Postprandial monocyte activation in response to meals with high and low glycemic loads in overweight women

Deborah D Motton, Nancy L Keim, Fatima A Tenorio, William F Horn, and John C Rutledge

ABSTRACT
Background: Recent data show that atherosclerosis is initiated and perpetuated by inflammatory events. Activation of immune cells such as monocytes initiates inflammation, a key step in atherosclerosis.

Objective: We hypothesize that a high–glycemic load meal activates inflammatory cells, and that this is mediated by elevated circulating triacylglycerol-rich lipoproteins.

Design: Sixteen women [body mass index (in kg/m²): 25.7–29.6], aged 20–48 y, consumed meals with a high or a low glycemic load in a crossover fashion. Blood samples were collected before and up to 8 h after the meals. Samples were measured for glucose, insulin, triacylglycerols, and circulating cytokines, and expression of tumor necrosis factor α (TNF-α) and interleukin 1β (IL-1β) was measured by flow cytometry.

Results: At 3.5 h after the test meals, we observed a significant increase in monocytes expressing TNF-α with both high- and low-glycemic load meals. Also, expression of IL-1β in monocytes tended to increase, but the change was not significant. The glycemic load of the meal did not influence circulating cytokines and had only a minimal effect on postprandial triacylglycerol concentrations despite marked postprandial changes in glyceria and circulating insulin concentrations.

Conclusions: In the postprandial state, monocytes can be activated by both high- and low–glycemic load meals. The glycemic load of a single meal did not have a significant effect on the degree of activation of the monocytes in women who displayed only a modest increase in circulating triacylglycerols in response to test meals. Future studies should examine the effect of glycemic load in subjects who have a hyperlipemic response to dietary carbohydrate. Am J Clin Nutr 2007;85:60–5.

KEY WORDS Monocytes, inflammation, tumor necrosis factor α, interleukin 6, glycemic index, insulin, glucose, triacylglycerols, obesity, carbohydrates

INTRODUCTION

Inflammation plays a key role in atherosclerotic cardiovascular disease. Certain risk factors for atherosclerosis (smoking, hypertension, dyslipidemia, and the presence of diabetes mellitus) are associated with increased inflammation. In addition, markers of systemic inflammation are predictors for both cardiovascular disease and diabetes (1, 2).

Much attention has been given to the role that carbohydrates play in cardiovascular health and disease. To understand the role carbohydrates play in metabolic diseases, a classification system was developed that is based on the digestibility and absorption of carbohydrates, as well as the quality and quantity of carbohydrate in the food item. The glycemic index (GI) is a measure of the postprandial glucose response to the available carbohydrate in the food. The glycemic load (GL) refers specifically to an individual food item and is the product of the GI value of a food and its carbohydrate content (3). Epidemiologic evidence shows that diets high in easily absorbed carbohydrates are associated with the development of type 2 diabetes mellitus and that such diets are linked with an increased risk of cardiovascular disease (3–5). We hypothesize that ingesting carbohydrates with a high GI, such as highly refined grains, leads to an increased state of inflammation postprandially, compared with low-GI carbohydrates, such as whole grains.

Increased consumption of diets high in carbohydrates also provokes hypertriacylglycerolemia, similar to what is seen in consumption of high-fat diets (6–8). In addition, it has been shown that diets high in refined sugars have the ability to reduce circulating HDL (9, 10), whereas diets with a low GI can lower LDL cholesterol (11). High-carbohydrate diets also lead to increased concentrations of the plasma soluble cell adhesion markers E-selectin and vascular cell adhesion molecule, as well as the inflammatory markers C-reactive protein, and interleukin 6 (IL-6) (2, 12). Furthermore, risk of coronary heart disease is increased in women consuming high-GI diets (13). These results point toward a relation among high-GI diets, hyperlipemia, and systemic inflammation, all strong risk factors for atherosclerosis and diabetes.

A previous study in our laboratory has shown that persons given a moderate–fat meal (40% of calories from saturated fat) respond postprandially with increased measures of inflammation, specifically, increases in monocyte and platelet activation.

