The purpose of this study was to determine if light physical activity (LPA) minimizes the impairment of cardiometabolic risk factors following a typical meal in adolescents. Eighteen adolescents (50% male, 14.8 ± 2.3 yrs) consumed a meal (32% fat, 14% protein, 53% carbohydrate), then completed a walking (1.5mph for 45 min of each hour) or sitting treatment for 3 hr in randomized order on separate days. Following the meal, HDL cholesterol declined 4.8% but remained higher during walking at 3 hr (42.1mg/dl ± 9.3) than sitting (8.4% decline; 40.5mg/dL ± 9.9; treatment × time interaction, $p < .03$). The 3-hr insulin was lower after walking (24.8μIU/ml ± 33.4) than sitting (37.8μIU/ml ± 34.7; treatment × time interaction, $p < .0001$). Triglycerides increased by ~40% above baseline at 1 and 2 hr, with higher values for walking (treatment × time interaction, $p < .02$). However by 3 hr, triglycerides were not different from baseline. Area under the curve (AUC) analyses were not significantly different between treatments for any outcomes. Although minor, LPA appears to mitigate the undesirable postprandial changes in HDL cholesterol and insulin but not triglycerides, following a typical meal in adolescents.

The acute responses to consumption of a high-fat meal in adolescents includes increased triglycerides (20,33), total cholesterol (39), serum-free fatty acids (33), decreased high- and increased low-density lipoproteins (39), and impaired endothelial function (20,33). When short-term responses following a high-fat meal were compared, overweight adolescent boys had a higher postprandial lipemia versus normal-weight controls (19). Furthermore, cardiometabolic risk factors (i.e., triglycerides [12,19,33], endothelial function [11], glucose [11] and insulin [11]) were still higher than fasting levels 3 or more hours after the meal was consumed (i.e., had not yet returned to fasting states). Slower lipid metabolism, as evidenced by higher postprandial triglycerides, predicts the presence of coronary artery disease (29), is atherogenic (28), and is further impaired in those with existing metabolic disease and obesogenic behaviors (2). Furthermore, postmeal impairment in triglycerides is an independent cardiometabolic-disease risk factor (17). Taken together, these
studies support the hypothesis of Anderson et al. (3), which is that fatty meals cause transient endothelial dysfunction, acute increases in oxidative stress on the vasculature and acute increased exposure of vessel walls to harmful triglyceride-rich lipoproteins and other cardiometabolic risk factors.

Volume of sitting is directly associated with the rates of all-cause and cardiovascular-related mortality in adults (16,40) in a dose-response manner that is independent of physical activity (16,40). In children, high volumes of self-reported sitting and objectively measured sedentary behavior are detrimentally associated with cardiometabolic-disease risk factors, including insulin resistance (30), waist circumference (34), fat mass (34), systolic blood pressure (23), triglycerides (23), and fasting glucose levels (23). Moderate to vigorous exercise reduces these risks (10,19,35,36), although it may not be feasible for adolescents to exercise at relatively high intensity following a calorically dense meal. While performing light physical activity may be a suitable alternative, its potential impact on acute changes in cardiometabolic risk factors following a typical meal is not well understood.

Therefore, the purpose of this project was to determine if participation in light physical activity (i.e., slow walking) following a typical meal affects the acute impairment in cardiometabolic risk factors in adolescents. We hypothesized that the atherogenic effects of the meal would be blunted when light physical activity was performed postprandially compared with a control condition of sitting.

**Methods**

**Subjects and Study Design**

A group of 18 10- to 18-year-olds that included those being classified as normal weight and overweight participated in this study. All participants were healthy and without previous medical history of cardiovascular or metabolic disease. Before participation, written and verbal informed consent/assent was obtained from parents and children, and this study was approved by university institutional review board. A complete physical examination was conducted by a nurse practitioner or physician. Tanner staging was assessed for pubertal status, and height, weight, waist circumference, and blood pressure were obtained using standard methods. All participants were pubertal Tanner Stage 2 or greater (21,22). Body composition was determined using dual energy x-ray absorptiometry (DEXA, QDR 4500A/Delphi model, HOLOGIC, Bedford, MA).

