

## Research Article

# Development of a Standardized Clinical Protocol for Ranking Foods and Meals Based on Postprandial Triglyceride Responses: The Lipemic Index

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Postprandial triglyceride levels are being increasingly recognized as an independent risk factor for the development of cardiovascular disease (CVD). There is a need for developing a standardized clinical protocol which allows foods and meals to be ranked based on the resulting postprandial triglyceride response. This pilot study offers a novel approach to standardize such testing based on equicaloric intakes, allowing for increased flexibility in comparing different food and meal offerings, as well as a high potential for public knowledge transfer. Our laboratory has developed a standardized 2100 kJ beverage, consisting of fat, protein, and simple carbohydrates (LIXR) with the goal of eliciting a reference postprandial triglyceride response. As we hypothesized, a certain commercial product which gave favourable glycemic responses yielded significantly higher triglyceride responses than our reference solution, indicating an important gap in current methods of identifying low-risk foods for subjects at risk for CVD. The lipemic index may eventually be used in combination with other nutritional tools to provide an enhanced overall assessment of health risks associated with consuming certain foods.

## 1. Background

In North American clinical practice, predictive risk assessment for cardiovascular disease (CVD) is based on a combination of traditional risk factors, including the assessment of fasting lipid panels [1]. Preventive CVD intervention strategies focus on evidence-based targets for modifiable risk factors, including the maintenance of fasting triglyceride levels to less than 1.70 mmol/L [2]. Hypertriglyceridemia as measured and defined in the fasted state has long held a strong association to the development of atherosclerosis [3]. However, there is increasing evidence that postprandial triglycerides provide a more accurate prognostic measure as a substantial risk marker for CVD [4–8]. Reasons for this prognostic advantage include the variable responses to food intake not seen in fasting serum triglycerides, as well as their inconsistent ability to predict individual postprandial

triglyceride response [9, 10]. Furthermore, it is stressed that fasting lipid profiles do not accurately reflect the norm in Western societies, where people are typically in the postprandial state for up to 18 hours per day [11]. Therefore, measurement of postprandial triglycerides may give a more appropriate reflection of individual risk for CVD than the measurement of fasting triglycerides [12, 13].

A strong positive association has been established between the levels of postprandial triglycerides and the risk for myocardial infarction, ischemic stroke, and early death [2–8]. Measurement of postprandial triglycerides has shown to be highly discriminatory in CVD risk reclassification of subjects after adjustment for fasting levels of triglycerides, HDL<sub>2</sub> cholesterol, apolipoprotein B, and age [6]. Data obtained over a median period of 11.5 years from the Women's Health study (a cohort of 26509 initially healthy women) demonstrated that fasting triglyceride levels had

little independent association with future cardiovascular events [13]. However, a strong association was found for women in higher quartiles of postprandial triglycerides, independent of baseline cardiac risk factors, markers of insulin resistance, and levels of other lipids. The Copenhagen City Heart study demonstrated that every 1 mmol/L increase in nonfasting triglycerides gave an adjusted risk of 1.5 for myocardial infarction, ischemic heart disease, and all-cause mortality [12]. A number of mechanisms have been proposed for how increased postprandial triglycerides lead to atherosclerosis and CVD, including endothelial dysfunction and the activation of inflammatory processes [14–20].

Nutritional intervention to help maintain control over postprandial levels can help support CVD risk management. It is well recognized that dietary fat, level and type of protein, and simple carbohydrates—particularly fructose—are known to be significant contributors to the postprandial triglyceride response [19, 21–24]. Our research goal was to develop a standardized clinical protocol, called the “**Lipemic Index**”, in which foods and meals can be tested and compared based on their lipemic response as measured by serum postprandial triglyceride surges. While this concept has been explored by other research groups, we have chosen to standardize testing based on fixed energy intakes in order to remove the limitation of a single component-based system (e.g., 50 g glucose regardless of energy content as is done with the glycemic index) [25–27].

This pilot study introduces the concept of ranking foods or meals according to an absolute “**Lipemic Index**” using a reference standard, or via relative “**Lipemic Indexing**” for cross-comparison purposes based on *equivalent caloric intakes*.

In addition to this innovative movement, we also wanted to highlight the limitations of assessing the health implications of foods and meals based solely on their glycemic response, without also looking at the lipemic response of those foods in tandem. We hypothesized that certain commercially available foods and meals which give a favourably low glycemic response could generate a significant rise in TG levels postprandially, based on their listed macronutrient content. In order to assess this hypothesis, we tested a meal replacement beverage marketed directly to diabetic individuals (thanks to its ability to maintain control over postprandial glycemia) against our standard beverage (formulated to elicit a maximal TG response).

