Lack of Effect of a High-Calorie Dextrose or Maltodextrin Meal on Postprandial Oxidative Stress in Healthy Young Men

Kelsey H. Fisher-Wellman and Richard J. Bloomer

**Background:** Carbohydrate powder in the form of maltodextrin is widely used by athletes for postexercise glycogen resynthesis. There is some concern that such a practice may be associated with a postprandial rise in reactive oxygen and nitrogen species production and subsequent oxidation of macromolecules. This is largely supported by findings of increased oxidative-stress biomarkers and associated endothelial dysfunction after intake of dextrose.

**Purpose:** To compare the effects of isocaloric dextrose and maltodextrin meals on blood glucose, triglycerides (TAG), and oxidative-stress biomarkers in a sample of young healthy men. **Methods:** 10 men consumed isocaloric dextrose and maltodextrin powder drinks (2.25 g/kg) in a random-order, crossover design. Blood samples were collected premeal (fasting) and at 1, 2, 4, and 6 hr postmeal and assayed for glucose, TAG, malondialdehyde, hydrogen peroxide, nitrate/nitrite, and Trolox-equivalent antioxidant capacity. **Results:** Significant meal effects were noted for glucose total area under the curve ($p = .004$), with values higher for the dextrose meal. No other statistically significant meal effects were noted ($p > .05$). With respect to the 2 (meal) × 5 (time) ANOVA, no significant interaction, time, or meal effects were noted for any variable ($p > .05$), with the exception of glucose, for which a main effect for both meal ($p < .0001$) and time ($p = .0002$) was noted. **Conclusions:** These data indicate that carbohydrate meals, consumed as either dextrose or maltodextrin, pose little postprandial oxidative insult to young, healthy men. As such, there should be minimal concern over such feedings, even at high dosages, assuming adequate glucose metabolism.

**Keywords:** carbohydrate metabolism, free radicals, reactive oxygen species, lipid peroxidation

Acute ingestion of protein (Mohanty et al., 2002), lipid (Mohanty et al., 2002), and carbohydrate administered alone (Mohanty et al., 2000) or in a wide variety of lipid- and carbohydrate-rich meals (Sies, Stahl, & Sevanian, 2005) has been shown to promote an increase in the formation of reactive oxygen and nitrogen species (RONS). This nutrient-induced increase in RONS is referred to as postprandial oxidative stress and appears to be mediated in part by the degree of elevation in both triglycerides (TAG; Bae et al., 2001; Bloomer, Ferebee, Fisher-Wellman, Quindry, & Schilling, 2009) and glucose (Monnier et al., 2006) after feeding. Both hypertriglyceridemia and hyperglycemia have been suggested to be independent risk factors for the development of diabetes (Ceriello, 2005; Pastromas, Terzi, Tousoulis, & Koulouris, 2007) and cardiovascular disease (O’Keefe & Bell, 2007), apparently because such conditions can initiate a harmful biochemical cascade throughout the circulation, inducing inflammation, endothelial dysfunction, hypercoagulability, and sympathetic hyperactivity, all of which may promote further RONS generation and oxidative damage (O’Keefe & Bell, 2007). Thus, repeated exposure to postprandial RONS generation may be a causal factor in the development of ill health or disease (Houstis, Rosen, & Lander, 2006).

With respect to hyperglycemia-induced oxidative damage, numerous investigators have reported an increase in various markers of oxidative stress after carbohydrate intake (Ceriello et al., 2002; Lee, Kim, & Bae, 2002; Miyazaki et al., 2007; Mohanty et al., 2000; Sampson, Gopaul, Davies, Hughes, & Carrier, 2002; Serin, Konukoglu, Firtina, & Mavis, 2007; van Oostrom et al., 2003). These findings have been observed in both diseased (Ceriello et al., 2002; Lee et al., 2002; Miyazaki et al., 2007; Sampson et al., 2002; Serin et al., 2007) and healthy (Ceriello et al., 2002; Miyazaki et al., 2007; Mohanty et al., 2000; van Oostrom et al., 2003) individuals alike, with the diseased experiencing an exacerbated response (Ceriello et al., 2002; Miyazaki et al., 2007; Serin et al., 2007). In either case, the resultant oxidative insult appears contingent on the magnitude and duration of glycemia experienced postprandially, because significant positive correlations between blood glucose and oxidative-stress biomarkers have previously been observed (Cavalot et al., 2006; Monnier et al., 2006).

