressed interest in participating and were then screened for eligibility. Forty-six twin pairs (92 individuals) aged between 18 and 68 y were recruited into the study, and co-twins from each pair were randomly assigned to receive either an LF or an HF diet. Before recruitment, a block-randomized sequence

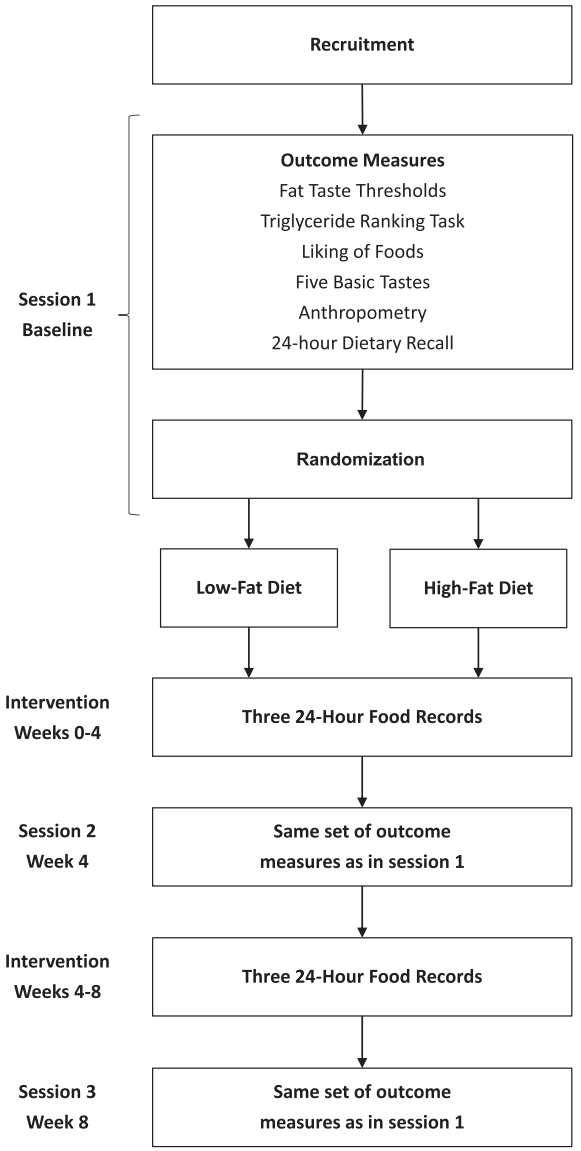

FIGURE 1 Outline of trial timeline. Food records were collected on 2 weekdays and 1 weekend day at any time during weeks 0–4 and weeks 4–8. A 24-h dietary recall was not repeated at sessions 2 and 3.

was generated with block sizes of 2. TRA was responsible for recruitment, and therefore characteristics of the participants were blinded to the researchers. Participants were allocated to the randomly assigned sequence on the basis of their TRA twin number; therefore, allocation of participants to diet group was concealed. Due to the nature of the intervention, blinding of participants was not feasible. Ethical approval was obtained by the Deakin University Human Research Ethics Committee in accordance with the Declaration of Helsinki, and informed written consent was obtained by all participants before participation.

Study outline

Participants attended three 2-h laboratory sessions at the Centre for Advanced Sensory Science at Deakin University, Burwood. Sessions were conducted 4 wk apart (**Figure 1**). Tests

were conducted in a temperature- and sound-controlled environment, with a 15-min break in the middle of their session to prevent fatigue. Participants were asked to avoid eating or drinking anything but water and to avoid brushing their teeth or using mouthwash ≤1 h before each tasting session. Tasting sessions measured for *1*) detection threshold to oleic acid (FATT), *2*) ability to rank the amount of fat in food, *3*) liking ratings for high-fat and reduced-fat foods, and *4*) intensity ratings to 5 prototypical tastants (21). Liking and intensity ratings were collected with the use of the computer software Compusense Cloud as part of the Compusense Academic Consortium (Compusense, Inc.). Anthropometric measurements were taken at the beginning of each session. A 24-h food recall was collected by a nutritionist during the first session. Between tasting sessions, participants recorded three 24-h diet records (2 weekdays, 1 weekend day) to determine dietary compliance.

Dietary intervention

The LF diet was defined as <20% of energy from fats and the HF diet was defined as >35% of energy from fats. These values were chosen because they fall outside the Acceptable Macronutrient Distribution Range (AMDR) (20–35%) for fat intake, which is used as a recommendation in the US, Canadian, and Australian dietary guidelines (22). Participants receiving the HF diet were encouraged to choose foods higher in monounsaturated and polyunsaturated fats rather than saturated fats in order to maintain a healthy diet. A booklet for each diet was created with the aid of an accredited practicing dietitian, which described the parameters of each diet; a list of foods that should and should not be eaten; and some sample recipes that adhere to the diet. Participants were given the HF- or LF-diet booklet, their assigned diet was explained, and they were taught how to interpret a nutrition information panel in order to identify which foods were acceptable for their diet.

They were requested to start the assigned diet the day after baseline measurement. Because foods were not provided in this study, food choice was up to the participants. To maximize adherence to the diets, participants were contacted via phone every 2 wk and questioned on their dietary habits. If the researcher felt that participants were not following the diet adequately, they were provided with suggestions and encouragement to aid in diet adherence. Participants were also asked a series of questions to ensure that they did not experience any negative effects from the diet. These questions included, "Do you feel like you have less energy since starting the diet," "Do you feel like your weight has changed significantly since starting the diet," and "Is the diet affecting your day-to-day activities?" If the researcher felt that participants were suffering from major negative effects due to the diet (e.g., severe nausea, inability to work), they were asked to stop the diet and were dropped from the trial.

Completed diet diaries, as described below, were inspected at the beginning of sessions 2 and 3 and reviewed for adherence to the assigned diet. Participants were encouraged to maintain their baseline weight throughout the study. A target of <2 kg of change in body weight over the trial was set. Participants were asked to stop eating once they were satiated to prevent overconsumption. Weight-maintenance guides for each diet, including tips and suggested recipes (e.g., LF guide contained LF, high-energy recipes to prevent weight loss), were provided to participants at the start of the trial to help maintain weight. If weight changed by >1 kg from the baseline and week 4 (session 2), participants were given further advice on how to maintain their weight for the latter half of the study.

Dietary assessment

A 24-h dietary recall was used to assess short-term dietary intake. A single 3-pass 24-h dietary recall (23) of the day before session 1 was conducted by a trained nutritionist. Participants also completed three 24-h diet records between sessions 1 and 2 and between sessions 2 and 3 (Figure 1), because three 24-h diet records are optimal for estimating energy and macronutrient intakes (24). Diet was recorded for 2 weekdays and 1 weekend day, chosen by the participant. Participants were asked to avoid filling out diet records on a nonstandard day (e.g., if they attended a wedding reception). They were taught to quantify foods in standard serving sizes (cups, teaspoons, tablespoons, etc.) with the use of a food model booklet and asked to weigh their food and drinks wherever possible. Details such as brand, cooking method, and food additives (e.g., sugar added to coffee) were included in the diet records. Food recall and records were analyzed for energy intake (kilojoules), total consumption (grams) of protein, fat (total fat, saturated fat, monounsaturated fat, and polyunsaturated fat), carbohydrate and alcohol, and percentage of energy from protein, fat, carbohydrate, and alcohol with the use of the computer software FoodWorks (version 8; Xyris).