With further testing, subjects at risk for CVD may consider foods and meals with a low “**Lipemic Index**” as an important and modifiable risk factor to optimize vascular health.

## 2. Methods

This study was approved as a Phase 1 Human Clinical Trial by the CSERB (Canadian SHIELD Ethics Review Board) on November 18, 2008 (study number 2008NDI001).

**2.1. Subjects.** Subjects were recruited via newspaper advertisement and gave written informed consent prior to testing.

TABLE 1: Subject characteristics.

Parameters	Mean $\pm$ standard error
Age (years)	56.3 $\pm$ 4.7
Weight (kg)	99.9 $\pm$ 2.9
Body mass index (kg/m <sup>2</sup> )	33.9 $\pm$ 1.5
Waist circumference (cm)	119.1 $\pm$ 4.8
Waist-to-hip ratio	1.02 $\pm$ 0.02
Fasting triglyceride (mmol/L)	2.42 $\pm$ 0.37
Fasting glucose (mmol/L)	5.32 $\pm$ 0.16
Fasting total cholesterol (mmol/L)	6.07 $\pm$ 0.30
Fasting LDL-cholesterol (mmol/L)	3.92 $\pm$ 0.29
Fasting HDL-cholesterol (mmol/L)	1.07 $\pm$ 0.11

The group was made up of six males and one postmenopausal female, not taking hormone replacement therapy. Subject characteristics are outlined in Table 1. Exclusion criteria were abnormal fasting glucose levels ( $>6.0$  mmol/L), history of drug abuse, regular consumption of omega-3 supplements, regular strenuous exercise, and certain medications [28, 29]. All subjects reported good overall health with no presence of diagnosed disease. Exclusion criteria and informed consent were reviewed with each subject by one of the study coordinators.

**2.2. Treatments.** All three test products were standardized to target 2100 kJ in order to accommodate potential future ranking of both individual foods and/or meals based on equivalent energy intakes. The first treatment was a reference beverage (2100 kJ/serving), consisting of fat, protein and simple carbohydrates (**LIXR**). The **LIXR** was compared to a popular and commercially available meal replacement product, marketed directly to a diabetic population for postprandial glycemic control, as well as to a commercial fast food breakfast. As seen in Table 2, the standard beverage, diabetic beverage, and fast food breakfast provided essentially identical intakes of total energy (kJ), and energy as fat, with differences in the amounts of carbohydrate and protein. Products were given to subjects in a random order and tested in duplicate with at least two days between each test day.

Based on work by Robertson et al. [30], a standard high, carbohydrate meal was given for consumption to the subjects the evening before each test day. Subjects were advised to abstain from alcohol and strenuous exercise for 24 hours prior to testing and arrived at the research facility in the morning after a 12-hour fast. A catheter was inserted into the subject's arm and a fasting blood sample was taken. The test product was ingested in approximately 10 min. Ten blood samples were obtained at standard time points (0, 15, 30, 45, 60, 90, 120, 180, 240, 300 minutes) during the subsequent five hours and centrifuged to provide serum samples for analysis. Serum glucose (at 0, 15, 30, 45, 60, 90, and 120 min) and triglycerides were analyzed independently by Gamma-Dynacare Medical Laboratories in Brampton, ON [Roche Modular P, Laval, QC].

TABLE 2: Macronutrient composition of test products.

Test product	Total kJ	Fat (g)	kJ from fat	CHO (g)	kJ from CHO	Protein (g)	kJ from protein
Standard beverage	2100	18	678	75	1255	10	167
Diabetic beverage	2084	18	678	42	703	42	703
Fast Food breakfast	2088	19	716	69	1155	13	218

**2.3. Analysis.** Data were collected for analysis, and average values from the duplicate tests were obtained for glucose and triglycerides for each time point and subject. Positive incremental areas under the curve (PIAUCs) were calculated [25] based on these averaged time points for glucose and triglyceride responses. A repeated-measures ANOVA was performed using SAS 9.1 (<http://www.sas.com/index.html-1976>).

### 3. Results

Typical serum glucose responses to each test product are demonstrated in Figure 1 (one subject representative for the group) and typical serum triglyceride responses to each test product are demonstrated in Figure 2 (same subject as shown in Figure 1, as a group representative). A significant difference was found between treatments, both for serum glucose PIAUC ( $P < 0.005$ , repeated measures ANOVA) and for serum triglyceride PIAUC ( $P < 0.05$ ). As seen in Table 3, the PIAUC obtained for serum glucose after the fast food breakfast test were significantly higher (by 317% and 247%) than those obtained after the standard beverage test ( $P < 0.005$ ), and after the diabetic beverage test ( $P < 0.005$ ), respectively. No significant difference was found in the PIAUCs between the standard beverage and the diabetic beverage. Interestingly, the PIAUCs for postprandial triglyceride were significantly higher (by 71% and 59%) for the diabetic beverage than for the fast food breakfast ( $P < 0.05$ ) and for the standard beverage ( $P < 0.05$ ), respectively. No significant difference was found in the PIAUCs for postprandial triglyceride between the standard beverage and the fast food breakfast.

