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Assessment of postprandial triglycerides in clinical practice: Validation in a general population and coronary heart disease patients

Pablo Perez-Martinez, MD, PhD^{1,*},

Lipid and Atherosclerosis Unit, Department of Internal Medicine, IMIBIC/Reina Sofia University Hospital/University of Cordoba, Cordoba, Spain

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

Juan F. Alcala-Diaz, MD¹,

Lipid and Atherosclerosis Unit, Department of Internal Medicine, IMIBIC/Reina Sofia University Hospital/University of Cordoba, Cordoba, Spain

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

Edmon K. Kabagambe, MD,

Division of Epidemiology, Department of Medicine, Vanderbilt University Medical Center, Nashville, TN, USA

Antonio Garcia-Rios, MD PhD,

Lipid and Atherosclerosis Unit, Department of Internal Medicine, IMIBIC/Reina Sofia University Hospital/University of Cordoba, Cordoba, Spain

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

Michael Y. Tsai, MD, PhD,

Department of Laboratory Medicine & Pathology, University of Minnesota, Minneapolis, MN, USA

Javier Delgado-Lista, MD, PhD,

Lipid and Atherosclerosis Unit, Department of Internal Medicine, IMIBIC/Reina Sofia University Hospital/University of Cordoba, Cordoba, Spain

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

*Corresponding author. Reina Sofia University Hospital, Lipids and Atherosclerosis Research Unit, Avda. Menéndez Pidal, s/n., Córdoba 14004, Spain. pablopermar@yahoo.es.

¹Equal contribution.

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Genovefa Kolovou, MD, PhD,

1st Cardiology Department, Onassis Cardiac Surgery Center, Athens, Greece

Robert J. Straka, PhD,

Department of Experimental and Clinical Pharmacology, College of Pharmacy, University of Minnesota, Minneapolis, MN, USA

Francisco Gomez-Delgado, MD, PhD,

Lipid and Atherosclerosis Unit, Department of Internal Medicine, IMIBIC/Reina Sofia University Hospital/University of Cordoba, Cordoba, Spain

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

Paul N. Hopkins, MD, PhD,

Department of Internal Medicine, University of Utah Health Sciences Center, Salt Lake City, UT, USA

Carmen Marin, PhD,

Lipid and Atherosclerosis Unit, Department of Internal Medicine, IMIBIC/Reina Sofia University Hospital/University of Cordoba, Cordoba, Spain

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

Ingrid Borecki, PhD,

Division of Statistical Genomics in the Center for Genome Sciences of the Washington University, St. Louis, USA

Elena M. Yubero-Serrano, PhD,

Lipid and Atherosclerosis Unit, Department of Internal Medicine, IMIBIC/Reina Sofia University Hospital/University of Cordoba, Cordoba, Spain

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

James E. Hixson, PhD,

Human Genetics Center, University of Texas Health Science Center at Houston, Houston, TX, USA

Antonio Camargo, PhD,

Lipid and Atherosclerosis Unit, Department of Internal Medicine, IMIBIC/Reina Sofia University Hospital/University of Cordoba, Cordoba, Spain

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

Michael A. Province, PhD,

Department of Genetics Division of Statistical Genomics, Washington University School of Medicine, St. Louis, MO, USA

Javier Lopez-Moreno, MD,

Lipid and Atherosclerosis Unit, Department of Internal Medicine, IMIBIC/Reina Sofia University Hospital/University of Cordoba, Cordoba, Spain

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

Fernando Rodriguez-Cantalejo, MD,

Biochemical Laboratory, Hospital Universitario Reina Sofia, Cordoba, Spain

Francisco J. Tinahones, MD, PhD,

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

Servicio de Endocrinologia y Nutricion, Hospital Clinico Virgen de la Victoria, Malaga, Spain

Dimitri P. Mikhailidis, MD, PhD,

Department of Clinical Biochemistry, Royal Free Hospital Campus, University College London Medical School, University College London, London, UK

Francisco Perez-Jimenez, MD, PhD,

Lipid and Atherosclerosis Unit, Department of Internal Medicine, IMIBIC/Reina Sofia University Hospital/University of Cordoba, Cordoba, Spain

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

Donna K. Arnett, PhD,

Department of Epidemiology, University of Alabama at Birmingham, Birmingham, AL, USA

Jose M. Ordovas, PhD,

Jean Mayer U.S. Department of Agriculture Human Nutrition Research Center on Aging, Tufts University School of Medicine, Boston, MA, USA

Department of Epidemiology, National Center of Cardiovascular Investigations, Madrid, Spain

Madrid Institute of Advanced Studies–Food, Madrid, Spain

Jose Lopez-Miranda, MD, PhD

Lipid and Atherosclerosis Unit, Department of Internal Medicine, IMIBIC/Reina Sofia University Hospital/University of Cordoba, Cordoba, Spain

CIBER Fisiopatologia Obesidad y Nutricion (CIBEROBN), Instituto de Salud Carlos III, Madrid, Spain

Abstract

BACKGROUND—Previous studies have suggested that for clinical purposes, subjects with fasting triglycerides (TGs) between 89–180 mg/dl (1–2 mmol/l) would benefit from postprandial TGs testing.