Anthropometric measurements

Body weight was measured after the removal of shoes, heavy clothing, and any items in participants' pockets with the use of electronic scales (OHAUS NV4101), and height was measured by using a free-standing stadiometer (Seca). BMI was calculated as weight (kilograms)/height (meters) squared. Hip and natural waist (midway between the lowest rib and the iliac crest) circumferences were measured according to WHO guidelines (25).

FATT

Although intensity of the traditional tastes—sweet, sour, salty, bitter, and perhaps umami—is the most relevant taste dimension for diet (26), evidence suggests that detection threshold is most relevant to diet for FT (1–7, 9–13).

Detection threshold to oleic acid (18:1) was measured with the use of established methods (21). Food-grade oleic acid was obtained from Sigma Aldrich and was stored under nitrogen gas below 4°C. Oleic acid was added at varying concentrations (0.02, 0.06, 1.00, 1.40, 2.00, 2.80, 3.80, 5.00, 6.40, 8.00, 9.80, 12.00, and 20.00 mM) to long-life fat-free milk (Devondale). All of the preparations were mixed with 5% (wt:vol) gum arabic (prehydrated FT Powder, TIC Gums; Alchemy Agencies) and 5% (vol:vol) liquid paraffin (Faulding Remedies) to produce perceptually identical textural attributes, including viscosity and lubricity between oleic acid and control samples. To prevent oxidation of oleic acid, all samples were mixed with 0.01% wt:vol EDTA (Merck). Samples were homogenized at room temperature for 30 s/100 mL at 12,000 rpm (Average centrifugal force, 3220 × *g*) (Silverston L4RT homogenizer), prepared ≤2 h before testing, and served at room temperature. Control samples were prepared in the same way but without added oleic acid. Participants were

TABLE 1
Fat taste ranks and their corresponding fatty acid taste threshold concentration ranges

Fatty acid taste threshold, mM	Fat taste rank
0.02–0.99	0
1.00–1.39	1
1.40–1.99	2
2.00–2.79	3
2.80–3.79	4
3.80–4.99	5
5.00–6.39	6
6.40–7.99	7
8.00–9.79	8
9.80–11.99	9
12.00–19.99	10
20.00	11
>20.00	12

asked to rinse their mouths with water before beginning the task and between sample sets. To prevent confounding nontaste sensory inputs, participants wore nose clips and all of the tests were conducted under red light.

FATT was determined by using ascending series 3-alternate forced choice (3-AFC) methodology (21), which is a test to select a sample among a set of 3 that differs in a known attribute. To familiarize participants with the taste attribute of oleic acid and to reduce sensory fatigue, participants were initially provided with warm-up sets before the 3-AFC test. A warm-up set contained an oleic acid sample (initially, 3.8 mM) and a control sample. If participants were unable to perceive a difference between the control and oleic acid sample during the first warm-up set, then they were provided with a new warm-up set at 8 mM.

The 3-AFC test began with the highest oleic acid concentration sample set that could not be differentiated from the control during warm-up (i.e., the 3-AFC test began with 0.02 mM if the participant was able to differentiate the 3.8-mM sample from the control sample). Participants were provided with multiple sample sets each containing 3 randomly ordered samples per set, 2 controls, and 1 containing oleic acid. Participants were asked to taste each sample in the set and identify the sample that matched the taste quality from the warm-up sets. Correct identification of the oleic acid sample resulted in the participants repeating the same sample set. Incorrect identification of the oleic acid sample resulted in new sample set with a higher concentration of oleic acid. This continued in an ascending order from the initial concentration to the highest (20 mM) concentration. The endpoint was defined as the concentration of oleic acid correctly identified in 3 consecutive sample sets of the same concentration, in line with commonly established sensory testing procedures (21). The 3-AFC test was performed in duplicate with participants consuming an LF plain water cracker (Manassen Foods) between tests to reduce sensory fatigue. FATT was defined as the mean of the 2 endpoints, because 2 measures on the same day have shown test-retest reliability (12). If participants were unable to correctly identify the oleic acid sample at the highest concentration (20 mM) for ≥1 of their trials, then they were given a detection threshold of >20 mM. Of note, due to the range of concentrations of fatty acid tested (**Table 1**), the outcome FATT is an interval-censored variable (i.e., a threshold of 20 mM indicates that the participant's actual threshold is anywhere between 12 and 20 mM). FATT was transformed to an ordinal variable—FT rank—ranging from 0 to 12, with higher ranks implying lower sensitivity to FT (Table 1) (1).

TG ranking task

This task was designed to evaluate the participants' ability to discriminate different amounts of fat content between food samples (21). Four food samples were made with the use of LF custard (Parmalat) and varying amounts of canola oil (0%, 2%, 6%, and 10% oil, wt:wt; Woolworths). All of the samples were stirred vigorously. Custard samples were presented in a randomized order, and participants were asked to taste and rank them according to their fat content. The custard samples were served at room temperature and prepared ≤2 h before testing. To prevent visual cues, the test was conducted under red light. TG ranking score was calculated with the use of the following formula:

$$(-2 \times c1) - (1 \times c2) + (1 \times c3) + (2 \times c4) \qquad (1)$$

where c1–c4 were the concentrations of the samples assigned by the participant as having the lowest to the highest fat content (27, 28). The values of the concentrations were −2 for 0%, −1 for 2%, 1 for 6%, and 2 for 10% (wt:wt). Accurately ranking samples in ascending or descending order are both considered to be correct; therefore, negative scores were converted to positive values. Final TG ranking score ranged from 0 to 10, with 10 indicating the participant was able to correctly discriminate the samples on the basis of fat content and 0 being unable.

Fatty food liking

Liking of food on the basis of fat content was measured by rating 7 HF foods and 7 LF counterparts. Participant liking was measured by rating "liking" on a hedonic labeled magnitude scale (LMS) with anchors −100 (extremely dislike), 0 (neither like nor dislike), and 100 (extremely like). Participants were trained in the use of the hedonic LMS by rating nontaste sensations on a validated liking questionnaire (29) and were instructed that the ends of the scale (extremely like) were intended for hedonic experiences beyond the context of food. Foods were presented under red light to reduce visual differences between samples. Savory biscuits (Arnott's), peanut butter (Mondelez), hummus (Black Swan), salad dressing (Goodman Fielder), processed cheese (Mondelez), cream cheese (Mondelez), and chocolate mousse (Fonterra) were tested. Foods were always presented in the order listed above to simulate normal eating behavior. LF and HF counterparts for each food were presented at the same time, side-by-side, in a randomized order (left or right side).

HF and LF liking scores were calculated as the mean of the 7 HF food and 7 LF food liking ratings, respectively. The differences between the HF and LF scores (HF − LF liking score) were also calculated to control for individual preferences for each food item.

