ORIGINAL RESEARCH

CURRENT DEVELOPMENTS IN NUTRITION

Maternal and Pediatric Nutrition

Spillover Effects of a Family-Based Childhood Weight-Management Intervention on Parental Nutrient Biomarkers and Cardiometabolic Risk Factors

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ABSTRACT

Background: Parental involvement has been shown to favorably affect childhood weight-management interventions, but whether these interventions influence parental diet and cardiometabolic health outcomes is unclear.

Objectives: The aim was to evaluate whether a 1-y family-based childhood weight-management intervention altered parental nutrient biomarker concentrations and cardiometabolic risk factors (CMRFs).

Methods: Secondary analysis from a randomized-controlled, parallel-arm clinical trial (NCT00851201). Families were recruited from a largely Hispanic population and assigned to either standard care (SC; American Academy of Pediatrics overweight/obesity recommendations) or SC + enhanced program (SC+EP; targeted diet/physical activity strategies, skill building, and monthly support sessions). Nutrient biomarkers (plasma carotenoids and fat-soluble vitamins, RBC fatty acid profiles) and CMRFs (BMI, blood pressure, glucose, insulin, lipid profile, inflammatory and endothelial dysfunction markers, adipokines) were measured in archived samples collected from parents of participating children at baseline and end of the 1-y intervention.

Results: Parents in both groups (SC = 106 and SC+EP = 99) had significant reductions in *trans* fatty acid (–14%) and increases in MUFA (2%), PUFA n–6 (ω-6) (2%), PUFA n–3 (7%), and β-carotene (20%) concentrations, indicative of lower partially hydrogenated fat and higher vegetable oil, fish, and fruit/vegetable intake, respectively. Significant reductions in high-sensitivity C-reactive protein (hsCRP; –21%) TNF-α (–19%), IL-6 (–19%), and triglycerides (–6%) were also observed in both groups. An additional significant improvement in serum insulin concentrations (–6%) was observed in the SC+EP parents. However, no major reductions in BMI or blood pressure and significant unfavorable trajectories in LDL-cholesterol and endothelial dysfunction markers [P-selectin, soluble intercellular adhesion molecule (sICAM), thrombomodulin] were observed. Higher carotenoid, MUFA, and PUFA (n–6 and n–3) and lower SFA and *trans* fatty acid concentrations were associated with improvements in circulating glucose and lipid measures, inflammatory markers, and adipokines.

Conclusions: The benefits of a family-based childhood weight-management intervention can spill over to parents, resulting in apparent healthier dietary shifts that are associated with modest improvements in some CMRFs. *Curr Dev Nutr* 2022;6:nzab152.

Keywords: childhood obesity, parent spill-over, family-based intervention, fatty acids, carotenoids, nutrient biomarkers, cardiometabolic risk factors

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Abbreviations used: CLA, conjugated linolenic acid; CMRF, cardiometabolic risk factor; D5D, delta-5-desaturase; D6D, delta-6-desaturase; DNL, de novo lipogenesis; EP, enhanced program; hsCRP, high-sensitivity C-reactive protein; SC, standard care; SCD, stearoyl Co-A desaturase; sICAM, soluble intercellular adhesion molecule; TC, total cholesterol; TG, triglyceride.

Introduction

Childhood overweight/obesity is a major public health problem in the United States and is associated with adverse health outcomes throughout the life span (1). Current recommended strategies to prevent/treat excess weight gain during childhood include a combination of dietary modification, increased physical activity, and behavioral therapy (2–5). The majority of childhood weight-management interventions have

been implemented in school and community settings with modest success (6–9). More recently, focus has shifted to the family/home environment, since parental involvement has been shown to be a key mediator in the effectiveness of childhood obesity interventions, especially in young children (10–16). These family-based interventions have sought to involve parents in various ways, ranging from solely targeting them as "agents of change" in their child's weight loss (17, 18) to participating in educational modules that support fostering a home environment that promotes healthy dietary habits, increases physical activity, and reduces sedentary behaviors (19–31).

Using the latter approach, we have documented that providing targeted family-based behavioral counseling as part of standard care (American Academy of Pediatrics overweight/obesity recommendations) (31) can help children with overweight/obesity adopt healthier eating patterns that are associated with modest improvements in BMI *z* score (BMI score standardized for age and sex) and several cardiometabolic risk factors (CMRFs) (30). The majority of parents who participated in this intervention were female (94% mothers) and nearly all of them (92%) had a BMI that classified them as being in the overweight or obese categories. This is consistent with the observation that children with BMI *z* scores over the 85% percentile tend to have home environments where either one or both parents are overweight/obese (23). Interestingly, maternal rather than paternal weight status (23) and nutrient intake (27) are stronger predictors of their child's dietary intake and weight status. This finding suggests that overall family lifestyle is predominantly driven by maternal outcomes (32, 33). Yet, very few family-oriented interventions (13, 19, 20, 22, 25, 27, 28, 34) have measured parental diet and their relation to health outcomes. Given the high prevalence of children as well as adults with obesity, a family-centered weight-management intervention that has beneficial effects for both the children and parents could have a significant public health impact (35, 36). Thus, the goal of the present study was to investigate whether a family-based weight-management intervention influenced parental nutrient intake patterns as well as CMRFs. We hypothesized that adoption of the lifestyle recommendations by the parents of the participating children would be reflected in circulating nutrient biomarker concentrations and lead to an improvement in their CMRF profile.

Methods

Study subjects and design

Detailed descriptions of the family-based management trial (NCT00851201 registered on clinicaltrials.gov), including design, intervention, and primary outcomes in the children, have been previously published (30, 31). This study focused on the parents ($n = 205$) of participating children (aged 7–12 y with baseline BMI *z* score $\geq$85th percentile) who had an archived fasting plasma, serum, and RBC sample at both baseline and end of the 1-y intervention. All study procedures were approved by the Institutional Review Board of the Albert Einstein College of Medicine. Approval to analyze de-identified samples and data was obtained from Tufts University/Tufts Medical Center Institutional Review Board.

Briefly, the study was a 2-arm, randomized, controlled, parallel-group trial comparing standard care alone (SC) with SC + enhanced program (SC + EP), and was conducted in a pediatric primary clinical care urban setting at Jacobi Medical Center (Bronx, NY). The SC intervention was based on the American Academy of Pediatrics' evidence-based recommendations (37) and included an initial comprehensive visit to assess weight-related issues and to engage both the children and parents/guardians in developing intervention goals collaboratively. The pediatricians utilized the 35-item Pediatric Symptom Checklist to screen for emotional and behavioral dysfunction (38, 39) and the 5-item Habits questionnaire to assess dietary, physical activity, and sedentary behaviors (40) and made referrals to a registered dietitian. The Habits questionnaire addressed meals (e.g., eating as a family and avoid eating while watching TV), fruit and vegetable intake (e.g., increasing serving, excluding juices), beverage intake (e.g., decreasing sugar-sweetened beverages, choosing 1%-fat milk and water), fast food (e.g., decreasing frequency, avoiding super-sizing, and choosing healthier options), and physical activity/sedentary behavior (e.g., increasing moderate and vigorous physical activity and decreasing screen time). Families also received a dietary booklet targeting behaviors associated with excess body weight (soda, sugary beverages, junk food, fast foods) as well as the federal 2005 Dietary Guidelines, recipes, physical activity booklet (listing recreational facilities, tips to reduce TV viewing, and engage in 60 to 90 min of vigorous activity per day), and a monthly newsletter (tips for healthy living). During the quarterly follow-up pediatrician visits, the collaborative goals identified at the initial visit were reviewed and reiterated. The pediatricians who provided the SC to both study groups were blinded to treatment allocation.

The EP added a behavioral change component (8 weekly skill-building core sessions, each 1.5 to 2 h in duration), and subsequent monthly post-core support sessions focused on improving dietary behaviors and increasing engagement in physical activities provided by bilingual multidisciplinary staff. As described previously (31), the skill-building core sessions included alternating in-person groups and parent phone consultations. The in-person core group sessions consisted of food preparation or other skill activity for parents and children, followed by a physical activity session for the children and discussion session for parents to enhance parenting and problem-solving skills related to the themes covered in the joint family sessions. The monthly post-core support sessions consisted of engagement activities that were designed to provide ongoing support to parents/guardians and children during the intervention program. A group "meet up" approach was used to provide families with the opportunity to "check in" with EP multidisciplinary staff. Post-core session themes included "boot camp" circuit training, holiday themes with active games, and outing/field trips to a local park or within the campus grounds. Development of the EP components was guided by evidence-based recommendations and interventions and clinical experience in the target communities. Motivational enhancement based on motivation interviewing principles was used to engage both parent and child to evoke "their" reasons for changing unhealthy lifestyle behaviors. All intervention components were available in Spanish and English. The newsletter (provided to both groups) included healthful versions of Latinx recipes and featured information regarding popular Latin American fruits and vegetables sold in the local farmers' market sponsored by the health system. Likewise, the physical activity sessions included popular Latin American dance steps.

Outcome variables and assessment

Nutrient biomarkers.

