Effects of meals rich in either monounsaturated or saturated fat on lipid concentrations and on insulin secretion and action in subjects with high fasting triglyceride concentrations\(^1\)–\(^4\)

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ABSTRACT

Background: The nature of dietary fats and fasting concentrations of triglycerides affect postprandial hypertriglyceridemia and glucose homeostasis.

Objectives: The objectives were to examine the effects of meals enriched in monounsaturated fatty acids (MUFAs) or saturated fatty acids (SFAs) on postprandial lipid, glucose, and insulin concentrations and to examine the extent of \(\beta\) cell function and insulin sensitivity in subjects with high fasting triglyceride concentrations.

Design: Fourteen men with fasting hypertriglyceridemia and normal glucose tolerance were given meals (~10 kcal/kg body weight) containing MUFAs, SFAs, or no fat. Blood samples were collected at baseline and hourly over 8 h for analysis.

Results: The high-fat meals significantly increased postprandial concentrations of triglycerides, nonesterified fatty acids, and insulin and postprandial indexes of \(\beta\) cell function. However, postprandial indexes of insulin sensitivity decreased significantly. These effects were significantly attenuated with MUFAs relative to SFAs.

Conclusions: MUFAs postprandially buffered \(\beta\) cell hyperactivity and insulin intolerance relative to SFAs in subjects with high fasting triglyceride concentrations. These data suggest that, in contrast with SFAs, MUFAs-based strategies may provide cardiovascular benefits to persons at risk by limiting lipid and insulin excursions and may contribute to optimal glycemic control after meal challenges. *Am J Clin Nutr* 2011;93:494–9.

INTRODUCTION

Fasting hypertriglyceridemia is one of the risk factors for the metabolic syndrome, which is a cluster of metabolic abnormalities related to insulin resistance and the risk of development of type 2 diabetes (1). The Third Report of the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) (2) has advised the use of lower cutoffs for the categorization of fasting triglycerides than the ATP II guidelines (3), which reflects a growing awareness of the importance of even moderate increases in triglyceride concentrations. Recently, it was postulated that insulin resistance might also be involved in the acute metabolism of dietary fats (4). Exaggerated nonfasting concentrations of triglycerides, via higher peak concentrations or delayed clearance, is an inherent feature of diabetic dyslipidemia and is frequently found even in diabetic patients with normal fasting triglycerides (5). In the ATP III recommendations, fasting hypertriglyceridemia management recapitulates different lines of therapy, including lifestyle changes and triglyceride-lowering drugs. However, less attention has been devoted to nonfasting hypertriglyceridemia, despite growing recognition that postprandial triglyceride concentrations may be more significant than fasting concentrations in the assessment of cardiovascular disease risk (6).

Postprandial triglyceride concentrations are highly influenced by fasting concentrations and by the nature of the dietary fats in the meal (7). In healthy subjects, there is also consistent evidence that the early insulin response becomes less pronounced in the postprandial state as the proportion of saturated fatty acids (SFAs) with respect to monounsaturated fatty acids (MUFAs) in dietary fats increases (8). Therefore, we sought to test the hypothesis that MUFAs, when compared with SFAs, can improve physiologic parameters of postprandial lipid and glucose metabolism in subjects with high fasting triglyceride concentrations. These findings could be of clinical relevance for the development of prevention strategies in the population at risk of type 2 diabetes and prediabetic states. We conducted a single-blind, randomized, within-subject crossover study to determine the acute effects of 2 dietary fats rich in MUFAs (olive oil) or SFAs (butter) on plasma lipids [triglycerides and nonesterified fatty acids (NEFAs)], glucose, and insulin. The effects of MUFAs and SFAs on the extent of \(\beta\) cell function and insulin sensitivity were also examined.

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SUBJECTS AND METHODS

Subjects and design

Ethics approval was obtained from the Human Clinical Research and Ethics Committee of the University Hospital Virgen del Rocío (UHVR, Seville), and the study complied with the current revision of the Declaration of Helsinki. All subjects gave written informed consent. Recruitment for this study began in January 2008.

