Contribution of Visceral Adiposity to the Exaggerated Postprandial Lipemia of Men With Impaired Glucose Tolerance

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OBJECTIVE — Impaired glucose tolerance (IGT) has been associated with alterations in numerous coronary heart disease risk factors, including postprandial hyperlipidemia. An excess visceral adipose tissue accumulation is also predictive of IGT and of an exaggerated postprandial lipemia. The objective of the present study was therefore to compare the respective contributions of visceral adipose tissue accumulation versus IGT with the variation in postprandial lipemia.

RESEARCH DESIGN AND METHODS — Potential differences in postprandial triglyceride (TG)-rich lipoprotein (TRL) levels following a standardized breakfast with a high fat content were examined among men characterized by normal glucose tolerance (NGT) or IGT. Sixty-seven men were classified according to their glucose tolerance status (<7.8 mmol/l [NGT] or between 7.8 and 11.1 mmol/l [IGT] 2 h after a 75-g oral glucose test).

RESULTS — Men with IGT showed the highest TRL-TG concentrations ($P < 0.05$) at the 4-, 6-, and 8-h time points compared with men with NGT. These higher postprandial TRL-TG levels among men with IGT were also accompanied by a greater postprandial TG total area under the incremental curve in all TRL fractions (large, medium, and small) ($P < 0.05$). Furthermore, subjects characterized by IGT had also the highest visceral adipose tissue accumulation ($P < 0.009$). When subgroups of IGT and NGT men were individually matched ($n = 11$) for similar visceral adipose tissue accumulation, no significant difference was found in postprandial responses of all TRL-TG fractions between the two groups.

CONCLUSIONS — These results provide evidence that visceral adipose tissue accumulation is an important factor involved in the deterioration of postprandial lipemia noted among men with IGT.

Diabetes Care 26:3303–3309, 2003

The interest for the study of postprandial lipemia was renewed in 1979 when Zilversmit (1) hypothesized that the development of atherosclerosis could largely be a postprandial phenomenon. Since then, several studies (2) have shown that postprandial hyperlipidemia may be a better discriminant of the presence of coronary heart disease (CHD) than fasting triglyceride (TG) levels. Despite the fact that no study has demonstrated that postprandial hyperlipidemia is an independent risk factor for CHD, case-control studies have suggested that postprandial hypertriglyceridemia was more strongly correlated to carotid intima-media thickness than fasting TG concentrations (3,4). Disturbances in postprandial lipemia have also been observed in type 2 diabetic patients (5) and in individuals with visceral obesity or features of the metabolic syndrome (6).

In this regard, impaired glucose tolerance (IGT) has been reported to represent an intermediate stage predictive of an increased susceptibility to type 2 diabetes (7). Hyperglycemia and IGT have also been suggested to be predictive of an increased risk of CHD (8). As IGT has been associated with visceral obesity (9), the presence of postprandial hyperlipidemia among IGT individuals is to be expected. However, we do not know whether the postprandial hyperlipidemia of IGT subjects would be the consequence of the hyperglycemic state per se or rather explained by the concomitant presence of the expanded visceral fat depot.

Thus, the objective of the present study was to compare the respective contributions of visceral adipose tissue accumulation versus IGT status to the variation in postprandial lipemia in middle-aged men. For that purpose, potential differences in postprandial TG-rich lipoprotein (TRL) levels were examined in a sample of 67 men characterized by normal glucose tolerance (NGT) or IGT.

RESEARCH DESIGN AND METHODS

— Sixty-seven men, aged 19–67 years (mean age ± SD: 45.8 ± 10.7 years) were recruited from the Québec City metropolitan area by solicitation through the media. Participants were selected to cover a wide range of BMI values (20.2–41.0 kg/m^2). Subjects gave their written consent to participate in the study, which was approved by the Medi-

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Received for publication 5 May 2003 and accepted in revised form 9 September 2003.