Dietary and endogenous metabolism biomarkers were measured in fasting plasma and RBC samples collected from the parents both pre- and post-intervention. Dietary biomarkers included plasma carotenoid concentrations (pigmented fruit and vegetable intake) (41); fat-soluble vitamins A, D, E, and K (animal foods, fortified foods, supplements, and/or vegetable oils) (42); and RBC fatty acid profiles including linoleic [18:2n–6] and α-linolenic [18:3n–3] (vegetable oils) (43); eicosapentaenoic [EPA, 20:5n–3], docosapentaenoic [DPA, 22:5n–3], and docosahexaenoic [DHA, 22:6n-3] (fish) (44); pentadecanoic [15:0] (products containing dairy fat) (45); and *trans* fatty acids (ruminant/partially hydrogenated fat) (46). Endogenously synthesized SFA, MUFA, and PUFA n–6 profiles were also measured, and desaturase enzyme indices estimated to reflect de novo lipogenesis (DNL) (47).

HPLC was used to determine plasma carotenoid (lutein, zeaxanthin, cryptoxanthin, β-carotene, and lycopene) including vitamin A and vitamin E, (48) as well as vitamin K concentrations (49), as previously described. Vitamin D (25-hydroxyvitamin D) was measured using a commercially available kit (DiaSorin). The respective intra-assay and inter-assay CVs were 4% and 3.9% for carotenoids and 9% and 10% for vitamin D. For vitamin K, 2 pooled plasma samples were run as low (CV: 12%) and high (CV: 8%) controls with every batch. RBC fatty acid profiles were quantified using an established GC method (50–52). The inter-assay CVs ranged from 0.5% to 4.3% for fatty acids with concentrations >5 mol%, 1.8–7.1% for fatty acids between 1 and 5 mol%, and 2.8–11.1% for fatty acids <1 mol%. Desaturase enzyme activities were calculated as product to precursor ratios of individual fatty acids and included the following: stearoyl-CoA-desaturase [SCD1; palmitoleic (16:1n–7)/palmitic (16:0) and SCD2; oleic (18:1n–9)/ stearic (18:0)], delta-6-desaturase (D6D; dihomo-gamma-linolenic (20:3n–6)/linoleic (18:2n–6), and delta-5-desaturase (D5D; arachidonic (20:4n–6)/20:3n-6 (47).

Cardiometabolic risk factors.

Available CMRF data for the parents from the primary clinical trial (31) and ancillary study (30) were divided into 7 broad categories: BMI, blood pressure (systolic and diastolic), glucose metabolism (fasting glucose and insulin), lipid profile [total cholesterol (TC), LDL cholesterol, HDL cholesterol, triglycerides (TGs)], markers of inflammation [high-sensitivity C-reactive protein (hsCRP), TNF-α, IL-6], vascular adhesion [E-selectin, P-selectin, soluble intercellular adhesion molecule (sICAM)] and coagulation (thrombomodulin), and adipokines (leptin and adiponectin).

Fasting TC, LDL cholesterol, HDL cholesterol, TGs, insulin, and glucose were assessed using standard methods, as described for the primary study (31). Serum TNF-α, IL-6, E-selectin, P-selectin, sICAM, thrombomodulin, leptin and adiponectin concentrations were measured using commercially available multiplex assays (electrochemiluminescence detection sandwich immunoassay: V-PLEX Human Cytokine Assays; V-PLEX Human Biomarker Assays; Human Metabolic Assays) from Meso Scale Discovery using a Meso Scale Discovery SECTOR Imager 2400. Serum hsCRP was measured by solid-phase, 2-site chemiluminescent immunometric assay using the IMMULITE 2000 (Siemens Healthcare Diagnostics). All CMRFs were measured in the fasted state.

Study sample.

Sample size estimates for the primary clinical trial that provided the samples for the present study have been reported previously (31). For the 321 children in the primary trial, there were 287 parents after accounting for siblings, with 205 parents having an archived blood sample to perform the nutrient biomarker and CMRFs reported in the present study. With this given sample size of 106 in SC and 99 in SC + EP groups, there was 80% power to detect between-group differences of 0.39 SD with a 2-sided type I error rate of 5%. Additionally, to account for multiple comparisons, with a type I error rate = 0.005 under a conservative Bonferroni adjustment with 80% power, the minimum detectable standardized effect size was 0.52 SD.

Statistical analysis

The analysis was based on an intention-to-treat approach. Data from each parent were analyzed as per their initial assignment in the primary clinical trial to the SC or SC + EP group. Only parents with nutrient biomarkers and CMRF data at both baseline and 1-y were included in the analysis. Data were checked to identify and resolve reasons for missing values, inconsistencies, and out-of-range values.

Descriptive analyses of baseline characteristics of the SC and SC + EP groups were summarized using medians (IQR) or proportions. Nutrient biomarkers and CMRF data at baseline and 1-y were summarized for each group using geometric means and SD estimated from log-transformed values.

Differences in nutrient biomarker and CMRFs, as dependent variables, were assessed using a mixed-effects random intercept linear model with group, time, and group × time interaction as fixed effects. Participant was included as a random effect within the model and P values presented from the corresponding F-test for each fixed effect. Robust SEs were used to account for possible model misspecification. Dependent variables were log-transformed to facilitate reporting differences as mean % difference (95% CIs) and were calculated from back-transformed model-based least-square means as {2.72^[LSMEANS(1 y − baseline)] − 1} × 100%. Additionally, given the lack of intervention effect, least-square means for 1-y change in outcomes were reported from the mixed-effects model to represent pooled results across all parents among combined groups.

Spearman's ρ correlation between 1-y change in CMRFs with 1-y change in nutrient biomarkers was presented for each outcome pair. Correlation estimates were adjusted for sex, age, group, and baseline CMRFs (and additionally adjusted for baseline BMI for correlations not with BMI). Sex differences were not presented due to the small number of fathers in the sample (7 males in the SC and 6 males in the SC + EP group). All statistical analyses were performed with SAS software (version 9.4; SAS Institute, Inc.). Significance level was set at $P < 0.05$.

Results

Baseline characteristics

The baseline characteristics of the parents are listed in **Table 1**. Their ages ranged between 23 and 68 y and 93% were women (mothers). The

TABLE 1 Characteristics of the parents at baseline[1]

Variables	SC ($n = 106$)	SC + EP ($n = 99$)
Age,[2] y	37 (32–42)	38 (33–43)
Sex, females/males, *n/n*	99/7	93/6
BMI,[3] kg/m^2	33.5 (6.9)	32.5 (7.0)
BMI classification, *n* (%)		
Normal weight (<25)	2 (2%)	10 (10%)
Overweight (≥25 to <30)	25 (24%)	30 (30%)
Obesity (≥30)	76 (74%)	59 (60%)
Blood pressure classification, *n* (%)		
Normal (<120/<80 mmHg)	63 (61%)	71 (72%)
Elevated (120–129/<80 mmHg)	20 (19%)	11 (11%)
Stage 1 hypertension (130–139/80–80 mmHg)	9 (9%)	9 (9%)
Stage 2 hypertension (≥140/≥90 mmHg)	12 (11%)	8 (8%)
Fasting plasma glucose, *n* (%)		
<100 mg/dL	64 (60%)	69 (70%)
100–125 mg/dL	24 (23%)	19 (19%)
≥125 mg/dL	18 (17%)	11 (11%)
Education, %		
No formal schooling	1.8%	1.0%
Grades 1–11	50.0%	54.5%
High school/GED	29.3%	20.2%
Some college/technical school certificate	9.5%	15.2%
Associate's/Bachelor's degree	9.4%	9.1%
Race/ethnicity, %		
Hispanic/Latino	70.8%	78.8%
Non-Hispanic Black	20.7%	14.1%
White, Asian, and multiracial	8.5%	7.1%
Occupation, %		
Homemaker	56.6%	52.5%
Employed full time	12.3%	12.1%
Employed part time	20.8%	21.2%
Unemployed/retired	10.3%	14.1%
Income, %		
$0–$9999	39.6%	37.4%
$10,000–$29,999	34.9%	35.4%
$30,000 or above	5.6%	10.1%
Prefer not to answer	19.8%	17.2%
Marital status, %		
Married/living as married	59.4%	54.6%
Widowed	2.8%	1.0%
Divorced/separated	18.9%	14.2%
Never married	15.1%	24.2%
Prefer not to answer	3.8%	6.1%

[1] EP, enhanced program; GED, general educational development; SC, standard care.
[2] Median (IQR)
[3] Mean (SD)

mean BMI (kg/m^2) was 33.0, with 7% being classified as normal weight, 27% in the overweight category, and 66% in the obese category. The majority of the parents in both groups had normal fasting glucose concentrations (85%) and blood pressure (67%), but approximately 20% met either the stage 1 or 2 hypertension classification (53). Approximately 70% of the parents self-identified as Hispanic/Latino, approximately 50% had less than a high school education, and over 70% reported an annual income less than $30,000.