We recruited 14 men with newly diagnosed type IIb or IV hyperlipoproteinemia (fasting triglycerides >200 mg/dL), according to the NCEP ATP III (2), with a mean (±SD) age of 33 ± 7 y, BMI (in kg/m²) of 24.2 ± 5.1, and normal glucose tolerance after a 75-g oral-glucose-tolerance test. They were required to have no evidence of established coronary heart disease and were excluded if there was any evidence of renal impairment, hypothyroidism, or liver dysfunction based on clinical chemistry testing. None of the subjects used tobacco, consumed special diets, or took medication known to alter gastric emptying, lipoprotein metabolism, insulin secretion, or insulin activity.

The study was designed as a within-subject crossover in which the subjects attended the Clinic Experimental Research Unit for Vascular Risk at the UHVR on 3 separate occasions. The investigators were blinded throughout the study. Fasting blood samples (t = 0) were taken at 0800 after a 12-h overnight fast. The test meals were given in random order with an interval of 1 wk between meals. The meals consisted of a portion of plain pasta (30 g/m² body surface area), one slice of brown bread, one skim yogurt, and either olive oil or butter (50 g/m² body surface area). The average total energy provided by the high-fat meals was ≈800 kcal (=10 kcal/kg), with a macronutrient profile of 72% fat, 22% carbohydrate, and 6% protein (8). The fatty acid composition was 14.9% SFAs, 81.0% MUFAs, and 4.1% polyunsaturated fatty acids (PUFAs) for the meals enriched with olive oil and 65.3% SFAs, 31.3% MUFAs, and 3.4% PUFAs for the meals enriched with butter (7). The subjects also consumed a test meal containing the same foods, but no fat, as a control meal. After the ingestion of the meals, blood samples were collected each hour in tubes containing EDTA for the measurement of triglycerides, NEFAs, glucose, and insulin concentrations over 8 h. In this study, each participant served as his own control.

Biochemical determinations

Plasma glucose and triglyceride concentrations were measured with a Hitachi Modular Analytics D-2400 analyzer (Roche Diagnostics, Basel, Switzerland) by using commercially available reagents and an enzyme-based kit. Plasma insulin was measured by using a specific enzyme-linked immunosorbent assay (Dako, Cambridge, United Kingdom) with a Hitachi Modular Analytics E-170 analyzer. Plasma NEFAs were measured with an ACS-ACOD assay (Wako Chemicals GmbH, Neuss, Germany) with a COBAS Mira-Plus analyzer.

Calculations

Fasting β cell function was estimated by 2 methods: 1) the homeostasis model assessment of insulin secretion (HOMA-B), by using the formula HOMA-B = I₀ × 3.33/(G₀ – 3.5), and 2) the basal disposition index (DI₀), which gives an adjusted measure by insulin sensitivity according to the HOMA of insulin resistance (HOMA-IR), by using the formula DI₀ = HOMA-B × (1/HOMA-IR) (9).

Fasting insulin resistance and its reciprocal (fasting insulin sensitivity) were estimated by 4 methods: 1) HOMA-IR = I₀ × G₀ divided by 22.5, 2) the revised-quantitative insulin sensitivity check index [rQUICKI = 1/(log I₀ + log G₀ + log NEFA₀)], 3) the basal insulin sensitivity index (ISI₀) for glycemia {ISI₀(G₀) = 2/[I₀ × G₀ + 1]}, and 4) the basal ISI₀ for blood NEFAs {ISI₀ (NEFA₀) = 2/[I₀ × NEFA₀ + 1]}. I₀, G₀, and NEFA₀ refer to fasting concentrations (t = 0) of insulin, glucose, and NEFAs, respectively.

Postprandial β cell function was estimated by 4 methods: 1) the insulinogetic index (IGI), which is a surrogate measure of first-phase insulin secretion and was calculated by using the difference between the postprandial insulin peak (t = 60 min) and the fasting insulin concentration in relation to the difference in glucose concentrations (IGI = ΔI₀,G₀/ΔG₀); 2) the ratio of the IGI to the HOMA-IR, which gives an adjusted measure of β cell function that accounts for variations in insulin sensitivity; 3) the ratio of the insulin to glucose areas under the curve (AUCins/AUCGLU), which significantly correlates with glucose sensitivity and early-phase insulin secretion, calculated by using the trapezoidal method from 0 to 120 min; and 4) the total disposition index from 0 to 120 min (DI₁₂₀) by using the product of AUCins/AUCGLU with the Matsuda insulin sensitivity index (10), which incorporates both the hepatic and muscle components of insulin resistance and correlates well with the euglycemic insulin clamp.