1 From the Division of Endocrinology, Clinical Nutrition, and Vascular Medicine, Department of Internal Medicine, University of California, Davis (DDM, FAT, and JCR), and the Western Human Nutrition Research Center, Agricultural Research Service, US Department of Agriculture, University of California, Davis (NLK and WFH)
2 Supported by grants from the National Institutes of Health (NHLBI HL71488 and HL55667) and the USDA Current Research Information System (CRIS) Project 5306-51000-002-00D.
3 Address reprint requests to NL Keim, Western Human Nutrition Research Center, ARS, USDA, 213 Surge IV, University of California, Davis, CA 95616. E-mail: nkeim@whnrc.usda.gov.
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(14), both strongly associated with the development of atherosclerosis (15, 16). We hypothesize that measures of systemic inflammation will increase postprandially in response to a high-GL meal compared with a low-GL meal. In the present study we examine the level of activation of monocytes and soluble markers of inflammation in women consuming both low- and high-GL diets to assess their state of inflammation preprandially and postprandially. This study compares 2 high- and low-GL diets, which were specifically formulated so that the contents of available carbohydrate, total protein, and fat were identical, in the same population and also provides information about an understudied but highly prevalent population, overweight women.

SUBJECTS AND METHODS

Study subjects

Healthy overweight women were recruited from the greater Sacramento area which includes the University of California, Davis, campus. Twenty-four women were admitted to the study, meeting the following inclusion criteria: premenopausal with regular menstrual cycles; age between 18 and 48 y; body mass index (in kg/m²) between 25.0 and 29.9; and fasting blood glucose, serum cholesterol, and triacylglycerols in the normal range. Exclusion criteria were smoking, pregnancy, lactation, and chronic illness, including a history of liver, kidney, heart, or thyroid disease; elevated thyroid-stimulating hormone; or current use of prescription medications or over-the-counter antiinflammatory medications. Approval for this study was obtained by the Institutional Review Board of the University of California, Davis, and informed consent was obtained before all data collection. During the course of the study, 8 subjects dropped out (6 had personal reasons, 1 became pregnant, and 1 was unable to comply with the diet). The remaining 16 subjects successfully completed the study, and their data are reported here.

Study design

Subjects were studied under 2 postprandial conditions in a crossover design. In one test condition, a meal with a high GL was used, and in the other test condition a meal with a low GL was used. Subjects were randomly assigned to the order in which the GL test conditions were presented, and a wash-out period of 8–12 wk fell between the 2 tests. Before each test day, subjects consumed a standardized run-in diet for 3 d that matched the test meal or run-in diet.

Diet interventions

The energy and macronutrient composition of the test meals and run-in diets are listed in Table 1. Although the diet interventions were designed to elicit divergent postprandial glycemic responses, they were equivalent in the amount of energy provided and in the distribution of energy from protein, fat, and carbohydrate. To achieve divergent glycemic responses, different sources of carbohydrate-containing foods were used. The high-GL run-in diet provided carbohydrates from refined grain sources, whereas the low-GL run-in diet provided carbohydrates from whole-grain sources with higher fiber content. The high-GL test meal was based on a refined rice and corn cereal, whereas the low-GL test meal was based on a multigrain bran cereal. Other foods included in both test meals were low-fat milk and low-fat yogurt. Small quantities of cream and whey protein powder were used to equalize the fat and protein contents of the 2 test meals.

Blood sample processing

Blood samples obtained for measurements of glucose, triacylglycerol, and cytokine were immediately centrifuged (1300 × g, 10 min, 4 °C) to separate plasma from cells. Samples obtained for measurement of insulin concentration was held at room temperature for 30 min to allow a clot to form and then centrifuged (1300 × g, 10 min, 4 °C) to separate sera from the clotted cells. Plasma and serum aliquots were frozen (−80 °C) until use in assays.

Plasma concentrations of glucose, insulin, and triacylglycerol

Plasma concentrations of glucose and triacylglycerol were measured enzymatically (Roche Diagnostics, Manheim, Germany; kit 12146029) on a Hitachi 902 Automatic Analyzer (Boehringer Mannheim Corp, Indianapolis, IN) according to the manufacturer’s instructions. The within-run CVs for glucose

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### Table 1

<table>
<thead>
<tr>
<th></th>
<th>High–glycemic load test condition</th>
<th>Low–glycemic load test condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test meal</td>
<td>Run-in diet</td>
</tr>
<tr>
<td>Energy (kcal)</td>
<td>833</td>
<td>2091</td>
</tr>
<tr>
<td>Carbohydrate (%)</td>
<td>54.2</td>
<td>56.4</td>
</tr>
<tr>
<td>Protein (%)</td>
<td>15.0</td>
<td>13.9</td>
</tr>
<tr>
<td>Fat (%)</td>
<td>30.8</td>
<td>29.7</td>
</tr>
<tr>
<td>Fiber (g)</td>
<td>2.4</td>
<td>9.9</td>
</tr>
<tr>
<td>Glycemic index</td>
<td>76.7</td>
<td>76.6</td>
</tr>
<tr>
<td>Glycemic load</td>
<td>86.3</td>
<td>225.7</td>
</tr>
</tbody>
</table>