Nutrient biomarkers

No significant effect of the intervention (group effect) was observed between parents in either the SC or SC + EP groups (**Table 2**). However, significant differences were observed in several nutrient biomarkers at the end of the 1-y intervention (time effect) in both groups. Thus, nutrient biomarker data from parents in both groups were combined and the pooled change over 1-y is summarized in **Figure 1**. Results indicate a significant increase in plasma concentrations of β-carotene (20%; predominantly yellow/orange fruits and vegetables) and lycopene (7%; predominantly tomatoes and derived products). There was a significant decrease in vitamin D (–7%) and vitamin E (–14%) concentrations. No significant changes were observed in plasma vitamin A or K concentrations. Among the fatty acids, total SFA was significantly decreased in both groups (–3%), primarily due to lower proportions of palmitic (–9%), with compensatory higher proportions of the minor

TABLE 2 Nutrient biomarker concentrations and desaturase enzyme activities at baseline and end of the 1-y intervention by study group[1]

	SC			SC + EP			*P*[2]		
Biomarkers	**Baseline**[3]	**1-y**[3]	**Mean percent difference**[4]	**Baseline**[3]	**1-y**[3]	**Mean percent difference**[4]	**Group**	**Time**	**Group × time**
Carotenoids, μg/dL									
Lutein	10.0 (5.6)	10.3 (5.7)	2.8 [–4.0, 10.0]	11.1 (6.4)	10.4 (5.9)	–6.7 [–13.8, 0.98]	0.319	0.427	0.069
Zeaxanthin	3.4 (2.1)	3.5 (2.0)	4.5 [–2.9, 12.5]	3.7 (2.4)	3.7 (2.2)	0.1 [–7.5, 8.3]	0.319	0.412	0.426
Cryptoxanthin	9.1 (12.7)	10.3 (14.0)	12.8 [1.6, 25.4]	10.8 (15.5)	10.2 (16.0)	–5.9 [–16.2, 5.7]	0.483	0.448	0.023
β-Carotene	15.3 (25.8)	19.3 (32.7)	26.3 [12.9, 41.3]	16.6 (18.8)	19.6 (22.6)	17.8 [3.4, 34.1]	0.652	<0.001	0.422
trans-Lycopene	17.6 (11.3)	18.7 (13.7)	6.4 [-3.4, 17.2]	20.4 (11.3)	19.6 (11.8)	4.2 [–13.8, 6.4]	0.114	0.792	0.148
Fat-soluble vitamins									
Vitamin A, μg/dL	48.4 (14.9)	47.2 (12.7)	–2.5 [–6.0, 1.1]	46.5 (12.0)	44.8 (11.5)	–3.5 [–8.5, 1.8]	0.154	0.064	0.757
Vitamin D, ng/mL	17.9 (8.9)	17.2 (7.2)	–4.8 [–11.9, 2.9]	18.5 (9.6)	16.9 (7.0)	–8.2 [–15.1, –0.8]	0.915	0.017	0.513
Vitamin E, μg/dL	184 (120)	168 (122)	–8.8 [–17.1, 0.3]	188 (128)	158 (121)	–16.3 [–24.8, –6.8]	0.783	<0.001	0.241
Vitamin K, nM/L	0.54 (0.69)	0.55 (0.63)	0.8 [–13.7, 17.7]	0.54 (0.58)	0.49 (0.52)	–8.1 [–22.1, 8.5]	0.533	0.508	0.424
Fatty acids, mol%									
SFAs	41.1 (1.9)	39.8 (2.0)	–3.3 [–4.4, –2.2]	41.1 (2.1)	39.8 (1.7)	–3.4 [–4.5, –2.2]	0.934	<.001	0.938
12:0	0.13 (0.12)	0.12 (0.14)	–3.6 [–21.3, 18.0]	0.14 (0.13)	0.12 (0.12)	–10.8 [–25.2, 6.3]	0.415	0.266	0.569
14:0	0.43 (0.16)	0.53 (0.20)	24.0 [14.5, 34.3]	0.46 (0.18)	0.56 (0.19)	21.7 [12.3, 31.9]	0.138	<.001	0.746
15:0	0.35 (0.08)	0.38 (0.11)	10.8 [3.5, 18.6]	0.36 (0.09)	0.38 (0.14)	7.1 [–1.3, 16.2]	0.750	0.002	0.529
16:0	22.2 (2.1)	20.1 (1.8)	–9.2 [–11.1, –7.2]	22.1 (2.3)	20.1 (1.6)	–8.9 [–10.8, –7.0]	0.732	<.001	0.86
18:0	16.2 (1.1)	16.6 (1.1)	2.4 [1.0, 3.8]	16.2 (1.2)	16.6 (1.1)	2.4 [1.0, 3.8]	0.873	<.001	0.965
20:0	0.16 (0.03)	0.19 (0.05)	20.1 [14.5, 25.9]	0.17 (0.03)	0.20 (0.05)	18.8 [12.8, 25.2]	0.245	<.001	0.772
22:0	0.46 (0.09)	0.49 (0.11)	8.3 [3.1, 13.7]	0.46 (0.10)	0.49 (0.11)	5.3 [–0.3, 11.2]	0.956	<.001	0.453
24:0	1.07 (0.23)	1.13 (0.31)	5.6 [–0.1, 11.7]	1.09 (0.24)	1.15 (0.30)	4.9 [–1.0, 11.1]	0.398	0.012	0.869
MUFAs	16.3 (1.6)	16.48 (1.5)	1.5 [–0.3, 3.3]	16.2 (1.5)	16.5 (1.4)	1.9 [0.1, 3.7]	0.868	0.010	0.748
16:1n–7	0.49 (0.21)	0.55 (0.21)	11.9 [5.6, 18.5]	0.50 (0.21)	0.57 (0.28)	13.3 [5.8, 21.4]	0.54	<0.001	0.772
16:1n–9	0.1 (0.03)	0.12 (0.03)	19.5 [12.5, 26.8]	0.11 (0.03)	0.13 (0.03)	19.2 [11.9, 26.9]	0.389	<0.001	0.955
18:1n–7	1.72 (0.66)	1.81 (0.77)	5.5 [–3.4, 15.2]	1.77 (0.73)	1.71 (0.71)	–3.4 [–12.7, 6.8]	0.736	0.784	0.192
18:1n–9	12.39 (1.3)	12.32 (1.1)	–0.5 [–2.2, 1.2]	12.2 (1.3)	12.4 (1.2)	1.3 [–0.3, 3.0]	0.83	0.517	0.125
20:1n–9	0.21 (0.05)	0.24 (0.06)	16.4 [11.3, 21.7]	0.20 (0.05)	0.24 (0.05)	14.9 [10.7, 19.3]	0.751	<0.001	0.659
22:1n–9	0.046 (0.02)	0.049 (0.02)	5.4 [–3.9, 15.7]	0.05 (0.02)	0.05 (0.02)	3.5 [–5.9, 13.9]	0.923	0.197	0.791
24:1n–9	1.12 (0.23)	1.19 (0.28)	6.2 [0.8, 11.9]	1.10 (0.23)	1.16 (0.26)	5.8 [0.0, 12.0]	0.421	0.003	0.922
PUFA n–6	34.9 (2.4)	35.7 (2.8)	2.1 [0.9, 3.3]	35.2 (2.3)	36.0 (2.1)	2.3 [1.2, 3.4]	0.299	<0.001	0.834
18:2n–6	13.7 (1.6)	13.7 (1.7)	–0.1 [–1.7, 1.7]	14.1 (2.2)	13.9 (1.9)	–1.0 [–3.0, 1.0]	0.187	0.444	0.459
18:3n–6	0.07 (0.04)	0.08 (0.04)	6.8 [–2.2, 16.6]	0.07 (0.04)	0.08 (0.05)	17.8 [5.9, 31.0]	0.671	0.001	0.163
20:2n–6	0.33 (0.08)	0.38 (0.08)	14.1 [11.1, 17.2]	0.34 (0.08)	0.39 (0.08)	12.6 [9.3, 16.0]	0.271	<0.001	0.509
20:3n–6	1.86 (0.57)	2.08 (0.58)	12.0 [7.8, 16.4]	1.86 (0.49)	2.07 (0.52)	11.5 [7.1, 16.0]	0.956	<0.001	0.858
20:4n–6	14.5 (1.3)	14.8 (1.5)	1.8 [0.1, 3.6]	14.3 (1.7)	14.7 (1.5)	3.5 [1.5, 5.5]	0.423	<0.001	0.213
22:2n–6	0.072 (0.03)	0.073 (0.04)	1.9 [–10.1, 15.5]	0.08 (0.04)	0.07 (0.05)	–6.6 [–18.8, 7.4]	0.289	0.601	0.362
22:4n–6	3.17 (0.69)	3.54 (0.77)	11.4 [8.2, 14.8]	3.25 (0.70)	3.61 (0.68)	10.9 [7.3, 14.6]	0.402	<0.001	0.828
22:5n–6	0.97 (0.33)	0.79 (0.29)	–18.6 [–24.5, –12.3]	0.92 (0.34)	0.80 (0.33)	–13.2 [–20.1, –5.7]	0.595	<0.001	0.258
PUFA n–3	6.34 (1.5)	6.77 (1.7)	6.7% [3.5, 10.0]	6.06 (1.5)	6.51 (1.6)	7.4 [3.7, 11.3]	0.171	<0.001	0.775
18:3n–3	0.19 (0.06)	0.22 (0.07)	18.0 [10.5, 25.9]	0.19 (0.07)	0.22 (0.07)	15.2 [8.2, 22.7]	0.464	<0.001	0.609

(*Continued*)

TABLE 2 (*Continued*)

