The role of postprandial insulin in the regulation of postprandial lipid metabolism is still poorly understood. The roles of hyperinsulinemia and insulin resistance in the alteration of postprandial lipid metabolism are not clear either. To improve knowledge in this area, we submitted healthy men to acute hyperinsulinemia in two different ways. In the first study, we compared in 10 men the effects of four isolipidic test meals that induce different degrees of hyperinsulinemia on postprandial lipid metabolism. Three different carbohydrate sources were compared according to their glycemic indexes (GIs; 35, 75, and 100 for white kidney bean, spaghetti, and white bread test meals, respectively); the fourth test meal did not contain any carbohydrates. Postprandial plasma insulin levels were proportional to the GIs (maximal plasma insulin concentrations: 113 ± 16 to 266 ± 36 pmol/l). We found a strong positive correlation during the 6-h postprandial period between apolipoprotein (apo) B-48 plasma concentration and insulin plasma concentration ($r^2 = 0.70; P = 0.0001$). In a second study, 5 of the 10 subjects again ingested the carbohydrate-free meal, but during a 3-h hyperinsulinemic- (550 ± 145 pmol/l plasma insulin) euglycemic (5.5 ± 0.8 mmol/l plasma glucose) clamp. A biphasic response was observed with markedly reduced levels of plasma apoB-48 during insulin infusion, followed by a late accumulation of plasma apoB-48 and triglycerides. Overall, the data obtained showed that portal and peripheral hyperinsulinism delays and exacerbates postprandial accumulation of intestinally derived chylomicrons in plasma and thus is involved in the regulation of apoB-48-triglyceride–rich lipoprotein metabolism, in the absence of insulin-resistance syndrome. *Diabetes* 50:462–469, 2001

The role of insulin in the regulation of postprandial lipemia still remains misunderstood. However, abnormalities in postprandial lipid and lipoprotein metabolism are increasingly involved in the etiology of cardiovascular diseases (1,2) and in hypertriglyceridemic patients (3–5), type 2 diabetic patients (6–10), and patients affected by polymetabolic syndrome (5, 11,12). In the latter two situations particularly, an abnormal postprandial lipid metabolism is observed and acknowledged to favor atherogenesis (13). Because these patients display both hyperinsulinism and insulin resistance, it is difficult to evaluate the respective role of hyperinsulinism and insulin resistance in the abnormalities of the postprandial lipemia observed. Several studies have shown that the observed accumulation of exogenous triglyceride-rich lipoproteins (TRLs) after a fat-containing test meal positively correlates with both fasting and postprandial plasma insulin levels in normal or obese subjects (5,12,14,15). Some reports, on the basis of indirect arguments, have suggested that insulin resistance is the main factor in this phenomenon (5,12,14,15), and that the amplitude of postprandial lipemia correlates with the severity of insulin resistance (14). Thus, even if a clear consensus can be derived from these studies that hyperinsulinemia and insulin resistance are associated with exacerbated postprandial lipid accumulation, the causal relationship is not yet established.

It is widely accepted that the hypertriglyceridemic state found in the postprandial period results from accumulation of both hepatically derived triglyceride-rich lipoproteins (VLDL and LDL) and intestinally derived ones (chylomicrons and remnants) (5,16), although the relative importance and fate of the two kinds of lipoprotein particles is still being debated.

The aim of this study was to determine the role of postprandial hyperinsulinemia per se on postprandial lipemia and, especially, on TRLs from both origins. To achieve this goal, healthy normal-weight subjects with normal fasting insulin and lipid levels were recruited. In the nutritional study, they were given four regular fat-containing mixed test meals with different carbohydrate sources selected according to their glycemic/insulinemic indexes (17,18) to induce graded physiological insulin responses. In a complementary mechanistic approach, five subjects were submitted or not to a hyperinsulinemic-euglycemic clamp after the ingestion of a carbohydrate-free fat-containing meal during the first part (0–3 h) of the postprandial phase, to model the transient
hyperinsulinism observed under high-glycemic-index meals. Under the two hyperinsulinemic states, the accumulation of apolipoprotein (apo) B-48–intestinally derived and apoB-100–hepatically derived TRLs in plasma was determined. The data obtained indicate that acute hyperinsulinism (in the absence of insulin-resistance syndrome) modulates the concentration of intestinally derived TRLs in plasma.