*Test meal values represent an energy intake prescription of 2100 kcal/d. The energy content of the test meal was adjusted on an individual basis and provided 40% of the person’s daily energy requirement for weight maintenance. The amounts of foods served in the test meal were adjusted proportionately to maintain the same macronutrient ratios at all energy levels. Run-in diet values were reported as average of intake per day, based on an energy intake prescription of 2100 kcal/d. The energy content of the run-in diet was adjusted on an individual basis to meet the person’s daily energy requirement for weight maintenance.*

2 Glycemic index values are based on the glucose standard and represent an average weighted by available carbohydrate in each food item constituting the test meal or run-in diet.
triacylglycerol were 0.6% and 1.4%, respectively, and the corresponding between-run CVs were 3.5% and 2.5%. Serum insulin was determined by a solid-phase, 2-site, chemiluminescent enzyme-labeled immunometric assay with the use of an Immulite analyzer (Diagnostic Products Corp, Los Angeles, CA). For insulin, the within-run CV was 3.8% and the between-run CV was 4.4%. For these assays, all samples from one subject were analyzed within the same run.

**Insulin resistance**

Insulin resistance was calculated by using the homeostasis model assessment of (IRHOMA), based on the Matthews et al (17) computer model of insulin-glucose interactions. The insulin resistance index value is calculated by using fasting insulin concentrations (in mIU/L) and fasting glucose concentrations (in mmol/L) (18). The equation used is as follows:

\[
IR_{HOMA} = (I_0 \times G_0)/22.5
\]

**Plasma cytokine concentrations**

Plasma concentrations of TNF-α, IL-1β, and IL-6 were measured with the Bio-Plex Multiplex Suspension Array System (Bio-Rad Laboratories, Hercules, CA) with the use of Beadlyte kits and reagents (Upstate Serologicals, Waltham, MA). The within-plate CVs for the cytokine assays ranged between 1% and 14%, averaging 6%. The between-plate CVs were 19.7%, 24.0%, and 29.8% for TNF-α, IL-1β, and IL-6, respectively. All samples from one subject were analyzed within the same run.

**Monocyte intracellular TNF-α and IL-1β**

Blood was collected in sodium heparin anticoagulant and then incubated for 4 h at 37 °C in 5% CO₂, with either lipopolysaccharide (a positive control) or sterile phosphate-buffered saline (PBS) in the presence of Brefeldin A (to inhibit Golgi function and thus exocytosis; Sigma, St Louis, MO). After incubation, blood was incubated with fluorescent labeled anti-CD14 (monocyte-specific marker) for 20 min at room temperature. Red blood cells were lysed, and blood was centrifuged at 500 × g for 5 min at room temperature. The supernatant fluid was removed, and the remaining cell pellet was permeabilized with the use of fluorescence-activated cell sorting permeabilizing solution (BD Biosciences, San Diego, CA) for 5 min at room temperature. The cell pellet was again centrifuged at 500 × g for 5 min at room temperature, then washed with sterile PBS and centrifuged again. The resulting pellet was incubated with fluorescent-labeled anti-TNF-α and anti-IL-1 antibodies (BD Biosciences) for 30 min at room temperature in the dark. After incubation, cells were washed with sterile PBS and centrifuged at 500 × g for 5 min at room temperature. Cells were fixed in and resuspended in 1% paraformaldehyde before analysis on the fluorescence-activated cell sorter Calibur flow cytometer (Becton Dickinson, Franklin Lakes, NJ) within 24 h after completion of the assay. Data were analyzed with the use of the CELLQUEST software (version 3.3; Becton Dickinson).

**Statistical analysis**

All data are expressed as the mean ± SEM, unless indicated otherwise in footnotes to tables. Statistical analyses were performed with the use of PC-SAS (version 8.2; SAS Institute, Cary, NC). Data distributions were evaluated with the use of the Bartlett’s test (19), and variables displaying heterogeneous variance were transformed before tests for statistical significance. For analysis of serial data, mixed models analysis of variance was performed with the use of a crossover model with repeated measures. Factors included diet condition (high GL and low GL), time (fasting, 3.5 h, and 8.0 h), order (high GL followed by low GL and low GL followed by high GL), and the interaction of diet condition × time. It was determined that there were no main effects of order, and the model was collapsed to examine the effects of diet condition, time, and diet condition × time interaction. Post hoc comparison of means was accomplished with Fisher’s least significant difference multiple comparison tests, with P values adjusted with the use of the Bonferroni method. Mixed models analysis of variance was also performed to determine the effects of diet condition and order on variables such as peak values and area under the curve. A 2-tailed value of P < 0.05 was considered statistically significant.