Postprandial serum glucose in response to each test item is shown for Subject 6 as a representative for the group. All time points have been averaged from duplicate trials. Respective PIAUC for the standard, the diabetic beverage, and the fast food breakfast are 42.3, 37.0, and 169.5 mmol glucose·min.

Postprandial serum triglyceride in response to each test item is shown for Subject 6 as a representative for the group. All time points have been averaged from duplicate trials. Respective PIAUC for the standard, the diabetic beverage, and the fast food breakfast are 86.6, 153.8, and 120.0 mmol triglyceride·min.

### 4. Discussion

Postprandial triglyceride surges are now being recognized as major risk factors for CVD and mortality and may even be more favourable than fasting levels with respect to individual

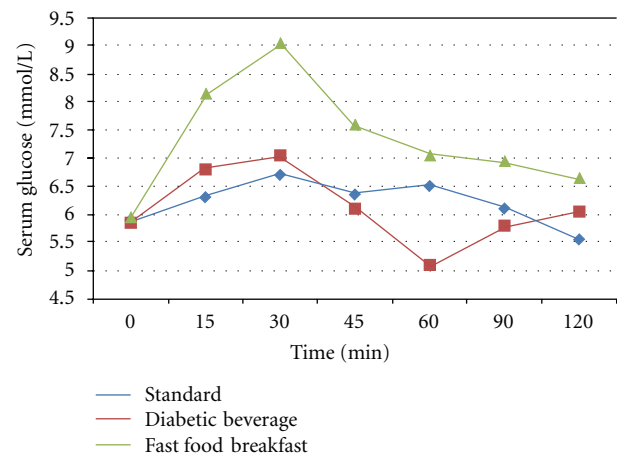


FIGURE 1: Serum glucose response to 2100 kJ standard, diabetic beverage, and fast food breakfast.

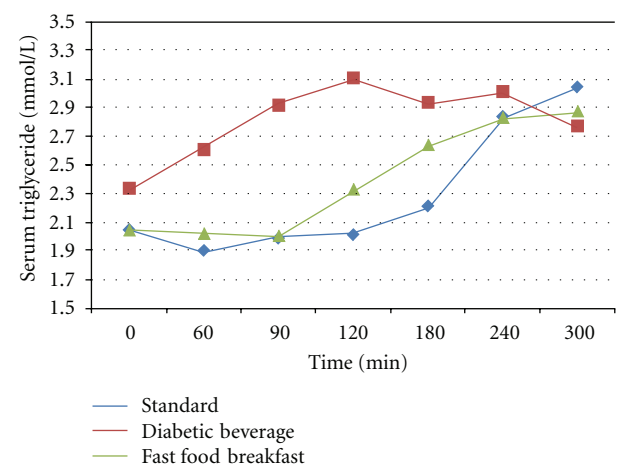


FIGURE 2: Serum triglyceride response to 2100 kJ standard, diabetic beverage, and fast food breakfast.

risk identification [7, 8]. It is recognized that many factors, including nutritional, may influence the postprandial triglyceride response of an individual [28]. Thus, certain measures were followed to recruit subjects who would be expected to give significant postprandial triglyceride responses to the dietary regimens. All seven subjects were overweight and carried body fat in their abdominal regions, with an average waist circumference of 119.1 cm (Table 1). The same evening meal was given to the subjects to be consumed before a 12-hour fast prior to each test. The macronutrient composition of this meal was based on the work of Robertson et al. [30]

TABLE 3: Average positive incremental areas under the curve (PIAUC) for serum glucose and total triglycerides—Standard versus Diabetic Beverage versus Fast Food Breakfast<sup>1</sup>.

PIAUC	Standard	Diabetic Beverage	Fast Food Breakfast
Serum glucose (mmol <i>glucose</i> ·min)	40.5 ± 26.4 <sup>a</sup>	48.7 ± 26.6 <sup>a</sup>	169.0 ± 26.4 <sup>b*</sup>
Total serum triglyceride (mmol <i>triglyceride</i> ·min)	87.5 ± 17.6 <sup>a</sup>	149.3 ± 18.2 <sup>b**</sup>	94.0 ± 17.6 <sup>a</sup>

<sup>1</sup>Values are means ± SE,  $n = 7$ ; <sup>a,b</sup>Different superscripts indicate a statistically-significant difference between the corresponding groups. \* $P < 0.005$ ; \*\* $P < 0.05$  ( $P$  values based on difference of least squares means test).

and meant to encourage increased postprandial triglyceride responses the following morning.

