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Human Kinetics
ORIGINAL RESEARCH

Participation in a Social-Support Physical Activity Intervention Modestly Improves Lipoprotein Cholesterol Distribution Among Postpartum Sedentary Hispanic Women

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***Background*:** The effects of moderate intensity walking on lipoprotein remodeling in postpartum Hispanic women are unknown. ***Methods*:** Sedentary postpartum Hispanic women (28.2 ± 5.6 y; BMI = 29.3 ± 3.3 kg/m^2) participating in a social support physical activity (PA) intervention, were randomly assigned to a 12-month walking program (walkers; n = 22; target 150 min/wk, moderate intensity) or a control group (nonwalkers; n = 22). Fasting lipids and cholesterol distribution within low-density lipoprotein (LDL) and high-density lipoprotein (HDL) particles were measured at baseline (BL), 6 months, and 12 months. ***Results*:** Walkers had an 11% increase and nonwalkers a 7% decrease in HDL cholesterol from 6 to 12 months (P = .0367) without an effect on LDL cholesterol. Whereas nonwalkers had virtually no change in mean LDL particle size, walkers had a borderline reduction in LDL size from BL (268.7 ± 4.1 Å) to 6 months (266.9 ± 4.9 Å), followed by a significant increase in size by 12 months (269.7 ± 4.1 Å; P = .011). The proportion of cholesterol in large LDL particles decreased by 15% from BL to 6 months, but subsequently increased 25% by 12 months among walkers; changes among nonwalkers were smaller and in opposite direction (4% and –3%, respectively; P = .0004). ***Conclusions*:** Participation in the social-support PA intervention resulted in slightly increased HDL cholesterol concentrations and a modest and beneficial shift toward larger, less atherogenic LDL particles.

***Keywords*:** HDL particles, LDL particles, randomized controlled trial, walking

Among Hispanics, increased body weight has been associated with presence of the metabolic syndrome, hypertension, insulin resistance, diabetes, hyperlipidemias, and cardiovascular disease (CVD).[1–3] In particular, Mexican Americans have been reported to have greater prevalence of metabolic syndrome components than non-Hispanic whites and blacks.[4,5] However, there is limited information regarding the presence of other atherogenic factors, such as smaller, denser low-density lipoprotein (LDL), in this population.

Excess weight gain during pregnancy and failure to lose weight postpartum have been associated with long-term obesity and the risk of developing dyslipidemia, diabetes, hypertension and CVD.[6] Postpartum Hispanic women are at particular risk due to higher prepregnancy obesity rates[7] and the presence of many contributors to excessive weight retention after childbirth (eg, role changes, cultural norms that promote sedentary behavior and overeating, living in obesogenic environments, postpartum depression, negative body image).[8] Specific information on how postpartum weight retention affects metabolic biomarkers among Hispanic women is scarce.

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Late pregnancy is characterized by complex physiologic changes including alterations in lipid metabolism. These changes include an increase in circulating triglycerides[9,10] and an accumulation of triglycerides in high-density lipoprotein (HDL) particles,[11] resulting in the formation of small, dense LDL and large buoyant HDL particles.[9,10] Lipoprotein changes are partially reverted at 4 to 6 weeks postpartum, with further improvements after the lactation period.[9,12]

The role of physical activity (PA) in modulating lipoprotein changes in the postpartum period has not been thoroughly studied, and information related to changes in different lipoprotein subfractions with PA is lacking. The current study was conducted to examine cardiometabolic disease risk factors, particularly those associated with an unfavorable remodeling of the lipoprotein profile, in a subsample of women from the efficacy of *Madres para la Salud (*Mothers for Health; *Madres)* study, a theory-driven social support program to promote moderate-intensity PA, to reduce body fat in postpartum Hispanic women. It was hypothesized that enhancing social support from participation in *Madres para la Salud* would increase moderate-intensity PA and result in beneficial changes associated with a less atherogenic lipoprotein profile.

Methods

Participants

Study participants were enrolled in *Madres para la Salud* (Mothers for Health), a community-based social support intervention to promote PA in Hispanic postpartum women.[13] Participants were habitually sedentary Hispanic young women (18–40 years), 6 weeks to less than 6 months postpartum, and physically able to participate in moderate intensity walking recruited from the Phoenix metropolitan area. Exclusion criteria were a) participation in regular, strenuous PA (> 150 minutes/week of moderate PA); b)

severe musculoskeletal or cardiorespiratory problems that would preclude PA; c) pregnancy or planning to become pregnant within 12 months; d) current use of antidepressants, anticoagulants, oral steroids, nonsteroidal anti-inflammatory drugs, or hypolipidemic medications; e) infectious illness, chronic systemic inflammatory diseases (eg, rheumatoid arthritis), or other acute illness that may affect systemic inflammation at baseline; f) BMI less than 25 or greater than 35 kg/m^2; and h) presence of osteoporosis at baseline (bone mineral density > 2.5 SD below the average).

Study Design

Madres para la Salud (Mothers for Health) was a 12-month randomized controlled social support PA intervention for sedentary Hispanic postpartum women designed to increase moderate intensity walking. Detailed study design and intervention outcomes have been reported elsewhere.[13–15] The study was approved by Arizona State University's Institutional Review Board and the Maricopa Integrated Health System Human Subjects Review Board. All participants provided written consent to participate.

Following informed consent, participants were randomly assigned to a 12-month social support PA intervention (*walkers*) or an attention-control group (*nonwalkers*) using a SAS Proc Plan to generate the randomization schedule. Because recruitment and the intervention were done in waves or cohorts, participants were randomized in blocks using a seed to generate a randomization list for each wave or cohort with equal numbers for the intervention and the control group. Because of cost, a subsample of 44 women randomly selected by using a random number generator, were invited to complete additional data collection procedures for the assessment of cardiometabolic disease risk factors (see below) and were included in the current analysis. Data were collected at baseline, 6 and 12 months post randomization. These included measures of cardiometabolic disease risk factors (anthropometric measurements, blood pressure, and biomarkers of lipoprotein and glucose metabolism), dietary intake, and physical activity. Of the 44 women selected for this analysis, 33 and 31 participants completed 6-month and 12-month data collection procedures, respectively.