Intensity ratings to 5 prototypical tastants

Participants rated the intensities of sweet, salty, sour, bitter, and umami solutions at concentrations prepared on the basis

of Webb et al. (26). Solutions were prepared at suprathreshold concentrations (weak, moderate, and strong) by using sucrose (Woolworths), sodium chloride (Woolworths), citric acid (Ward McKenzie), caffeine (Sigma-Aldrich), and monosodium glutamate (Ajinomoto Cooperation), respectively. To prevent confounding nontaste sensory inputs, participants wore nose clips and the test was conducted under red light. Participants tasted each sample and rated its intensity on an LMS, with anchors of 0 (no taste) to 100 (strongest imaginable taste). Participants were trained in the use of the LMS following published standard procedures (26) that involved culturally appropriate remembered or imagined sensations, such as the coolness of an ice-cold beverage or the sweetness of fairy floss (known as candy floss in the United Kingdom, or cotton candy in the United States). All of the solutions were prepared within 5 d of testing, stored at 4°C, and served at room temperature. Participants were presented all 3 concentrations (weak, moderate, and strong) of 1 taste at a time in random order. The sequence of the tastants was also randomized.

Statistical analysis

Numerical variables are reported as mean ± SDs, and categorical data as n (%). Null hypotheses were rejected at $P < 0.05$.

The effect of the diet on anthropometric measures, dietary compliance, FT rank, TG ranking score, food liking, and intensity ratings to 5 prototypical tastants was assessed with the use of linear mixed models including diet (LF and HF), time (baseline, week 4, and week 8), and time × diet interaction as fixed effects, with twin pair as a random effect to account for the correlation between co-twins, and participant as the subject with repeated measures. The same analysis was repeated for FT rank in the subset of participants whose weight changed by <2 kg. Post hoc Sidak's P values and CIs are reported.

Further analysis explored the effect of genetics on diet-mediated changes in FT rank, the only variable that showed a significant diet effect with the use of the linear mixed model described above, further including zygosity (MZ and DZ) and all double and triple interactions. The effect of diet on FT rank for MZ and DZ pairs is reported at each time. A greater diet effect in DZ pairs than in MZ pairs would suggest some degree of genetic effect that regulates changes in FATT.

To explore the strength of the association ($\hat{\beta}$) between change in FT rank and overall change in fat intake (grams) and energy from fat (percentage) over the 8-wk period, a linear mixed model was used including Δ FT rank (Δ, week 8 − baseline) as the outcome and Δ fat intake or Δ energy from fat as a fixed effect, with twin pair as a random effect. Pearson's correlations (r) between Δ FT rank and Δ fat intake are also reported for descriptive purposes.

Twin concordance for anthropometric measures and FT rank was estimated through the intraclass correlation coefficient (ICC) separately for MZ and DZ twin pairs. ICC was additionally estimated controlling for co-twin living status (together or apart). A Wald test was used to compare MZ and DZ pair ICC estimates (30). Correlation between twin pairs is assumed to be due to 2 factors, an additive genetic effect (A) and a common environmental effect shared by the twin pair (C), with residual variance in an individual attributed to unique environment effects (E) (19). These effects can be calculated to provide an estimate of heritability, which is the degree of variation of a phenotype that is attributable to additive genetic effects (19). Heritability (h^2) of baseline FT rank was estimated by using a variance components model including zygosity as a fixed effect and twin pair as a random effect. Under the assumption that individual variance can be modeled as $\sigma^2 = \mathrm{A} + \mathrm{C} + \mathrm{E}$, MZ covariance as $\sigma^2{}_{\mathrm{MZ}} = \mathrm{A} + \mathrm{C}$, and DZ covariance as $\sigma^2{}_{\mathrm{DZ}} = \frac{A}{2} + \mathrm{C}$ because DZ pairs share 50% of genes (19, 31, 32), heritability can be estimated as $h^2 = \frac{A}{A+C+E}$. Analyses were conducted with the use of SPSS (version 22.0), except for twin concordance and heritability analyses, which were conducted by using STATA (version 15.0; StataCorp).

Sample size calculation

Data from an unpublished nontwin dietary intervention informed the sample size calculations. The computer software PASS version 15.0 (NCSS) was used to calculate sample size and power estimates. A sample size of 38 participants/diet group achieves 82% power to detect a mean difference in FATT of 5.5 mM oleic acid between diet groups when the SD of change is 8.5 mM oleic acid and the correlation between co-twins is 0.1 (significance level: 0.05; 2-sided, 2-sample paired means analysis using simulation). Under the same assumptions, a correlation between co-twins of 0.3 or 0.5 achieves a power of 92% and 97%, respectively. In addition, the target sample size (38 pairs) achieves 85% power for detecting an FATT change of 3 mM oleic acid between baseline and week 8 in 1 of the groups, assuming an SD of 6 mM oleic acid (significance level: 0.05; 2-sided, 1-sample t test). Forty-six pairs were recruited to allow for 15% attrition.

To assess if 34 MZ and 10 DZ pairs from the final sample had adequate power to estimate heritability for baseline FT rank, we performed a post hoc sample size calculation to detect A and C for the equation $h^2 = \frac{A}{A+C+E}$ (33, 34). The calculations are based on the proportion of MZ twin pairs in the sample. On the basis of Visscher (33, 34), the ideal proportion of MZ pairs in a sample is 0.61 for detecting A and 0.06 for detecting C, whereas the proportion of MZ pairs in this study was 0.77. To detect the contribution of additive genetic effects (A) to a trait with 80% power required ≥16 MZ pairs and 10 DZ pairs, and to detect the contribution of common environmental effects (C) to a trait with 80% power required ≥10 MZ pairs and 149 DZ pairs. Because this study was only powered to estimate A, we present the heritability estimate for baseline FT rank but not the full model that contains estimates for A, C and E (ACE model).

RESULTS

Sixty-six pairs of twins expressed interest in participating; 46 pairs (70%) were eligible and randomly assigned to the study (**Figure 2**). Two twin pairs dropped out of the study after baseline measurements. One female individual dropped out due to difficulty adhering to the LF diet, whereas another male individual receiving the HF diet did not give a reason for dropping out. Accordingly, their co-twins were excluded from the study. The trial was completed by 44 twin pairs (35 MZ, 9 DZ; 33 female pairs, 10 male pairs, 1 gender-discordant pair). There were 34 female participants (77.3%) in the LF-diet group and 33 female