Abbreviations: apo, apolipoprotein; AUIC, area under the incremental curve; CHD, coronary heart disease; IGT, impaired glucose tolerance; NGT, normal glucose tolerance; OGTT, oral glucose tolerance test; TG, triglyceride; TRL, triglyceride-rich lipoprotein.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

cal Ethics Committee of Laval University. All subjects were asymptomatic, nonsmoking volunteers who were not under treatment for CHD, diabetes, dyslipidemias, or endocrine disorders.

Anthropometric and body composition measurements

Body weight, height (10), and waist circumference (11) were measured following standardized procedures. Body density was measured by the hydrostatic weighing technique (12) and percent body fat derived from body density using the equation of Siri (13). Fat mass was obtained by multiplying body weight by percent body fat. Abdominal adipose tissue accumulation was assessed by computed tomography using previously described procedures (14).

Oral glucose tolerance test

A 75-g oral glucose tolerance test (OGTT) was done in the morning after an overnight fast. Blood samples were collected in EDTA-containing tubes (Miles Pharmaceuticals, Rexdale, Ontario, Canada) through a venous catheter placed in an antecubital vein at −15, 0, 15, 30, 45, 60, 90, 120, 150, and 180 min for the measurement of plasma glucose and insulin concentrations. Plasma glucose was measured enzymatically (15), whereas plasma insulin was measured by radioimmunoassay with polyethylene glycol separation (16). Men were classified according to their 2-h glucose concentrations (NGT: 2-h glucose levels <7.8 mmol/l; IGT: 2-h glucose levels between 7.8 and 11.1 mmol/l) as recommended by the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus (17).

Oral lipid tolerance test

After a 12-h overnight fast, an intravenous catheter was inserted into a forearm vein for blood sampling. Each participant was given a test meal containing 60 g fat/m^2 body surface area, as previously described (6). The meal consisted of eggs, cheese, toast, peanut butter, peaches, whipped cream, and milk. Composition of the meal was 64% fat, 18% carbohydrates, and 18% protein. The test meal was well tolerated by all subjects. After the meal, subjects were not allowed to eat for the next 8 h but were given free access to water. Blood samples were drawn before the meal and every 2 h after the meal over an 8-h period.

Table 1—*Physical characteristics and metabolic profile of subjects classified on the basis of 2-h glucose levels*

	NGT	IGT
n	54	13
Age (years)	44.0 ± 10.6	53.5 ± 7.7*
BMI (kg/m^2)	28.2 ± 3.9	31.1 ± 2.9*
Weight (kg)	85.2 ± 11.6	94.1 ± 10.9*
Fat mass (kg)	22.9 ± 7.8	28.8 ± 6.1*
Waist circumference (cm)	96.0 ± 9.0	106.3 ± 8.0*
Abdominal adipose tissue areas (cm^2)		
Total	391.8 ± 139.6	512.2 ± 141.3*
Visceral	134.3 ± 55.3	200.9 ± 67.2*
Subcutaneous	257.5 ± 104.8	311.2 ± 88.5
Cholesterol (mmol/l)	5.21 ± 0.77	5.36 ± 0.44
Triglycerides (mmol/l)	1.84 ± 1.04	2.51 ± 1.29*
LDL cholesterol (mmol/l)	3.50 ± 0.70	3.51 ± 0.77
HDL cholesterol (mmol/l)	1.00 ± 0.23	0.93 ± 0.10
ApoB (g/l)	1.05 ± 0.21	1.14 ± 0.17
LDL apoB (g/l)	0.94 ± 0.19	1.00 ± 0.19
Cholesterol/HDL cholesterol	5.45 ± 1.40	5.83 ± 0.79

Data are means ± SD. *Different from NGT group ($P < 0.05$).