In addition to the assessment of oxidative stress, several studies also incorporated measures of postprandial endothelial dysfunction in their study design.
Endothelial dysfunction refers to a decrease in nitric oxide (NO)-mediated vasodilation (Cai & Harrison, 2000) and is commonly assessed by a reduction in flow-mediated dilation (Al-Qaisi, Kharbanda, Mittal, & Donald, 2009) or in the plasma concentration of nitrate/nitrite (NOx; Kawano et al., 1999; Neri et al., 2005). In either case, the reduction in flow-mediated dilation and NOx is believed to result from the interaction of NO with superoxide in the vasculature, which forms the harmful peroxynitrite radical at the expense of decreasing NO bioavailability (Ceriello, 2003). Similar to that observed with oxidative stress, a reduction in flow-mediated dilation (Ceriello et al., 2002; Kawano et al., 1999; Lee et al., 2002; Title et al., 2000; Cummings, Giddens, & Nassar, 2000; Zhu, Zhong, Yu, & Li, 2007) and NOx (Weiss et al., 2004) has been observed after an oral glucose tolerance test (OGTT) in both diseased (Ceriello et al., 2002; Kawano et al., 1999; Lee et al., 2002) and healthy (Ceriello et al., 2002; Kawano et al., 1999; Lee et al., 2002; Title et al., 2000; van Oostrom et al., 2003; Zhu et al., 2007) individuals, with diseased individuals experiencing an exacerbated response (Ceriello et al., 2002; Kawano et al., 1999). Moreover, significant positive correlations have also been reported between endothelial dysfunction and postprandial glycemia (Kawano et al., 1999; Title et al., 2000).

Taken together, the deleterious effects associated with carbohydrate meals appear to depend on the ability of such meals to induce an increase in blood glucose. These data are in relation to an OGTT, which is commonly used in clinical settings as a diagnostic tool for classifying diabetes or prediabetes. It involves 75 g of dextrose in solution administered orally. After administration, postprandial glucose clearance is measured for a period of 2 or more hours and results are used to diagnose diabetes. Dextrose is a naturally occurring form of glucose with a dextrose equivalent of 100 (Chronakis, 1998). The dextrose equivalent is a measure of the total reducing power of all sugars present in a substance relative to dextrose (Chronakis, 1998).

Maltodextrin is a complex carbohydrate source (chain of glucose polymers) made from corn starch and is defined as having a dextrose equivalent less than 20 (Chronakis, 1998). It is widely used in the food industry as a texture modifier, as well as a fat substitute (Chronakis, 1998). Moreover, maltodextrin is readily used in the sports nutrition industry as a carbohydrate supplement in the hope of maximizing glycogen storage (i.e., “carbohydrate loading”; Jeukendrup & Jentjens, 2000). In such cases, it is not uncommon for endurance athletes to consume as much as 220 g of maltodextrin before (Wright, Sherman, & Dernbach, 1991) and during (Jeukendrup & Jentjens, 2000) prolonged exercise, as well as during the postexercise period to assist with glycogen resynthesis (Friedman, Neuffer, & Dohm, 1991; Piehl Aulin, Soderlund, & Hultman, 2000). Ingesting such high dosages of maltodextrin may be expected to result in a significant glycemic response and thus induce a postprandial oxidative insult, albeit to a lesser degree than that of dextrose.

To our knowledge, no study has compared the postprandial oxidative-stress response induced by dextrose and maltodextrin. Therefore, the purpose of the current investigation was to compare the effects of isocalorically administered dextrose and maltodextrin meals on the induction of postprandial oxidative stress in a sample of young healthy men. We hypothesized that both carbohydrate meals would result in an increase in oxidative stress, with greater responses observed after the dextrose meal.

**Methods**

**Participants**

Ten young, healthy men were recruited from the University of Memphis and the surrounding community. All participants were nonsmokers, normolipidemic (fasting triglycerides < 200 mg/dl), not using antioxidant supplements or drugs, and free of any cardiovascular or metabolic disorders. For a description of participant characteristics, please see Table 1.