Postprandial insulin sensitivity was estimated by 4 methods: 1) the insulin sensitivity index IS₀; 2) the oral glucose IS index (OGIS₀); 3) the postprandial insulin sensitivity index (ISI₀) for glycemia [ISI₀(G₀)]; and 4) the ISI₀ for blood NEFA [ISI₀ (NEFA₀)]. The infinity symbol represents the values at any postprandial time. The details of the equations and additional references using this multisampling protocol were previously described (8).

Statistical analyses

The summary data (the fasting and postprandial response) were analyzed by using one-factor repeated-measures ANOVA. The postprandial time courses after the test meals were analyzed by using 2-factor repeated-measures ANOVA, and Bonferroni correction was applied for the post hoc detection of significant pairwise differences. The data were analyzed by using STATVIEW for WINDOWS (SAS Institute, Cary, NC). P < 0.05 was considered significant.

RESULTS

No significant differences were observed between baseline values for plasma triglyceride, NEFA, glucose, and insulin concentrations at the beginning of each of the 3 treatment periods (Table 1). Likewise, we found no significant differences in the basal values for HOMA-B, DI₀, HOMA-IR, rQUICKI, and the basal Belfiore indexes for glycemia and blood NEFAs (Table 2). These data indicated that the hypertriglyceridemic subjects had...
similar basal β cell function and insulin sensitivity before the ingestion of the high-fat meals.

The plasma concentrations of postprandial triglycerides, NEFAs, glucose, and insulin induced by the meal with no fat and the meals enriched in MUFAs or SFAs are shown in Figure 1, A–D. As expected, no changes in plasma triglyceride concentrations were observed when fat was not included in the meal (Figure 1A). The postprandial glucose response was similar after ingestion of any of the meals, increasing from a basal concentration to a peak at 60 min (7.31 ± 0.59 mmol/L with MUFAs and 7.51 ± 0.24 mmol/L with SFAs compared with 6.96 ± 0.94 mmol/L with no fat; \( P > 0.05 \)) and returning to basal values between 120 and 180 min (Figure 1C). However, the fat-enriched meals markedly increased (\( P < 0.05 \)) mean plasma insulin concentrations to a peak at 60 min (264 ± 25 pmol/L with MUFAs and 503 ± 45 pmol/L with SFAs compared with 190 ± 26 pmol/L with no fat) (Figure 1D), and the iAUC values (0–8 h) for insulin also increased (+262% with MUFA and +638% with SFA compared with no fat, 100%) (Table 1). Likewise, the high-fat meals increased (\( P < 0.05 \)) mean plasma triglyceride concentrations to a peak at 120 min (6.55 ± 0.67 mmol/L with MUFAs and 7.06 ± 0.82 mmol/L with SFAs compared with 4.46 ± 0.73 mmol/L with no fat) (Figure 1A), and the iAUC values (0–8 h) for triglycerides also increased (+404% with MUFAs and +711% with SFAs compared with no fat, 100%). All of the meals also induced a decrease in mean plasma NEFA concentrations at 120 min (0.34 ± 0.03 mmol/L with MUFAs and 0.46 ± 0.07 mmol/L with SFAs compared with 0.25 ± 0.02 mmol/L with no fat; \( P < 0.05 \)) (Figure 1B). After remaining suppressed for >180–240 min, plasma NEFA concentrations rebounded, reaching basal values in the subjects given the meals with MUFAs (0.50 ± 0.05 mmol/L) and no fat (0.53 ± 0.04 mmol/L) \( (P > 0.05) \) at 360 min. The plasma NEFA concentrations rose markedly (\( P < 0.05 \)) to a peak at 360 min (0.79 ± 0.06 mmol/L) in the subjects given the meals containing SFAs. Accordingly, the high-fat meals increased (\( P < 0.05 \)) the iAUC values (0–8 h) for NEFAs (+276% with MUFAs, 896% with SFAs, and 100% for no fat) (Table 1).