OBJECTIVE—To determine the postprandial TG response in 2 independent studies and validate who should benefit diagnostically from an oral-fat tolerance test (OFTT) in clinical practice.

METHODS—A population of 1002 patients with coronary heart disease (CHD) from the CORDIOPREV clinical trial and 1115 white US subjects from the GOLDN study underwent OFTTs. Subjects were classified into 3 groups according to fasting cut points of TGs to predict the usefulness of OFTT: (1) TG < 89 mg/dl (<1 mmol/l); (2) TG, 89–180 mg/dl (1–2 mmol/l); and (3) TG > 180 mg/dl (>2 mmol/l). Postprandial TG concentration at any point > 220 mg/dl (>2.5 mmol/l) has been pre-established as an undesirable postprandial response.

RESULTS—Of the total, 49% patients with CHD and 42% from the general population showed an undesirable response after the OFTT. The prevalence of undesirable postprandial TG in the CORDIOPREV clinical trial was 12.8, 50.3, and 89.7%, in group 1, 2, and 3, respectively ($P < .001$) and 11.2, 58.1, and 97.5% in group 1, 2, and 3, respectively ($P < .001$) in the GOLDN study.

CONCLUSIONS—These two studies validate the predictive values reported in a previous consensus. Moreover, the findings of the CORDIOPREV and GOLDN studies show that an OFTT is useful to identify postprandial hyperlipidemia in subjects with fasting TG between 1–2 mmol/l (89–180 mg/dL), because approximately half of them have hidden postprandial hyperlipidemia, which may influence treatment. An OFTT does not provide additional information regarding postprandial hyperlipidemia in subjects with low TG (<1 mmol/l, <89 mg/dL) or increased TG (>2 mmol/l, >180 mg/dl).

Keywords

Triglycerides; Postprandial lipemia; Oral-fat tolerance test; Coronary heart disease; CORDIOPREV study; GOLDN study

Current recommendations propose that therapeutic targeting of elevated fasting plasma triglycerides (TGs) 1.7 mmol/L or 150 mg/dL, a marker of TG-rich lipoprotein (TRL) and their remnants, may provide further cardiovascular benefit.¹ However, we spend most of the time in the postprandial state because of several meals and occasional “snacking”. Postprandial lipemia is a physiological response occurring 2 to 12 hours after consuming a fat-enriched meal, and it is defined by the extent and duration of the increase in plasma TGs.^{2,3} Thus, the evaluation of postprandial lipemia becomes more physiologically relevant, particularly since nonfasting TGs are independent predictors of the risk of atherosclerosis and cardiovascular disease (CVD).⁴ Furthermore, postprandial hypertriglyceridemia is associated with increased inflammation and oxidation that influences vascular function.^{5–7}

Currently, there is no definitive consensus or enough evidence to sustain the further development of routine nonfasting/postprandial TGs measurements for clinical and research purposes. In this context, an Expert Panel of scientists and clinicians together with a meta-analysis of 113 studies conducted in healthy white subjects (without clinical or physician-diagnosed CVD or metabolic disease, with baseline TGs <2.0 mmol/l [<177 mg/dL], with body mass index <30 kg/m² and not on chronic medication) has suggested that subjects with fasting TGs between 1–2 mmol/L (89–180 mg/dL) would benefit from the additional clinical information provided by an oral-fat tolerance test (OFTT).^{8,9} In contrast, individuals with fasting TGs <1 mmol/L (89 mg/dL) rarely have an exaggerated or delayed TG response after an OFTT, even in the presence of dyslipidemia or obesity. Conversely, individuals with fasting TGs >2 mmol/L (180 mg/dL) usually have an exaggerated and delayed TG response,

and an OFTT will not add clinically relevant information. Based on this evidence, the Expert Panel⁸ suggested that an OFTT should not be performed in the latter 2 subgroups. However, other studies have demonstrated the heterogeneity of the response of TGs after fat load.^{10,11} In this context, Wojczynski et al. observed that the pattern of postprandial change in TGs was qualitatively similar for normo-triglyceridemic and hypertriglyceridemic individuals; however, the magnitude of the response was exaggerated among hypertriglyceridemic when compared with normo-triglyceridemic individuals.¹²

To confirm the postprandial TGs limits set by the Expert Panel,⁸ we evaluated the results of 2 studies. The CORonary Diet Intervention with Olive Oil and Cardiovascular PREvention (CORDIOPREV) study (NCT00924937) is an ongoing prospective, controlled trial with a mean follow-up of 5 years, including 1002 patients with coronary heart disease (CHD). In this cohort of high-risk patients, our primary aim was to explore the degree of undesirable postprandial TG response in patients with CHD based on an OFTT. We also assessed the postprandial TG response in a second population drawn from a multicenter, population-based study comprising a large group of US white subjects from the Genetics of Lipid Lowering Drugs and Diet Network (GOLDN) study (NCT00083369).