RESEARCH DESIGN AND METHODS

Study 1: Postprandial lipemia responses to test meals with different glycemic indexes

Subjects. The 10 adult male volunteers in this study, average age 24 years (range 21–30), participated after giving written informed consent according to a protocol approved by the local medical ethics committee (Comite Consultatif pour la Protection des Personnes se Prétendant à des Recherches Bio-Médicales, Marseille 1). No volunteer suffered from any digestive or metabolic disease, as confirmed by medical history and fasting blood parameters, nor was any subject on any drug treatment that could influence carbohydrate or lipid metabolism.

Fasting blood concentrations were in the normal range, with the following overall mean ± SE values: serum triacylglycerol, 0.84 ± 0.07 mmol/l; total cholesterol, 4.6 ± 0.20 mmol/l; glucose, 5.20 ± 0.90 mmol/l; and insulin, 54.90 ± 5.64 pmol/l. No volunteer was obese (BMI, 22.8 ± 1.4 kg/m²). All subjects had E/E, apoE genotypes. The subjects relied on a typical Mediterranean diet with a moderate energy intake (mean ± SD: 10,120 ± 950 kcal/d; 2,421 kcal/d), with protein, carbohydrate, and fat accounting for 17, 44, and 39% energy, respectively. They were instructed to avoid alcohol consumption and intensive exercise for 36 h before being tested, and to reduce carbohydrate consumption the day before the study. The subjects were asked to have a light standard dinner before 9:00 p.m. the evening before the experiment. Each subject ingested the four test meals at a 1-week interval in a random order.

Test meals. Four different test meals were designed. Three carbohydrate components (90 g) were chosen according to their glycemic indexes (GI) (18): white bread (GI 100; B test meal), spaghetti (GI 70; S test meal), or white kidney beans (GI 35; KB test meal). The fourth test meal was free of carbohydrates, containing only lipids and proteins (CF test meal). The four meals were isocaloric (48 g) and isoinert (33 g) and were composed of commercially available food. The fat was provided as olive oil incorporated into tomato sauce. Protein was provided as cooked egg whites to adjust the protein level. After an overnight fast, an antecubital vein was catheterized with intravenous canulas equipped with disposable obturators (Jelco-Critikon, Chatenay-Malabry, France). A baseline fasting blood sample was collected at 0 h. The subjects then ingested the test meal within 20 min. The countdown was fixed at the middle of food intake. Blood samples were collected every half hour during the first 2 h and then every hour until 6 h after the meal.

Analytical determinations. Plasma and serum were immediately separated from whole blood by centrifugation (10°C, 910 g) and then peroxidase-labeled immunoglobulins. The color was developed and measured as described above. ApoB-100–containing VLDLs that reacted with the monoclonal antibody were quantified after addition of an anti-apoB-100 polyclonal antibody (Calbiocol, Meudon, France) and then peroxidase-labeled immunoglobulins. The color was developed and measured as described above. ApoB-100 containing LDLs was used as the standard.

After 6-h postprandial blood sampling, each subject was injected with 80 U/kg heparin (Choay, Gentilly, France); a blood sample was drawn 10 min after heparin administration for subsequent determination of lipoprotein lipase (LPL) and hepatic lipase (HL) activities, according to the method of Krauss et al. (24).