Biomarkers	SC			SC + EP			*P*[2]		
	Baseline[3]	1-y[3]	Mean percent difference[4]	Baseline[3]	1-y[3]	Mean percent difference[4]	Group	Time	Group × time
20:5n–3	0.41 (0.21)	0.43 (0.26)	7.1 [0.3, 14.5]	0.37 (0.21)	0.42 (0.24)	15.8 [8.0, 24.2]	0.29	<0.001	0.112
22:5n–3	2.03 (0.33)	2.17 (0.41)	6.9 [3.5, 10.4]	2.03 (0.43)	2.20 (0.41)	8.3 [4.0, 12.7]	0.817	<0.001	0.631
22:6n–3	3.59 (1.2)	3.79 (1.3)	5.7 [1.9, 9.6]	3.33 (1.1)	3.54 (1.1)	6.1 [1.7, 10.7]	0.08	<0.001	0.883
trans-Fatty acids	0.99 (0.25)	0.88 (0.27)	–11.7 [–16.4, –6.7]	1.00 (0.29)	0.86 (0.27)	–14.5 [–18.6, –10.3]	0.899	<0.001	0.382
16:1n–7*t*	0.084 (0.03)	0.076 (0.02)	–8.9 [–13.6, –4.0]	0.08 (0.02)	0.07 (0.02)	–10.4 [–14.8, –5.8]	0.874	<0.001	0.653
16:1n–9*t*	0.03 (0.01)	0.02 (0.01)	–13.7 [–19.6, –7.4]	0.03 (0.01)	0.02 (0.01)	–16.1 [–22.0, –9.7]	0.809	<0.001	0.594
18:1n–7*t*	0.16 (0.06)	0.15 (0.07)	–7.3 [–15.0, 1.0%]	0.18 (0.06)	0.14 (0.07)	–19.2 [–26.6, –11.0]	0.829	<0.001	0.038
18:1n–9*t*	0.3 (0.11)	0.26 (0.10)	–14.0 [–18.7, –9.1]	0.31 (0.12)	0.26 (0.11)	–15.6 [–19.9, –11.1]	0.974	<0.001	0.627
18:1n–10–12*t*	0.20 (0.08)	0.17 (0.08)	–14.2 [–21.6, –6.1]	0.20 (0.09)	0.16 (0.07)	–18.0 [–25.5, –9.7]	0.427	<0.001	0.502
18:2*t*	0.11 (0.06)	0.10 (0.05)	–11.3 [–21.3, –0.1]	0.11 (0.05)	0.10 (0.06)	–10.5 [–20.7, 0.9]	0.995	0.008	0.921
18:2CLA	0.07 (0.03)	0.07 (0.03)	–9.9 [–15.9, –3.4]	0.08(0.03)	0.07(0.03)	–9.2 [–15.2, –2.8]	0.724	<0.001	0.885
Desaturase activity[5]									
SCD1 (16:1n–7/16:0)	0.02 (0.01)	0.02 (0.01)	7.1 [1.1, 13.4]	0.02 (0.01)	0.02 (0.01)	8.8 [1.9, 16.3]	0.513	<0.001	0.713
SCD2 (18:1n–9/18:0)	0.77 (0.11)	0.74 (0.09)	–2.9 [–5.2, –0.5]	0.76 (0.12)	0.75 (0.10)	–1.0 [–3.6, 1.6]	0.817	0.03	0.299
D6D (20:3n–6/18:2n–6)	0.14 (0.04)	0.15 (0.04)	12.1 [7.4, 16.9]	0.13 (0.03)	0.15 (0.03)	12.6 [7.5, 17.9]	0.414	<0.001	0.876
D5D (20:4n–6/20:3n–6)	7.80 (2.62)	7.09 (2.13)	–9.1 [–12.7, –5.4]	7.66 (2.63)	7.11 (2.27)	–7.2 [–11.0, –3.2]	0.832	<0.001	0.473

[1]Number of parents with both baseline and 1-y values: carotenoids (106 in SC and 99 in SC + EP); fat-soluble vitamins (91–106 in SC and 95–99 in SC + EP); fatty acids and desaturase activity (106 in SC and 99 in SC + EP). CLA, conjugated linolenic acid; D5D, delta-5-desaturase; D6D, delta-6-desaturase; EP, enhanced program; SC, standard care; SCD, stearoyl Co-A desaturase.
[2]F-tests on fixed effects of study group, time, and group by time interaction from a mixed-effects random intercept linear model.
[3]Values are geometric means (SD); SD was estimated from log-transformed values and based on equations described in Quan et al (54).
[4]Values are % mean difference [95% CI], calculated from model-based least-square means as {2.72^[LSMEANS(1 y − baseline)] − 1} × 100%.
[5]Values are calculated as fatty acid product:precursor ratios.

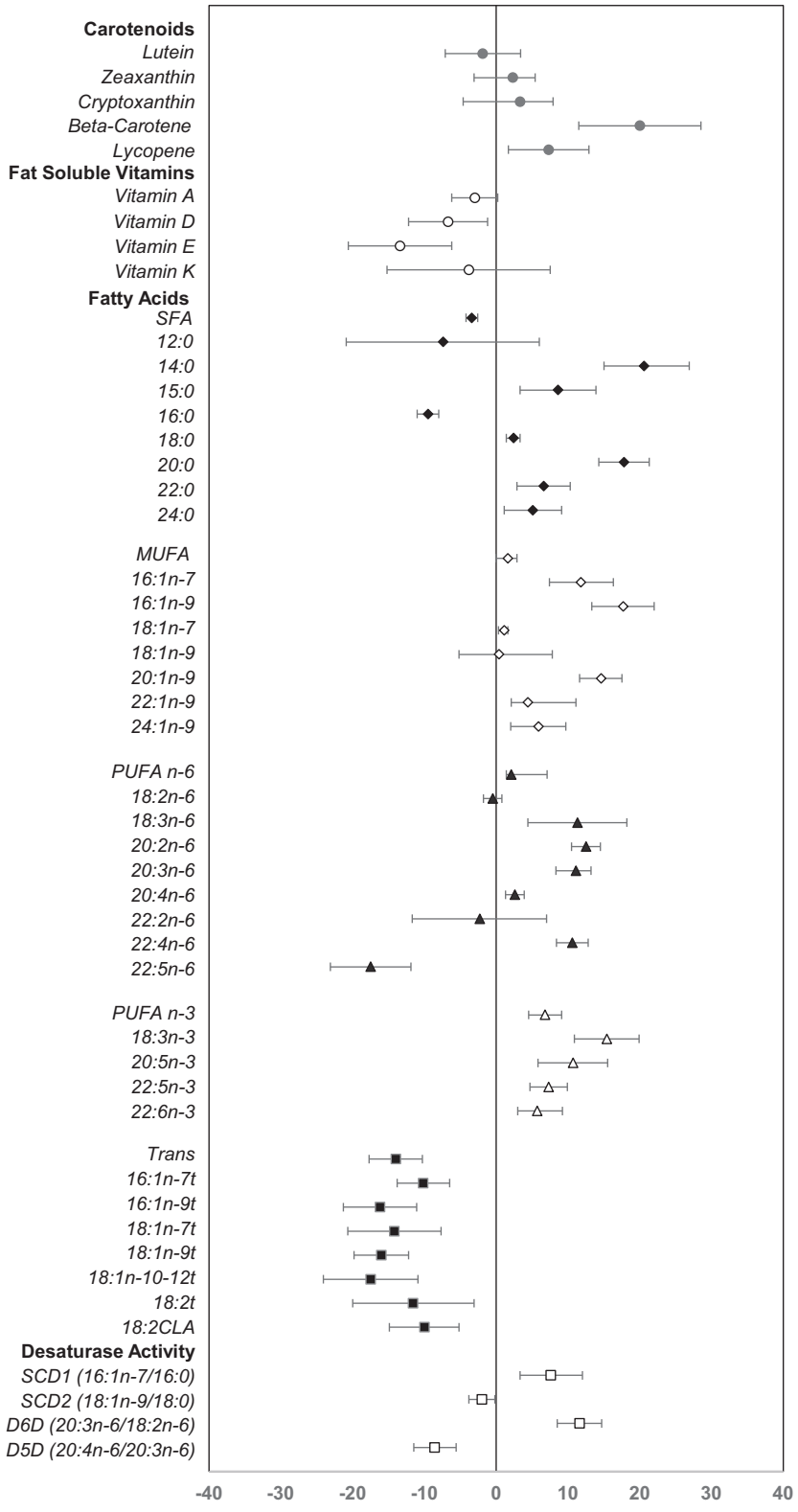

FIGURE 1 Pooled 1-y change in nutrient biomarker concentrations and desaturase enzyme activities. For each individual nutrient biomarker, the mean % difference is plotted as the symbol and the 95% CIs displayed as the bars. The mean % difference value and 95% CIs were derived from least-square means calculated from a mixed-effects random intercept model with time (baseline or 1-y) as a fixed effect and a random intercept for subject correlations. A separate model was fitted for each log-transformed outcome. $n = 205$ and included parents in the SC and SC + EP groups with both a baseline and 1-y nutrient biomarker value. CLA, conjugated linolenic acid; D5D, delta-5-desaturase; D6D, delta-6-desaturase; EP, enhanced program; SC, standard care; SCD, stearoyl Co-A desaturase.

SFAs (2% to 20% for myristic [14:0], stearic, arachidic [20:0], and lignoceric [24:0]). MUFAs significantly increased, especially those in the DNL pathway (6% to 18% for palmitoleic, hypogeic [16:1n–9], gondoic [20:1n–9], and nervonic [24:1n–9]). Total PUFA n–6, gamma-linolenic [18:3n–6], eicosadienoic [20:2n–6], dihomo-gamma-linolenic, arachidonic, and adrenic [22:4n–6] were significantly increased (2% to 13%), with the exception of docosapentaenoic [22:5n–6], which was significantly decreased (−18%). All PUFA n–3, including alpha-linolenic (15%) from vegetable oils and EPA, DPA, and DHA (6% to 11%) from fish and seafood, were significantly increased in the parents. Conversely, all *trans*-fatty acids, indicators of ruminant fat (palmitelaidic [16:1n–7 *t*], *trans*-7-hexadecenoic [16:1n–9 *t*], *trans*-vaccenic [18:1n–7 *t*], linoelaidic [18:2 *t*], conjugated linolenic acid (CLA)], and partially hydrogenated fat typically found in traditional margarines, commercially prepared fried foods, and savory snacks (elaidic [18:1n–9 *t*], petroselinic [18:1n–10 to 12 *t*]) were significantly decreased (–10% to –18%). Desaturase enzyme activity indices, SCD1 (8%) and D6D (12%) were significantly increased, while SCD2 (–2%) and D5D (–9%) were significantly decreased.