Material and methods

CORDIOPREV population

The CORDIOPREV study is an ongoing prospective, randomized, open, controlled trial including 1002 patients with CHD and with a last coronary event >6 months before enrollment into 2 dietary models (Mediterranean or low-fat diet).¹³ The diet was followed for a period of 5 years in addition to conventional treatment for CHD. Patients were recruited from November 2009 to February 2012, mostly at the Reina Sofia University Hospital (Cordoba, Spain), but other centers from the Cordoba and Jaen provinces also participated. The inclusion and exclusion criteria have been published.¹³ In summary, patients were eligible if they were >20 years, but <75 years, had established CHD without clinical events in the last 6 months, agreed to follow a long-term dietary intervention and did not have severe diseases or expected life expectancy <5 years. Patients were classified in 3 groups according to fasting TG concentrations: (1) <1 mmol/L (89 mg/dl), (2) 1–2 mmol/L or (89–180 mg/dL), and (3) >2 mmol/L (180 mg/dl). Postprandial TG concentration at any point >2.5 mmol/L (220 mg/dL) has been proposed as an undesirable response. These limits were recommended by the Expert Panel consensus.⁸ The participants in CORDIOPREV study were on treatment for CHD, which could influence the results of TG concentrations after an OFTT. All patients gave their written informed consent to participate in the study. The local ethics committees, after the Helsinki declaration and the good clinical practices, approved the trial protocol and all amendments.

At the entry to the CORDIOPREV study, participants had an OFTT using a weight-adjusted meal (0.7-g fat and 5-mg cholesterol per kg body weight) with 12% saturated fatty acids (SFAs), 10% polyunsaturated fatty acids (PU-FAs), 43% monounsaturated fatty acids (MUFAs), 10% protein, and 25% carbohydrates (CHO).¹⁴ After that, they were enrolled into 2 different dietary models (Mediterranean or low-fat diet). Meal preparation for the OFTT was performed by nutritionists using olive oil, skimmed milk, white bread, cooked egg

yolks, and tomatoes. Previous to starting the test, the patients had been fasting for 12 hours and were asked to refrain from smoking during the fasting period and from alcohol intake during the preceding 7 days. They were also asked to avoid strenuous physical activity the day before the test. At 8:00 AM, patients attended the laboratory, completed anthropometric (weight, height, waist circumference, body mass index [BMI], blood pressure), and biochemical measurements, donated a fasting blood sample and under supervision, ingested the fatty meal. The meal was consumed in 20 minutes. After the meal, volunteers were rested and consumed no food for 5 hours but were allowed to drink water. Blood samples for biochemical testing were collected before the meal and every hour during the next 4 h, after recommendations for an OFTT proposed in a meta-analysis.⁹

GOLDN population

This US study sample comprised of 533 men and 582 women selected from those who participated in the GOLDN study (Minneapolis, MN and Salt Lake City, UT) and who had full data record for the OFTT. Detailed design and methodology for the GOLDN study have been described.¹⁵ Briefly, exclusion criteria included recent history (6 months) of myocardial infarction, coronary bypass surgery, coronary angioplasty (percutaneous coronary intervention, PCI), self-report of a positive history of liver, kidney, pancreas, or gallbladder disease, or a history of malabsorption of nutrients; current use of insulin or warfarin; pregnant women; or women of childbearing potential not using contraception. Individuals who reported current use of prescription and/or over-the-counter hypolipidemic drugs or dietary supplements known to influence lipid values (e.g., fish oils, flaxseed oil, niacin, and so forth) were required to consult their physician for approval to discontinue these lipid-lowering agents for 4 weeks before study participation. Criteria for inclusion in the subgroup analyses included a BMI <25 kg/m² to match the anthropometric characteristics of the participants in the primary study. Participants were asked to fast for >12 h and abstain from using alcohol for >24 h before visiting the clinic. The OFTT consisted of a meal after the protocol of Patsch et al.¹⁶ This high-fat meal was consumed after fasting laboratory measurements were collected. The meal was consumed within 15 minutes and had 700 calories/m² body surface area (2.93 MJ/m², body surface area); 3% of calories were derived from protein, 14% from carbohydrates, and 83% from fat sources. The cholesterol content and the ratio of polyunsaturated to saturated fat were 240 mg and 0.06, respectively. The average person consumed 175-mL heavy whipping cream (39.5% fat) and 7.5 mL of chocolate-flavored or strawberry-flavored syrup to increase palatability. Blood samples were drawn immediately before (time 0) and 3.5 and 6 h after consuming this high-fat meal. Participants were allowed to drink water but they did not ingest any other food or beverage. In addition, they abstained from exercise and strenuous physical work until the final blood sample was obtained. All meals were administered in the early morning, usually between 06.30 and 09.00 hour. Written informed consent was obtained from each participant at his/her screening visit. This protocol was approved by the Institutional Review Boards at the University of Minnesota, the University of Utah, and Tufts University.