Fasting and postprandial plasma lipoprotein concentrations

Plasma was separated immediately after blood collection by centrifugation at 3,000 rpm for 10 min at 4°C. TG and cholesterol concentrations in total plasma were determined enzymatically on a Technicon RA-500 (Bayer, Tarrytown, NY), as previously described (18). Each plasma sample (4 ml) was then subjected to a 12-h ultracentrifugation (50,000 rpm) in a Beckman 50.3 Ti rotor (Beckman, Palo Alto, CA) at 4°C, in a 6-ml Beckman Quickseal tube, which yielded two fractions: the top fraction containing TRLs (total TRLs; density (d) <1.006 g/ml) and the bottom fraction (d >1.006 g/ml) containing TG-poor lipoproteins. Using the distilled water–layering technique and the modified method of Ruotolo et al. (19), the total TRL fraction was further separated through a 5-min spin (40,000 rpm) at 4°C using the same tubes and rotor into three subclasses of TRLs, namely large, medium, and small, as previously described (6). Large TRLs consist of lipoproteins of S_f >400, whereas the medium and small TRLs are within a spectrum of particles of S_f 20–400 (19). HDL particles were isolated from the bottom fraction (d >1.006 g/ml) after precipitation of apolipoprotein (apo) B–containing lipoproteins with heparin and $MnCl_2$ (20). The TG contents of the large, medium, and small fractions were quantified enzymatically, as described above. All lipoprotein isolation procedures were completed within 2–3 days of the fat load. Total apoB concentration was measured in plasma and in the LDL fraction by the rocket immunoelectrophoretic method of Laurell (21). The lyophilized serum standard for apoB measurement was prepared in our laboratory and calibrated with reference standards obtained from the Centers for Disease Control and Prevention (Atlanta, GA).

Statistical analyses

Differences between men with NGT and men with IGT were examined by Student's unpaired t tests. Areas under the incremental curve (AUICs) of TG, insulin, and glucose concentrations were determined by the trapezoid method. We also wanted to verify whether differences in TRL-TG responses could be attributed to variation in the absolute amount of fat grams consumed by subjects. Adjustment by ANCOVA of TRL-TG responses for amount of fat consumed during the oral lipid tolerance test was therefore performed. Partial correlation coefficients were also computed to examine the contribution of visceral adipose tissue to TRL-TG responses after adjustment for 2-h glucose levels. Finally, partial correlations between 2-h glucose levels and TRL-TG responses were also calculated after adjustment for visceral adipose tis-