Participants assigned to the PA intervention (*walkers*) were allocated to *Promotora* (community health worker)-led walking groups that met at select community-based locations convenient to group members' area of residence. The first 3 months of the intervention consisted of 12 weekly support sessions immediately followed by walking sessions through which *Promotoras* provided training to ensure proper stretching and cooling down procedures, and to ensure participants sustained a walking pace of moderate intensity; women in the intervention group completed walking records of the days and times of PA. After the 12 support sessions concluded, the *Promotoras* held weekly 30-minute walking sessions with group members for the remainder of the study, after which they assessed adherence to the walking dose, and checked participants' walking records. Participants in the control group received weekly newsletters with information unrelated to PA, such as breast-feeding, sun protection, and scripted follow-up phone calls throughout the duration of the study. Neither of the study groups received information about diet or dietary change.

The prescribed walking dose was consistent with the 2008 Physical Activity Guidelines for Adults, which recommend that adults engage in at least 150 minutes/week of moderate-intensity PA.[16] Participants in the PA intervention group were instructed to complete a minimum of 30 minutes/day of brisk walking for at least 5 days of the week, with the option of completing the prescribed dose by accumulating bouts of at least 10 minutes each.

Measures

At each data collection time point, participants arrived to the study site after a 12-hour overnight fast. Upon arrival, height and weight, and waist circumference were measured in triplicate following standard procedures.[13] Body composition (percent body fat) was measured using a portable 4-terminal bioelectric impedance (BIA) system (Tanita Corporation of America, Inc., Arlington Heights, IL). After a 5-minute rest, blood pressure (BP) was measured 3 times on the left arm using an Omron IntelliSense HEM-907XL automated blood pressure monitor (Omron Healthcare, Inc., Bannockburn, IL). Fasting blood was collected from the antecubital vein into EDTA- or EDTA/sodium fluoride-containing evacuated tubes for the measurement of biomarkers and glucose, respectively. Plasma was separated by centrifugation at 1100 × g at 4°C for 20 minutes and stored at –80°C until analysis.

Biomarkers of Cardiometabolic Disease Risk

Plasma glucose and lipids (total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides) were measured using an automated chemistry analyzer (Cobas C111; Roche Diagnostics, Indianapolis, IN). Plasma insulin was measured using Human Insulin Specific RIA kit (Millipore, Billerica, MA).

The cholesterol distribution in LDL and HDL subfractions and mean LDL particle diameter were determined by the Lipoprint/Lipoware system (Quantimetrix Co., Redondo Beach, CA).[17] The relative percentage of cholesterol in large and small LDL particles was calculated by adding the proportion of cholesterol in fractions LDL_1 plus LDL_2, and LDL_3 through LDL_7, respectively. The relative percentage of cholesterol in large, intermediate, and small HDL particles was calculated by adding the proportion of HDL cholesterol in fractions HDL_1 through HDL_3, HDL_4 through HDL_6, and HDL_7 through HDL_{10}, respectively.

The number of metabolic syndrome components was assessed using ATP III criteria[18] based on the presence of any of the following components: 1) abdominal obesity defined as waist circumference ≥ 102 cm in men and ≥ 88 cm in women, 2) plasma triglycerides ≥ 150 mg/dL, 3) HDL-cholesterol ≤ 40 mg/dL in men and ≤ 50 mg/dL in women, 4) hypertension defined as blood pressure ≥ 130/85 mm Hg, and 5) fasting plasma glucose ≥ 100mg/dL.

Physical Activity Assessment

Participants completed the Stanford Brief Activity Survey (SBAS),[19] and were asked to wear an Omron HJ-720ITC pedometer (Omron Healthcare, Inc., Bannockburn, IL) for 7 consecutive days immediately after each data collection point. Pedometer-generated data included total steps/day and moderate-intensity (aerobic) walking in min/day, corresponding to the amount of time spent walking at a cadence associated with moderate-intensity exercise (> 100 steps/min).

Diet Assessment

Dietary intake data were obtained by trained bilingual staff using 3 unannounced 24-hour recalls that included a weekend day at each time point, following the 5-step multiple pass method.[20] Dietary data were analyzed using the Nutrition Data System for Research (NDSR, version 2009; Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN).

Statistical Analysis

Statistical analyses were carried out using the Statistical Program for the Social Sciences (SPSS) 17.0 for Windows (SPSS, Inc., Evanston, IL), SAS version 9.3 (SAS Institute, Cary, NC), and R version 3.1.0 (R Core Team). Before the analyses, descriptive statistics and graphs were used to summarize variable distributions. Data are presented in text and tables as mean ± standard deviation (SD). The general approach we used for modeling changes in waist circumference, metabolic markers, blood lipids, diet composition, and PA followed a 2 × 3 Group (Intervention vs. Control) × Time (repeated measures taken at baseline, 6 months, and 12 months) analysis of variance (ANOVA) framework. Tests of overall main effects and the Group × Time interaction were followed by planned contrasts among cell means, testing 1) Group differences at each time point and 2) Group differences in the degree of change a) from baseline to 6 months, b) from 6 months to 12 months, and c) from baseline to 12 months. Statistical significance was considered at $\alpha < 0.05$.

Results

Baseline Participant Characteristics

Most participants were either married (46%) or living as married (43%; Table 1). Most participants had completed at least high school education (64%); 36% of participants had only completed elementary or middle school. Yearly household income was below $20,000 for 73% of participants, and most participants were unemployed (66%) or had never been employed (16%). Mean time in the U.S. was 12.0 ± 8.0 years; 38 participants (86%) were foreign-born.

Table 1 Sociodemographic Characteristics of Study Participants at Baseline (n = 44)

Characteristics	n (%)
Marital status	
Single	5 (11.4)
Married	20 (45.5)
Living with a significant other	19 (43.2)
Household income	
< $10,000	12 (27.3)
$10,000–$20,000	20 (45.5)
$20,000–$30,000	5 (11.4)
> $30,000	6 (13.6)
Employment status	
Never employed	7 (15.9)
Unemployed	29 (65.9)
Part-time work	6 (13.6)
Full-time work	2 (4.5)
Education (highest level completed)	
Elementary school	8 (18.2)
Middle school	8 (18.2)
High school	27 (61.4)
College	1 (2.3)
Number of children at home (mean ± SD)	3.3 ± 1.6
1	3 (6.8)
2	15 (34.1)
3	9 (20.5)
4	7 (15.9)
5 or more	10 (22.7)
Time in the U.S. (mean ± SD)	12.0 ± 8.0
Place of birth	
U.S.	6 (14)
Outside the U.S.	38 (86)

Mean age of study participants was 28.2 ± 5.6 years (Table 2). The mean BMI of 29.3 ± 3.3 kg/m^2 was in the overweight range, and participants had a mean 38.2 ± 4.7% of body fat. Mean values for BP, fasting plasma glucose, and fasting triglycerides were within desirable values. Systolic and diastolic BPs were significantly higher in the intervention group than in the control group (P-values = 0.002 and 0.005, respectively). Participants had elevated waist circumference (99.8 ± 9.0 cm) and low HDL-cholesterol concentrations (43 ± 9 mg/dL). Per National Cholesterol Education Program Adult Treatment Panel (NCEP ATP) III criteria,[21] participants had optimal and near-optimal concentrations of total- and LDL-cholesterol at 164 ± 37 mg/dL, and 106 ± 33 mg/dL, respectively, with levels on both total- and LDL-cholesterol being higher in the intervention group than in the control group (P-values = 0.049 and 0.045, respectively).