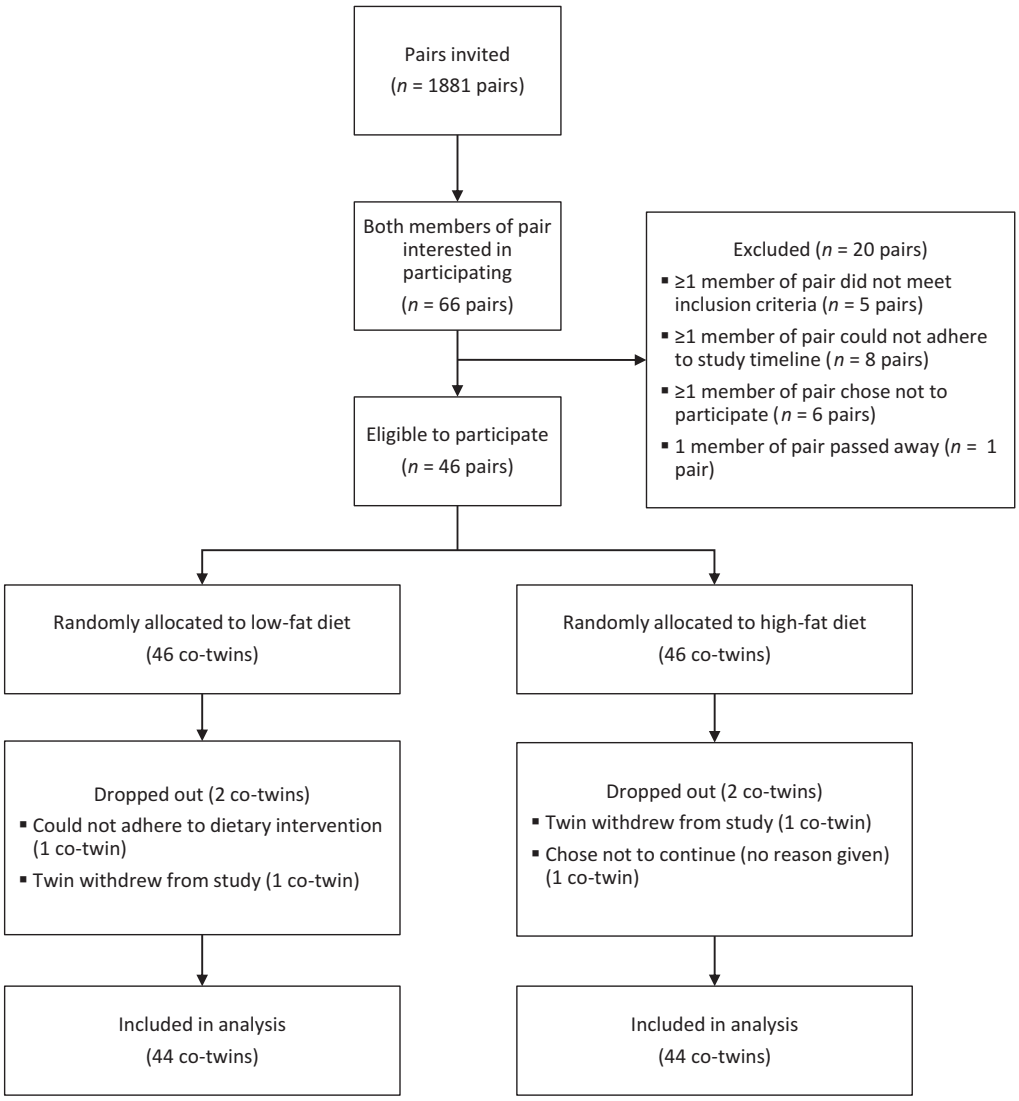

FIGURE 2 Study CONSORT flow chart diagram. CONSORT, Consolidated Standards of Reporting Trials.

participants (75%) in the HF-diet group. The mean ± SD age for both groups was 43.7 ± 15.5 y.

Anthropometric measurements

There was a significant time × diet interaction for both weight and BMI (**Table 2**). There was a significant decrease in weight and BMI from baseline to week 4 and from baseline to week 8 in the LF group. There were no significant changes observed in weight, BMI, waist circumference, or waist-to-hip ratio in the HF group over the 8 wk. At baseline, the HF group had a significantly higher waist-to-hip ratio than the LF group. However, this was no longer significantly different between groups after baseline. No other between-group differences were observed over the trial.

There were 21 individuals who were not able to maintain baseline body weight (<2.0 kg variation) over the 8-wk trial. In the LF-diet group, 11 individuals lost >2 kg and 1 gained >2 kg. In the HF-diet group, 4 individuals lost >2 kg and 5 gained >2 kg.

FATT

Compared with baseline, in the LF group, mean FT rank significantly decreased from 6.8 to 3.6 at week 4 [mean change (95% CI): −3.2 (−4.3, −2.0); $P < 0.001$] and to 2.6 at week 8 [mean change (95% CI): −4.2 (−5.4, −3.0); $P < 0.001$]. In the HF group, FT rank significantly increased from 6.9 to 8.3 at week 8 [mean change (95% CI): +1.4 (0.2, 2.6); $P = 0.017$], but not at week 4. There was a significant between-group difference at week 4 [mean difference (95% CI): 4.3 (3.3, 5.4); $P < 0.001$] and at week 8 [mean difference (95% CI): 5.7 (4.6, 6.9); $P < 0.001$] (**Figure 3**). There was a significant time × diet interaction ($P < 0.001$).

The analysis of FT rank was also conducted including only participants who maintained body weight (LF: $n = 32$; HF: $n = 35$). The change in FT rank in this subgroup was similar to that in the full analysis (**Supplemental Figure 1**).

We explored the association between change in FT rank and change in total dietary fat intake over the 8 wk. The estimated association between Δ FT rank (Δ, week 8 − baseline) and Δ fat

TABLE 2
LF and HF diet within- and between-group mean differences in anthropometric measurements over the 8-wk trial[1]

	LF diet ($n = 44$)	HF diet ($n = 44$)	Between-group difference	Time × diet interaction, P
Weight, kg				0.003
Baseline	72.6 ± 16.9[2]	74.7 ± 18.1	2.1 (−1.9, 6.1)[3]	
Week 4 vs. baseline	−0.9 (−1.4, −0.4)***	0.2 (−0.4, 0.7)	3.1 (−1.0, 7.3)	
Week 8 vs. baseline	−1.3 (−1.9, 0.6)***	0.1 (−0.6, 0.7)	3.4 (−0.8, 7.7)	
BMI, kg/m^2				0.003
Baseline	26.3 ± 5.1	26.8 ± 5.8	0.5 (−0.7, 1.7)	
Week 4 vs. baseline	−0.3 (−0.5, −0.1)***	0.0 (−0.1, 0.2)	0.9 (−0.4, 2.1)	
Week 8 vs. baseline	−0.5 (−0.7, −0.2)***	0.0 (−0.2, 0.2)	1.0 (−0.3, 2.3)	
WC, cm				0.767
Baseline	83.6 ± 15.4	86.7 ± 16.1	3.1 (−0.7, 6.9)	
Week 4 vs. baseline	−0.8 (−2.4, 0.7)	−1.0 (−2.6, 0.5)	2.9 (−1.1, 6.9)	
Week 8 vs. baseline	−0.7 (−2.3, 0.9)	−0.1 (−1.8, 1.4)	3.6 (−0.2, 7.5)	
WHR				0.529
Baseline	0.814 ± 0.09	0.838 ± 0.09	0.025 (0.000, 0.049)*	
Week 4 vs. baseline	0.003 (−0.013, 0.018)	−0.005 (−0.020, 0.011)	0.017 (−0.007, 0.042)	
Week 8 vs. baseline	−0.001 (−0.016, 0.013)	−0.011 (−0.026, 0.003)	0.015 (−0.006, 0.035)	

[1]Between-group differences were calculated as HF diet − LF diet; means (95% CIs) and P values were estimated by using a linear mixed model including diet, time, and time × diet interaction as fixed effects, with twin pair as a random effect to account for the correlation between co-twins, and participant as the subject with repeated measures. Post hoc Sidak's test and CIs are reported. *$P < 0.05$, ***$P < 0.001$. HF, high-fat; LF, low-fat; WC, waist circumference; WHR, waist-to-hip ratio.
[2]Mean ± SD (all such values).
[3]Mean; 95% CI in parentheses (all such values).

intake (grams) was $\hat{\beta} = 0.041$ (95% CI: 0.034, 0.048; $P < 0.001$, $r = 0.475$) (**Figure 4**) and between Δ FT rank and Δ energy from fat (percentage) was $\hat{\beta} = 0.15$ (0.12, 0.19; $P < 0.001$). This means that for every 10 g of fat intake or 1% change in energy from fat, FT rank changed in the same direction by 0.41 or 0.15, respectively.