Health history and drug and dietary supplement use were provided, and physical activity questionnaires were completed by all participants to determine eligibility. Before participation, each participant was informed of all procedures, potential risks, and benefits associated with the study in both verbal and written form in accordance with the procedures approved by the university institutional review board for research on human subjects. Participants signed an informed-consent form before being admitted into the study.

During the initial visit to the laboratory, participants completed the informed-consent form and the health and physical activity questionnaires. Their height, weight, and body composition (seven-site skinfold assessment) were measured. Heart rate and blood pressure were recorded after a 10-min period of quiet rest. A full explanation of dietary data recording was provided, along with data-collection forms. An overview of study procedures was also provided.

**Table 1 Descriptive Characteristics of Participants, M ± SD**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>27 ± 7</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>180 ± 9</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>81 ± 19</td>
</tr>
<tr>
<td>Body-mass index (kg/m²)</td>
<td>25 ± 4</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>15 ± 6</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>83 ± 12</td>
</tr>
<tr>
<td>Hip circumference (cm)</td>
<td>102 ± 10</td>
</tr>
<tr>
<td>Waist:hip</td>
<td>0.82 ± 0.06</td>
</tr>
<tr>
<td>Resting heart rate (beats/min)</td>
<td>67 ± 19</td>
</tr>
<tr>
<td>Resting systolic blood pressure (mm Hg)</td>
<td>124 ± 12</td>
</tr>
<tr>
<td>Resting diastolic blood pressure (mm Hg)</td>
<td>71 ± 7</td>
</tr>
</tbody>
</table>
Meal Testing

All participants reported to the laboratory in the morning after a 10-hr overnight fast. They rested for 10 min and then a premeal blood sample was collected. Participants then consumed one of two carbohydrate meals, in random order, separated by 1 week. The meals were composed of dextrose (NOW Foods, Bloomingdale, IL) or maltodextrin powder (Carbo Gain; NOW Foods, Bloomingdale, IL). The size of the meals was based on participant body mass and was equivalent to 2.25 g of carbohydrate per kilogram of body mass. This provided an average caloric load of approximately 730 kcal per participant. Portions were weighed on a laboratory-grade balance and mixed with water in a blender. All meals were isovolumetric and contained approximately 10 ml of fluid per kilogram of body mass.

The postprandial observation period lasted 6 hr. Participants remained in the laboratory during this period and expended as little energy as possible. No additional meals or calorie-containing beverages were allowed during this period. Water was allowed ad libitum.

Blood Sampling

Venous blood samples (~20 ml) were taken from each participant’s forearm via needle and Vacutainer by a trained phlebotomist. Blood samples were collected premeal (0 hr) and 1, 2, 4, and 6 hr postprandial. After collection, blood samples were immediately processed and then centrifuged at 2,000 g, and the plasma/serum was stored at –80 °C until analyzed. All blood samples were assayed for glucose, TAG, malondialdehyde, hydrogen peroxide, Trolox-equivalent antioxidant capacity, and NOx.

Assays for glucose and TAG were performed following standard enzymatic procedures described by the reagent manufacturer (Thermo Electron Clinical Chemistry). Standard curves for all assays were developed for determination of unknown samples, and known normal and abnormal samples were used for quality-control purposes. Malondialdehyde was analyzed in plasma using a commercially available colorimetric assay (Northwest Life Science Specialties, Vancouver, WA) with previously described methods (Jentzsch, Bachmann, Furst, & Biesalski, 1996). Hydrogen peroxide was measured in plasma using the Amplex Red reagent method as described by the manufacturer (Molecular Probes, Invitrogen Detection Technologies, Eugene, OR). Antioxidant capacity was measured in serum using the Trolox-equivalent antioxidant capacity assay with procedures outlined by the reagent provider (Sigma Chemical, St. Louis, MO). NOx was measured using a commercially available assay (Caymen Chemical, Ann Arbor, MI). All assays were performed in duplicate on first thaw.