On average, participants were 12.1 ± 7.1 weeks postpartum and had 2.8 ± 1.2 births. There was no significant difference in weeks postpartum at baseline between intervention (12.9 ± 7.2 weeks) and control groups (11.4 ± 7.0 weeks). There were no group differences in baseline feeding methods based on responses to a 5-category measure (breastfeeding only, breastfeeding more than bottle feeding, breastfeeding and bottle feeding about equally, bottle feeding more than breastfeeding, bottle feeding only; n = 43, P = .69).

Physical Activity and Dietary Intake

Physical activity was generally low among all participants throughout the duration of the study. At baseline, participants performed 4.5 ± 7.1 min/day of aerobic walking time and 5575 ± 2373 steps/day (Table 2). As assessed by the SBAS, the level of PA was classified as inactive, light, and moderate for 52.3%, 31.8%, and 15.9% of participants, respectively.

As objectively assessed by use of pedometers, there was a significant increase in total step counts over time for both groups ($P_{time} < 0.001$; Table 3). From baseline to 6 months there was an increase in mean step counts of 2171 steps/day (38%) among walkers and 1845 steps/day (34%) among nonwalkers. This was followed by a decrease in mean step counts from 6 to 12 months of 1238 steps/day (16%) among walkers and 1446 steps/day (25%) for nonwalkers. Thus, the net increase in step counts from baseline to 12 months was small. Changes in aerobic walking significantly differed over time between groups ($P_{time \times group} = 0.001$); walkers achieved more aerobic steps than nonwalkers at 6 and 12 months (P-values < 0.01). Subjective assessment of PA indicated significantly different changes in SBAS score over time between groups ($P_{time \times group} = 0.003$). SBAS score was significantly higher for walkers than nonwalkers at 6 and 12 months (P-values < 0.01).

There were no significant group differences or group x time interactions on total energy intake per day, or the percentages of energy from fats, total carbohydrates, sugars, and protein, and grams of fiber consumed (Table 3). There was a main effect of time on percent of energy from protein (an increase from baseline to 6 months, followed by a decrease, P = .045) and on fiber consumption (monotonic decrease from baseline to 12 months, P = .006).

Cardiometabolic Disease Risk Factors

There were no significant differences in weight, waist circumference, or body fat percentage over time between walkers and nonwalkers (Table 4). Change in systolic BP over time differed significantly

Table 2 Physiologic Characteristics, Parity, Child Feeding Method, and Physical Activity Levels, of Study Participants at Baseline

	Mean ± SD		
Characteristics	**All (n = 44)**	**Walkers (n = 22)**	**Nonwalkers (n = 22)**
Age (years)	28.2 ± 5.6	28.7 ± 5.4	27.6 ± 5.8
BMI (kg/m^2)	29.3 ± 3.3	29.7 ± 2.6	28.9 ± 3.8
Body fat (%)	38.2 ± 4.7	38.9 ± 3.8	37.5 ± 5.5
Waist circumference (cm)	99.8 ± 9.0	98.7 ± 6.9	98.9 ± 10.9
Systolic blood pressure (mm Hg)**	113 ± 11	118 ± 13	108 ± 6
Diastolic blood pressure (mm Hg)**	70 ± 10	74 ± 11	66 ± 6
Glucose (mg/dL)	90 ± 10	91 ± 11	89 ± 8
Total cholesterol (mg/dL)*	164 ± 37	175 ± 34	153 ± 38
LDL-cholesterol (mg/dL)*	106 ± 33	116 ± 29	96 ± 35
HDL-cholesterol (mg/dL)	43 ± 9	42 ± 8	44± 11
Triglycerides (mg/dL)	117 ± 64	131 ± 63	102 ± 63
Weeks postpartum	12.1 ± 7.1	12.9 ± 7.2	11.4 ± 7.0
Parity	2.8 ± 1.2	2.6 ± 1.2	3.0 ± 1.2
Number of children at home (mean ± SD)	3.3 ± 1.6	3.3 ± 1.8	3.3 ± 1.3
Pedometer-measured physical activity			
Aerobic walking time (min/day)	4.5 ± 7.1	4.8 ± 6.8	4.3 ± 7.6
Total steps (per day)	5575 ± 2373	5778 ± 1745	5373 ± 2899
	n (%)		
Stanford Brief Activity Survey category	**All (n = 44)**	**Walkers (n = 22)**	**Nonwalkers (n = 22)**
Inactive	23 (52.3)	10 (45.5)	13 (59.1)
Light	14 (31.8)	9 (40.9)	5 (22.7)
Moderate activity	7 (15.9)	3 (13.6)	4 (18.2)
Hard/very hard activity	0 (0.0)	0 (0.0)	0 (0.0)
Child feeding method (n = 43)			
Breastfeeding only	6 (14.0)	3 (13.6)	3 (14.3)
Breastfeeding > bottle feeding	10 (23.3)	4 (18.2)	6 (28.6)
Breastfeeding = bottle feeding	6 (14.0)	2 (9.1)	4 (19.0)
Bottle feeding > breastfeeding	5 (11.6)	3 (13.6)	2 (9.5)
Bottle feeding only	16 (37.2)	10 (45.4)	6 (28.6)

* $P < 0.05$; ** $P < .01$ for comparisons of means for walkers vs. nonwalkers at baseline.

between groups ($P_{time \times group} = 0.002$); walkers had higher systolic BP than nonwalkers at baseline and 6 months postrandomization (P-values < 0.05). Linear change (decrease) in systolic BP from baseline to 12 months postrandomization was significantly different between groups (6% reduction for walkers and 1% increase for nonwalkers; $P < .01$), and while systolic BP in walkers showed a modest decrease from 6 months to 12 months, it increased in nonwalkers over the same time. Walkers had higher diastolic BP than nonwalkers at baseline and 6 months postrandomization, but change in diastolic BP over time did not differ significantly between groups.