**RESULTS**

Body weight and body composition remained stable between the tests (Table 2). The high-GL test condition led to higher fasting concentrations of blood glucose (P < 0.05), but none of the subjects had fasting hyperglycemia. Fasting triacylglycerol concentrations were not different between tests (Table 2), and all subjects had fasting values < 200 mg/dL for both test conditions.

**Glycemic response to test meals**

As expected, the peak glycemic response and the 2-h area under the curve (both absolute and incremental) values were higher for the high-GL test meal than for the low-GL test meal (Table 3). Also, the postprandial insulin response differed between the test conditions (Table 3).

**Postprandial triacylglycerol concentrations**

Mean plasma triacylglycerol concentrations were not different from fasting (0 h) values at 3.5 h or 8 h, and no differences were observed between diet conditions at 3.5 h or at 8.0 h (Figure 1). Triacylglycerol concentrations peaked at 3.1 ± 0.5 h and 3.3 ± 0.4 h for the high- and low-GL conditions, respectively. The peak times were not different between diet conditions, but the peak value for the high-GL meal, 129.0 ± 62.3 mg/dL, was greater than that of the low-GL meal, 117.0 ± 63.5 mg/dL, (P < 0.05).

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**TABLE 2**

Characteristics of subjects before consumption of test meal

<table>
<thead>
<tr>
<th></th>
<th>High–glycemic load test condition</th>
<th>Low–glycemic load test condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>30.9 ± 8.6</td>
<td>30.9 ± 8.7</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>75.6 ± 6.9</td>
<td>76.3 ± 6.9</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.0 ± 1.1</td>
<td>27.2 ± 1.1</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>39.0 ± 3.1</td>
<td>39.3 ± 3.0</td>
</tr>
<tr>
<td>Fasting glucose (mg/dL)</td>
<td>88.1 ± 4.0</td>
<td>85.2 ± 4.9</td>
</tr>
<tr>
<td>Fasting triacylglycerols (mg/dL)</td>
<td>88.2 ± 40.7</td>
<td>82.8 ± 44.6</td>
</tr>
</tbody>
</table>

1 All values are ± SD; n = 16. All measurements were taken after the 3-d run-in diet, after an overnight fast, before consumption of the test meal.

2 Significant effect of diet condition on fasting glucose concentrations, P < 0.05 (mixed models analysis of variance).
### Plasma cytokine concentrations

Fasting plasma concentrations of TNF-α, IL-1β, and IL-6 in this population were within the normal range (<20 pg/mL for TNF-α, and <4 pg/mL for IL-1β), and no statistically significant changes were observed in plasma cytokines postprandially (Table 4). No significant difference was observed between diet conditions on plasma cytokine concentrations (Table 4).

### Insulin resistance

The IRHOMA for this population of subjects ranged between 0.6 and 4.1. An IRHOMA of 1 is considered normal for healthy persons with 100% β-cell function. Mean IRHOMA values did not differ between GL conditions: 1.72 ± 0.21 and 1.43 ± 0.15 for high-GL and low-GL conditions, respectively. IRHOMA values were not correlated with monocyte TNF-α or IL-1β responses at 3.5 h or 8.0 h for the high-GL or low-GL condition (data not shown).

### Monocytes expressing intracellular TNF-α

Concentrations of monocytes expressing intracellular TNF-α were not different between diet conditions (Figure 2). A significant increase was observed in the number of monocytes expressing TNF-α after meal ingestion, regardless of meal type ($P < 0.02$). At 3.5 h, the increase was significant compared with fasting (0 h) ($P < 0.02$). At 8 h, the number of monocytes expressing TNF-α had decreased to an intermediate value between that of 3.5 h and 0 h and was not statistically different from values measured at either of these time points.

### Monocytes expressing intracellular IL-1β

Concentrations of monocytes expressing intracellular IL-1β were not different between the high- and low-GL conditions (Figure 3). At 3.5 h and 8 h postprandially, a tendency was observed for the number of monocytes expressing IL-1β to increase in response to both meal conditions, but because of the large individual variation, the effect of time was not significant ($P = 0.07$).