Genetic variation on fatty acid taste threshold

All of the twins in this study were reared together. At the time of the study, 11 pairs lived at the same address (7 MZ twins, 4 DZ twins) and 33 pairs lived apart (27 MZ twins, 6 DZ twins). Mean baseline within-pair difference and ICC estimates, a measure of co-twin correlation, of each zygosity group are detailed in **Table 3**. For the sake of comparison, we include the analysis for anthropometric measures alongside FT rank. As expected, MZ pairs had a significantly higher ICC than DZ pairs for all anthropometric measurements. However, the ICC estimated for FT rank was low for both MZ and DZ pairs, with no difference between zygosity. In addition, when controlling for co-twin living status (together or apart), ICC estimates for FT rank remained similar (MZ ICC: 0.306; DZ ICC: 0.278). Baseline FT rank heritability was estimated as 8%.

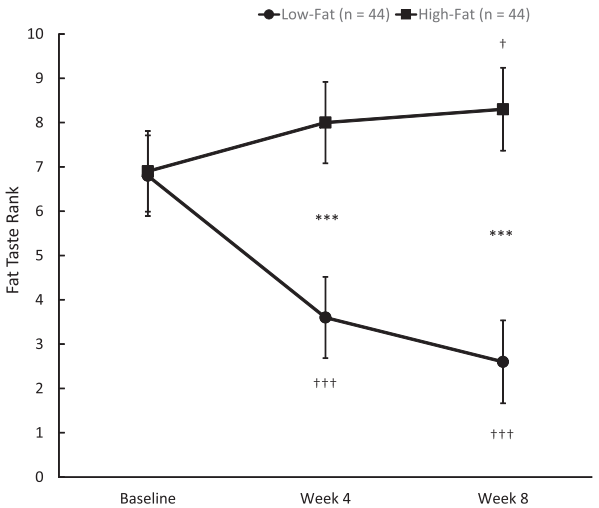

FIGURE 3 Means (95% CIs) for fat taste rank by diet group. Means (95% CIs) and P values were estimated by using a linear mixed model including diet, time, and time × diet interaction as fixed effects, with twin pair as a random effect to account for the correlation between co-twins, and participant as the subject with repeated measures. Time × diet interaction, $P < 0.001$. †,†††Within-group difference from baseline: †$P < 0.05$, †††$P < 0.001$. ***Between-group difference, $P < 0.001$.

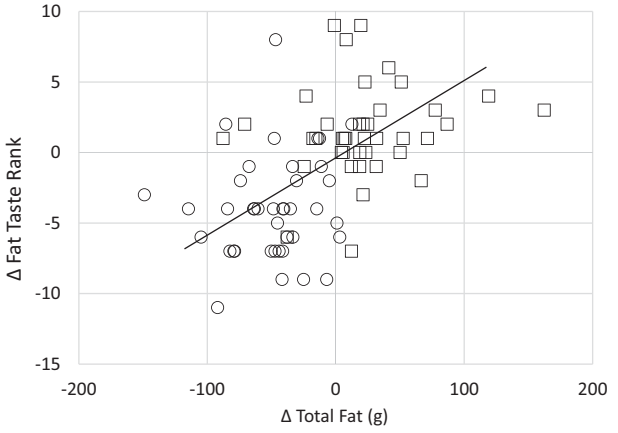

FIGURE 4 Δ in FT rank compared with Δ fat intake at week 8. Slope ($\hat{\beta}$, represented by the diagonal line) and P values were estimated by using a linear mixed model including Δ FT rank as the outcome and Δ fat intake as a fixed effect, with twin pair as a random effect to account for the correlation between co-twins. r Values were estimated by using Pearson's correlation for descriptive purposes. Circles (○) indicate participants in the low-fat group ($n = 44$); squares (□) indicate participants in the high-fat group ($n = 44$). $\hat{\beta} = 0.041$, $P < 0.001$, $r = 0.475$. FT, fat taste; Δ, change.

TABLE 3
Mean within-pair differences and ICCs of twin pairs for each zygosity at baseline[1]

	MZ pairs ($n = 34$)		DZ pairs ($n = 10$)		
	Within-pair difference	ICC	Within-pair difference	ICC	Comparison of ICCs, P
FT rank	3.1 ± 2.8	0.334	2.8 ± 2.7	0.294	0.405
Height, cm	1.9 ± 1.4	0.972	6.0 ± 9.2	0.315	<0.001
Weight, kg	5.7 ± 6.3	0.859	17.1 ± 16.4	0.433	0.008
BMI, kg/m^2	2.0 ± 2.2	0.852	4.7 ± 4.2	0.350	0.030
WC, cm	7.0 ± 6.4	0.798	14.3 ± 14.6	0.373	0.023
WHR	0.05 ± 0.05	0.679	0.09 ± 0.09	0.263	0.040

[1] Values are means ± SDs unless otherwise indicated. P values were obtained by using a Wald test to compare MZ and DZ ICCs. Larger ICCs in MZ pairs than in DZ pairs suggest some genetic contribution to a trait under the assumption that MZ twins share 100% of genes and DZ twins share ~50% of genes. DZ, dizygotic; FT, fat taste; ICC, intraclass correlation coefficient; MZ, monozygotic; WC, waist circumference; WHR, waist-to-hip ratio.

Effect modification by zygosity was explored by comparing the diet effect on FT rank in MZ pairs and DZ pairs. The greater the difference of the diet effect on FT rank between DZ pairs and MZ pairs, the greater degree genetic effects have on regulating changes to FATT. The effect of the diet was not significantly different between zygosities at any time point (**Table 4**) and there was no evidence of time × diet × zygosity interaction ($P = 0.892$) (i.e., the pattern of FT rank in each diet group was similar for MZ and DZ pairs) (**Figure 5**).

Dietary compliance

Compared with baseline, there was a significant reduction in energy (kilojoules) intake in the LF group at weeks 4 and 8 and a significant increase in energy intake in the HF group at week 8 (**Table 5**). Energy intake was significantly higher in the HF group than in the LF group at weeks 4 and 8.