Mean fasting glucose concentrations remained within normal ranges throughout the study and did not significantly differ in concentration or change over time between groups (Table 4). There were no significant between-group differences in levels or change in insulin. However, insulin concentrations were consistently elevated throughout the study for both groups.

There were no significant changes in total- or LDL-cholesterol concentrations throughout the intervention (Table 4). Participants in the walking group had significantly more total and LDL-cholesterol concentrations at all 3 time points ($P_{group} < 0.01$), however all values were within near optimal concentrations. Although HDL-cholesterol concentrations remained below desirable concentrations (<50 mg/dL for women)[21] for both groups throughout the study, walkers had an 11% increase and nonwalkers a 7% decrease in HDL cholesterol from 6 to 12 months ($P < .05$). Walkers had significantly higher triglycerides than nonwalkers ($P_{group} = 0.019$), although all participants had normal triglycerides concentrations.

Distribution of Cholesterol in LDL and HDL Subfractions

There were no intervention effects on LDL-cholesterol concentrations (see Table 4), but change in LDL particle size and the proportion of cholesterol in large LDL particles over time did differ between groups ($P_{time \times group} = 0.011$ and 0.028, respectively; Table 5). Whereas nonwalkers had virtually no change in mean LDL particle diameter, walkers had a borderline significant reduction in LDL diameter from BL (268.7 ± 4.1 Å) to 6 months (266.9 ± 4.9

Table 3 Mean ± SD for Physical Activity and Dietary Composition at Baseline, 6 Months, and 12 Months Postrandomization for Walkers and Nonwalkers

					P		
Variable	**Group**	**Baseline**	**6 months**	**12 months**	**Time**	**Group**	**Interaction**
Total steps (per day)	Walkers	5778 ± 1745	7949 ± 2650	6711 ± 2784	**<.001**	.250	.828
	Nonwalkers	5373 ± 2899	7218 ± 3068	5772 ± 3127			
Aerobic walking (min/day)	Walkers	4.8 ± 6.8	24.4 ± 14.6**,†	17.0 ± 13.3**,§§	**<.001**	**.001**	**.001**
	Nonwalkers	4.3 ± 7.6	7.3 ± 11.7	4.7 ± 7.1			
SBAS score	Walkers	1.68 ± 0.72	2.93 ± 0.86**	3.20 ± 0.94**,‡‡	**<.001**	**<.001**	**.003**
	Nonwalkers	1.59 ± 0.80	2.09 ± 0.61	1.85 ± 0.81			
Energy intake (kcal/day)	Walkers	1502 ± 541	1442 ± 439	1346 ± 359	.101	.760	.884
	Nonwalkers	1457 ± 495	1334 ± 421	1302 ± 406			
Energy from carbohydrate (%)	Walkers	53.04 ± 6.44	50.48 ± 8.50	49.73 ± 10.65	.155	.163	.998
	Nonwalkers	55.53 ± 7.68	52.82 ± 7.32	52.60 ± 7.32			
Energy from protein (%)	Walkers	15.95 ± 2.94	18.17 ± 4.06	16.14 ± 5.77	**.045**	.109	.726
	Nonwalkers	14.86 ± 3.02	16.18 ± 2.90	15.30 ± 3.58			
Energy from fat (%)	Walkers	30.70 ± 5.63	31.34 ± 5.83	33.41 ± 7.62	.192	.527	.876
	Nonwalkers	29.48 ± 5.79	31.02 ± 7.06	31.76 ± 5.91			
Total sugars (%)	Walkers	25.97 ± 7.80	27.38 ± 9.73	24.46 ±8.03	.514	.997	.584
	Nonwalkers	26.92 ± 5.96	26.41 ± 7.04	26.10 ± 10.01			
Total fiber (g/day)	Walkers	14.30 ± 7.05	13.44 ± 8.03	11.44 ± 5.59	**.006**	.944	.680
	Nonwalkers	13.30 ± 6.10	12.12 ± 4.81	11.09 ± 3.94			

* $P < 0.05$, ** $P < .01$ for comparisons of means for walkers vs. nonwalkers by time point.

† $P < .05$ for comparisons of change from baseline to 6 months for walkers vs. nonwalkers.

‡ $P < .05$, ‡‡ $P < .01$ for comparisons of change from baseline to 12 months for walkers vs. nonwalkers.

§ $P < .05$, §§ $P < .01$ for comparisons of change from 6 months to 12 months for walkers vs. nonwalkers.

Å; $P = .065$), followed by a significant increase in diameter by 12 months (269.7 ± 4.1 Å; $P = .005$). The proportion of cholesterol in the largest LDL particles decreased by 15% from BL to 6 months, but subsequently increased 25% by 12 months among walkers; changes among nonwalkers were smaller and in opposite direction (4% and –3%, respectively; $P < .001$). As a result, the proportion of cholesterol in large LDL particles among walkers was 11% greater at baseline ($P = .019$) and 10% greater at 12 months ($P = .048$) relative to that of nonwalkers. There were no significant differences in the proportion of cholesterol in small LDL particles.

There was a significant time effect on the proportion of cholesterol in different HDL subfractions for all participants, but this effect did not differ between groups. Relative to baseline, the proportion of HDL cholesterol in large HDL particles was 11% higher at 12 months postrandomization ($P_{time} = 0.005$). In contrast, the proportion of HDL cholesterol in intermediate HDL particles was 4% and 6% lower at 6 months and 12 months, respectively ($P_{time} = 0.012$).

Discussion

Madres para la Salud was a randomized controlled trial designed to reduce postpartum body weight among Latinas using a social support mediated walking intervention. Despite the weight status of participants who were overweight/obese, the subsample in this analysis was comprised of young postpartum women with relatively few metabolic abnormalities that primarily included elevated waist circumference and low HDL-cholesterol concentrations. Although for the most part the cardiometabolic risk factors assessed, including weight, remained unchanged, walkers experienced favorable, albeit small, changes in BP, distribution of cholesterol in large LDL particles and LDL particle size, and HDL cholesterol.