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### Table 3: Glucose and insulin responses to high- and low-glycemic load test meals

<table>
<thead>
<tr>
<th>Glucose response</th>
<th>High-glycemic load condition</th>
<th>Low-glycemic load condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak glucose (mg/dL)</td>
<td>125.3 ± 7.3</td>
<td>108.7 ± 3.4^2</td>
</tr>
<tr>
<td>Glucose 2-h AUC</td>
<td>213.4 ± 9.3</td>
<td>196.0 ± 6.7^2</td>
</tr>
<tr>
<td>Glucose 2-h incremental</td>
<td>37.3 ± 8.4</td>
<td>25.5 ± 5.4^2</td>
</tr>
<tr>
<td>AUC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak insulin (IU/mL)</td>
<td>138.3 ± 24.2</td>
<td>71.2 ± 9.1^2</td>
</tr>
<tr>
<td>Insulin 2-h AUC</td>
<td>176.0 ± 30.3</td>
<td>88.5 ± 12.0^2</td>
</tr>
<tr>
<td>Insulin 2-h incremental AUC</td>
<td>160.3 ± 29.4</td>
<td>74.9 ± 11.3^2</td>
</tr>
</tbody>
</table>

^1 All values are $\bar{x} \pm$ SEM; $n = 16$. AUC, area under the curve.

^2-4 Significant effects of diet condition (mixed models analysis of variance): $^2P < 0.005$, $^3P < 0.001$, $^4P < 0.05$.

### Table 4: Plasma cytokine concentrations before and after consumption of test meal

<table>
<thead>
<tr>
<th>Cytokine</th>
<th>Fasting</th>
<th>3.5 h</th>
<th>8.0 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>TNF-α (pg/mL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High GL</td>
<td>1.18 ± 0.16</td>
<td>0.81 ± 0.20</td>
<td>0.86 ± 0.25</td>
</tr>
<tr>
<td>Low GL</td>
<td>1.29 ± 0.23</td>
<td>1.30 ± 0.35</td>
<td>1.06 ± 0.22</td>
</tr>
<tr>
<td>IL-1β (pg/mL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High GL</td>
<td>1.08 ± 0.49</td>
<td>0.61 ± 0.20</td>
<td>0.49 ± 0.15</td>
</tr>
<tr>
<td>Low GL</td>
<td>1.01 ± 0.26</td>
<td>0.86 ± 0.29</td>
<td>1.15 ± 0.36</td>
</tr>
<tr>
<td>IL-6 (pg/mL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High GL</td>
<td>1.86 ± 0.31</td>
<td>3.15 ± 0.78</td>
<td>2.49 ± 0.45</td>
</tr>
<tr>
<td>Low GL</td>
<td>2.15 ± 0.47</td>
<td>3.57 ± 1.24</td>
<td>4.94 ± 1.72</td>
</tr>
</tbody>
</table>

^1 All values are $\bar{x} \pm$ SEM; $n = 16$. TNF-α, tumor necrosis factor α, IL-1β, interleukin 1β, GL, glycemic load. Mixed models analysis of variance was performed with diet condition, time, and the interaction of diet condition $\times$ time as predictor variables. There were no significant effects of diet condition, time, or diet condition $\times$ time.
DISCUSSION

This is the first report that examines a potential link between the GL of test meals and measures of monocytic inflammation in vivo. We found no significant differences in monocyte activation in the fasting state in response to the run-in diets; similarly, there was no differential activation of monocytes in the postprandial state when high- and low-GL meals were compared. However, both the high- and low-GL meals did lead to increased activation of monocytes expressing TNF-α at 3.5 h after ingestion of the meals. We observed a similar pattern of monocytes expressing IL-1β in the postprandial state, but variability in the response was too large to produce a statistically significant postprandial effect. These data add to the increasing amount of evidence that purport an increased inflammatory state in the postprandial period, even when the fat content of meals is moderate (≈30% of energy).