Participants completed a total of three visits to the laboratory: one screening visit and two counter-balanced treatments (sitting and light physical activity). Participants arrived in the morning after an overnight fast and without having participated in any exercise in the previous 8 hr. After at least 5 min of rest in a reclined position, resting baseline (i.e., fasting) measures were collected. Participants consumed a breakfast meal within 15 min. Immediately following completion of the meal, participants began either the light physical activity or sitting treatment, which lasted for 3 hr. Venous blood samples were obtained from a forearm vein during each visit at baseline (0 hr) and then at 1, 2, and 3 hr after the meal. A trained technician measured endothelial function at baseline and 3 hr after the meal. Multiple measures of endothelial function during the treatment could not be made due to the time (~15 min) and nature of this measure (i.e., participants must be supine and resting).
Test Meal

A meal that was high in fat percentage (e.g., biscuit with cheese, egg and meat-substitute; MorningStar Farms veggie sausage, egg, and cheese biscuit) was prepared in the OUHSC Clinical Research Center Metabolic Kitchen by a registered dietitian. The meal’s energy content of 580 kcals was comprised of 32% fat (20.5 g), 48% of which was saturated fat (9.8g), 14% protein (20.0 g) and 53% carbohydrate (78.0 g). This level of fat content is slightly higher than the average school lunch served to American children (8) and is consistent with meals served in previous studies that measured cardiometabolic effects of high fat meals (33).

Treatments

The light physical activity treatment consisted of slow walking (1.5 mph) on a treadmill for 45 min, followed by 15 min rest, following which the participant resumed walking and the pattern was repeated for 3 hr. The walking treatment was designed to operate within the established guidelines for light physical activity, defined as less than 3 and more than 1.5 metabolic equivalents (METs; 27). 1.5 mph on a treadmill is estimated to be equivalent to 2.0 METs (1). Water was available ad lib throughout all visits. Participants wore an ActiGraph GT3-X accelerometer; average counts per minute was 385, which is within the range (101–3,000) for light physical activity (37). The 3 hr duration of the study was selected because it fell between ranges reported in previous studies, in which adverse changes in lipids, insulin, and/or vascular function outcomes in children and adults were reported following a meal (2 (13,25) to 8 (29) hr).

In the sitting treatment, the participant sat or reclined quietly with minimal movements but remained awake. Sitting or lying down is defined as less than 1.5 METs (27). The ActiGraph detected a mean of 24 counts per minute, which is within the sedentary range (<100 cpm; 37). Participants were able to read and watch television for the duration of both treatment visits.

Vascular Function

Endothelial function was measured using the Endo-PAT2000 (Itamar Medical, Caesarea, Israel). Pneumatic probes were placed on each forefinger and a blood pressure cuff on the testing arm. Five minutes of each baseline, occlusion, and postocclusion pulse pressure measurements were recorded on both arms and used to calculate endothelial function as an RHI value (38). This method is a valid (6) and reliable measurement of endothelial function (31) and is well tolerated by children ages 13–19 years (31). Testing was administered with the participant in the supine position in a dimly lit ~22°C exam room.

Blood Analysis

Blood samples were centrifuged at 4°C and plasma aliquots were stored at −80°C until further analysis was performed by the OU Medical Center Laboratory in Oklahoma City, OK. Measurements included triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol using the time endpoint method, plasma insulin determined by insulin reagent kit from Siemens Diagnostics performed on the Immulite 2000 (intra-assay coefficient of variation 2.3–2.7%,...
interassay coefficient of variation 4.5–5.4%), and plasma glucose assessed using the oxygen rate electrode method.

Statistical Analysis

Descriptive characteristics of the participants were calculated using means ± SD and frequencies. Insulin measurements were highly skewed, so analyses were conducted on log-transformed values (figures present untransformed values). Paired $t$ tests were used to determine if baseline values differed between treatment days. The difference in measurements ($\Delta$) between post 3 hr and baseline was calculated for each variable. Area under the curve (AUC) was calculated for all outcome measures that were obtained at hourly intervals using the trapezoidal method (32). For all variables a 2 × 2 (treatment × time) repeated-measures ANOVA was calculated to determine the effect of light physical activity on each outcome over time (pre, 1 hr, 2 hr, 3 hr). Post hoc analyses (least significant difference) were conducted for each time point. A secondary approach to understand the effects of the treatments on the outcomes included paired $t$ tests to compare the $\Delta$ and AUC across treatments for each variable. Percent increase or decrease from baseline was calculated by determining the difference between the value of interest and the baseline value; that difference was then divided into the baseline value. Statistical significance was set at $p < .05$. Analyses were conducted using SPSS version 18.0 and SAS version 9.3.