The intervention resulted in increased PA, as indicated by greater aerobic walking time and SBAS score among walkers. Nevertheless, PA among study participants was low throughout the study; based on step counts, women were classified as low active (5000–7499 steps/day) at baseline, and walkers only reached the "somewhat active" category (7500–9999 steps/day) at the 6-month time point. In general, postpartum women have been documented to spend most of their time in sedentary activities.[14,22,23] The increase in PA observed over time in both groups could be in part attributed to intervention effects as well as recruitment attention to health mailed messages to the control groups.

Overall, PA increased slightly in the control group and showed limited sustainability in the intervention group. The possible reasons for limited treatment effects may be related to the notion that new mothers were adjusting to the challenges of caring for an infant, multiple other small children in the home, and only gradually being able to incorporate more activities to their daily life. Although the possibility that women in both groups walked more when they were wearing the pedometer cannot be ruled out, the differential increase in aerobic walking between groups suggests a small effect of the intervention. Nevertheless, the increase in aerobic walking was small (about 12 min/day); among walkers PA declined once *Promotoras* were no longer holding support sessions.

Table 4 Mean ± SD for Cardiometabolic Disease Risk Factors at Baseline, 6 Months, and 12 Months Postrandomization for Walkers and Nonwalkers

						P	
Variable	**Group**	**Baseline**	**6 months**	**12 months**	**Time**	**Group**	**Interaction**
Weight (kg)	Walkers	75.4 ± 9.6	74.2 ± 9.3	73.3 ± 10.3	.128	.133	.454
	Nonwalkers	71.1 ± 11.3	68.3 ± 12.0	70.3 ± 12.6			
Body fat (%)	Walkers	38.9 ± 3.8	38.5 ± 3.8	38.7 ± 3.2	.237	.324	.111
	Nonwalkers	37.5 ± 5.5	36.6 ± 7.2	36.5 ± 6.2			
Waist circumference (cm)	Walkers	98.7 ± 6.9	98.4 ± 6.3	99.4 ± 7.3	.545	.391	.212
	Nonwalkers	98.9 ± 10.9	97.7 ± 8.5	95.9 ± 9.1			
Systolic BP (mm Hg)	Walkers	118 ± 13**	112 ± 13*	111 ± 15 [‡,§]	**.014**	**.048**	**.002**
	Nonwalkers	108 ± 6	104 ± 9	109 ± 8			
Diastolic BP (mm Hg)	Walkers	74 ± 11**	71 ± 11**	72 ± 12	**.034**	**.003**	.272
	Nonwalkers	66 ± 6	63 ± 4	67 ± 6			
Glucose (mg/dL)	Walkers	91 ± 11	92 ± 12	89 ± 9	.521	.963	.272
	Nonwalkers	89 ± 8	90 ± 11	95 ± 24			
Insulin (μU/mL)	Walkers	19.3 ± 8.4	22.8 ± 10.3	21.4 ± 10.5	.104	.775	.319
	Nonwalkers	18.2 ± 9.7	22.1 ± 14.7	30.3 ± 37.4			
Total cholesterol (mg/dL)	Walkers	175 ± 34*	171 ± 24*	171 ± 25**	.343	**.010**	.589
	Nonwalkers	153 ± 38	153 ± 34	146 ± 31			
LDL-cholesterol (mg/dL)	Walkers	116 ± 29*	112 ± 24*	117 ± 23**	.142	**.009**	.393
	Nonwalkers	96 ± 35	96 ± 30	94 ± 27			
HDL-cholesterol (mg/dL)	Walkers	42 ± 8	42 ± 8	47 ± 10[§]	*.063*	.692	*.097*
	Nonwalkers	44± 11	46 ± 11	43 ± 11			
Triglycerides (mg/dL)	Walkers	131 ± 63	138 ± 56**	122 ± 54	.996	**.019**	.116
	Nonwalkers	102 ± 63	93 ± 51	99 ± 49			
Metabolic syndrome components (count)	Walkers	2.57 ± 0.81*	1.93 ± 0.80**	1.86 ± 0.53	**<.001**	**.003**	.414
	Nonwalkers	1.95 ± 0.84	1.22 ± 0.88	1.47 ± 0.87			

* $P < 0.05$, ** $P < .01$ for comparisons of means for walkers vs. nonwalkers by time point.

† $P < .05$ for comparisons of change from baseline to 6 months for walkers vs. nonwalkers.

‡ $P < .05$, ‡‡ $P < .01$ for comparisons of change from baseline to 12 months for walkers vs. nonwalkers.

§ $P < .05$, §§ $P < .01$ for comparisons of change from 6 months to 12 months for walkers vs. nonwalkers.

Table 5 Mean ± SD for LDL Peak Particle Size and Distribution of Cholesterol in LDL and HDL Subfractions at Baseline, 6 Months, and 12 Months Post Randomization for Walkers and Nonwalkers

						P	
Variable	**Group**	**Baseline**	**6 months**	**12 months**	**Time**	**Group**	**Interaction**
LDL peak diameter (Å)	Walkers	268.73 ± 4.06	266.87 ± 4.93	269.71 ± 4.08[§§]	.308	.477	**.011**
	Nonwalkers	269.45 ± 5.11	269.44 ± 5.12	268.71 ± 6.69			
% cholesterol in large LDL	Walkers	29.03 ± 4.01*	27.07 ± 3.96[†]	28.84 ± 3.53*	.347	*.058*	**.028**
	Nonwalkers	26.08 ± 4.22	27.20 ± 3.46	26.29 ± 4.48			
% cholesterol in small LDL	Walkers	2.47 ± 3.02	3.88 ± 3.73	2.36 ± 2.73	.640	.836	*.093*
	Nonwalkers	2.55 ± 4.89	2.62 ± 4.88	3.25 ± 6.20			
% HDL cholesterol in large HDL	Walkers	24.97 ± 9.06	24.29 ± 8.40	29.93 ± 8.07	**.005**	.886	.259
	Nonwalkers	24.98 ± 9.69	25.61 ± 9.00	25.77 ± 9.04			
% HDL cholesterol in intermediate HDL	Walkers	57.98 ± 8.20	56.21 ± 5.43	53.94 ± 4.11	**.012**	.383	.632
	Nonwalkers	58.64 ± 5.22	56.35 ± 5.00	56.52 ± 6.47			
% HDL cholesterol in small HDL	Walkers	16.95 ± 8.00	19.52 ± 8.00	16.12 ± 7.55	*.058*	.529	.563
	Nonwalkers	16.15 ± 8.16	17.96 ± 7.02	17.71 ± 6.83			

Abbreviations: LDL, low-density lipoprotein; HDL, high-density lipoprotein.