Cardiometabolic risk factors

At the end of the 1-y intervention period, parents in both groups had significant decreases in circulating markers of inflammation (hsCRP, TNF-α, and IL-6) and increases in LDL-cholesterol, P-selectin, sICAM, and thrombomodulin concentrations (**Table 3**). Parents in the SC + EP group had additional significant decreases in insulin concentrations compared with parents in the SC group. No significant changes were observed in BMI, blood pressure, or glucose, TC and HDL-cholesterol, and adipokine concentrations. When both groups of parents were combined (**Figure 2**), significant improvements in TGs (–6%), hsCRP (–21%), TNF-α (–19%), and IL-6 (–19%) concentrations were observed. However, significant unfavorable increases in LDL cholesterol (3%), P-selectin (21%), sICAM (20%), and thrombomodulin (–8%) were also observed.

Correlation between nutrient biomarkers and CMRFs

A heatmap of Spearman correlations between the change in nutrient biomarkers and change in CMRFs over the 1-y intervention period is presented in **Figure 3**.

Carotenoids

The changes in plasma carotenoid concentrations (adjusted by TG concentrations) were generally associated with an improvement in CMRFs, including an inverse association with BMI (lutein), insulin (lutein), TGs (all), TNF-α (zeaxanthin, cryptoxanthin, and β-carotene), and IL-6 (β-carotene), and a positive association with HDL-cholesterol concentrations (lutein and zeaxanthin). An exception was lycopene (found in tomatoes and derived products), which was positively associated with LDL-cholesterol concentrations.

Fat-soluble vitamins

Positive associations were observed between the fat-soluble vitamins and glucose (vitamin K), TC (all), LDL-cholesterol (vitamins A and D), HDL-cholesterol (vitamin A), and TG (vitamins E and K) concentrations. Vitamin E also showed a positive association with inflammatory (IL-6), vascular adhesion (E-selectin, P-selectin, sICAM), and coagulation (thrombomodulin) markers. In contrast, vitamin A was negatively associated with hsCRP and TNF-α concentrations.

RBC fatty acids

Among the SFAs, associations with CMRFs varied by fatty acid type. Total SFAs, lauric [12:0], myristic, and palmitic were positively associated with insulin, TC, TG, hsCRP, IL-6, P-selectin, sICAM, thrombomodulin, and adiponectin concentrations. Longer-chain SFAs (stearic to lignoceric) were generally inversely associated with BMI (arachidic), TC and HDL cholesterol (stearic), hsCRP (arachidic and behenic), IL-6 (arachidic), E-selectin (arachidic), P-selectin (arachidic, behenic, and lignoceric), sICAM (arachidic), and thrombomodulin (arachidic). Leptin concentrations were negatively associated with stearic but positively associated with behenic. The odd-chain fatty acid pentadecanoic (biomarker of dairy fat) was inversely associated with hsCRP.

Among the MUFAs, hypogenic, palmitoleic, and cis-vaccenic, which are synthesized from carbohydrates via the DNL pathway, were positively associated with BMI, TC, TG, and P-selectin concentrations. Conversely, the n–9 fatty acid concentrations were negatively associated with BMI (erucic [22:1n–9] and nervonic), diastolic blood pressure (erucic), glucose (erucic), insulin and leptin (gondoic [20:1n–9], erucic, and nervonic), hsCRP (gondoic, erucic), IL-6 and P-selectin (gondoic, nervonic), and sICAM and thrombomodulin (oleic, gondoic). Total MUFAs, predominantly driven by nervonic were positively associated with adiponectin concentrations.

Among the n–6 class of fatty acids, total PUFA n–6, eicosadienoic, arachidonic, adrenic, and docosapentaenoic were negatively associated with several CMRFs including BMI, glucose, insulin, TC, TGs, IL-6, P-selectin, sICAM, thrombomodulin, and leptin. Interestingly, DHA was positively associated with sICAM, thrombomodulin, and adiponectin. Positive associations were also observed between linoleic and gamma-linolenic and BMI, insulin, TC and LDL-cholesterol, TG, and leptin concentrations.

The plant-derived n–3 PUFAs (alpha-linolenic) were positively associated with TC, TGs, and adiponectin and negatively associated with TNF-α, sICAM, and thrombomodulin. Among the marine-derived PUFAs, DHA and DPA but not EPA were negatively associated with insulin, P-selectin, and leptin.

The majority of the *trans* fatty acids were positively associated with inflammatory, vascular adhesion, and coagulation markers, as well as leptin. Surprisingly, they were negatively associated with blood pressure. CLA was the only *trans* fatty acid that showed a weak but significant positive association with BMI.

Desaturase enzyme indices

SCD1 was positively associated with BMI, TC, and TGs. SCD2 was also positively associated with TC, TGs, and adiponectin but negatively associated with sICAM and thrombomodulin concentrations. D6D was positively associated with HDL cholesterol and negatively with P-selectin. Conversely, D5D was negatively associated with the lipid profile and positively with P-selectin.

TABLE 3 Cardiometabolic risk factors at baseline and end of the 1-y intervention by study group[1]

	SC			SC + EP			*P*[2]		
Cardiometabolic risk factors	**Baseline**[3]	**1-y**[3]	**Mean percent difference**[4]	**Baseline**[3]	**1-y**[3]	**Mean percent difference**[4]	**Group**	**Time**	**Group × time**
BMI, kg/m^2	33.0 (6.2)	32.9 (6.0)	–0.4 [–1.7, 0.9]	31.8 (6.5)	31.6 (6.8)	–0.7 [–2.1, 0.7]	0.176	0.233	0.738
Blood pressure, mmHg									
Diastolic	67.3 (8.8)	66.8 (8.9)	–0.8 [–2.9, 1.3]	66.5 (8.8)	65.6 (8.4)	–1.2 [–3.5, 1.1]	0.158	0.200	0.786
Systolic	115.7 (14.9)	114.9 (13.2)	–0.7 [-2.6, 1.2]	114.6 (16.4)	113.2 (14.2)	–1.1 [–3.3, 1.1]	0.170	0.206	0.779
Glucose metabolism									
Glucose, mg/dL	103.4 (31.7)	103.8 (46.3)	0.3 [–5.7, 6.8]	98.8 (26.6)	101.1(27.3)	2.3 [–1.0, 5.7]	0.348	0.469	0.593
Insulin, μU/mL	19.19 (9.91)	19.23 (10.3)	0.2 [–8.0, 9.1]	17.5 (9.39)	16.5 (9.68)	–5.5 [–12.8, 2.4]	0.030	0.357	0.328
Lipid metabolism, mg/dL									
Total cholesterol	179.2 (39.1)	181.4 (35.2)	1.2 [–1.8, 4.4]	184.0 (39.8)	184.2 (34.2)	0.2 [–2.6, 3.1]	0.472	0.499	0.636
LDL cholesterol	104.0 (35.1)	106.9 (32.1)	2.9 [–1.4, 7.5]	107.0 (32.0)	111.1 (28.9)	3.7 [0.0, 7.6]	0.270	0.024	0.801
HDL cholesterol	48.6 (10.7)	48.4 (11.3)	0.5 [–3.1, 2.2]	48.9 (11.8)	47.4 (10.4)	–3.1 [–5.7, –0.4]	0.781	0.062	0.168
Triglycerides	115.2 (81.9)	108.5 (70.0)	–5.8 [–12.8, 1.8]	113.6 (87.4)	107.7 (73.4)	–4.9 [–12.0, 2.9]	0.776	0.051	0.862
Inflammatory markers									
hsCRP, mg/L	4.06 (20.4)	3.64 (18.9)	–10.3 [–22.0, 3.2]	3.86 (10.2)	2.82 (9.41)	–26.9 [–37.0, –15.2]	0.343	<0.001	0.049
TNF-α, pg/mL	4.82 (1.52)	3.85 (1.44)	–20.1 [–25.4, –14.4]	4.75 (1.71)	4.08 (1.57)	–14.2 [–20.2, –7.8]	0.63	<0.001	0.164
IL-6, pg/mL	1.35 (2.86)	1.12 (1.99)	–16.9 [–28.9, –3.0]	1.17 (1.95)	0.97 (1.27)	–17.6 [–30.0, –3.0]	0.291	0.001	0.946
Vascular adhesion and coagulation markers									
E-selectin, pg/mL	4.12 (4.18)	4.31 (3.92)	3.7 [–9.7, 19.2]	4.19 (6.06)	4.68 (4.41)	12.1 [–5.4, 32.9]	0.564	0.176	0.485
P-selectin, pg/mL	37.4 (24.9)	45.9 (28.4)	23.1 [9.7, 38.2]	36.5 (24.1)	45.0 (26.1)	24.6 [9.6, 41.7]	0.887	<0.001	0.891
sICAM-3, ng/mL	0.51 (0.38)	0.57 (0.29)	15.3 [0.8, 31.9]	0.49 (0.36)	0.64 (0.29)	30.3 [13.8, 49.1]	0.419	<0.001	0.207
Thrombomodulin, ng/mL	1.76 (0.87)	1.83 (0.72)	4.6 [–4.8, 14.8]	1.64 (0.85)	1.84 (0.68)	11.4 [0.0, 24.2]	0.466	0.037	0.382
Adipokines									
Adiponectin, mg/mL	87.3 (48.4)	84.1 (48.5)	–4.0 [–11.0, 3.6]	89.4 (52.1)	84.4 (47.1)	–5.7 [–12.6, 1.7]	0.778	0.069	0.738
Leptin, ng/mL	25.6 (33.0)	24.7 (34.1)	–3.4 [–14.1, 8.7]	18.7 (34.4)	18.5 (47.0)	–1.3 [–14.5, 13.9]	0.015	0.613	0.823