Results

Baseline characteristics of the participants are presented in Table 1. Baseline and post 3 hr risk factors by treatment for the total sample are presented in Table 2. There were no significant differences between baseline risk factor values between

<table>
<thead>
<tr>
<th>Table 1 Participant Demographic Characteristics</th>
</tr>
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<tbody>
<tr>
<td>Variable</td>
</tr>
<tr>
<td>---------------------------------</td>
</tr>
<tr>
<td>Sample size</td>
</tr>
<tr>
<td>Age (years)</td>
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<tr>
<td>Sex (%)</td>
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<tr>
<td>male</td>
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<tr>
<td>Race (%)</td>
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<tr>
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<tr>
<td>Black</td>
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<td>Height (cm)</td>
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<td>Weight (kg)</td>
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<td>BMI</td>
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<tr>
<td>Body mass index percentile</td>
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<tr>
<td>Percent body fat</td>
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<tr>
<td>Percent overweight/obese</td>
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</table>

Note. Values are means ± SD.
<table>
<thead>
<tr>
<th>Variable</th>
<th>LPA Treatment</th>
<th></th>
<th></th>
<th></th>
<th>Sitting Treatment</th>
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<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>1 hr</td>
<td>2 hr</td>
<td>3 hr</td>
<td>Baseline</td>
<td>1 hr</td>
<td>2 hr</td>
<td>3 hr</td>
</tr>
<tr>
<td>Reactive hyperemic index (AU)</td>
<td>1.81 ± 0.41</td>
<td>n/a</td>
<td>n/a</td>
<td>1.81 ± 0.59</td>
<td>1.86 ± 0.48</td>
<td>n/a</td>
<td>n/a</td>
<td>1.79 ± 0.52</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)*</td>
<td>94.7 ± 53.9</td>
<td>132.1 ± 70.3</td>
<td>129.6 ± 72.8</td>
<td>101.2 ± 70.0</td>
<td>91.4 ± 59.4</td>
<td>120.2 ± 66.5</td>
<td>110.1 ± 63.9</td>
<td>103.7 ± 73.1</td>
</tr>
<tr>
<td>HDL-C (mg/dl)*</td>
<td>44.2 ± 9.9</td>
<td>41.9 ± 9.2</td>
<td>41.3 ± 9.0</td>
<td>42.1 ± 9.3</td>
<td>44.2 ± 9.3</td>
<td>40.1 ± 9.1</td>
<td>40.3 ± 9.9</td>
<td>40.5 ± 9.9</td>
</tr>
<tr>
<td>LDL-C (mg/dl)</td>
<td>84.8 ± 20.1</td>
<td>74.1 ± 20.9</td>
<td>72.4 ± 19.4</td>
<td>77.7 ± 19.2</td>
<td>86.1 ± 22.3</td>
<td>76.7 ± 25.1</td>
<td>77.4 ± 23.7</td>
<td>78.6 ± 24.6</td>
</tr>
<tr>
<td>LDL-C/HDL-C ratio</td>
<td>2.05 ± 0.83</td>
<td>1.90 ± 0.82</td>
<td>1.87 ± 0.80</td>
<td>1.97 ± 0.81</td>
<td>2.07 ± 0.84</td>
<td>2.04 ± 0.92</td>
<td>2.04 ± 0.94</td>
<td>2.01 ± 0.96</td>
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<tr>
<td>Insulin (μIU/ml)*</td>
<td>10.0 ± 13.4</td>
<td>76.1 ± 65.3</td>
<td>43.7 ± 36.5</td>
<td>24.8 ± 33.4</td>
<td>7.3 ± 9.5</td>
<td>64.0 ± 78.9</td>
<td>52.0 ± 67.7</td>
<td>37.8 ± 34.7</td>
</tr>
<tr>
<td>Glucose (mg/dl)</td>
<td>92.9 ± 8.8</td>
<td>115.0 ± 25.0</td>
<td>106.8 ± 14.2</td>
<td>94.3 ± 12.6</td>
<td>92.9 ± 8.8</td>
<td>106.7 ± 34.1</td>
<td>99.9 ± 19.3</td>
<td>94.6 ± 13.9</td>
</tr>
</tbody>
</table>

* Significant Treatment × Time interaction; HDL-cholesterol (p < 0.03), Insulin (p < 0.0001), Triglycerides (p < 0.02).
treatment days. Sitting and light physical activity treatment AUC values for all participants are presented in Table 3.