* $P < 0.05$, ** $P < .01$ for comparisons of means for walkers vs. nonwalkers by time point.

† $P < .05$, for comparisons of change from baseline to 6 months for walkers vs. nonwalkers.

‡ $P < .05$, ‡‡ $P < .01$ for comparisons of change from baseline to 12 months for walkers vs. nonwalkers.

§ $P < .05$, §§ $P < .01$ for comparisons of change from 6 months to 12 months for walkers vs. nonwalkers.

Physical Activity and Lipids

Existing evidence suggests that aerobic exercise results in small LDL-cholesterol reductions (about 3%) especially when combined with weight loss.[24–26] Although exercise training has been reported to raise HDL-cholesterol concentrations (by about 2% to 5%), evidence is inconsistent; effects, when present, seem to be greater with greater triglycerides or lower HDL concentrations at baseline.[24,25] Previous PA interventions resulting in lipid changes[27,28] have mainly consisted of supervised training sessions in which the dose of PA (frequency, intensity, and/or duration) was greater than that achieved by participants of the current study.

In contrast to supervised structured PA interventions in controlled environments, behavioral interventions conducted in free-living conditions have had less consistent effects on lipoproteins. Among adults with type 2 diabetes, a pedometer-based intervention delivered in 1 face-to-face session followed by 7 phone calls over 24 weeks resulted in no significant differences in lipids, glucose or body weight despite a significant increase in about 2700 steps/day among participants in the intervention group (vs. a reduction of about 1250 steps/day among those in the control group).[29] In *Pasos Adelante*, a 12-week chronic disease prevention program in which community health workers led walking groups among Hispanic adults (92% women), no significant prepost changes in LDL- or HDL-cholesterol, triglyceride, and glucose concentrations were observed despite increased self-reported PA relative to baseline.[30] It is possible that, similar to the current study, the magnitude of PA changes attained by participants was of smaller magnitude than what would cause physiologically meaningful changes in lipid concentrations. Interestingly, a meta-analysis of PA interventions targeting minority adults documented that neither supervised exercise interventions nor behavioral interventions resulted in significant changes in lipid concentrations despite small effects on anthropometric measurements, potentially due to small effects on PA.[31] In the current study, favorable changes in triglycerides, HDL cholesterol, and LDL particle size were achieved from 6 to 12 months, and suggest benefits from longer-term interventions.

Although lipid concentrations are susceptible to alterations due to dietary changes, participants in the current study did not change their intake of nutrients likely to affect lipoprotein metabolism (eg, energy, fat, carbohydrates, sugars, fiber). Moreover, observed differences in diet over time occurred in both walkers and nonwalkers.

Physical Activity and Lipoprotein Subfractions

Lipoproteins are heterogeneous particles regarding their size, density and composition, with size being inversely correlated to cholesterol content and atherogenicity.[32,33] The presence of small, dense LDL particles has been associated with type 2 diabetes, metabolic syndrome, and coronary artery disease.[34,35] Larger HDL particles are regarded as cardioprotective, and their presence has been associated with lower cardiovascular disease risk.[32,36] In contrast, small HDL particles have been reported as a trait of abdominal obesity-related dyslipidemias.[37]

In the current study the proportion of cholesterol in large LDL particles among walkers was greater at baseline and at 12 months postrandomization relative to that of nonwalkers. In cross-sectional studies regular PA, particularly aerobic activity, has been associated with the presence of larger LDL and HDL particles.[38,39] In randomized controlled interventions, PA has been reported to favorably modify LDL particle size even in the absence of LDL cholesterol concentration changes.[40] Results from a 6-month randomized trial with overweight and obese adults with mild dyslipidemia demonstrated that even low amounts of PA (equivalent to walking 12 miles per week) resulted in LDL particle size increase regardless of intensity (moderate, 40% to 55% VO_2max vs. high, 65% to 80% VO_2max).[40] However, high amounts of PA (equivalent of 20 miles per week) were necessary to observe a reduction in cholesterol in small LDL particles, and an increase in HDL-cholesterol, cholesterol in large HDL particles, and HDL particle size.[40]

Lipids and Lipoproteins in the Postpartum Period

There is limited information regarding the role of PA in modulating lipoprotein changes and lipoprotein subfractions in the postpartum period. Despite an improvement in cardiovascular fitness, a 12-week aerobic exercise intervention (45 min/day, 5 days/week) among women 6 to 8 weeks postpartum resulted in only marginally significant increases in HDL cholesterol, with no changes in other lipid concentrations.[41] Conversely, in a 16-week weight loss intervention starting at 7 to 8 weeks postpartum that included supervised walking sessions 3 to 4 times/week, low- and moderate-intensity PA induced significant reductions in fasting glucose (–5%) and LDL cholesterol (–9%) concentrations relative to sedentary controls.[42] In contrast to previous reports, women in the current study were recruited later in the postpartum period (mean 12 weeks) and more likely to be at a stage at which pregnancy-related lipid changes would have already reverted.[9,12] In animal studies early lactation was associated with lower triglycerides and VLDL particles, and more abundant larger LDL particles; once lipoprotein lipase from mammary gland is downregulated because of lactation cessation, triglyceride-rich particles became more abundant and LDL particles increased in size.[43] Although breastfeeding duration data were not collected, only a small percentage of participants were exclusively breastfeeding at baseline and would have been susceptible to major lipoprotein fluctuations because of breastfeeding cessation. Moreover, there were no differences between groups in child feeding method at baseline.

The current study has several limitations worth discussing. Overall, nonwalkers had a more favorable metabolic profile than walkers as indicated by lower BP, total-, LDL-cholesterol, and triglyceride concentrations. Although the subsample of participants for this analysis was randomly selected, the small sample size may have limited the ability to select weighted subgroups or to provide adequate statistical power to detect differences. Nevertheless, the differences between walkers and nonwalkers were maintained throughout the study and did not seem to result in unexpected differential responses between groups. The intervention was targeted toward overweight/obese postpartum women, regardless of their metabolic health. It is possible that greater effects could have been observed among individuals with a metabolic profile indicative of greater risk at baseline. Because the study was not conducted in a controlled environment, the walking dose attained by participants in the intervention group was low, and potentially insufficient to elicit changes in lipids and other cardiovascular risk factors. It is also possible that factors others than those accounted for in the current study (eg, breastfeeding duration, hormonal changes) could have affected results.