Estimates of postprandial β cell function, including IGI (0–60 min), IGI/HOMA-IR, AUCINS/AUCGLU (0–120 min), and \( DI_{120} \) (0–120 min), were higher \( (P < 0.05) \) after the high-fat meals than after the meal containing no fat, and these estimates were higher \( (P < 0.05) \) after the SFA meal than after the MUFA meal (Table 3). In addition, estimates of postprandial insulin sensitivity, including \( OGIS_{0–80} \) (0–180 min), \( IS_{0–80} \) (0–480 min), and the postprandial Belfiore indexes for glycemia \( [IS/G_{0}^{s}] \) and for blood nonesterified fatty acids \( [IS/NEFA_{0}^{s}] \) were higher \( (P < 0.05) \) after the high-fat meals than after the meal containing no fat. These estimates were also lower \( (P < 0.05) \) after the SFA meal than after the MUFA meal.

**DISCUSSION**

This randomized and within-subject crossover study is the first to our knowledge to show the time course of changes in plasma

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**TABLE 1**

Fasting values on the day the test meals were administered and postprandial responses [incremental area under the curve (iAUC)] of triglycerides, nonesterified fatty acids (NEFAs), glucose, and insulin to the test meals over 8 h

<table>
<thead>
<tr>
<th></th>
<th>Meal with no fat</th>
<th>MUFA meal</th>
<th>SFA meal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting triglycerides (mmol/L)</td>
<td>4.22 ± 0.72</td>
<td>4.10 ± 1.11</td>
<td>4.31 ± 0.88</td>
</tr>
<tr>
<td>Triglyceride iAUC0–480 min (mmol · min⁻¹ · L⁻¹)</td>
<td>−140 ± 34</td>
<td>286 ± 97*</td>
<td>716 ± 146*</td>
</tr>
<tr>
<td>Fasting NEFA (μmol/L)</td>
<td>500 ± 73</td>
<td>511 ± 51</td>
<td>515 ± 60</td>
</tr>
<tr>
<td>NEFA iAUC0–480 min (μmol · min⁻¹ · L⁻¹)</td>
<td>−12,649 ± 2642</td>
<td>9626 ± 1920*</td>
<td>88,001 ± 8552*†</td>
</tr>
<tr>
<td>Fasting glucose (mmol/L)</td>
<td>5.77 ± 0.64</td>
<td>5.80 ± 0.91</td>
<td>5.81 ± 0.88</td>
</tr>
<tr>
<td>Glucose iAUC0–480 min (mmol · min⁻¹ · L⁻¹)</td>
<td>−6.0 ± 3.1</td>
<td>−8.4 ± 2.8</td>
<td>−9.6 ± 3.5</td>
</tr>
<tr>
<td>Fasting insulin (pmol/L)</td>
<td>89.6 ± 10.2</td>
<td>87.4 ± 12.7</td>
<td>91.3 ± 14.1</td>
</tr>
<tr>
<td>Insulin iAUC0–480 min (pmol · min⁻¹ · L⁻¹)</td>
<td>6306 ± 1307</td>
<td>16,506 ± 2070*</td>
<td>40,247 ± 4406*†</td>
</tr>
</tbody>
</table>

† All values are means ± SDs; \( n = 14 \). Participants ingested either a control meal (containing no fat) or a meal enriched in either monounsaturated fatty acids (MUFAs) or saturated fatty acids (SFAs). Statistical differences are based on repeated-measures ANOVA with Bonferroni correction. *Significantly different from the meal with no fat, \( P < 0.05 \). †Significantly different from the MUFA meal, \( P < 0.05 \).
lipid (triglyceride and NEFA), glucose, and insulin concentrations and the extent of β cell function and insulin sensitivity dysregulation after MUFA and SFA meals in subjects with high fasting triglyceride concentrations. All of the subjects had similar fasting β cell function and insulin sensitivity, and all of the meals elicited similar postprandial glucose responses. However, the high-fat meals increased postprandial β cell function and decreased postprandial insulin sensitivity relative to the meal containing no fat. Most importantly, our results showed that hypertriglyceridemic subjects had lower lipemic and insulinemic responses and were less insulin resistant postprandially when given a meal containing MUFAs rather than SFAs.