Total fat (grams), saturated fat (grams), monounsaturated fat (grams), polyunsaturated fat (grams), and protein (grams) were significantly different between diet groups at weeks 4 and 8 (**Table 6**). The LF group participants significantly reduced their intake of total fat, saturated fat, monounsaturated fat, and polyunsaturated fat at weeks 4 and 8, whereas the HF group significantly increased their intake of total fat and monounsaturated fat at weeks 4 and 8, and intakes of saturated fat and polyunsaturated fat only at week 4.

As expected, the percentage of energy from total fat, saturated fat, monounsaturated fat, polyunsaturated fat, protein, and carbohydrate differed significantly between groups at weeks 4 and 8 (Table 6). The LF group significantly decreased intakes of energy derived from total fat, saturated fat, monounsaturated fat, and polyunsaturated fat, and significantly increased intakes of energy derived from protein and carbohydrate at weeks 4 and 8. The HF group significantly increased intakes of energy derived from total fat and monounsaturated fat at weeks 4 and 8. The mean percentages of energy from total fat in the LF group were 16.7% and 17.2% at weeks 4 and 8, respectively. Similarly, mean percentages of energy from total fat in the HF group were 39.6% and 38.4% at weeks 4 and 8, respectively. Both diet groups complied, on average, with the required amounts of energy from fat: <20% and >35%.

TABLE 4
Diet effect on fat taste rank by zygosity and time[1]

	Fat taste rank		
	MZ pairs ($n = 34$)	DZ pairs ($n = 10$)	Time × diet × zygosity contrasts
Baseline	0.00 (−1.54, 1.4)	0.40 (−2.43, 3.23)	0.40 (−2.84, 3.64)
Week 4	4.23 (3.05, 5.41)	4.63 (2.37, 6.89)	0.40 (−2.17, 2.96)
Week 8	5.56 (4.34, 6.78)	6.65 (4.12, 9.18)	1.09 (−1.73, 3.92)

[1] Values are means (95% CIs). Estimates (95% CIs) were obtained by using a linear mixed model including diet, time, zygosity, and all double and triple interactions as fixed effects. Greater differences within DZ pairs than within MZ pairs indicate a greater genetic contribution to diet-mediated change in fat taste rank. DZ, dizygotic; MZ, monozygotic.

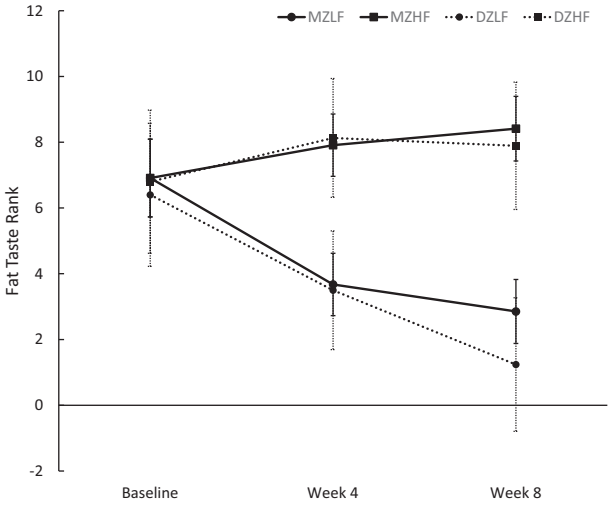

FIGURE 5 Means (95% CIs) for fat taste rank by diet group and zygosity. Means (95% CIs) and P values were estimated by using a linear mixed model including diet, time, zygosity, and all double and triple interactions as fixed effects. Time × diet × zygosity interaction, $P = 0.892$. DZHF, high-fat dizygotic; DZLF, low-fat dizygotic; MZHF, high-fat monozygotic; MZLF, low-fat monozygotic.

TABLE 5
LF and HF diet within- and between-group mean differences in energy intake over the 8-wk trial[1]

	Energy, kJ		
	LF diet ($n = 44$)	HF diet ($n = 44$)	Between-group differences
Baseline	9076 ± 4293[2]	8375 ± 3224	−700 (−1907, 506)[3]
Week 4 vs. baseline	−2810 (−7079, −1541)***	1276 (−3, 2555)	3385 (2546, 4225)***
Week 8 vs. baseline	−2159 (−3335, −983)***	1257 (75, 2440)*	2716 (1682, 3750)***

[1]Between-group differences were calculated as HF diet − LF diet; means (95% CIs) and P values were estimated by using a linear mixed model including diet, time, and time × diet interaction as fixed effects, with twin pair as a random effect to account for the correlation between co-twins, and participant as the subject with repeated measures. Post hoc Sidak's test and CIs are reported. *$P < 0.05$, ***$P < 0.001$. HF, high-fat; LF, low-fat.
[2]Mean ± SD (all such values).
[3]Mean; 95% CI in parentheses (all such values).

TG ranking task

There was no significant time × diet interaction for TG ranking score (**Table 7**).

Liking of foods

There was no significant time × diet interaction for LF liking score, HF liking score, or HF-LF liking score (Table 7).

Intensity ratings to 5 prototypical tastants

There were no significant time × diet interactions for intensity ratings to any of the prototypical tastants ($P > 0.05$) (**Supplemental Table 1**).

DISCUSSION

This study assessed changes in FATT after 8 wk of LF and HF dietary intakes. After the LF diet, FT rank decreased from

TABLE 6
LF and HF diet within- and between-group mean differences in nutrient intakes over the 8-wk trial[1]