The study also has several strengths worth highlighting. Because of its community-based nature, this study is more likely to reflect PA behavior changes that could be attained in real-life circumstances. Aside from providing social support, *Promotoras* provided supervised walking, which was instrumental in ensuring that participants walked at an intensity that would increase aerobic PA, as well as providing an additional source for social support.

Moreover, in addition to the use of subjective measures (SBAS), PA was assessed using objective measures (pedometers), which allowed for identifying attainment of health-enhancing aerobic walking. Finally, the current study was carefully designed to incorporate elements of cultural sensitivity and ensure intervention fidelity.[13,44]

In conclusion, participation in the social-support PA intervention resulted in slightly increased HDL cholesterol concentrations, a modest and beneficial shift toward larger, less atherogenic LDL particles, and small reductions in systolic BP. Whether a higher amount and intensity of exercise than those attained in the current and previous studies can provide additional benefits in lipoprotein profile among postpartum women needs to be further investigated. Future research is also needed to better understand how naturally occurring postpartum- and lactation-related fluctuations in lipoprotein metabolism are modulated by PA.

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References

1. Lorenzo C, Williams K, Hunt KJ, Haffner SM. Trend in the prevalence of the metabolic syndrome and Its impact on cardiovascular disease incidence: the San Antonio Heart Study. *Diabetes Care*. 2006;29(3):625–630. PubMed doi:10.2337/diacare.29.03.06.dc05-1755
2. Torres M, Azen S, Varma R. Prevalence of obesity and associated comorbid conditions in a population-based sample of primarily urban Mexican Americans. *Ethn Dis*. 2006;16(2):362–369. PubMed
3. Burroughs VJ, Nonas C, Sweeney CT, et al. Self-reported comorbidities among self-described overweight African-American and Hispanic adults in the United States: results of a national survey. *Obesity (Silver Spring)*. 2008;16(6):1400–1406. PubMed doi:10.1038/oby.2008.61
4. Cowie CC, Rust KF, Byrd-Holt DD, et al. Prevalence of diabetes and impaired fasting glucose in adults in the U.S. population: National Health and Nutrition Examination Survey 1999-2002. *Diabetes Care*. 2006;29(6):1263–1268. PubMed doi:10.2337/dc06-0062
5. Ong KL, Cheung BMY, Wong LYF, Wat NMS, Tan KCB, Lam KSL. Prevalence, treatment, and control of diagnosed diabetes in the U.S. National Health and Nutrition Examination Survey 1999-2004. *Ann Epidemiol*. 2008;18(3):222–229. PubMed doi:10.1016/j.annepidem.2007.10.007
6. Rooney BL, Schauberger CW, Mathiason MA. Impact of perinatal weight change on long-term obesity and obesity-related illnesses. *Obstet Gynecol*. 2005;106(6):1349–1356. PubMed doi:10.1097/01.AOG.0000185480.09068.4a
7. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999-2004. *JAMA*. 2006;295(13):1549–1555. PubMed doi:10.1001/jama.295.13.1549
8. Records K, Keller C, Ainsworth B, Permana PA. Overweight and obesity in postpartum Hispanic women. *Health Care Women Int*. 2008;29(6):649–667. PubMed doi:10.1080/07399330802089214
9. Silliman K, Shore V, Forte TM. Hypertriglyceridemia during late pregnancy is associated with the formation of small dense low-density lipoproteins and the presence of large buoyant high-density lipoproteins. *Metabolism*. 1994;43(8):1035–1041. PubMed doi:10.1016/0026-0495(94)90186-4
10. Belo L, Caslake M, Santos-Silva A, et al. LDL size, total antioxidant status and oxidised LDL in normal human pregnancy: a longitudinal study. *Atherosclerosis*. 2004;177(2):391–399. PubMed doi:10.1016/j.atherosclerosis.2004.07.023
11. Winkler K, Wetzka B, Hoffmann MM, et al. Low density lipoprotein (LDL) subfractions during pregnancy: accumulation of buoyant LDL with advancing gestation. *J Clin Endocrinol Metab*. 2000;85(12):4543–4550. PubMed doi:10.1210/jcem.85.12.7027
12. Alvarez JJ, Montelongo A, Iglesias A, Lasuncion MA, Herrera E. Longitudinal study on lipoprotein profile, high density lipoprotein subclass, and postheparin lipases during gestation in women. *J Lipid Res*. 1996;37(2):299–308. PubMed
13. Keller CS, Records K, Ainsworth BE, et al. Madres para la Salud: design of a theory-based intervention for postpartum Latinas. *Contemp Clin Trials*. 2011;32(3):418–427. PubMed doi:10.1016/j.cct.2011.01.003
14. Ainsworth BE, Keller C, Herrmann S, et al. Physical activity and sedentary behaviors in postpartum Latinas: Madres para la Salud. *Med Sci Sports Exerc*. 2013;45(7):1298–1306. PubMed doi:10.1249/MSS.0b013e3182863de5
15. Keller C, Todd M, Ainsworth B, *et al.* Overweight, obesity, and neighborhood characteristics among postpartum Latinas. *J Obes*. 2013;2013:916468. doi:10.1155/2013/916468
16. U.S. Department of Health and Human Services. *Physical Activity Guidelines for Americans, 2008*. Washington, DC: U.S. Government Printing Office; 2008.
17. Hoefner DM, Hodel SD, O'Brien JF, et al. Development of a rapid, quantitative method for LDL subfractionation with use of the Quantimetrix Lipoprint LDL system. *Clin Chem*. 2001;47(2):266–274. PubMed
18. Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. *Circulation*. 2005;112(17):2735–2752. PubMed doi:10.1161/CIRCULATIONAHA.105.169404
19. Taylor-Piliae RE, Fair J, Haskell W, et al. Validation of the Stanford Brief Activity Survey: examining psychological factors and physical activity levels in older adults. *J Phys Act Health*. 2010;7(1):87–94. PubMed
20. Conway JM, Ingwersen LA, Moshfegh AJ. Accuracy of dietary recall using the USDA five-step multiple-pass method in men: an observational validation study. *J Am Diet Assoc*. 2004;104(4):595–603. PubMed doi:10.1016/j.jada.2004.01.007
21. NCEP Expert Panel. Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*. 2001;285(19):2486–2497. PubMed doi:10.1001/jama.285.19.2486
22. Durham HA, Morey MC, Lovelady CA, Namenek Brouwer RJ, Krause KM, Ostbye T. Postpartum physical activity in overweight and obese women. *J Phys Act Health*. 2011;8(7):988–993. PubMed
23. Walker L, Freeland-Graves JH, Milani T, et al. Weight and behavioral and psychosocial factors among ethnically diverse, low-income women after childbirth: II. Trends and correlates. *Women Health*. 2004;40(2):19–34. PubMed doi:10.1300/J013v40n02_02
24. Eckel RH, Jakicic JM, Ard JD, et al. AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;129(25 Suppl 2):S76–99. PubMed
25. Kelley GA, Kelley KS, Roberts S, Haskell W. Comparison of aerobic exercise, diet or both on lipids and lipoproteins in adults: a meta-analysis of randomized controlled trials. *Clin Nutr*. 2012;31(2):156–167. PubMed doi:10.1016/j.clnu.2011.11.011