Laboratory methods in the CORDIOPREV population

Venous blood from an antecubital vein and collected into Vacutainer tubes with no anticoagulant and tubes containing EDTA, and immediately transferred to 4°C. To minimize

proteolytic degradation, plasma was supplemented with protease inhibitor cocktail (Roche Diagnostic, Germany) 40 μ L/mL of plasma. Plasma and serum samples were frozen at -80°C for further analysis. Serum parameters were measured using Architect c-16000 analyzers (Abbott, Chicago, Illinois, USA) by spectrophotometric techniques (enzymatic colorimetric methods): hexokinase method for glucose and oxidation-peroxidation for total cholesterol, high-density lipoprotein cholesterol (HDL-C), and TGs. Apolipoprotein (apo) A-1 and apo B were determined by immunoturbidimetry using mouse-specific antibodies. Plasma levels of insulin were measured by chemiluminescent microparticle immunoassay using an analyzer (i-2000 Abbott Architect, Chicago, Illinois). Homeostatic model assessment insulin resistance (HOMA-IR) was derived from fasting glucose and insulin levels $[(\text{fasting plasma glucose} \times \text{fasting serum insulin})/22.5]$.¹⁷ As HOMA-IR takes into account both insulin and glucose levels, it may be a more complete index than plasma insulin. Plasma concentrations of high-sensitivity C-reactive protein (hsCRP) were determined by high-sensitivity enzyme-linked immunosorbent assay (Bio-Check, Inc., Foster City, CA, USA) at the University College Dublin.

Laboratory methods in the GOLDN population

All samples were centrifuged within 20 minutes of collection at $2000\times g$ for 15 minutes at 4°C and stored frozen at -70°C . For each analyte, specimens from each participant were assayed in the same batch to eliminate inter-assay imprecision. Biochemical analyses were performed as previously described.^{18,19} Briefly, TGs were measured using a glycerol-blanked enzymatic method (Trig/GB, Roche Diagnostics, Indianapolis, IN), and cholesterol was measured using a cholesterol esterase, cholesterol oxidase reaction (Chol R1, Roche Diagnostics) on the Roche/Hitachi 911 Automatic Analyzer (Roche Diagnostics Corporation). For HDL-C, the non-HDL-C was first precipitated with magnesium/dextran. Low-density lipoprotein-cholesterol (LDL-C) was measured by a homogeneous direct method (LDL Direct Liquid Select Cholesterol Reagent, Equal Diagnostics, Exton, PA). Fasting glucose was measured using the hexokinase-mediated reaction on a Hitachi 911 analyzer (Roche Diagnostics), whereas fasting insulin was measured using the human insulin-specific RIA kit (Linco Research, St. Charles, MO). hsCRP was measured on the Hitachi 911 using a latex particle-enhanced immunoturbidimetric assay (Kamiya Biomedical Company, Seattle, WA).²⁰

Statistical analysis

Biochemical variables were assessed for normality of distribution, and skewed variables were normalized by log 10 or square root transformation as appropriate. Continuous variables were compared using the analysis of variance (ANOVA), and qualitative variables were compared using chi-square test. The odd ratios for undesirable postprandial response, defined as TG concentrations at any point during the OFTT >2.5 mmol/L or 220 mg/dL, were calculated through a logistic regression with groups according to fasting TGs as covariates. A two-tailed $P < .05$ was considered significant. A logistic regression using backward elimination method was performed to identify those independent predictors of undesirable postprandial response in the group of patients with fasting TGs between 1–2 mmol/L (89–180 mg/dL). As covariables to evaluate, we included age, gender, alcohol intake (non-drinkers, <8 , 8–16, or >16 g/day), smoking status (never, former, or active

smoker), anthropometric variables such as waist circumference, systolic blood pressure, diastolic blood pressure, BMI, and fasting blood variables such as HDL-C, TGs, non-HDL-C, glucose, insulin, HOMA-IR, hsCRP, and also the presence of metabolic syndrome.²¹ To evaluate the discriminatory capability of the models, we calculated the area under the receiver operation characteristic (ROC) curves. All statistical analyses were carried out using SPSS, version 18.0, for Windows (SPSS Inc, Chicago, IL).

Results

We explored the dynamic response during the OFTT to identify those subjects with postprandial TG concentrations at any point >2.5 mmol/L or >220 mg/dL, considered as an undesirable response. Thus, 49% patients with CHD from the CORDIOPREV and 42% white US subjects from the GOLDN showed an undesirable response after the OFTT.

To predict the utility of OFTT, patients were classified into 3 groups according to fasting TG concentrations: (group 1) TGs: <1 mmol/L or 89 mg/dL; (group 2) TGs: 1–2 mmol/L or 89–180 mg/dL; and (group 3) TGs >2 mmol/L or 180 mg/dL. These limits were recommended by the expert panel consensus.⁸ Baseline demographic and metabolic differences according to these groups are shown in Table 1 for the CORDIOPREV and GOLDN studies. The percentage of patients on lipid-lowering drugs (fibrates or statins) was not significantly different between groups (Table 1).