Dietary Records

All participants were instructed to maintain their normal diet and record their food and beverage intake during the 6-day period before each test-meal day. Nutritional records were analyzed for total calories, protein, carbohydrate, fat, and a variety of micronutrients (Food Processor SQL, version 9.9, ESHA Research, Salem, OR). Participants were instructed to maintain their normal physical activity habits during the 6 days before each test-meal day. They were given specific instructions regarding abstinence from alcohol consumption, in addition to avoidance of strenuous exercise during the 24 hr immediately before the test-meal day, because such activity may have affected the chosen biomarkers, as previously reported (McClean et al., 2007).

Statistical Analysis

For all biochemical variables, area under the curve (AUC) was calculated using the trapezoidal method as described in detail by Pruessner, Kirschbaum, Meinlschmidt, and Hellhammer (2003). All AUC data were analyzed using a one-way analysis of variance (ANOVA). In addition, all variables were analyzed using a 2 (meal) × 5 (time) repeated-measures ANOVA. Significant interactions and main effects were further analyzed using Tukey’s post hoc tests when necessary. Dietary variables were analyzed using a t test. All analyses were performed using JMP statistical software (version 4.0.3, SAS Institute, Cary, NC). Statistical significance was set at \( p \leq .05 \). The data are presented as \( M \pm \text{SEM} \), except for participants’ descriptive characteristics, which are presented as \( M \pm SD \).

Results

No differences were noted between meals for total kilocalorie intake or grams and percentage of protein, carbohydrates, or fat, as well as vitamin A, vitamin C, or vitamin E consumption \( (p > .05) \). Data are presented in Table 2.

Significant meal effects were noted for glucose AUC \( (p = .04) \), with greater values after the dextrose meal. No statistically significant meal effects were noted for the AUC for TAG \( (p = .92) \), malondialdehyde \( (p = .75) \), NOx \( (p = .72) \), hydrogen peroxide \( (p = .79) \), or Trolox-equivalent antioxidant capacity \( (p = .18) \). AUC data are presented in Table 3.

### Table 2 Dietary Data of Participants During 6 Days Before Each Test Meal, \( M \pm \text{SEM} \)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Dextrose</th>
<th>Maltodextrin</th>
</tr>
</thead>
<tbody>
<tr>
<td>kcal</td>
<td>2,274 ± 158</td>
<td>2,219 ± 135</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>84 ± 7</td>
<td>85 ± 7</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>292 ± 21</td>
<td>307 ± 22</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>85 ± 9</td>
<td>69 ± 6</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>91 ± 16</td>
<td>84 ± 21</td>
</tr>
<tr>
<td>Vitamin E (mg)</td>
<td>12 ± 5</td>
<td>9 ± 3</td>
</tr>
<tr>
<td>Vitamin A (retinol equivalents)</td>
<td>4,240 ± 845</td>
<td>4,351 ± 1,699</td>
</tr>
</tbody>
</table>

Note. No statistically significant differences noted \( (p > .05) \).
With respect to the 2 (meal) × 5 (time) ANOVA, no significant interaction, time, or main effects were noted for any variable \((p > .05)\), with the exception of glucose, for which main effects for both meal \((p < .0001)\) and time \((p = .0002)\) were reported. Specifically, glucose was higher for dextrose than for maltodextrin, and values were lower 2, 4, and 6 hr postmeal than 1 hr postmeal \((p < .05)\). Data for each variable are presented in Figures 1–6 (Figure 1, glucose; Figure 2, TAG; Figure 3, malondialdehyde; Figure 4, NOx; Figure 5, hydrogen peroxide; and Figure 6, Trolox-equivalent antioxidant capacity), with \(p\) values for all main effects and interactions presented in Table 4.