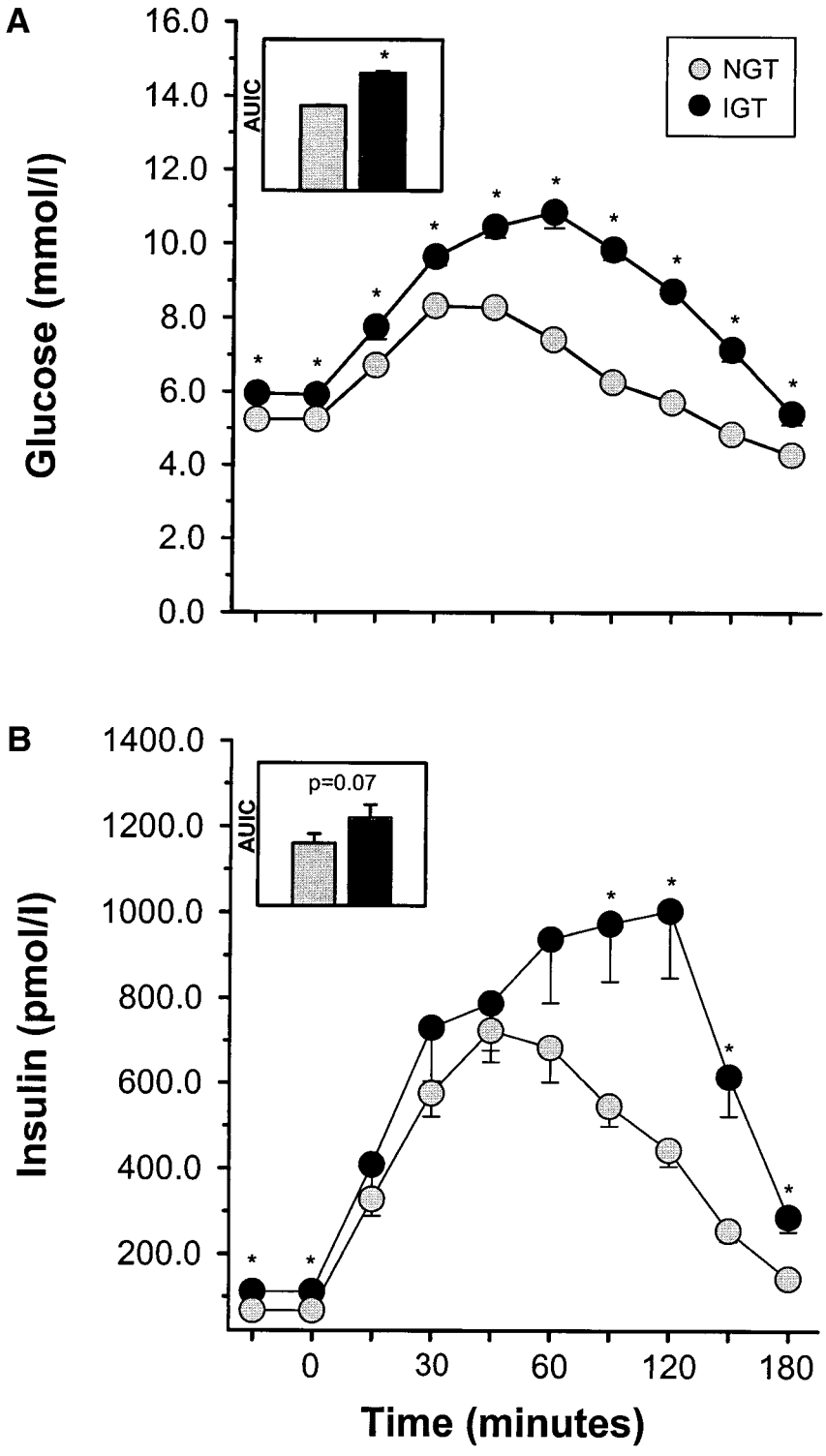

Figure 1— *Plasma glucose (A) and insulin (B) concentrations in the fasting state and after the 75-g oral glucose load among men with NGT (2-h glucose levels <7.8 mmol/l) or IGT (2-h glucose levels between 7.8 and 11.1 mmol/l). Bars represent the total AUICs for the two groups of men. Values are expressed as means ± SE.* *P *< 0.02 different from men with NGT.*

sue. All statistical analyses were performed with the SAS package (SAS Institute, Cary, NC).

RESULTS — Physical and metabolic variables of men classified according to their 2-h glucose values are shown in Table 1. Subjects with IGT were older than men characterized by NGT ($P < 0.003$). In addition, adiposity indexes and abdominal fat distribution variables, such as waist circumference and visceral adipose tissue accumulation, were higher in men with IGT than in men with NGT ($P < 0.02$). Subjects characterized by IGT also displayed greater fasting TG levels than men with NGT ($P < 0.05$). As expected, fasting glucose concentrations, AUIC of glucose, and 2-h post-OGTT glucose concentrations were higher in men with IGT than in men with NGT ($P < 0.008$) (Fig. 1). In addition, subjects with IGT showed higher insulin levels ($P < 0.02$) and a trend for higher AUIC of insulin than men with NGT ($P = 0.07$) (Fig. 1).

Figure 2 illustrates postprandial TG concentrations in total as well as large, medium, and small TRL fractions before and after the fat meal test among men with either NGT or IGT. Overall, men with IGT showed the highest postprandial TG concentrations in total, large, medium, and small TRL fractions ($P < 0.05$), which resulted in higher AUIC-TG in TRL fractions compared with men with NGT ($P < 0.05$). Furthermore, subjects characterized by IGT displayed greater glucose and insulin AUICs throughout the 8-h postprandial period than men with NGT ($P < 0.03$).