The relative effects of MUFA and SFA meals on postprandial triglyceride and NEFA concentrations have been investigated in only a small number of studies to date (8, 11–13), and none of these studies included subjects with high fasting triglyceride concentrations. Our study showed a significant attenuation of

### TABLE 3

Postprandial indexes of β cell function [insulinogenic index (IGI), HOMA-IR, AUCINS/AUCGLU, ratio of the area under the curve for insulin and glucose (AUCINS/AUCGLU), and basal disposition index (DI120)] and insulin sensitivity (oral glucose insulin sensitivity index [OGIS0-N] and insulin sensitivity index [IS0-N] for glycemia [ISI(G)0-N] and for blood nonesterified fatty acids [ISI(NEFA)0-N]) after the test meals

<table>
<thead>
<tr>
<th></th>
<th>Meal with no fat</th>
<th>MUFA meal</th>
<th>SFA meal</th>
</tr>
</thead>
<tbody>
<tr>
<td>IGI, 0–60 min (pmol/mmol)</td>
<td>101.9 ± 8.0</td>
<td>118.7 ± 6.2*</td>
<td>241.7 ± 12.3*†</td>
</tr>
<tr>
<td>IGI/HOMA-IR (L/mmol)</td>
<td>4.44 ± 0.31</td>
<td>5.43 ± 0.23*</td>
<td>10.25 ± 0.58*†</td>
</tr>
<tr>
<td>AUCINS/AUCGLU, 0–120 min (pmol/mmol)</td>
<td>22.4 ± 2.1</td>
<td>26.0 ± 1.7*</td>
<td>49.2 ± 2.9*†</td>
</tr>
<tr>
<td>DI120, 0–120 min</td>
<td>121.8 ± 6.4</td>
<td>130.5 ± 7.2*</td>
<td>174.0 ± 9.7*†</td>
</tr>
<tr>
<td>OGIS0-N, 0–180 min (mL·m⁻²·min⁻¹)</td>
<td>273.0 ± 2.2</td>
<td>268.6 ± 1.9*</td>
<td>250.7 ± 1.8*†</td>
</tr>
<tr>
<td>ISI(G)0-N, 0–480 min (min⁻¹·dL⁻¹·kg⁻1·min⁻1)</td>
<td>197.1 ± 8.5</td>
<td>144.1 ± 9.0*</td>
<td>54.6 ± 6.3*†</td>
</tr>
<tr>
<td>Postprandial-Belfiore index for glycemia [ISI(G)0-N]</td>
<td>1.00 ± 0.00</td>
<td>0.91 ± 0.02*</td>
<td>0.72 ± 0.05*†</td>
</tr>
<tr>
<td>Postprandial-Belfiore index for blood NEFA</td>
<td>1.00 ± 0.00</td>
<td>0.86 ± 0.03*</td>
<td>0.54 ± 0.04*†</td>
</tr>
</tbody>
</table>

† All values are means ± SDs; n = 14. Participants ingested either a control meal (containing no fat) or a meal enriched in either monounsaturated fatty acids (MUFAs) or saturated fatty acids (SFAs). Statistical differences are based on repeated-measures ANOVA with Bonferroni correction. *Significantly different from the meal with no fat, P < 0.05. †Significantly different from the MUFA meal, P < 0.05.
incremental triglyceride and NEFA responses after the MUFA meal relative to the SFA meal, which is similar to the findings observed in healthy subjects (8, 11). Differences in the apolipoprotein (apo CII, CIII, and E) content of postprandial triglyceride-rich lipoproteins (TRLs) have been proposed to influence postprandial triglycerides and NEFAs via their roles in regulating TRL lipolysis and hepatic removal of remnant TRL (14). Because each participant served as his own control and no differences were found in fasting triglycerides before ingestion of the MUFA and SFA meals, we excluded any influence of fasting triglycerides on postprandial lipid concentrations and clearly established that MUFAs are superior to SFAs in reducing the dysregulation of postprandial lipid metabolism in hypertriglyceridemic subjects.