### Table 3 Postprandial Area-Under-the-Curve Data After a Dextrose and Maltodextrin Meal in Young Healthy Men, \(M \pm SEM\)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Dextrose</th>
<th>Maltodextrin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malondialdehyde (μmol · L⁻¹ · 6 hr⁻¹)</td>
<td>6.84 ± 1.48</td>
<td>6.30 ± 0.86</td>
</tr>
<tr>
<td>Hydrogen peroxide (μmol · L⁻¹ · 6 hr⁻¹)</td>
<td>77.68 ± 24.32</td>
<td>71.98 ± 10.50</td>
</tr>
<tr>
<td>Triglyceride (mg · dl⁻¹ · 6 hr⁻¹)</td>
<td>245.47 ± 53.93</td>
<td>251.65 ± 36.73</td>
</tr>
<tr>
<td>Glucose (mg · dl⁻¹ · 6 hr⁻¹)</td>
<td>468.04 ± 28.52*</td>
<td>365.80 ± 21.26</td>
</tr>
<tr>
<td>TEAC (mmol · L⁻¹ · 6 hr⁻¹)</td>
<td>3.00 ± 0.19</td>
<td>3.40 ± 0.28</td>
</tr>
<tr>
<td>Nitrate/Nitrite (μmol · L⁻¹ · 6 hr⁻¹)</td>
<td>112.75 ± 23.74</td>
<td>108.97 ± 15.47</td>
</tr>
</tbody>
</table>

*Note. TEAC = Trolox-equivalent antioxidant capacity.

*Significant meal effect for glucose \((p = .04)\).

**Discussion**

We found no significant increase in postprandial oxidative stress in response to high-dose dextrose or maltodextrin meals consumed by young, healthy men. Although we did note an overall lower blood glucose response for the maltodextrin meal, this was not associated with any reduction in oxidative stress.

With respect to both meals, we found no increase in our selected markers of oxidative stress, nor did we note any decrease in NOx, a commonly measured biochemical marker of endothelial dysfunction. We also failed to note any significant increase in blood glucose after feeding.
Although it is possible that we may have missed the peak in blood glucose by not including additional measurements between the premeal and 1-hr time points, we do not feel that this had any significant impact on our chosen biomarkers of oxidative stress. In support of our position, we have found in our work that although blood glucose is higher at 30 versus 60 min after ingestion of a dextrose meal, oxidative-stress biomarkers do not peak until 1–2 hr postingestion (unpublished data). Therefore, because oxidative-stress biomarkers were of main interest in the current study, we did not see any reason to include an additional blood sample 30 min postingestion.

Although it is possible that we may have missed the peak in blood glucose by not including additional measurements between the premeal and 1-hr time points, we do not feel that this had any significant impact on our chosen biomarkers of oxidative stress. In support of our position, we have found in our work that although blood glucose is higher at 30 versus 60 min after ingestion of a dextrose meal, oxidative-stress biomarkers do not peak until 1–2 hr postingestion (unpublished data). Therefore, because oxidative-stress biomarkers were of main interest in the current study, we did not see any reason to include an additional blood sample 30 min postingestion.

The aforementioned null findings for both meals were surprising, given the relatively large dose of carbohydrate administered. In the current investigation, both meals were administered relative to the participant’s body mass and provided 2.25 g of carbohydrate per kilogram of body mass (mean dose = 182 g carbohydrate, 728 kcal). This dose represents a greater than twofold increase in carbohydrate load compared with previous studies reporting an increase in oxidative stress and endothelial dysfunction using an OGTT, which provides 75 g of carbohydrate (300 kcal). We chose this dose in an effort to mimic that used by athletes during carbohydrate supplementation, in which the ingestion of as much as 200 g of carbohydrates before, during, and after exercise is not uncommon (Friedman et al., 1991; Jeukendrup & Jentjens, 2000; Piehl Aulin et al., 2000; Wright et al., 1991). The discrepancies between our data and those of other investigators can likely be primarily attributed to the age and health status of our participant population; young (27.3 ± 7.0 years), healthy men were used in the current design. It seems that in this population, rapid
clearance of blood glucose occurs before the development of any significant increase in RONS formation. Therefore, ingestion of relatively large doses of rapidly or more slowly digestible carbohydrate sources as a supplementation strategy for young, healthy individuals does not appear to induce any deleterious effects in terms of postprandial oxidative stress. Although this sample size was similar to that used by other investigators in the field, the recruitment of additional participants would certainly have increased statistical power. This is a limitation of the current investigation, and caution should be exercised when attempting to generalize these findings to other populations.