We also analyzed the prevalence of undesirable TG postprandial concentrations by groups. In the CORDIOPREV study, the prevalence of TG concentration at any point >2.5 mmol/L or 220 mg/dL was 12.8, 50.3, and 89.7%, in groups 1, 2, and 3, respectively, suggesting undesirable response ($P < .001$; Fig. 1A). Thus, group 2 showed the more heterogeneous response. The odd ratios (ORs) for undesirable postprandial response, defined as TG concentration at any point during the OFTT >2.5 mmol/L or 220 mg/dL, were calculated through a logistic regression with groups according to fasting TGs as covariates (odds ratio [OR] = 6.932, 95% confidence intervals [CI], 4.616–10.411 for fasting TG between 1–2 mmol/L and 89–180 mg/dL group; OR = 62.013, 95% CI, 34.717–110.769 for fasting TG >2 mmol/L or 180 mg/dL group).

In the GOLDN cohort, prevalence of undesirable postprandial TGs was 11.2, 58.1, and 97.5% in groups 1, 2, and 3, respectively ($P < .001$). The OR for undesirable postprandial response were significantly higher for group 2 (OR, 5.1; 95% CI, 3.6–7.2; $P < .001$) and for group 3 (OR, 99.2; 95% CI, 56.2–174.9; $P < .001$; Fig. 1B).

In both cohorts, a logistic regression using backward elimination method was performed to identify the independent predictive variables of undesirable postprandial response in the group of patients with fasting TGs between 1–2 mmol/L and 89–180 mg/dL. In the CORDIOPREV study, only fasting TGs (OR = 1.04, $P < .001$) and age (OR = 0.972, $P = .019$) remained in the final step. Receiver operating characteristics (ROC) analysis of the predicted probabilities by this model showed an area under the curve (AUC) ROC = 0.748. In the GOLDN study, fasting TGs (OR = 1.01, $P < .001$), fasting glucose (OR = 0.983, $P = .02$), age (OR = 1.03, $P < .001$), gender (OR = 2.31 for males, $P < .001$), metabolic syndrome

(OR = 2.172, $P = .004$), systolic blood pressure (OR = 0.986, $P = .042$), and HDL-C (OR = 1.017, $P > .05$) remained in the final step. ROC analysis of the predicted probabilities by this model showed an AUC ROC = 0.706. Tables 2 and 3 summarize the final models and coefficients in each cohort.

Discussion

These 2 studies validate the predictive values reported in a previous consensus,⁸ where patients with fasting TGs <1 mmol/L or 89 mg/dL commonly do not have exaggerated response and those with >2 mmol/L or 180 mg/dL do have and therefore will not benefit diagnostically from an OFTT. Postprandial hypertriglyceridemia appears to be common; thus, 49% patients with CHD (CORDIOPREV study) and 42% from a general population (GOLDN study) showed an undesirable response after the OFTT. Based on our findings, we recommend that subjects with fasting TG concentrations between 1–2 mmol/L and 89–180 mg/dL should be tested to identify an exaggerated postprandial TG response.

In the last decade, evidence has emerged supporting the role of nonfasting TGs or TRL as an additional predictor of CVD.^{22,23} Moreover, exaggerated postprandial hypertriglyceridemia has also been considered a risk factor for CVD.²⁴ This metabolic abnormality is principally due to overproduction and/or decreased catabolism of TRLs and is a consequence of such factors as genetics,^{25–27} the environment,²⁸ and pathologic conditions¹⁴ that modify metabolism during the postprandial period. Accumulation of TRLs in the postprandial state promotes the formation of small, dense low-density lipoproteins, as well as oxidative stress, inflammation, and endothelial dysfunction, all of which compound the risk of CVD.⁴ In modern society, postprandial lipid metabolism is relevant, given that this is a common metabolic state (due to frequent meals/snacking).

However, the assessment of postprandial hypertriglyceridemia in the clinical setting has been hampered by technical complexity and the lack of well-established clinical protocols for evaluating postprandial lipemia. Nevertheless, there is now enough evidence to sustain further development of routine nonfasting/postprandial TG measurements for clinical and research purposes. Although at this point, there is no internationally agreed management for postprandial hypertriglyceridemia, a previous consensus⁸ has suggested a simple clinical protocol for investigating postprandial TG measurements and has pointed out cutoffs for exaggerated and delayed response (TG concentration <2.5 mmol/L (220 mg/dL) at any time after an OFTT meal should be considered as a desirable postprandial TG response; higher levels would be undesirable). Based on this definition, in the present study, we assessed the frequency of undesirable postprandial TG response in 2 large populations. Furthermore, we explored several independent predictive variables of undesirable postprandial response in the group of patients with fasting TGs between 1–2 mmol/L and 89–180 mg/dL and in both cohorts, fasting TGs remained in the final step. In the GOLDN study, systolic blood pressure, HDL-C, and the presence of metabolic syndrome remained in the final step. In this context, we have previously reported in the CORDIOPREV cohort that fasting TGs, fasting glucose, and waist circumference were significant independent contributors of postprandial TGs levels, with fasting TGs as the major contributors. In the same study, we also showed that, in nonhypertriglyceridemic patients, the magnitude of postprandial response is related