Postprandial studies previously showed the potential of high-fat meals to induce β cell dysfunction and insulin resistance in healthy individuals (4, 8) and in subjects with type 2 diabetes (15) or the metabolic syndrome (16). In addition, Monnier et al (17) found that the evolution of type 2 diabetes is related to the progressive deterioration of glucose homeostasis that starts during the postprandial period in subjects whose glycemic profiles are normal at fasting. More recently, we hypothesized (18) that the transition from normal to impaired glucose tolerance and then to overt diabetes hinges on the nature of the postprandial lipid excursions. However, little is currently known about the postprandial insulin secretion and action after specific fatty acid intake in humans. In earlier studies, Rasmussen et al (19) showed that insulin release was stimulated by SFAs, but not by MUFA, in subjects with type 2 diabetes. The authors attributed these results, in part, to differences in the chain lengths of the fatty acids contained in the meals. In the studies by Robertson et al (11) and Lopez et al (8), higher postprandial insulin concentrations were observed after an SFA meal than after a MUFA meal in healthy subjects, but there was no effect on glucose responses. These findings agree with the data reported in the present study. The mechanisms that might link high-fat meal consumption and acute insulin hypersecretion (or impaired insulin sensitivity) have not been fully elucidated. We speculated that the insulinotropic effects mediated by the abrupt delivery of fatty acids in the vicinity of the β cells during the early postprandial period may be involved (18), which agree with the finding that SFAs more effectively stimulate insulin release in human islets than do MUFA (20). Lipoprotein lipase activity could be an important control point for postprandial TRL lipolysis and the dispatch of fatty acids to β cells, because both β cell–specific deletion and overexpression of the lipoprotein lipase gene in mouse models has been shown to impair insulin secretion and glucose homeostasis (21). Likewise, the G protein–coupled receptor GPR40, also known as free fatty acid receptor 1 (FFAR1), is abundantly expressed in β cells and contributes to the fatty acid–mediated enhancement of glucose-stimulated insulin secretion. Recent data from FFAR1-null mice showed impaired insulin and incretin hormone secretion in response to a high-fat meal (22). A variety of fatty acids have been found to acutely activate FFAR1 within the physiologic range in vitro (23). However, the relative potency of MUFA and SFAs in vivo remains unclear. Our study contributes to the understanding of this issue and shows that postprandial β cell function (and insulin sensitivity) improved more after MUFA consumption than after SFA consumption and, therefore, extends the relation between MUFA-rich meals and the benefits on postprandial glucose homeostasis observed in subjects with normal fasting triglyceride concentrations (8) to a population of subjects with high fasting triglyceride concentrations. Furthermore, with regard to resistance to insulin-mediated glucose disposal, we found that SFA stimulated additional insulin secretion to maintain postprandial glucose homeostasis, which suggests a mechanism of lipid-induced deterioration of insulin sensitivity coupled with compensatory insulin secretion that is distinctively modulated by MUFA and SFAs. These findings agree with those of a previous study involving healthy subjects (8) and overweight and obese nondiabetic subjects (13), which showed that an acute decrease in insulin sensitivity was directly proportional to the proportion of SFA in the meal. These findings also agree with those of an in vitro study (24), in which postprandial TRLs containing SFAs induced skeletal muscle insulin resistance as evidenced by impaired insulin signaling and glucose metabolism. This in vitro effect was independent of the NEFAs in the media of the cells, but was absolutely dependent on the integrity of postprandial TRLs and their interactions with members of the LDL receptor family. Some of these observations were corroborated in a human postprandial study involving healthy subjects (4). Therefore, whether lipoprotein receptor–mediated signaling pathways in skeletal muscle and other insulin target tissues, such as adipose tissue and liver, are sensitive to the type of fatty acids in postprandial TRLs, which largely depend on the nature of dietary fats embodied in the meal, and whether they are connected to insulin signaling pathways should be established.

Collectively, the present data confirm that MUFA (mainly oleic acid) are superior to SFAs (mainly palmitic acid) at buffering β cell hyperactivity and insulin intolerance postprandially in subjects with high fasting triglyceride concentrations. These data also suggest that, as a replacement for SFAs, MUFA-based strategies may provide cardiovascular benefits to persons at risk by limiting lipid and insulin excursions and may contribute to optimal glycemic control after meal challenges.

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The authors' responsibilities were as follows—SL, BB, AO, LMV, YMP, JV, RA, and FJGM: participated in the study design and performed the data analysis; SL, BB, AO, LMV, and RA: assisted with the editing of the manuscript; SL, BB, AO, LMV, YMP, and JV: acquired the data; JV: recruited the volunteers and collected the samples; and FJGM: wrote the first draft and finalized the manuscript. All authors participated in the analytic discussion of the results and approved the final version of the manuscript. None of the authors had any personal or financial conflicts of interest.

REFERENCES