In opposition to our findings, other investigators have reported an increase in various markers of oxidative stress after an OGTT in healthy, insulin-sensitive participants (Ceriello et al., 2002; Miyazaki et al., 2007; Mohanty et al., 2000; van Oostrom et al., 2003). However, most of those investigations were carried out in older populations (53.5 ± 2.5 years, Ceriello et al., 2002; 68 ± 12 years Miyazaki et al., 2007). It is possible that the increased age of the participants in the studies was responsible for the observed increase in oxidative stress, because insulin secretion (Ioizzo et al., 1999), beta-cell function (Chiu, Lee, Cohan, & Chuang, 2000), and oxidative stress (Lee & Wei, 2007) all appear to be influenced by aging. Furthermore, although a decrease in thioredoxin (a critical thiol-disulfide oxidoreductase; Prinz, Aslund, Holmgren, & Beckwith, 1997) was reported by Miyazaki et al., it was not accompanied by any significant increase in oxidative damage to DNA (assessed via 8-hydroxydeoxyguanosine) in healthy participants.

In reference to younger populations, Mohanty et al. (2000) reported an increase in leukocyte superoxide production in young (25–42 years), healthy participants after an OGTT. However, this increase in superoxide did not result in any significant increase in lipid peroxidation (thiobarbituric acid-reactive substances). In a similar study, an increase in total lipid hydroperoxides was reported after an OGTT in young, healthy individuals (van Oostrom et al., 2003). In support of our findings, two other studies reported no increase in oxidative stress after an OGTT in younger, healthy men (47.3 ± 13.7 years, Serin et al., 2007; 22.6 ± 2.3 years, Zhu et al., 2007).

Regarding NOx, only two studies to our knowledge have included this measure as a biochemical surrogate of endothelial function by way of OGTT-induced changes in NOx, noting no change (Kawano et al., 1999), as well as a postprandial decrease (Weiss et al., 2004). However, the age of the participant population used by Weiss et al. was 58 ± 1 year. Therefore, our null findings in relation to NOx were likely a result of the use of young, healthy participants. It is possible that in our participants, NOx was maintained as a consequence of the rapid clearance of blood glucose in the vasculature, thus preventing any prolonged production of superoxide.

Regarding the maltodextrin meal, the observed attenuation in blood glucose was expected based on the assumed disparities in dextrose equivalence between the two meals (dextrose = 100; maltodextrin ≤ 20). Both dextrose and maltodextrin are regarded as easily digestible carbohydrates; however, the complex structure and lower dextrose-equivalent value for maltodextrin were likely responsible for this blunted glycemic response. Although an attenuation in blood glucose would also be expected to blunt the formation of RONS (Monnier et al., 2006) during the postprandial period, neither meal promoted a significant increase in postprandial oxidative stress. Therefore, we did not observe any significant difference between the dextrose and maltodextrin meals with regard to the oxidative-stress biomarkers. As stated previously, we believe that the lack of increase in our chosen biomarkers is related to our use of young, healthy men in the study design. It is possible that in this population, the maintenance of sufficient beta-cell function and insulin secretion allowed for rapid clearance of blood glucose after feeding. In addition, insulin has been shown to attenuate postprandial RONS production and inflammation after an OGTT in healthy participants (Dandona et al., 2001). However, no measures of insulin sensitivity or beta-cell function were included in the current design; this is a limitation of the study.

**Conclusion**

The current findings indicate that using either dextrose or maltodextrin powder as a carbohydrate supplement likely exerts no deleterious effects in terms of postprandial oxidative stress in young, healthy men. This is true despite what might be considered an excessive intake of carbohydrate in a single meal (i.e., 2.25 g/kg body mass). It is likely that efficient carbohydrate metabolism, inclusive of enhanced insulin sensitivity and responsiveness (Monnier et al., 2006), in such participants is responsible for these results. Therefore, based on our findings coupled with those of other investigators, it appears that glucose may only pose a significant threat in terms of RONS production and subsequent oxidative damage when it remains stagnant in the vasculature for an extended period of time, perhaps as a consequence of insulin resistance or glucose intolerance. However, this statement needs to be substantiated via additional related work, because we did not include any measures of insulin sensitivity in the current design. Moreover, the relatively small sample size and potential lack of statistical power should be considered when attempting to generalize these findings to other populations.

**Acknowledgments**

Both authors were involved in study design, data collection, and data analysis. Manuscript preparation was primarily carried out by K.F.W., with assistance from R.B. Both authors reviewed and approved of the final draft. The authors declare no conflicts of interest.
References