To quantify the specific contribution of visceral adipose tissue accumulation to differences in postprandial lipemia between NGT and IGT men, subgroups of subjects were individually matched for similar accumulation of visceral adipose tissue (within a variation of ± 18 cm^2) but with either NGT or IGT. After such a procedure, most fasting differences in metabolic risk variables were eliminated with the exception of glucose tolerance, which was an expected finding by design ($P < 0.03$). However, age, insulin concentrations, and AUIC of insulin during the OGTT were no longer different after having matched IGT and NGT men for visceral adipose tissue. Moreover, the two groups of men were characterized by similar adiposity indexes and fasting TG levels. Although a small residual difference at the 6-h time point ($P < 0.006$) in glucose concentrations between NGT and IGT men remained during the postprandial phase, the two groups were characterized by similar AUIC of insulin (data not shown). Adjustment for fasting glucose levels and for glucose levels measured 2 h

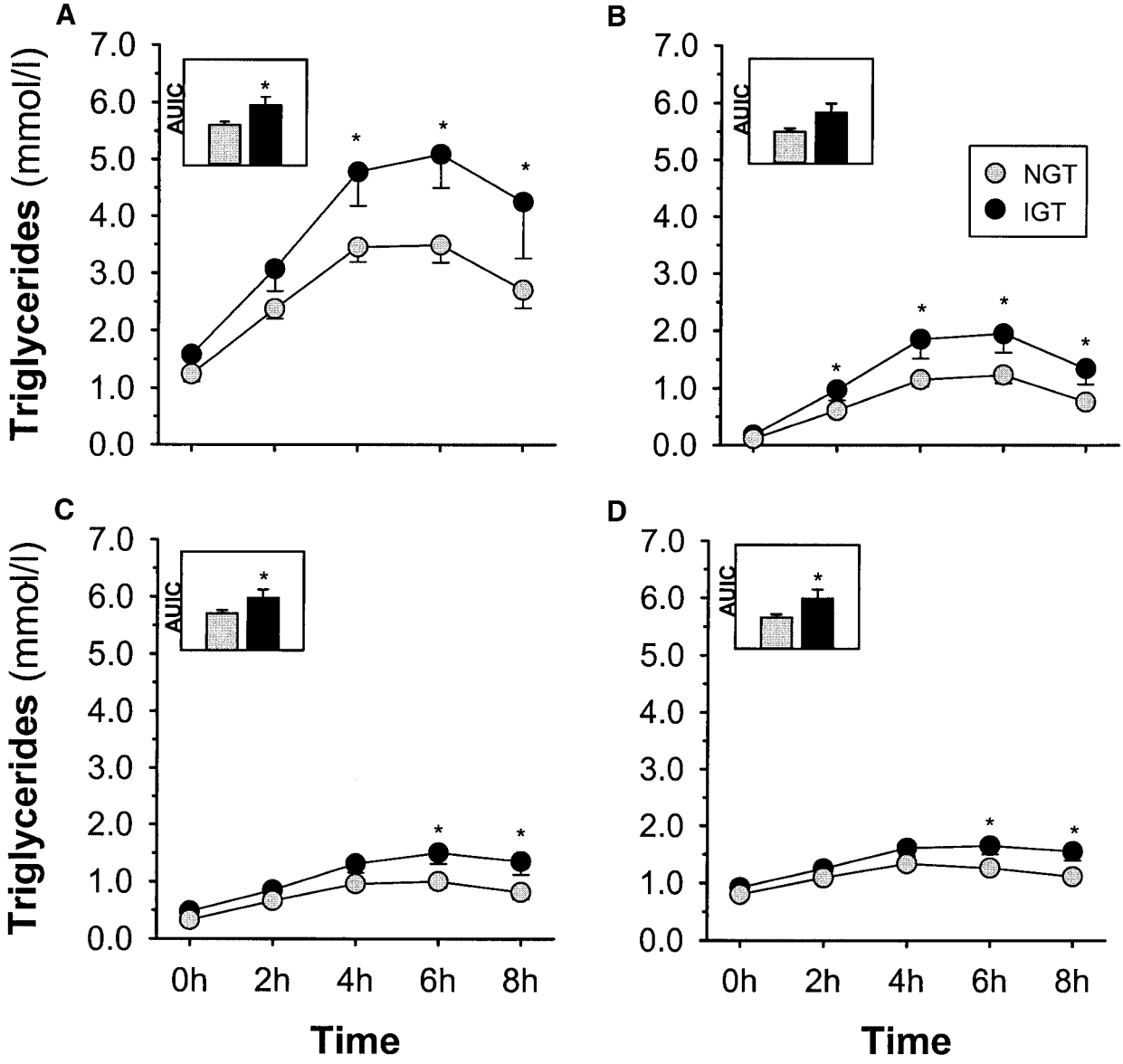

Figure 2— *Postprandial TG levels of total* (A)*, large* (B)*, medium* (C)*, and small* (D) *TRLs among men with NGT (2-h glucose levels <7.8 mmol/l) or IGT (2-h glucose levels between 7.8 and 11.1 mmol/l). Bars represent the total AUICs for the two groups of men. Values are expressed as means ± SE. *P < 0.05 vs. men with NGT.*