	LF diet ($n = 44$)		HF diet ($n = 44$)		Between-group differences	
	g	% of energy	g	% of energy	g	% of energy
Protein						
Baseline	100.5 ± 45.4[2]	19.6 ± 5.4	98.3 ± 39.1	20.5 ± 5.3	−2.2 (−17.3, 13.0)[3]	0.9 (−1.4, 3.2)
Week 4 vs. baseline	−12.7 (−27.5, 2.1)	4.7 (2.3, 7.1)***	8.3 (−6.6, 23.3)	−1.3 (−3.7, 1.1)	18.8 (8.5, 29.2)**	−5.1 (−7.2, −3.0)***
Week 8 vs. baseline	−9.5 (−24.9, 5.9)	3.8 (1.5, 6.1)**	5.5 (−10.0, 21.0)	−1.6 (−3.9, 0.8)	12.9 (2.4, 23.4)*	−4.5 (−7.0, −1.9)**
Total fat						
Baseline	86.1 ± 54.0	33.9 ± 6.5	81.2 ± 43.5	34.7 ± 9.1	−4.9 (−22.8, 13.1)	0.8 (−2.4, 3.9)
Week 4 vs. baseline	−57.2 (−75.1, −39.4)***	−17.3 (−20.7, −13.8)***	25.3 (7.3, 43.3)**	4.9 (1.4, 8.3)**	77.7 (63.1, 92.3)***	22.9 (20.2, 25.6)***
Week 8 vs. baseline	−55.2 (−72.6, −37.7)***	−16.8 (−20.3, −13.2)***	18.8 (1.2, 36.4)*	3.8 (0.2, 7.3)*	69.1 (57.2, 81.0)***	21.3 (18.5, 24.1)***
Saturated fat						
Baseline	33.7 ± 26.1	12.9 ± 3.3	31.6 ± 17.6	13.5 ± 4.4	−2.1 (−11.0, 6.9)	0.6 (−1.0, 2.3)
Week 4 vs. baseline	−24.7 (−32.9, −16.5)***	−7.7 (−9.3, −6.0)***	8.7 (0.4, 16.9)*	1.6 (−0.0, 3.3)	31.2 (25.6, 36.8)***	10.0 (8.6, 11.3)***
Week 8 vs. baseline	−24.8 (−32.7, −16.8)***	−7.9 (−9.6, −6.1)***	5.8 (−2.3, 13.8)	0.8 (−0.9, 2.6)	28.5 (23.7, 33.2)***	9.3 (8.0, 10.7)***
Monounsaturated fat						
Baseline	32.6 ± 18.8	13.0 ± 3.2	30.8 ± 17.9	13.1 ± 4.1	−1.8 (−8.6, 5.0)	0.1 (−1.4, 1.5)
Week 4 vs. baseline	−22.1 (−28.8, −15.5)***	−7.0 (−8.6, −5.5)***	9.6 (2.9, 16.3)**	1.9 (0.3, 3.4)*	29.9 (24.5, 35.4)***	9.0 (7.9, 10.1)***
Week 8 vs. baseline	−21.8 (−28.8, −15.5)***	−6.5 (−8.2, −4.9)***	7.8 (0.9, 14.6)*	1.7 (0.1, 3.4)*	26.8 (22.0, 31.7)***	8.3 (7.0, 9.6)***
Polyunsaturated fat						
Baseline	12.4 ± 7.7	5.1 ± 2.0	12.7 ± 10.5	5.2 ± 3.1	0.3 (−3.7, 4.3)	0.2 (−0.9, 1.3)
Week 4 vs. baseline	−6.9 (−9.6, −4.2)***	−1.9 (−2.7, −1.1)***	3.5 (0.8, 6.2)**	0.6 (−0.2, 1.5)	10.8 (7.5, 14.0)***	2.7 (2.0, 3.4)***
Week 8 vs. baseline	−6.0 (−9.4, −2.7)***	−1.6 (−2.5, −0.7)***	3.3 (−0.1, 6.7)	0.8 (−0.1, 1.8)	9.6 (7.0, 12.1)***	2.6 (1.8, 3.4)***
Carbohydrate						
Baseline	222.7 ± 105.9	40.8 ± 7.7	193.5 ± 90.8	37.8 ± 10.7	−29.2 (−63.8, 5.4)	−3.0 (−6.3, 0.3)
Week 4 vs. baseline	−25.4 (−57.9, 7.1)	10.3 (6.2, 14.4)***	17.5 (−15.3, 50.3)	−1.4 (−5.5, 2.6)	13.7 (−8.0, 35.4)	−14.7 (−18.1, −11.4)***
Week 8 vs. baseline	8.7 (−25.9, 43.3)	12.0 (8.2, 15.7)***	28.9 (−5.7, 63.6)	−0.3 (−4.1, 3.5)	−9.0 (−53.2, 35.2)	−15.3 (−18.6, −12.0)***
Alcohol						
Baseline	7.2 ± 16.5	2.6 ± 5.3	8.2 ± 23.0	3.1 ± 8.8	1.0 (−7.6, 9.6)	0.5 (−2.7, 3.8)
Week 4 vs. baseline	−2.9 (−10.6, 4.7)	−0.3 (−3.2, 2.6)	−3.3 (−11.1, 4.4)	−1.3 (−4.1, 1.6)	0.6 (−4.1, 5.4)	−0.4 (−2.7, 1.9)
Week 8 vs. baseline	−4.4 (−11.0, 2.1)	−1.0 (−3.0, 1.0)	0.4 (−6.1, 7.0)	−1.0 (−3.0, 1.0)	5.9 (−4.3, 16.1)	0.5 (−2.2, 3.2)

[1]Between-group differences were calculated as HF diet − LF diet; means (95% CIs) and P values were estimated by using a linear mixed model including diet, time, and time × diet interaction as fixed effects, with twin pair as a random effect to account for the correlation between co-twins, and participant as the subject with repeated measures. Post hoc Sidak's test and CIs are reported. *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$. HF, high-fat; LF, low-fat.
[2]Mean ± SD (all such values).
[3]Mean; 95% CI in parentheses (all such values).

TABLE 7
LF and HF diet within- and between-group mean differences in TG ranking score and food liking scores over the 8-wk trial[1]

	LF diet ($n = 44$)	HF Diet ($n = 44$)	Between-group differences	Time × diet interaction, P
TG ranking score				0.849
Baseline	6.3 ± 3.5[2]	5.6 ± 3.9	−0.7 (−2.2, 0.8)[3]	
Week 4 vs. baseline	−0.1 (−1.9, 1.8)	0.1 (−1.7, 2.0)	−0.5 (−1.9, 1.0)	
Week 8 vs. baseline	−0.5 (−2.3, 1.4)	−0.9 (−2.7, 1.0)	−1.1 (−2.6, 0.4)	
LF liking score				0.757
Baseline	13.8 ± 10.8	11.0 ± 12.1	−2.9 (−6.3, 0.6)	
Week 4 vs. baseline	0.0 (−3.0, 3.0)	1.3 (−1.6, 4.3)	−1.6 (−4.7, 1.6)	
Week 8 vs. baseline	0.4 (−2.5, 3.3)	1.5 (−1.4, 4.4)	−1.7 (−5.2, 1.8)	
HF liking score				0.645
Baseline	19.9 ± 11.5	18.9 ± 12.4	−1.0 (−4.8, 2.8)	
Week 4 vs. baseline	−1.8 (−5.2, 1.5)	−0.9 (−4.2, 2.5)	−0.0 (−4.2, 4.1)	
Week 8 vs. baseline	−3.4 (−6.3, −0.6)*	−1.8 (−4.7, 1.0)	0.6 (−3.4, 4.7)	
HF-LF liking score				0.851
Baseline	6.1 ± 6.3	7.9 ± 8.8	1.8 (−1.4, 5.1)	
Week 4 vs. baseline	−1.6 (−4.7, 1.6)	−2.0 (−5.2, 1.2)	1.4 (−1.7, 4.5)	
Week 8 vs. baseline	−3.7 (−6.7, −0.6)*	−3.3 (−6.3, −0.3)*	2.2 (−0.3, 4.8)	

[1]Between-group differences were calculated as HF diet − LF diet; means (95% CIs) and P values were estimated by using a linear mixed model including diet, time, and time × diet interaction as fixed effects, with twin pair as a random effect to account for the correlation between co-twins, and participant as the subject with repeated measures. Post hoc Sidak's test and CIs are reported. *$P < 0.05$. HF, high-fat; LF, low-fat; TG, triglyceride.

[2]Mean ± SD (all such values).

[3]Mean; 95% CI in parentheses (all such values).