[1]Number of parents with both baseline and 1-y values: BMI (99 in SC and 94 in SC + EP); blood pressure (100 in SC and 94 in SC + EP); glucose metabolism, lipid profile, and hsCRP (103–106 in SC and 97–99 in SC + EP); other inflammatory, vascular adhesion, and coagulation markers and adipokines (98–99 in SC and 92–93 in SC + EP). EP, enhanced program; hsCRP, high-sensitivity C-reactive protein; SC, standard care; sICAM-3, soluble intercellular adhesion molecule 3.
[2]F-tests on fixed effects of study group, time, and group by time interaction from a mixed-effects random intercept linear model.
[3]Values are geometric means (SD); SD was estimated from log-transformed values.
[4]Values are mean % difference [95% CI], calculated from model-based least-square means.

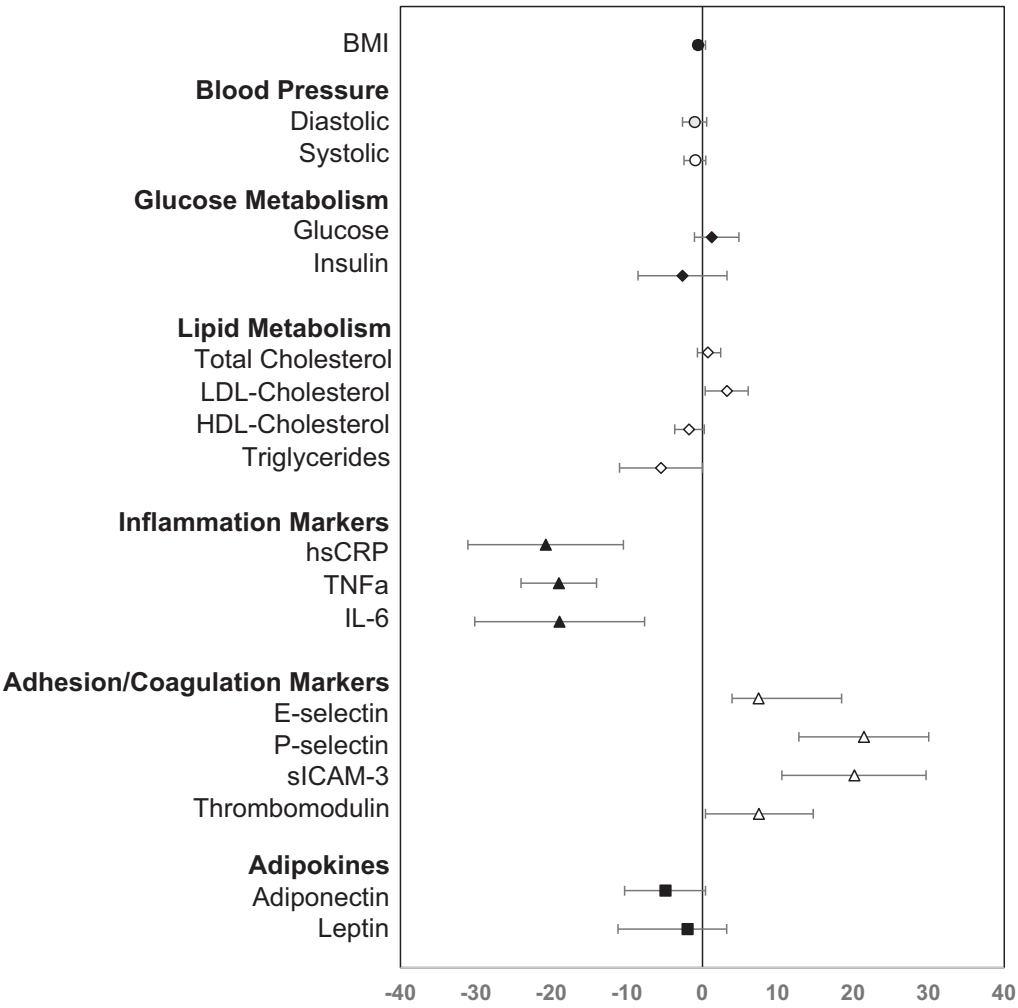

FIGURE 2 Pooled 1-y change in CMRFs. For each individual CMRF, the mean % difference is plotted as the symbol and the 95% CIs displayed as the bars. The mean % difference value and 95% CIs are derived from least-square means calculated from a mixed-effects random intercept model with time (baseline or 1-y) as a fixed effect and a random intercept for subject correlations. A separate model is fitted for each log-transformed outcome. Numbers of parents in the SC and SC + EP groups with both a baseline and 1-y values were as follows: BMI ($n = 193$); blood pressure ($n = 194$); glucose metabolism ($n = 205$); lipid profile ($n = 205$); inflammatory, vascular adhesion, coagulation markers, and adipokines ($n = 192$). CMRF, cardiometabolic risk factor; EP, enhanced program; hsCRP, high-sensitivity C-reactive protein; SC, standard care; sICAM-3, soluble intercellular adhesion molecule 3.

Discussion

To our knowledge, this study is the first to evaluate whether a 1-y family-based childhood weight-management intervention influenced parental nutrient patterns and cardiometabolic health outcomes. Results suggest an improvement in diet quality, as indicated by an increase in biomarkers of fruits and vegetables (carotenoids), dairy (pentadecanoic), vegetable oils (alpha-linolenic), and fish (EPA, DPA, DHA), and a decrease in biomarkers of ruminant and partially hydrogenated fat (*trans* fatty acids). Additionally, there were modest yet significant favorable improvements in 4 (TGs, hsCRP, TNF-α, and IL-6) of the 18 CMRFs measured at the end of the 1-y intervention. These improvements were weakly to moderately correlated with the shifts in nutrient patterns. However, we did not observe a significant reduction in BMI. Furthermore, the intervention did not slow the unfavorable trajectories observed in LDL cholesterol and markers of endothelial dysfunction (P-selectin, sICAM, and thrombomodulin). For the most part, changes in nutrient biomarkers and CMRFs in the parents were independent of the intervention group, suggesting limited added benefit of the enhanced program component. Nonetheless, these results document that SC alone, based on the American Academy of Pediatrics' evidence-based recommendations that target lifestyle behaviors associated with excess body weight in children, can result in beneficial dietary and cardiometabolic health benefits in their parents when implemented within the context of a family-based clinical setting.

There is limited research on change in parental diet quality as part of family-based weight-management interventions (13). This is partly