We were not able to show that ingestion of a meal with a high GL stimulated greater activation of monocytes than did a meal with a low GL. This may have been because, at the time of blood sampling (3.5 and 8.0 h after meal ingestion), no significant differences were observed in postprandial triacylglycerol concentrations between the meal conditions. Previous studies have shown that the degree of vascular inflammation is directly related to the postprandial triacylglycerol response (1, 14, 20). The similarity in triacylglycerol concentrations is not entirely unexpected because this study examined the effects of an acute meal response, and the triacylglycerol response may be dictated by the long-term, habitual diet. In addition, because subjects were given a run-in diet for 3 d before the test date, this may have stabilized the postprandial triacylglycerol response such that large differences in postprandial triacylglycerol concentrations were not observed. Finally, although our study subjects were overweight women, they all had normal fasting lipid concentrations and may have been less sensitive to the known effects of carbohydrate loads on triacylglycerol metabolism.

Nevertheless, this study showed that ingestion of meals increases the level of monocyte inflammation in vivo. Activation of circulating monocytes allows them to be recruited and sequestered into the arterial intima, one of the early steps in plaque formation (21, 22). A hallmark of monocyte activation is the production of cytokines, including TNF-α and IL-1β (23, 24), both of which are potent activators of the vascular endothelium (25, 26). Activation of the endothelium by these cytokines leads to increased vascular permeability and expression of cell adhesion molecules, key events in atherosclerosis. We found that there was an overall increase in the number of cells expressing measurable concentrations of TNF-α and IL-1β postprandially. However, the concentrations of circulating cytokines after the meals did not change from fasting concentrations with either the high- or low-GL meal. Nonetheless, both meals elicited significant increases in the postprandial monocyte response for TNF-α at 3.5 h. The increase in inflammatory marker production may be underestimated because this study examined postprandial effects of a single meal in the morning, after an overnight fast. Most persons eat ≥3 times during the day; therefore, the activation of monocytes might be repeatedly stimulated with each successive meal.

One important and concerning observation in this study is the longevity of the increase in monocyte TNF-α. For both meals, the values at 8 h after the meal still tended to be higher than fasting and were not significantly different from those at 3.5 h. This is in contrast to the results of our previous study in healthy, young men with normal body weight that showed a definite return of inflammatory markers to baseline 6 h after ingestion (14). In the US population most persons would have already consumed another meal or 2 in this time period. Additional studies are needed to determine synergistic effects of subsequent high-GL meals and to compare responses in lean, overweight, and obese men and women.

The relative amount of monocytes with elevated intracellular TNF-α and IL-1β did not correspond to changes in circulating concentrations of these 2 cytokines. The concentrations of these 2 markers in circulation were well within the normal range, and no significant changes were seen in the postprandial period when compared with fasting concentrations. This is not surprising, because the changes in the number of monocytes expressing these 2 cytokines, although increasing >3-fold from ≈2% to 6–8%, may not be sufficiently large enough to yield a systemic change in circulating concentrations. Moreover, the cytokines are produced by multiple cell types and are secreted into a large reservoir of extracellular fluid. However, the measurement of intracellular concentrations of TNF-α and IL-1β could be considered more important in that this measures a specific cellular response and a potential pathophysiologic pathway. Measurement of circulating concentrations of these cytokines reflect cytokine levels from many cellular sources, whereas the monocyte intracellular concentrations showed activation of a cell type key to inflammation and atherosclerosis. In addition, because activation of monocytes normally results in sequestration of these same cells in tissues such as the arterial intima (27, 28), it is possible that the small number of monocytes activated by the diets could have quickly entered tissues, such that secretion did not result in significant changes in plasma concentrations of the cytokines or activated monocytes.

Consumption of diets with a high GL have been implicated in obesity, insulin sensitivity, circulating lipid concentrations, endothelial function, and presumably risk of cardiovascular disease. The present study aimed to show a link between the GL of a test meal and some well-accepted markers of inflammation that are important in the initiation of atherosclerotic plaque formation.
and propagation. Data presented here indicate an increased monocyte inflammatory response to both high- and low-GL meals. However, it is possible that the GL of a meal or a diet might influence the inflammatory response in persons who are more sensitive to the carbohydrate content of the diet. Further studies should be undertaken to elucidate the inflammatory effects of chronic consumption of diets with a high GL, particularly in subjects who are at increased risk of metabolic disease and have elevated fasting triacylglycerol concentrations.

We thank Dr. Hank Schwartz for his help in this study. We would also like to thank the nursing and dietary staff of the USDA, Western Human Nutrition Research Center.

DDM participated in data collection and manuscript preparation; NLK participated in study design, data collection, statistical analysis, and editorial duties; FAT participated in data collection; WFH participated in data collection and editorial duties; JCR participated in study design, editorial duties, and funding. None of the authors had any financial or personal conflict of interest with the sponsors of the study.

REFERENCES