26. Ahmed HM, Blaha MJ, Nasir K, Rivera JJ, Blumenthal RS. Effects of physical activity on cardiovascular disease. *Am J Cardiol*. 2012;109(2):288–295. PubMed doi:10.1016/j.amjcard.2011.08.042
27. Altena TS, Michaelson JL, Ball SD, Guilford BL, Thomas TR. Lipoprotein subfraction changes after continuous or intermittent exercise training. *Med Sci Sports Exerc*. 2006;38(2):367–372. PubMed doi:10.1249/01.mss.0000185088.33669.fd
28. Butcher LR, Thomas A, Backx K, Roberts A, Webb R, Morris K. Low-intensity exercise exerts beneficial effects on plasma lipids via PPAR. [gamma] *Med Sci Sports Exerc*. 2008;40(7):1263–1270. PubMed doi:10.1249/MSS.0b013e31816c091d
29. Van Dyck D, De Greef K, Deforche B, et al. The relationship between changes in steps/day and health outcomes after a pedometer-based physical activity intervention with telephone support in type 2 diabetes patients. *Health Educ Res*. 2013;28(3):539–545. PubMed doi:10.1093/her/cyt038
30. Staten LK, Scheu LL, Bronson D, Peña V, Elenes J. Pasos Adelante: the effectiveness of a community-based chronic disease prevention program. *Prev Chronic Dis*. 2005;2(1): http://www.cdc.gov/pcd/issues/2005/jan/04_0075.htm. PubMed
31. Conn VS, Phillips LJ, Ruppar TM, Chase JA. Physical activity interventions with healthy minority adults: meta-analysis of behavior and health outcomes. *J Health Care Poor Underserved*. 2012;23(1):59–80. PubMed doi:10.1353/hpu.2012.0032
32. Asztalos BF, Schaefer EJ. High-density lipoprotein subpopulations in pathologic conditions. *Am J Cardiol*. 2003;91(7A):12E–17E. PubMed doi:10.1016/S0002-9149(02)03383-0
33. Mudd JO, Borlaug BA, Johnston PV, et al. Beyond low-density lipoprotein cholesterol: defining the role of low-density lipoprotein heterogeneity in coronary artery disease. *J Am Coll Cardiol*. 2007;50(18):1735–1741. PubMed doi:10.1016/j.jacc.2007.07.045
34. Krauss RM. Lipoprotein subfractions and cardiovascular disease risk. *Curr Opin Lipidol*. 2010;21(4):305–311. PubMed doi:10.1097/MOL.0b013e32833b7756
35. Ip S, Lichtenstein AH, Chung M, Lau J, Balk EM. Systematic review: association of low-density lipoprotein subfractions with cardiovascular outcomes. *Ann Intern Med*. 2009;150(7):474–484. PubMed doi:10.7326/0003-4819-150-7-200904070-00007
36. Yu S, Yarnell JW, Sweetnam P, Bolton CH. High density lipoprotein subfractions and the risk of coronary heart disease: 9-years follow-up in the Caerphilly Study. *Atherosclerosis*. 2003;166(2):331–338. PubMed doi:10.1016/S0021-9150(02)00361-1
37. Pascot A, Lemieux I, Prud'homme D, et al. Reduced HDL particle size as an additional feature of the atherogenic dyslipidemia of abdominal obesity. *J Lipid Res*. 2001;42(12):2007–2014. PubMed
38. Durstine JL, Grandjean PW, Davis PG, Ferguson MA, Alderson NL, DuBose KD. Blood lipid and lipoprotein adaptations to exercise: a quantitative analysis. / Lipides sanguins et adaptations des lipoproteines a l ' exercice: analyse quantitative. *Sports Med*. 2001;31(15):1033–1062. PubMed doi:10.2165/00007256-200131150-00002
39. Aadland E, Andersen JR, Anderssen SA, Kvalheim OM. Physical activity versus sedentary behavior: associations with lipoprotein particle subclass concentrations in healthy adults. *PLoS One*. 2013;8(12):e85223. PubMed doi:10.1371/journal.pone.0085223
40. Kraus WE, Houmard JA, Duscha BD, et al. Effects of the amount and intensity of exercise on plasma lipoproteins. *N Engl J Med*. 2002;347(19):1483–1492. PubMed doi:10.1056/NEJMoa020194
41. Lovelady CA, Nommsen-Rivers LA, McCrory MA, Dewey KG. Effects of exercise on plasma lipids and metabolism of lactating women. *Med Sci Sports Exerc*. 1995;27(1):22–28. PubMed doi:10.1249/00005768-199501000-00005
42. Davenport MH, Giroux I, Sopper MM, Mottola MF. Postpartum exercise regardless of intensity improves chronic disease risk factors. *Med Sci Sports Exerc*. 2011;43(6):951–958. PubMed doi:10.1249/MSS.0b013e3182051155
43. Smith JL, Lear SR, Forte TM, Ko W, Massimi M, Erickson SK. Effect of pregnancy and lactation on lipoprotein and cholesterol metabolism in the rat. *J Lipid Res*. 1998;39(11):2237–2249. PubMed
44. Keller C, Records K, Coe K, et al. Promotoras' roles in integrative validity and treatment fidelity efforts in randomized controlled trials. *Fam Community Health*. 2012;35(2):120–129. PubMed doi:10.1097/FCH.0b013e31824650a6