	BIOMARKERS	BMI	BLOOD PRESSURE		GLUCOSE METABOLISM		LIPID METABOLISM				INFLAMMATION, VASCULAR ADHESION AND COAGULATION							ADIPOKINES	
			DBP	SBP	Glucose	Insulin	TC	LDL-C	HDL-C	TG	CRP	TNFa	IL-6	E-sel	P-sel	s-ICAM	Throm	Adipo	Leptin
CAROTENOIDS	*Lutein*	**−0.15**	0.06	0.09	−0.09	**−0.15**	−0.10	0.14	**0.14**	**−0.60**	−0.11	−0.11	−0.05	−0.01	0.02	0.00	−0.05	0.12	−0.12
	Zeaxanthin	−0.11	0.08	0.11	−0.10	−0.06	−0.09	0.13	**0.19**	**−0.54**	−0.09	**−0.16**	−0.05	−0.04	0.05	−0.01	−0.08	0.13	−0.02
	Cryptoxanthin	−0.04	0.01	0.03	−0.05	−0.04	−0.14	0.07	0.10	**−0.48**	−0.05	**−0.21**	−0.03	0.07	0.05	0.00	(0.03	0.04	−0.07
	Beta–Carotene	−0.11	−0.05	0.04	−0.12	−0.07	**−0.15**	0.06	0.08	**−0.47**	−0.13	**−0.20**	**−0.20**	0.04	0.07	−0.12	−0.13	−0.03	−0.06
	Lycopene	−0.01	0.04	0.07	0.02	0.00	−0.04	**0.21**	0.09	**−0.52**	−0.11	−0.10	−0.10	−0.03	−0.04	−0.05	−0.06	−0.02	−0.04
FAT SOLUBLE VITAMINS	*Vitamin A*	−0.05	0.07	−0.04	−0.07	−0.09	**0.29**	**0.24**	**0.17**	0.13	**−0.15**	**−0.15**	−0.10	0.05	0.12	−0.07	−0.04	−0.07	0.09
	Vitamin D	−0.04	0.04	−0.12	0.04	−0.06	**0.16**	**0.17**	0.08	0.05	0.03	0.05	−0.10	−0.02	0.00	0.07	−0.01	0.03	0.14
	Vitamin E	0.13	0.02	−0.06	0.03	0.09	**0.19**	0.08	0.04	**0.29**	0.08	0.13	**0.15**	**0.16**	**0.18**	**0.23**	**0.20**	0.09	0.02
	Vitamin K	−0.01	0.09	−0.02	**0.19**	0.09	**0.22**	0.12	0.05	**0.28**	−0.07	−0.08	0.05	0.14	0.07	0.01	0.01	0.04	0.15
FATTY ACIDS	*SFA*	0.13	−0.07	−0.01	0.07	**0.19**	0.01	−0.04	−0.03	0.06	0.12	0.03	**0.26**	0.07	**0.24**	**0.21**	**0.28**	−0.13	**0.19**
	12:0	0.05	−0.03	−0.01	−0.09	−0.03	−0.07	−0.08	−0.09	−0.03	−0.13	0.06	**0.26**	0.11	0.07	**0.23**	**0.28**	0.09	−0.01
	14:0	0.06	−0.08	−0.06	−0.09	0.09	**0.14**	0.02	0.10	**0.22**	−0.11	−0.14	0.09	−0.04	0.06	0.05	0.02	0.04	0.13
	15:0	−0.01	−0.13	−0.03	−0.03	−0.03	−0.05	−0.02	0.02	−0.11	**−0.20**	−0.09	−0.08	−0.02	0.07	0.14	0.06	0.12	−0.10
	16:0	0.14	−0.04	0.02	0.03	**0.19**	0.09	0.00	0.03	**0.16**	**0.21**	0.04	**0.23**	0.09	**0.28**	0.09	**0.20**	−0.08	**0.17**
	18:0	−0.05	−0.09	−0.09	0.06	0.03	**−0.14**	−0.06	**−0.15**	−0.12	−0.11	0.07	0.05	0.03	0.02	0.12	0.13	**−0.22**	0.04
	20:0	**−0.14**	0.05	0.01	0.04	−0.13	−0.06	−0.06	0.11	−0.07	**−0.18**	−0.10	**−0.16**	**−0.25**	**−0.26**	**−0.23**	**−0.32**	0.09	−0.04
	22:0	−0.13	0.07	0.09	−0.04	−0.14	−0.14	−0.07	−0.04	**−0.16**	**−0.14**	−0.02	−0.13	−0.09	**−0.25**	0.11	−0.04	**0.17**	−0.11
	24:0	−0.06	0.13	0.05	0.00	−0.07	−0.13	−0.10	−0.08	−0.09	0.01	0.10	0.04	−0.04	**−0.20**	**0.27**	0.13	0.13	−0.06
	MUFA	−0.03	0.00	−0.02	−0.02	−0.10	0.01	−0.05	0.05	0.07	**−0.17**	−0.14	−0.05	−0.07	−0.08	**−0.18**	**−0.22**	**0.16**	−0.04
	16:1n−7	**0.18**	0.07	0.04	0.01	0.11	**0.19**	0.04	0.03	**0.35**	0.02	**−0.15**	0.04	0.09	−0.02	−0.01	0.04	0.01	0.07
	16:1n−9	0.05	−0.01	−0.02	0.00	−0.09	0.06	−0.04	0.09	**0.15**	−0.03	**−0.15**	0.00	−0.05	**−0.16**	**−0.19**	**−0.21**	0.10	0.02
	18:1n−7	−0.05	−0.01	0.04	−0.06	−0.07	−0.12	**−0.16**	0.06	−0.07	−0.07	−0.04	0.04	**−0.16**	**0.15**	−0.04	−0.08	0.02	0.05
	18:1n−9	−0.01	0.02	−0.04	0.00	−0.05	0.08	0.04	−0.04	0.13	−0.12	−0.12	−0.07	0.07	−0.12	**−0.19**	**−0.20**	0.12	−0.08
	20:1n−9	−0.13	−0.09	−0.07	−0.02	**−0.22**	−0.11	−0.10	0.13	−0.08	**−0.18**	−0.10	**−0.16**	−0.11	**−0.30**	**−0.25**	**−0.32**	0.11	**−0.15**
	22:1n−9	**−0.22**	**−0.15**	−0.08	**−0.16**	**−0.21**	−0.11	−0.07	−0.03	−0.09	**−0.17**	0.08	−0.04	−0.13	−0.10	−0.01	−0.05	0.10	**−0.21**
	24:1n−9	**−0.15**	0.04	0.03	−0.02	**−0.23**	−0.10	−0.07	0.01	−0.11	−0.11	0.03	**−0.22**	**−0.19**	**−0.33**	−0.10	**−0.23**	**0.21**	**−0.18**
	PUFA n−6	−0.11	0.04	0.01	−0.01	−0.11	0.03	0.09	0.07	−0.07	−0.08	−0.05	**−0.27**	−0.03	**−0.22**	−0.11	**−0.20**	0.06	**−0.15**
	18:2n−6	0.12	0.08	0.01	0.07	**0.15**	**0.21**	**0.16**	0.09	0.07	0.02	−0.11	0.03	0.00	0.07	0.06	0.06	0.08	**0.20**
	18:3n−6	**0.16**	−0.06	0.02	−0.02	**0.22**	**0.14**	−0.01	0.04	**0.34**	−0.10	**−0.15**	0.07	0.09	0.00	−0.07	−0.06	0.06	**0.23**
	20:2n−6	**−0.17**	−0.01	−0.01	−0.13	**−0.21**	0.00	−0.01	**0.16**	−0.04	−0.02	−0.10	**−0.16**	−0.10	**−0.28**	−0.11	−0.22	0.14	−0.07
	20:3n−6	0.02	0.07	−0.02	−0.10	−0.10	**0.16**	**0.15**	**0.22**	0.06	0.04	−0.06	−0.04	−0.05	**−0.28**	−0.03	−0.10	0.11	−0.01
	20:4n−6	**−0.18**	0.04	0.03	−0.02	**−0.17**	**−0.21**	−0.05	−0.12	**−0.29**	−0.11	0.08	**−0.29**	−0.05	−0.14	−0.13	**−0.17**	−0.06	**−0.26**
	22:2n−6	0.00	−0.04	−0.06	−0.01	−0.11	−0.04	−0.03	0.02	−0.04	0.08	0.05	−0.08	0.03	−0.04	**0.30**	**0.19**	**0.16**	0.00
	22:4n−6	**−0.21**	0.04	0.06	0.01	**−0.17**	−0.08	−0.04	0.01	−0.06	−0.08	−0.02	**−0.30**	−0.10	**−0.33**	**−0.15**	**−0.30**	0.08	**−0.26**
	22:5n−6	−0.05	−0.04	0.08	**−0.15**	−0.01	0.01	−0.02	0.07	0.03	0.04	−0.12	−0.08	0.10	−0.14	**−0.16**	−0.14	−0.10	−0.07
	PUFA n−3	−0.02	0.09	0.02	−0.05	−0.12	0.04	0.08	−0.06	−0.02	0.10	0.07	**−0.17**	−0.05	**−0.15**	−0.07	−0.12	−0.04	−0.10
	18:3n−3	0.07	0.05	−0.01	0.03	0.03	**0.16**	0.05	0.10	**0.21**	−0.09	**−0.17**	−0.01	−0.08	−0.12	**−0.16**	**−0.18**	**0.17**	0.13
	20:5n−3	0.09	0.09	0.04	0.10	0.07	0.09	0.13	0.01	0.01	0.07	−0.03	−0.13	−0.04	0.06	−0.06	−0.08	−0.05	0.04
	22:5n−3	−0.02	0.06	−0.02	−0.02	−0.13	−0.07	0.03	−0.04	−0.12	0.04	0.10	**−0.15**	−0.03	−0.12	0.06	−0.01	−0.02	−0.05
	22:6n−3	−0.06	0.11	0.03	−0.07	**−0.15**	0.02	0.06	−0.11	−0.04	0.09	0.10	−0.14	−0.02	**−0.16**	−0.10	−0.14	−0.05	**−0.16**
	Trans	0.06	**−0.21**	−0.07	0.02	0.10	0.01	0.00	0.05	0.02	−0.03	0.06	**0.18**	**0.15**	**0.24**	0.06	0.14	0.09	0.14
	16:1n−7t	0.02	**−0.19**	**−0.16**	0.08	0.00	−0.06	−0.04	0.04	−0.12	−0.06	−0.10	−0.01	0.02	**0.21**	−0.12	−0.11	−0.14	−0.03
	16:1n−9t	0.04	−0.13	−0.08	−0.04	0.00	0.04	−0.04	0.14	0.03	−0.07	−0.05	−0.04	−0.03	0.11	**−0.17**	**−0.16**	0.10	0.08
	18:1n−7t	0.02	0.04	0.05	−0.08	0.08	0.02	0.03	−0.01	−0.05	0.00	0.08	**0.22**	**0.19**	−0.06	**0.23**	**0.21**	**0.16**	0.00
	18:1n−9t	−0.03	**−0.20**	−0.08	0.00	0.09	0.00	0.01	0.11	0.01	−0.06	0.08	0.06	0.08	**0.23**	−0.06	0.00	−0.01	**0.16**
	18:1n−10 to12t	0.07	**−0.21**	−0.03	0.03	0.06	0.07	0.04	0.06	0.08	−0.03	0.03	0.12	**0.15**	**0.23**	−0.07	0.07	0.05	0.07
	18:2t	0.00	−0.08	0.00	−0.03	0.02	−0.05	−0.04	0.01	−0.06	−0.05	−0.03	0.14	0.07	**0.23**	0.14	**0.21**	0.09	0.11
	18:2CLA	**0.16**	−0.09	−0.01	0.05	**0.15**	0.11	0.05	0.08	**0.18**	0.10	0.07	0.14	0.01	0.06	−0.04	−0.01	0.11	0.06