to the number of altered MetS components.¹¹ Nevertheless, to our knowledge, the present study is the first to propose prediction models of impaired postprandial TGs based on a general population and CHD patients. These proposed models should be replicated and tested in different populations. In our study, sex was only a predictor of undesirable postprandial response in the population-based cohort. This could be related to the fact that the mean age of the CORDIOPREV cohort is higher than in the GOLDN study. Several studies have demonstrated that men have higher levels of fasting and postprandial TG than premenopausal women.²⁹ This fact has been related to a higher clearance capacity in women caused by an estrogen-induced increase in LPL activity.³⁰ However, this gender protective effect is mitigated in postmenopausal women.³¹

Although randomized trials showing cardiovascular benefit of TGs reduction are scarce, new molecules are being developed, such as incretin-based therapies, dual peroxisome-proliferator-activated receptor α/δ agonists, inhibitors of diacylglycerol acyltransferase 1, and microsomal triglyceride transfer protein, and antisense oligonucleotides for apo B-100 and apo C-III.³² Moreover, well-designed large-scale trials have been initiated that will hopefully provide conclusive evidence as to whether lowering TGs reduces the risk of CVD.³² Some of these new drugs may exert their effects by modifying the postprandial lipoprotein metabolism (i.e., microsomal triglyceride transfer protein inhibitors).³³ Thus, the nonfasting/postprandial testing of lipoproteins may be an important measurement.

Our 2 studies involved a larger number of participants ($n = 2115$) when compared with most studies evaluating postprandial lipemia, which involve <100 participants, with the exception of the EARS and EARS II studies ($n = 1445$ and $n = 822$ participants, respectively).^{34,35} This allows drawing more convincing conclusions. However, there are limitations. We included 2 independent studies with different inclusion criteria and a different protocol. The participants in CORDIOPREV study were on treatment for CHD, which could influence TG concentrations after OFTT. However, the percentage of patients on lipid-lowering drugs (fibrates or statins) was similar between the 3 groups according to the fasting TG. Thus, undesirable postprandial TG concentrations could be high before hypolipidemic treatment or if treatment was not adequate. Despite the differences between the 2 studies, the criteria we used still identified low and very similar percentages (12.8 and 11.2%) of postprandial lipemia in those with fasting TG levels < 89 mg/dL (<1 mmol/L) and high and very similar percentages (89.7 and 97.5%) of postprandial lipemia in those with TG levels > 180 mg/dL (>2 mmol/L). Those who were judged to need an OFTT (fasting TG, 89–180 mg/dL; 1–2 mmol/L) also had very similar percentages (50.3% and 58.1%) of postprandial lipemia. This suggests that despite differences between the CORDIOPREV and GOLDN studies, the criteria for defining the need of postprandial lipemia proposed by the consensus⁸ are likely to be useful in clinical practice and for planning research. Finally, we suggest a simplified OFTT for clinical use or for large-scale epidemiological studies. Thus, we recommend an FTT meal consisting of 75-g fat, 25-g carbohydrates, and 10-g protein. Furthermore, the fatty test meal should contain mixtures of saturated and unsaturated fatty acids in a digestible form and be easy to prepare. We suggest an option such as a commercially available whipped cream or Cream Cheese (about 250 g, based on actual nutrient contents) with addition of 15-g sugar (the cream will provide approximately 10-g protein and 10-g

carbohydrate). An alternative to dairy products could be commercially available liquid formulas for this purpose.

Conclusions

The findings of the CORDIOPREV and GOLDN studies show that an OFTT is useful to identify postprandial hyperlipidemia in subjects with fasting TG between 1–2 mmol/l (89–180 mg/dl) since approximately half of them have hidden postprandial hyperlipidemia, which may influence treatment. An OFTT does not provide additional information regarding postprandial hyperlipidemia in subjects with low TG (<1 mmol/l, <89 mg/dl) or increased TG (>2 mmol/l, >180 mg/dl).

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Figure 1.

Prevalence of undesirable postprandial triglycerides (TGs) in the CORDIOPREV (A) and GOLDN (B) population according to fasting TGs: (1) TG < 89 mg/dL (<1 mmol/l); (2) TG 89–180 mg/dL (1–2 mmol/l); and (3) TG ≥ 180 mg/dL (≥2 mmol/l). The black bars represent the percentage of patients with postprandial TG concentration at any point >220 mg/dL (>2.5 mmol/l) within each group. In the CORDIOPREV cohort (A), the odds ratio (OR) for undesirable postprandial response were significantly higher for group 2 (OR, 6.8; 95% CI, 4.5–10.3; P < .001) and for group 3 (OR, 58.8; 95% CI, 32–105; P < .001). In the GOLDN cohort (B), the OR for undesirable postprandial response was significantly higher for group 2 (OR, 5.1; 95% CI, 3.6–7.2; P < .001) and for group 3 (OR, 99.2; 95% CI, 56.2–174.9; P < .001).