6.8 to 2.6 (∼6.1 to 1.8 mM in FATT), indicating a large increase in FT sensitivity. This is likely due to increased expression of fatty acid receptors on lingual taste papillae. Rodent studies have shown that expression of 2 fatty acid taste receptors, CD36 and free fatty acid receptor 4, decreases after HF exposure (35, 36). After the HF diet, FT rank increased from 6.9 to 8.4 after 8 wk (∼6.3 to 8.7 mM in FATT), indicating a decrease in FT sensitivity. Although the magnitude of the reduction in the HF group was much lower than the increase seen in the LF group, this is likely because, in the HF group, total fat intake (grams) increased by 23%, whereas in the LF group there was a 64% reduction in total fat (grams) consumed. Overall, an increase (or decrease) in energy from fat by 1% resulted in an increase (or decrease) in FT rank by 0.15, similar to the cross-sectional analysis of these data ($\hat{\beta} = 0.11$) (1).

Previous intervention studies reported a 1.0% increase in body weight after increased intake of energy from fat (28–45%) over 4 wk (11) and a 2.3% reduction in body weight after reduced energy intake from fat (33–16%) over 4 to 6 wk (11, 12). In the current 8-wk trial, 39.0% of energy from fat led to a 0.1% body weight increase, and 16.8% of energy from fat led to a 1.8% reduction in body weight, which was of lower magnitude than the previous studies, especially considering the longer duration. Importantly, when we assessed the change in FATT in the subgroup who maintained body weight, the conclusions were unchanged, suggesting that weight loss is not a factor in altering FT sensitivity.

Dietary fat contributed to ∼33% of energy intake in participants at baseline, similar to the 31–32% energy intake from fat in the Australian adult population (37). Despite efforts to maintain body weight, 11 individuals who consumed the LF diet lost >2 kg. This shows the difficulty of maintaining weight under an LF dietary protocol in free-living individuals and was likely exacerbated by increasing satiety response to dietary fat. Diets that approach the lower AMDR for fat are likely to be useful in Western populations to aid in lowering energy intake and the risk of obesity. This is in line with evidence that fat intake is linked to obesity (38) due to being more energy dense than other macronutrients.

The heritability of FT rank was relatively low compared with the heritability of salty (22%) and sour (53%) detection thresholds (39). Garneau et al. (40) reported heritability of 8–19% for linoleic acid intensity ratings at various concentrations, which is similar to the current study. However, intensity rating and detection threshold are different taste dimensions and are not directly comparable. In addition, MZ and DZ ICCs were similar, suggesting little to no genetic contribution to detection threshold, but rather that familial environment (e.g., diet) is responsible for the concordance within pairs. In addition, no significant effect modification of zygosity on diet was observed, indicating little genetic contribution to diet-mediated change in FATT. It is important to recognize that this does not suggest that genes do not influence FATT at all, because previous studies have found that polymorphisms of *CD36* influence FT sensitivity (14, 17). Rather, the current study suggests that genetic variation does not make individuals any more or less susceptible to modifying FATT by diet. We hypothesize that genetic polymorphisms have a role in establishing baseline FATT, whereas dietary intake modifies it. Although genetic variation has little influence on FATT, there is evidence of genetic contributions to fat preference (41), giving evidence to the contrast between thresholds and hedonics (42).

The expression of FT receptors in the oral cavity and throughout the alimentary canal, when triggered by fatty acids, initiates the satiety cascade (2, 7). Individuals with impaired FT may have lower expression of these receptors (35, 36), and therefore have an attenuated satiety response after fatty food consumption (6, 7). In this way, an individual who has lower sensitivity will feel less full and consume a greater quantity of energy, independent of the hedonic system. LF dieting may aid in increasing the expression of FT receptors throughout the alimentary canal (7), leading to an increased postingestive satiety response to fatty food and reducing passive overconsumption. However, FT sensitivity may not be associated with obesity (13). This may be because, although impaired FT sensitivity does influence increased dietary fat intake, it does not necessarily lead to increased energy intake in all individuals. Furthermore, factors that influence obesity are multifaceted and complex. As shown in mice, exposure to obesogenic conditions may make FT-impaired individuals more susceptible to overconsumption of energy and subsequent weight gain (43).

In other taste modalities, taste thresholds are not associated with hedonics (42) and this was the same with FT. In the current study, there was no significant time × diet interaction for HF-LF liking score, indicating that FT sensitivity has no influence on fatty food liking. This is in line with a recent study that did not observe an association between FT sensitivity and liking of fatty food (44). There is evidence that reduced dietary fat intake decreases the preference for fatty foods (45, 46). It should be noted that changes in dietary fat intake may affect other sensory acuities. For example, 24 wk of HF dietary intake reduced olfactory sensation in rodents (47), which may be a contributor to changed preferences in the previous studies (45, 46). However, olfactory acuity was not measured in the current study so we cannot conclude this is the case. Similarly, TG perception was not affected by diet in the current study. This may have been because the concentrations of canola oil used were too low to yield free fatty acid concentrations above the detection threshold. A recently published study reported that average canola oil detection thresholds were 11.7% ± 1.8% (48), whereas the concentrations in the current study ranged from 0% to 10%, which were likely below detection for most participants. Although there may be some degree of fatty acid perception in foods containing TGs due to free fatty acids (48, 49) and lingual lipase activity (17, 50), this does not have a noticeable impact on preference in most cases. However, in foods that contain concentrations of free fatty acid that exceed suprathreshold concentrations (e.g., spoiled foods, some oils, and nuts), FT may act as a warning system to deter intake, similar to bitter or sour taste. Therefore, higher sensitivity may reduce liking for these specific foods.

This randomized controlled trial has limitations that should be noted when interpreting these results. First, because the trial could not be blinded, we cannot rule out some level of contamination with co-twins mutually discussing their diets. This type of contamination is expected to bias the estimated diet effect toward the null; therefore, the true effect of the diet on FATT should be even larger than that reported. Second, although the number of twin pairs was adequate to assess the dietary intervention and heritability, the small number of DZ twin pairs recruited is not powered to detect small differences in effect modification of zygosity on the diet, and these estimates should be interpreted as being only indicative of a minimal impact of genetic variation on FATT. Third, the use of one 24-h dietary recall as a baseline measure of dietary intake only provided a snapshot of a participant's usual diet. In addition, diet recalls and records are subject to bias and underreporting in many studies, especially in obese individuals, and we cannot rule out bias and underreporting in this study. Fourth, although there was no effect of diet on intensity ratings to the 5 prototypical tastants, it is acknowledged that intensity ratings are not directly comparable to FATT. Finally, although satiety can be inferred on the basis of findings from Stewart et al. (7), we did not measure satiety ratings so we cannot conclude whether this trial had an influence on satiety.

The current study shows that 8 wk of consumption of an LF diet increases sensitivity to FT and the same period with an HF diet attenuates sensitivity, regardless of body weight. There is little indication of genetic contribution on FT. Therefore, dietary fat intake is the most important influencer on FT sensitivity. Diets that approach the lower AMDR for fat may aid in increasing the satiety response to fatty food, decrease passive overconsumption, and subsequently reduce body weight.

The authors' responsibilities were as follows—AC, CN, DB, KD, and RK: designed the research; AC and DB: conducted the research; AC and LO: analyzed the data and performed the statistical analyses; AC: wrote the manuscript; RK: had primary responsibility for the final content; and all authors: read and approved the final manuscript. None of the authors declared a conflict of interest.

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