While surges in postprandial triglycerides may not be as extreme in active, healthy young subjects, it has been demonstrated that any rise in postprandial triglyceride is inversely associated with increased flow-mediated vasodilation—a measure of endothelial function [18]. Additional mechanisms by which postprandial triglyceride surges are thought to predispose towards cardiovascular disorders include the formation of highly atherogenic remnant lipoprotein particles and increases in interleukin-6, tumour necrosis factor- $\alpha$ , high sensitivity C-reactive protein, and cell adhesion molecules [20, 31]. Therefore, foods and meals that are assigned a high “**Lipemic Index**” may not elicit the same magnitude of postprandial triglyceride response in the general population as in our current and future stock subject group—but the importance of this nutritional information and modification thereof could benefit the former group for preventative CVD risk reduction. Notably, an expert panel statement by Kolovou et al. has recently provided a target for a desirable nonfasting TG concentration of  $<2.5$  mmol/L [32]. The American Heart Association has also recommended that nonfasting triglyceride levels below 2.28 mmol/L generally correspond with safe fasting levels of TGs [33].

A surprising and potentially important finding from this project is the marked postprandial triglyceride response seen after the ingestion of a “diabetic/glycemic friendly” commercial product marketed directly to diabetics—patients already at an increased risk for CVD [1]. Although ideal for patients in that it controlled postprandial levels of glucose (across all subjects studied, as seen in, e.g., Figure 1 and Table 3), the high lipemic response associated with this product (across all subjects studied, as seen in e.g. Figure 2 and in Table 3) highlights a need for a broader approach in the clinical evaluation of foods and meals with respect to health-related outcomes including assessment of the “**Lipemic Index**”. There is considerable evidence that, in addition to dietary fat, simple carbohydrates (especially fructose) as well as the level and type of dietary protein can significantly influence the magnitude of the postprandial triglyceride elevations [21–24]. Our findings herein emphasize a need for education about the importance of postprandial triglyceride surges as a strong independent risk factor for CVD among health care professionals, high-risk patients, and food industry professionals.

With future research into the development of a clinical protocol, a standard beverage may be (arbitrarily) assigned a “**Lipemic Index**” of 100 based on its average associated

PIAUCs in a specific group of subjects. This could create an absolute scale by which other foods and meals may be scored against, analogous to the approach taken for the glycemic index. In this regard, assigning an arbitrary “**Lipemic Index**” of 100 to the PIAUC for the standard beverage in Table 3 would lead to a calculated “**Lipemic Index**” value for the diabetic beverage of 171 ( $149.3/87.5 \cdot 100$ ). Additionally, foods and meals may be directly compared to each other in order to determine their relative “**Lipemic Index**” provided they are given in equicaloric quantities. In this way, the “**Lipemic Index**” could become an effective educational tool for individuals who wish to nutritionally manage CVD risk.

Finally, it is highly likely that most food products and meals exhibiting an unfavourable “**Lipemic Index**” can be readily reformulated by the food industry so as to reduce any health risks associated with postprandial triglyceride surges by such products.

## 5. Conclusions

Our research highlights that adherence to currently recommended nutritional management tools may not necessarily reduce total risk for CVD complications since, as found herein, a popular diabetic-targeted beverage which provided excellent blood glucose control led to a surprisingly exaggerated postprandial triglyceride response. It would therefore appear very important to develop a standard clinical protocol to define the lipemic response of food items or meals as has become popular for the glycemic index. The “**Lipemic Index**” may be a key modifiable risk factor for CVD risk reduction, which should be considered by health care professionals when making specific food and dietary recommendations. The “**Lipemic Index**” can readily be applied to whole foods, individual products, or meals because it is standardized for energy (regardless of the levels or types of macronutrients). This practical parameter should facilitate knowledge transfer to the general public (including high-risk individuals such as those with the metabolic syndrome and diabetics) thereby helping to promote positive choices for the nutritional prevention and management of CVD. The present study demonstrates a standardized approach, which can be further modified if desired, to evaluate, compare, or rank foods and meal offerings based on their “**Lipemic Index**”.

## Conflict of Interests

B. Holub and M. Wlodek both hold ownership shares in Nutrasource Diagnostics Inc.



## Authors' Contributions

E. M. O'Reilly acquired all data, participated in study design and interpretation as a graduate student at the University of Guelph, and subsequently drafted the paper. B. J. Holub conceived the study, participated in its design and coordination, and helped to draft the paper. M. Laidlaw participated in the design and coordination of the study and supervised sample collection and treatment. C. Garrioch assisted in paper review and preparation. M. G. Wlodek participated in study design.

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