Table 1

Baseline characteristics according to fasting TG groups

	Fasting TG, <89 mg/dL	Fasting TG (between 89–180 mg/dL)	Fasting TG, >180 mg/dL	P value
The CORDIOPREV study				
N	283	503	216	
Age (y)	59.9 ± 9.0 [‡]	60.3 ± 9.0 [‡]	57.3 ± 8.7 ^{*,‡}	<.001
Gender (% male)	82.6	83.1	85.6	NS
Weight (kg)	80.8 ± 12.2 ^{‡,‡}	85.8 ± 14.8 ^{*,‡}	89.4 ± 15.1 ^{*,‡}	<.001
BMI (kg/m ²)	29.7 ± 4.1 ^{‡,‡}	31.4 ± 4.6 [*]	32.1 ± 4.3 [*]	<.001
Waist circumference (cm)	102.2 ± 10.5 ^{‡,‡}	105.4 ± 11.6 ^{*,‡}	108.3 ± 12.2 ^{*,‡}	<.001
SBP (mm Hg)	136.7 ± 19.4	139.9 ± 20.6	138.7 ± 19.2	NS
DBP (mm Hg)	75.7 ± 10.5 [‡]	77.5 ± 11.3	78.6 ± 10.1 [*]	.01
Creatinine (mg/dL)	0.8 ± 0.1 ^{‡,‡}	0.9 ± 0.2 ^{*,‡}	1.0 ± 0.3 ^{*,‡}	<.001
LDL-C (mg/dL)	87.7 ± 22.4	90.1 ± 25.8	85.9 ± 29.8	NS
TC (mg/dL)	149.8 ± 27.2 ^{‡,‡}	157.7 ± 29.6 ^{*,‡}	174.5 ± 34.0 ^{*,‡}	<.001
HDL-C (mg/dL)	47.5 ± 11.4 ^{‡,‡}	41.6 ± 8.4 ^{*,‡}	36.8 ± 8.5 ^{*,‡}	<.001
TG (mg/dL)	71.6 ± 11.8 ^{‡,‡}	125.6 ± 24.3 ^{*,‡}	238.4 ± 49.4 ^{*,‡}	<.001
Non-HDL-C (mg/dL)	102.1 ± 22.4 ^{‡,‡}	115.9 ± 26.4 ^{*,‡}	137.9 ± 31.6 ^{*,‡}	<.001
MetS (%)	30.7	59.6	89.8	<.001
hsCRP (mg/L)	2.1 ± 1.9 [‡]	2.4 ± 1.9 [‡]	3.0 ± 2.2 ^{*,‡}	<.001
Insulin (mU/l)	8.3 ± 6.4 ^{‡,‡}	10.7 ± 10.9 ^{*,‡}	15.3 ± 14.7 ^{*,‡}	<.001
HOMA-IR	3.1 ± 3.6 ^{‡,‡}	4.3 ± 5.3 ^{*,‡}	6.1 ± 6.5 ^{*,‡}	<.001
Lipid-lowering drugs				
Fibrates (%)	1.4	1.3	2.3	NS
Statins (%)	83.3	86.2	87.0	NS
The GOLDN study				
N	401	459	255	