after the 75-g oral glucose load by ANCOVA revealed that subjects characterized by an elevated visceral adipose tissue (≥130 cm^2) accumulation still showed higher postprandial TG concentrations in total, large, medium, and small TRL fractions, which resulted in higher AUIC in total, medium, and small TRL fractions compared with men with a low visceral adipose tissue accumulation (<130 cm^2).

Figure 3 presents the postprandial TG concentrations in total as well as large, medium, and small TRL fractions among the two subgroups of men matched for visceral adipose tissue accumulation but with either NGT or IGT. After this matching procedure, differences initially found in postprandial response between IGT and NGT men were no longer significant. In addition, we investigated the relationship between AUIC of TRL-TG and visceral adipose tissue accumulation in the total cohort, and we found a highly significant positive correlation between these two variables ($r = 0.48$, $P < 0.0001$), suggesting an important contribution of visceral adipose tissue to the deterioration of postprandial metabolism.

To take into account the possible effect of the absolute amount of fat grams consumed during the oral lipid tolerance test, additional analyses controlling for this variable were performed. Such statistical adjustment for the absolute amount of fat consumed did not have any impact on our results and conclusion (data not shown). Finally, we also computed partial correlations to evaluate the impact of visceral adipose tissue on the relationship between 2-h glucose levels and TRL-TG responses. The association initially found between 2-h glucose levels and TRL-TG responses was no longer significant after adjustment for visceral adipose tissue ac-