The ANOVA detected a statistical interaction between treatment and time of blood draw on postprandial HDL cholesterol (Figure 1A), log-transformed insulin (Figure 1B), and triglycerides (Figure 1C). The following paragraphs interpret these results by outcome.

Table 3  Area Under the Curve (AUC) for Cardiometabolic Outcomes by Light Physical Activity (LPA) and Sitting Treatment for All Participants ($N = 18$)

<table>
<thead>
<tr>
<th>Variable</th>
<th>LPA Treatment</th>
<th>Sit Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triglycerides (mg/h/dL)</td>
<td>359.7 ± 195.6</td>
<td>327.8 ± 180.8</td>
</tr>
<tr>
<td>High-density lipoprotein (mg/h/dL)</td>
<td>126.4 ± 27.6</td>
<td>122.7 ± 28.4</td>
</tr>
<tr>
<td>Low-density lipoprotein (mg/h/dL)</td>
<td>227.8 ± 58.6</td>
<td>236.4 ± 70.1</td>
</tr>
<tr>
<td>LDL/HDL ratio (ratio/h)</td>
<td>5.79 ± 2.43</td>
<td>6.17 ± 2.73</td>
</tr>
<tr>
<td>Plasma insulin (µIU/h/mL)</td>
<td>136.8 ± 120.1</td>
<td>138.6 ± 165.3</td>
</tr>
<tr>
<td>Plasma glucose (mg/h/dL)</td>
<td>315.4 ± 42.8</td>
<td>300.0 ± 56.7</td>
</tr>
</tbody>
</table>

Note. Values are means ± SD. There were no statistically significant differences between sitting and LPA treatment for all participants.

Change in RHI between baseline and 3 hr did not differ between treatments in baseline post 3 hr or Δ analyses. Glucose was increased above baseline at 1 hr in both trials and values declined toward baseline thereafter. However, the change in plasma glucose between baseline and three hours, and the AUC for plasma glucose over four measurements did not differ between treatments.

HDL-cholesterol (Figure 1A, Table 2) had a significant treatment by time interaction in the ANOVA analyses ($p = .011$). Compared with the baseline fasting value, HDL cholesterol was significantly lower at 1, 2 and 3 hr after the meal ($p < .0001$) in both treatments. HDL cholesterol did not decrease as much in the light physical activity treatment as it did in the sitting treatment (4.8% vs. 8.4% decrease from baseline), however the treatment effect was not significant. The Δ between post 3 hr-baseline HDL cholesterol was significantly greater in sitting (Δ = −3.7 ± 2.4 mg/dl) than in walking (Δ = −2.2 ± 2.5 mg/dl, $t = −2.496$, $p = .023$). While changes in HDL between baseline and three hours differed between treatments, across all four measurements, the AUC for HDL cholesterol did not differ between treatments (Table 3).

Log transformed measures of insulin concentration (Figure 1B, Table 2 show untransformed values) also demonstrated significant treatment by time interactions; following a peak value at 1-hr, insulin concentration in the light physical activity treatment returned closer to baseline values by 3 hr than in the sitting treatment.
Figure 1 — Effect of light physical activity (LPA) and sitting treatment on postprandial concentrations of A) HDL cholesterol, B) insulin (untransformed), C) triglyceride. Data are presented as mean ± SE. Note. 8 denotes difference from baseline. § denotes difference between light physical activity and sitting treatment.
Each time point was significantly different from every other time point ($p < .0001$), however no treatment effect was observed. At 3 hr, insulin remained 1.5 times above baseline (i.e., 148%) in the light physical activity treatment but was 4.2 times above baseline (i.e., 418%) in the sitting treatment and Δ log-transformed insulin (sitting $Δ = 1.87 \pm 0.67 \mu$IU/ml, walking $Δ = 1.10 \pm 0.77 \mu$IU/ml, $t = 4.351, p < .0001$) was significantly different between treatments. While changes in plasma insulin between baseline and three hours differed between treatments, across all four measurements, the AUC for plasma insulin did not differ between treatments (Table 3).