	Fasting TG, <89 mg/dL	Fasting TG (between 89–180 mg/dL)	Fasting TG, >180 mg/dL	P value
Age (y)	42.9 ± 16.7 ^{†,‡}	50.2 ± 16.1 ^{*,‡}	52.8 ± 13.9 ^{*,‡}	<.001
Gender (% male)	39.9	50.5	55.3	<.001
Weight (kg)	74.9 ± 16.9 ^{†,‡}	85.2 ± 17.1 ^{*,‡}	90.5 ± 17.7 ^{*,‡}	<.001
BMI (kg/m ²)	25.7 ± 5.2 ^{†,‡}	29.1 ± 5.3 ^{*,‡}	30.6 ± 5.2 ^{*,‡}	<.001
Waist circumference (cm)	88.7 ± 15.4 ^{†,‡}	98.8 ± 15.8 ^{*,‡}	103.5 ± 15.8 ^{*,‡}	<.001
SBP (mm Hg)	110.8 ± 16.1	116.3 ± 16.2	120.7 ± 16.3	<.001
DBP (mm Hg)	65.4 ± 8.5 ^{†,‡}	68.9 ± 9.2 ^{*,‡}	71.0 ± 9.6 ^{*,‡}	<.001
Glucose (mg/dL)	92.1 ± 9.2 ^{†,‡}	100.1 ± 20.3 ^{*,‡}	105.9 ± 24.7 ^{*,‡}	<.001
Creatinine (mg/dL)	0.80 ± 0.1 ^{†,‡}	0.83 ± 0.2 [*]	0.85 ± 0.2 [*]	.002
LDL-C (mg/dL)	107.0 ± 29.2 ^{†,‡}	122.2 ± 29.6 [*]	122.7 ± 31.8 [*]	<.001
TC (mg/dL)	171.7 ± 34.9 ^{†,‡}	188.9 ± 34.7 ^{*,‡}	201.8 ± 37.0 ^{*,‡}	<.001
HDL-C (mg/dL)	54.8 ± 13.9 ^{†,‡}	48.0 ± 12.2 ^{*,‡}	41.7 ± 10.8 ^{*,‡}	<.001
TG (mg/dL)	72.7 ± 28.2 ^{†,‡}	130.3 ± 67.0 ^{*,‡}	228.8 ± 94.3 ^{*,‡}	<.001
Non-HDL-C (mg/dL)	116.9 ± 31.3 ^{†,‡}	140.9 ± 34.1 ^{*,‡}	160.1 ± 35.7 ^{*,‡}	<.001
MetS (%)	5.7	32.5	80.4	<.001
hsCRP (mg/L)	2.1 ± 0.7 ^{†,‡}	2.7 ± 0.4 [*]	2.5 ± 0.3 [*]	NS
Insulin (mU/l)	10.6 ± 5.8 ^{†,‡}	13.9 ± 7.6 ^{*,‡}	18.3 ± 9.8 ^{*,‡}	<.001
HOMA-IR	2.5 ± 1.7 ^{†,‡}	3.6 ± 2.5 ^{*,‡}	5.1 ± 3.2 ^{*,‡}	<.001

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; TG, triglycerides; Non-HDL-C, non-high-density lipoprotein cholesterol; MetS, metabolic syndrome; hsCRP, high-sensitivity C-reactive protein; HOMA-IR, homeostatic model assessment insulin resistance. Results are mean ± SD for quantitative variables and percentages for qualitative variables. Quantitative variables were compared using the analysis of variance (ANOVA). Qualitative variables were compared using chi-square test.

* Differences ($P < .05$) with fasting TG <89 mg/dL group.

[†] Differences ($P < .05$) with fasting TG between 89 and 180 mg/dL group.

[‡] Differences ($P < .05$) with fasting TG >180 mg/dL group.

The CORDIOPREV study. Model summary obtained by logistic regression to identify independent predictive variables of undesirable postprandial response (TG > 220 mg/dL at any point during OFTT) in the group of patients with fasting triglycerides between 89 and 180 mg/dL

Table 2

Variables	B	SE	Wald	df	Sig	Exp(B)	95% CI for Exp(B)	
							Lower	Upper
Age, y	-0.028	0.012	5.467	1	0.019	0.972	0.950	0.995
Fasting triglycerides, mg/dL	0.038	0.005	63.977	1	0.000	1.039	1.029	1.049
Constant	-3.126	0.942	11.018	1	0.001	0.044		

Variables entered on the model: age, gender, smoking status, alcohol intake, body mass index, waist circumference, systolic blood pressure, diastolic blood pressure, fasting glucose, HDL-C, non-HDL-C, triglycerides, insulin, hsCRP, HOMA-IR, and metabolic syndrome. R² = 0.237. AUC ROC = 0.748. For definition of abbreviations, see table 1.

Table 3

The GOLDN study. Model summary obtained by logistic regression to identify independent predictive variables of undesirable postprandial response (TG > 220 mg/dL at any point during OFTT) in the group of patients with fasting triglycerides between 89 and 180 mg/dL

Variables	B	S.E.	Wald	df	Sig.	Exp(B)	95% CI for Exp(B)	
							Lower	Upper
Age, y	0.025	0.007	12.308	1	0.000	1.026	1.011	1.040
Gender (Male)	0.838	0.232	13.004	1	0.000	2.311	1.466	3.643
SBP, mm Hg	-0.014	0.007	4.127	1	0.042	0.986	0.973	1.000
Glucose, mg/dL	-0.017	0.007	5.554	1	0.018	0.983	0.970	0.997
HDL-C, mg/dL	0.017	0.009	3.157	1	0.076	1.017	0.998	1.036
Fasting triglycerides, mg/dL	0.009	0.003	13.581	1	0.000	1.009	1.004	1.014
Metabolic syndrome	0.776	0.272	8.142	1	0.004	2.172	1.275	3.701
Constant	-0.366	1.108	0.109	1	0.741	0.693		

Variables entered on the model: age, gender, smoking status, alcohol intake, body mass index, waist circumference, systolic blood pressure (SBP), diastolic blood pressure, fasting glucose, HDL-C, non-HDL-C, triglycerides, insulin, hsCRP, HOMA-IR, and metabolic syndrome. $R^2 = 0.169$. AUC ROC = 0.706. For definition of abbreviations, see table 1.