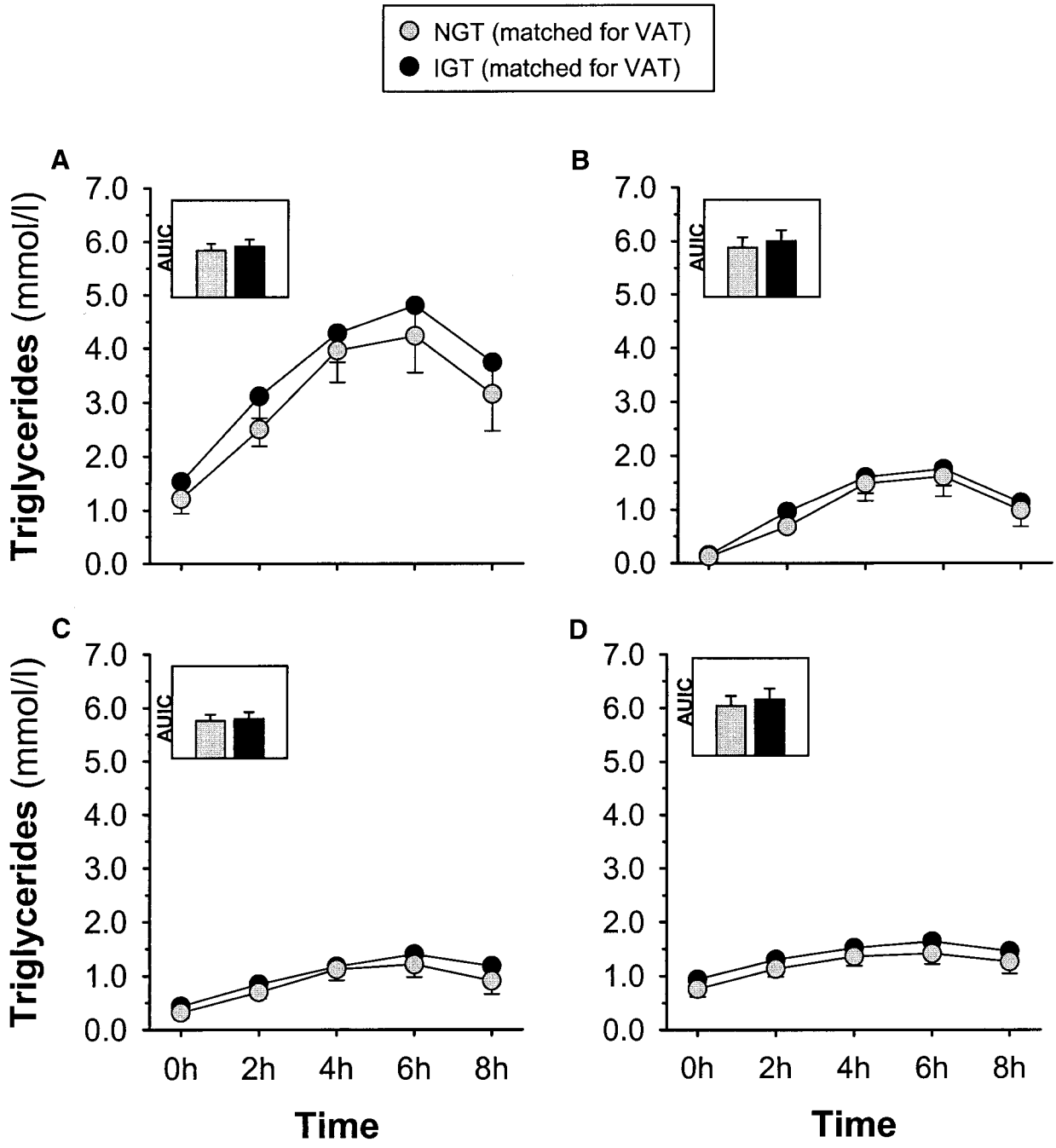

Figure 3— *Postprandial TG levels of total* (A)*, large* (B)*, medium* (C)*, and small* (D) *TRLs in the two groups of men* (n = *11) matched for similar visceral adipose tissue area but with NGT (2-h glucose levels <7.8 mmol/l) or with IGT (2-h glucose levels between 7.8 and 11.1 mmol/l). Bars represent the total AUICS for the two groups of men. Values are expressed as means ± SE.*

cumulation. However, adjustment for the 2-h glucose concentrations did not alter the relationship between visceral adipose tissue and TRL-TG responses.

CONCLUSIONS — Although type 2 diabetes is a major and well-established risk factor for CHD, there is accumulating evidence that the hyperglycemic state may only play a minor role in explaining the elevated CHD risk in these patients (9). For instance, numerous studies have suggested that a cluster of abnormalities referred to as the metabolic syndrome could largely contribute to the elevated CHD risk of type 2 diabetic patients (22). Because type 2 diabetes is invariably preceded by IGT, and because this stage has also been shown to be associated with an increased CHD risk (8), the same rationale could be used to question whether hyperglycemia per se plays a role in the etiology of CHD among individuals with IGT. For instance, subjects with IGT are often characterized by high levels of body fatness, especially by an increased accumulation of visceral adipose tissue. In this

regard, Pascot et al. (9) have suggested that visceral adipose tissue accumulation was an important contributor to the deterioration of the fasting plasma lipid-lipoprotein profile noted in men with IGT. After statistical adjustment for the amount of visceral adipose tissue accumulation, they reported that differences between men with IGT and NGT were eliminated for all variables of the fasting plasma lipid-lipoprotein profile (9). In accordance with these results, Nagaretani et al. (23) have suggested that visceral fat accumulation was a major contributor for the clustering of multiple risk factors such as dyslipidemia and hypertension in Japanese men with IGT and NGT. On that basis, we had already proposed that IGT only represents the tip of a huge atherothrombotic iceberg largely resulting from the presence of abdominal obesity (22).