Triglyceride concentration (Figure 1C, Table 2) had a significant treatment by time interaction. Triglyceride at 1 hr and 2 hr were significantly elevated relative to the fasting baseline ($p = .001$). However, the main effect of treatment was not significant. Between treatment comparisons across time revealed that triglyceride concentration was higher in the light physical activity treatment: 10% higher at 1 hr and 17% higher at 2 hr, when compared with sitting values at the same time point. However at 3 hr, the triglyceride concentration in the light physical activity treatment had dropped lower (2%) than the sitting treatment. Neither the Δ or AUC (Table 3) analyses demonstrated differences between treatment.

Changes over time in LDL and in the LDL-HDL ratio did not differ between treatments. Neither measure demonstrated Time × Treatment interaction in ANOVA models nor between-treatment differences in AUC.

**Discussion**

To our knowledge, this study is the first to report how light physical activity affects cardiometabolic-risk-factor responses following a typical meal consumed by adolescents, as most previous research focused on moderate and vigorous activity (19,35,36). The primary findings of this pilot study in adolescents support most (9,13,25) but not all (14) previous investigations in adults that participated in light physical activity via slow walking (1.5 mph). There were small differences in specific outcomes, insulin and HDL cholesterol, but overall exposure (AUC) was not significantly altered by light physical activity. In addition, we did not find any light-physical-activity response for LDL cholesterol, glucose, or endothelial function.

We found that insulin returned closer to baseline and HDL cholesterol decreased to a lesser degree during the 3 hr after the meal, when participants walked for 45 min per hour rather than remaining seated. However, the integrated response was not different by treatment, which tempers the support of light physical activity as an intervention to decrease postprandial-disease risk in adolescents. While the post 3 hr triglycerides were slightly lower in the light physical activity treatment versus the sitting treatment, the overall integrated response was higher, albeit not significantly so. It is unknown if the triglyceride response would have continued to drop in the light physical activity treatment more than the sitting treatment if this examination period had been longer (i.e., 4–6 hr rather than 3). The precise cause of this effect is not completely understood and the implications for health are not completely clear; perhaps, showing that the increase in energy expenditure associated with light physical activity could promote faster lipid metabolism. Evidence does support the health-enhancing effect of activity of at least moderate intensity to decrease postprandial triglycerides in adults (4,14,15) and adolescents (5,19,36). Activity at higher intensities did not show a dose response in healthy
adolescent boys (36). Further, there was no difference in the degree of decrement in postprandial triglycerides if the activity was accumulated in a single large bout or smaller intermittent bouts (5).

No previous studies, to our knowledge, have determined whether light physical activity performed after a typical meal can have a beneficial effect on changes in cardiometabolic disease risk factors in adolescents. Acute participation in moderate physical activity (53–65% VO₂peak) has been associated with lower postprandial triglycerides in adolescents (19,35,36). In adults, performing moderate to vigorous exercise before or immediately after a meal clearly has a healthful impact on postprandial cardiometabolic disease risk factors, as shown by attenuated responses in triglycerides (14,24), insulin (24), and endothelial function (26). Four studies in adults have examined light physical activity on postprandial metabolism (9,13,14,25). Two of the three used a high-carbohydrate meal, followed by either 30 min of cycling at ~70% HR max (13) or 15 and 40 min of slow, self-paced walking described as eliciting a “very light” rating of perceived exertion (25), respectively. A third, recently published trial used a high-fat meal (50 g fat, 75 g carbohydrate, 3195 kJ, approximately 59% fat; 9), followed on one day by 2-min walking bouts at 2 mph every 20 min for 5 hr (~28 total minutes of walking; 9). On a second day, the same participants walked on the same schedule at moderate intensity (3.5–4.0 mph). The postprandial elevations in glucose and insulin were blunted by both the light and moderate physical activity treatments when compared with a control day in which the participants remained seated throughout the postprandial period (9). Only one of the four studies reported that postprandial triglycerides and insulin responses to a high-fat meal followed by light physical activity were not different (238 min at ~25% peak VO₂) than sitting (14). Notably, in that study the measurement time lasted 8 hr. Although, immediately after the meal, the light physical activity treatment showed significantly lower insulin response, there was no difference at other time points or the integrated response (14), whereas significant benefits of physical activity were evident in studies using shorter observation periods ranging from 2 hr (13,25) to 5 hr (9).