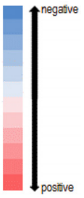

FIGURE 3 Heatmap of the correlation between nutrient biomarkers, desaturase enzyme activities, and CMRFs. The correlation between 1-y change in CMRFs with 1-y change in nutrient biomarkers for each outcome pair was estimated using Spearman ρ correlation, adjusted for sex, age, group, and baseline CMRFs (and additionally adjusted for baseline BMI for correlations not with BMI). Blue indicates a negative association, whereas redindicates a positive association, with the darkness of each color corresponding to the magnitude of the "*r*" value, with significant values ($P < 0.05$) in bold. The number of parents included ranged from 193 to 205. Adipo, adiponectin; CLA, conjugated linolenic acid; CMRF, cardiometabolic risk factor; CRP, C-reactive protein; DBP, diastolic blood pressure; E-sel, E-selectin; HDL-C, HDL cholesterol; LDL-C, LDL cholesterol; P-sel, P-selectin; SBP, systolic blood pressure; sICAM, soluble intercellular adhesion molecule; TC, total cholesterol; TG, triglyceride; Throm, thrombomodulin.

due to the challenges and inherent limitations associated with capturing dietary intake using subjective assessment tools (24-h recall, food-frequency questionnaires, food diaries) (55). To overcome this, we chose to objectively measure selected nutrients that reflected some of the dietary components of the intervention. We observed higher β-carotene and lycopene concentrations in the parents at the end of the intervention. This suggests increased consumption of foods such as carrots, tomato-based dishes, and fresh or canned fruits (apples, cherries, oranges). Of note, assessment of the dietary intake of the children in this study highlighted a "pizza and pasta"–based pattern, which was associated with their parents/guardian being born in the mainland United States and having a higher educational level (56). This highlights the important of parental acculturation status in influencing dietary behaviors of the family. The lower concentrations of the predominantly diet-derived *trans* fatty acids observed suggest that the parents in our study adhered to the lower fried foods/savory snack recommendations. Also noted were lower proportions of total SFAs and higher production of fatty acids in the DNL pathway. The lower SFA intake is most likely due to a combination of a lower intake of palmitic, which is the major dietary SFA in the diet, as well as increased endogenous desaturation to palmitoleic and conversion to downstream MUFA metabolites, as supported by the observation of higher SCD1 and lower SCD2 activities. The higher PUFA n−6 downstream metabolites observed reflect endogenous synthesis from linoleic via D6D. Additionally, the increase in diet-derived long-chain PUFA n−3 intake from both plant (alpha-linolenic) and marine (EPA, DPA, DHA) sources could account for the lower D5D activities, given the competition between PUFA n−6 and n−3 for D5D (47).

While it is difficult to contextualize our nutrient data given the dearth of comparable research, we identified a few studies that assessed dietary components using self-administered tools in parents participating in family-based intervention programs. Two studies, the High 5 for Preschool Kids (H5-KIDS) program, a home-based intervention to teach parents how to ensure a positive fruit-vegetable environment for their preschool child (25), and the Stoplight/Traffic-light Diet Treatment (20) both reported increases in fruit-vegetable servings in participating parents, which, in the latter study, was at the expense of high-fat/high-sugar foods. Among the 3 studies that reported dietary total fat intake of participating parents, 1 study (28) achieved significant reductions in total fat intake and to a smaller extent for sugar and complex carbohydrate intake following family dietary coaching to improve nutritional intake and weight control, while the other (19) observed a significant decrease in total fat and sodium intake, but only in non-Hispanic and not in Mexican-American families who participated in a family-based cardiovascular disease risk-reduction intervention. The third study (57) explored the efficacy of a 12-wk, culturally specific, obesity-prevention program in low-income, inner-city African-American girls and their mothers and showed significant differences between the treatment and control mothers for daily SFA intake and percentage of calories from fat. These data, including the present results, suggest that parent involvement in family-based interventions with a specific dietary component, whether indirect (targeting their child's dietary intake) or direct (targeting both parent and child), can result in modest shifts in their dietary behaviors.

An unexpected finding in this study was the decrease in concentrations of fat-soluble vitamins, most notably for vitamins D and E. These vitamins are transported in the circulation via TG-rich particles; however, similar results were still observed after correcting for TG concentrations. It is possible that an overall decrease in intake of fortified foods, such as cereals, and fried foods prepared with vegetable oils, major dietary sources of vitamins D and E, respectively, could account for this observation. Alternatively, evidence also suggests that, in the presence of obesity, there is a tendency for higher incorporation and storage of fat-soluble vitamins, especially in adipose tissue, which results in lower circulating concentrations (58).

Most family-based studies are designed to assess whether parent involvement enhanced the effectiveness of interventions that aimed to change their child's weight, with a few studies (20, 26, 29, 34, 59) also targeting parental weight. However, these latter studies had mixed results. Some studies (20, 26) that targeted weight loss as an outcome in both parents and children reported better success in parents. However, another family-based exploratory community study in low-income Latino mothers and daughters did not show any significant differences in BMI in mother–daughter dyads in the experimental versus control group, after adjusting for baseline BMI as a covariate (34). Among school- and community-based childhood weight-management programs, 1 study observed a spillover intervention effect on parents, resulting in a significant decrease in BMI (59), while the other study did not (29). We also did not observe a significant reduction in parental BMI, although there was a downward trend after adjustment for baseline variables. Nevertheless, we observed improvements in several obesity-related CMRFs, notably systemic inflammation markers and insulin and TG concentrations. Additionally, these favorable changes in CMRFs were associated with the changes in nutrient profiles. Whereas inverse associations between carotenoids and CMRFs (58) and positive associations between fatty acids, including *trans* fatty acids, and inflammation/endothelial dysfunction (60–62) have been previously documented in overweight and obese adults, our findings provide preliminary evidence that the shifts toward healthier eating patterns can extend beyond changes in BMI and improve cardiometabolic health outcomes, within a family-based childhood obesity intervention.

Excess body weight has also been associated with lower adiponectin and higher leptin concentrations (63). The absolute concentrations of these adipokines were not significantly altered by the intervention, and this is most likely due to the lack of an effect on BMI, as their concentrations tend to correlate with fat mass. However, the positive associations observed between both adipokines and several of the endogenously synthesized SFAs, MUFAs, and PUFA n–6 fatty acids at the end of the 1-y intervention suggest a potential modulatory effect that warrants further investigation (64).

Of note, there were unfavorable increases in LDL-cholesterol concentrations at the end of the study. This could reflect increased transfer of TGs from VLDL to LDL, as supported by the concomitant lowering of TG concentrations observed. Also observed were increases in concentrations of P-selectin, sICAM, and thrombomodulin, suggestive of endothelial injury (65), which may already be present in this group of parents with overweight and obesity, the trajectories of which the dietary components targeted in our intervention were unable to slow or reverse.

Strengths of this study include the randomized design, drawing on the social-ecological framework to develop intervention components and the principles of social cognitive theory, and social marketing to address the interaction of behavioral, environmental, and personal factors; collaborative goal-setting to empower families; and an expanded dataset of overweight- and obesity-related CMRF variables, in an underserved, high-obesity risk group of largely Hispanic mothers who participated in their child's weight-management intervention. Given the extremely low participation of fathers in our study, we were limited in our ability to analyze an intervention effect on paternal diet. Second, the clinical trial was not designed to address parental adherence to the lifestyle intervention per se. Consequently, the parental dietary and physical activity assessments were limited. We chose to utilize an objective biomarker approach, focusing on selected nutrients, since self-report dietary intake, especially in populations with overweight and obesity, has been associated with under- and overreporting of certain food groups (66, 67). However, there are a limited number of validated nutrients of dietary intake and none that capture total energy intake/balance, sugar-sweetened beverage intake, or quantity of the food consumed. Third, the possibility cannot be ruled out that the changes observed in CMRFs could potentially be mediated by increased physical activity levels of the parents and not solely related to modification of dietary behaviors. Finally, the majority of the changes in nutrient concentrations and CMRFs were observed in both groups of parents, which was contrary to our original hypothesis, and suggests limited added benefit of the enhanced module. While this could be because the dietary guidance/tools were provided to all participating families as part of SC and reinforced during the quarterly visits by the highly specialized study pediatricians and research staff, it is possible that other components, such as intensity of contacts, adherence, and/or resource sharing between groups, may have contributed to these observations.

In conclusion, this study documents a beneficial outcome of a family-based childhood weight-management intervention delivered within a primary care setting on parental nutrient patterns, which were associated with favorable changes in selected CMRFs, despite non-significant changes in BMI. These findings have significant public health implications since improving diet quality and health outcomes in a parent with overweight or obesity using the same guidelines that targeted their child could potentially result in a major cost–benefit of family-based interventions, which merits further investigation.

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Data Availability

Data described in the manuscript, code book, and analytic code will be made available upon request. Data sharing will require a signed agreement that addresses expenses for data transfer and protects participant confidentiality by exchanging de-identified data in protected formats.

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