Because IGT is also associated with postprandial hyperlipidemia (24), the main objective of our study was to verify the respective contributions of visceral adipose tissue accumulation and glycemia to the variation in postprandial lipemia. Following adjustment for visceral adipose tissue, all differences in the postprandial response observed between men with NGT and men with IGT were completely abolished, suggesting that the deterioration of the postprandial lipemia is more affected by visceral adipose tissue accumulation than by circulating glucose concentrations. Subjects with IGT have been reported to have increased postprandial TG levels that were, however, accompanied by the high TG and low HDL cholesterol fasting dyslipidemic state, which has been shown to affect the postprandial lipid response (24). Moreover, Henkel et al. (25) found that IGT and NGT subjects with similar levels of fasting TG did not exhibit any difference in postprandial lipid levels. Accordingly, another study reported that postprandial TG response was not increased among nonobese IGT subjects without fasting hypertriglyceridemia (26). Taken together, these results suggest that the IGT state per se does not directly affect postprandial lipemia.

Although the mechanisms leading to insulin resistance are not fully understood, it is known that impaired insulin action can be present in both obese and nonobese subjects (27). These observations suggest that insulin resistance is a heterogeneous condition and provide further evidence that some genetic factors are also likely involved. In the present study, IGT subjects were characterized by a higher accumulation of visceral adipose tissue compared with NGT subjects. To the best of our knowledge, our study is the first to assess postprandial lipemia in abdominally obese subjects with IGT. It has been suggested that visceral obesity was related to insulin resistance (27). In the present study, IGT men with visceral obesity showed higher insulin levels during the OGTT than NGT men. However, after matching for similar levels of visceral adipose tissue, plasma insulin levels during the OGTT were similar between NGT and IGT men. Moreover, the matching procedure also completely eliminated differences in postprandial insulin response to the fat load between the groups, whereas differences remained in glucose levels. These observations suggest that viscerally obese men characterized by IGT were insulin resistant and displayed a relative deficiency in insulin secretion.

In the postprandial state, no difference was observed in the TRL-TG response between subjects with IGT compared with those with NGT after matching for similar visceral adipose tissue accumulation. Thus, the major finding of our study is that subjects characterized by IGT in isolation do not exhibit postprandial hyperlipidemia compared with NGT men, suggesting that visceral obesity is an important variable involved in the deterioration of the postprandial lipemia noted among men with IGT. From a clinical perspective, if one accepts that postprandial hyperlipidemia represents a metabolic abnormality that may exacerbate CHD risk after a meal, our results suggest that it may be relevant to identify visceral adipose tissue as a more important therapeutic target than the normalization of glycemia per se among high-risk individuals with IGT.

Acknowledgments— This study was supported by the Canadian Institutes of Health Research (MOP-14014) and by the Québec Heart and Stroke Foundation.

P.B. is the recipient of a studentship from the Fonds de la Recherche en Santé du Québec (FRSQ); B.L. is chair professor of Nutrition, Functional Foods and Cardiovascular Health from the Canada Research program at Laval University; C.C. is a research scholar from the FRSQ-CHUQ/CHUL; A.T. holds the Canada Research Chair on Physical Activity, Nutrition and Energy Metabolism; and J.B. is a clinical research scholar from the FRSQ. J.-P.D. is chair professor of human nutrition, lipidology, and prevention of cardiovascular diseases, which is supported by Pfizer, Provigo, and the Foundation of the Québec Heart Institute.

We thank the staff of the Physical Activity Sciences Laboratory for the data collection and the staff of the Lipid Research Center for their excellent and dedicated work.

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