The current findings should be interpreted in light of their strengths and limitations. This study is the first of its kind to examine the effect of light physical activity on postprandial lipid and glucose metabolism in adolescents. This was a pilot study and therefore a relatively small sample size was used (N = 18). Although all participants were past Tanner Stage 2 (21,22), the wide age range (10–18 years old) may have contributed to the modest and nonsignificant differences between treatments. It is also possible that if the fat content of the meal was higher, a greater metabolic perturbation would have occurred; and as a result, the magnitude of impact of the light physical activity program would have also been larger. Our test meal contained 20.5g of fat (32% of energy content). Other studies have demonstrated significant changes in triglycerides with meals containing 30–50 g of fat (18). However, we selected the meal composition so that it was similar to current guidelines for school lunch and therefore the results might be generalizable to the majority of school-age adolescents. Another possible limitation was that our postprandial observation period was 3 hr, but differences in triglycerides in some high-fat meal studies did not emerge until at least 4 hr after eating (17). In the current study, however, triglyceride concentration was highest at 1–2 hr after the meal and had returned almost to baseline (fasting) level by 3 hr on both treatment days.
Thus, it seems unlikely that there would be a divergence in triglyceride concentration between treatments beyond the observation time that was used. This is also true for HDL-C and insulin. We acknowledge that diet and physical activity were not tightly controlled in the days before treatment visits—although, we confirmed that all participants refrained from food, beverage (except water), and exercise for a minimum of 8 hr before the treatment. Finally, the activity intensity during the walking treatment was not objectively confirmed via measurement of gas exchange during treatment. However, objective measurement of physical activity via waist-mounted accelerometer indicated the slow walking was indeed light intensity.

The clinical relevance of our study is that following a typical meal, adolescents’ cardiometabolic risk factors may be sensitive to improvement with a light-physical-activity intervention, although the effects were small and likely not clinically meaningful. In the current study, all participants were considered healthy. Further work is therefore needed to determine if light activity could be effectively used with at-risk populations, such as overweight adolescents who are at increased risk for cardiometabolic disease risk factor clustering (7) and have impaired glucose and lipid metabolism (19). Walking for 45 min of each hour for 3 hr following a meal is not an intervention that can be readily translated into classroom settings, but in this efficacy trial, our goal was to test the hypothesis that a large dose of light physical activity would benefit adolescent health. These findings can lead the way for future investigations and studies designed to understand more appropriate interventions for widespread implementation. More tightly controlled efficacy studies exploring dose response and variations in intensity and potential vulnerable, at-risk populations are prudent and needed before intervention recommendations can be generally applied to healthy or obese adolescents.

In conclusion, light physical activity minimally mitigated some of the undesirable changes in cardiometabolic risk factors following a typical meal in some analyses. Specifically, slow walking for 45 min per hour resulted in lower insulin concentration and less decline in HDL cholesterol measured 3 hr after the meal—although responses were small in this sample of healthy children of variable ages. Our findings are in agreement with prior studies performed with adults who performed light physical activity following a meal that insulin may be sensitive to light physical activity (9,13,25). Our findings do not support previous studies with adolescents that used moderate-to-vigorous exercise (19,35,36), which suggest that intensity of activity may be important. Light physical activity did not mitigate the change in postprandial triglycerides, LDL cholesterol, glucose, or endothelial function. The current investigation is, to our knowledge, the first to report the impact of light physical activity on postprandial responses following a meal in adolescents. Although the benefits of slow walking were statistically significant, the physiological effects were modest in this group of generally healthy adolescents.

Acknowledgments

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Conflict of Interest

The authors have no conflicts of interest to declare.

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