Study 2: Postprandial lipemia responses to a CF test meal during a euglycemic insulin clamp

Euglycemic insulin clamp. Five subjects from the first study were included in this study and instructed to maintain their regular food habits. The goal of the euglycemic insulin clamp, as previously described by de Fronzo et al. (25), was to acutely raise plasma insulin concentrations to a new plateau and to maintain them at that level to determine the role of hyperinsulinemia in the changes observed during the processing of a test meal. Each subject received a continuous intravenous infusion of insulin (40 mU/ml, NovoNordisk) via an electric syringe to obtain insulin concentrations in the range 460–620 pmol/l over 3 h. To avoid hypoglycemia, the plasma glucose concentration was maintained at its euglycemic level with a 20% glucose solution infused in the same way with variable rate. Venous blood samples were obtained from similar catheters inserted in the contralateral antecubital vein kept open with 0.9% NaCl infusion.

Test meal. Subjects ingested the CF test meal 20 min after beginning insulin infusion, as described above, (at time 0 h) within 20 min (euglycemic insulin clamp [EIC] test meal). The countdown was fixed at the middle of food intake. Blood samples were collected every half hour during the first 3 h (~30 min to 3 h) and then every hour until 6 h after the meal.

Analytical determinations. Plasma glucose, insulin, lipids, apoB-48, and apoB-100 concentrations were determined as described above for the first study. In the same way, lipoprotein fractions were isolated as described above.

Statistical analysis. In this randomized study, each subject experimented with the four (or five) test meals and served as his own control. Results are given as means ± SE. Absolute postprandial changes are given as concentration values. The 0- to 6-, 0- to 3-, and 3- to 6-h areas under the curve (AUCs) were calculated with the incremental postprandial variations (postprandial values minus baseline) by the trapezoidal method. Statistical significance (P < 0.05) of differences observed among test meals was assessed by analysis of variance for repeated values and Fisher's test.

RESULTS

Study 1: Postprandial lipemia responses to test meals with different GIs

Ingestion of the three carbohydrate-containing meals resulted in a significant increase in glyceremia (incremental 0- to 6-h AUC: 3.8 ± 0.94 [B] vs. 2.52 ± 0.4 [S] vs. 0.68 ± 0.57 [KB] vs. −1.03 ± 0.38 mmol · h⁻¹ · 1⁻¹ [CF]) and insulinemia, which lasted the first hour after the S and KB test meals and 2 h after the B test meal. The CF test meal resulted in a slight increase in insulinemia. Figure 1 shows the incremental insulin postprandial responses during the first 3 and 6 h after the four test meals. The 0- to 3-h postprandial insulinemia was higher (P < 0.05) after the B meal than after the S and KB meals, which had comparable values; values were lower after the CF meal than after the other three meals. The 0- to 6-h postprandial insulinemia ranked in a comparable order.

Postprandial serum TGs rose significantly over baseline after the four test meals, with no significant differences observed among the four test meals (Fig. 2). Incremental 0- to 3-, 3- to 6-, and 0- to 6-h AUCs were not different overall.

Absolute postprandial changes in plasma apoB-48 concentrations (µg/ml) are shown in Fig. 3. The four test meals resulted in a significant increase in plasma apoB-48 concentration over baseline. The apoB-48 peaked 4 h after the two high-GI test meals (B and S test meals) and peaked 3 h after the KB and CF test meals, as shown in Fig. 3. The 4-h
apoB-48 concentrations were significantly higher \( (P < 0.05) \) after the B and S test meals than after the KB test meal, and the 6-h apoB-48 concentrations were significantly higher \( (P < 0.05) \) after the B and S test meals than after the CF test meal. No differences were found among the incremental apoB-48 0- to 3-h AUCs after the four test meals (Fig. 4A). Conversely, incremental apoB-48 3- to 6-h AUCs after the KB test meal, and more markedly after the CF test meal, were lower than after the B and S test meals (Fig. 4B). Although no significant differences were observed for the incremental apoB-48 0- to 6-h AUCs after the four test meals, a strong positive correlation was found \( (r^2 = 0.70; \ P = 0.0001) \) between incremental 0- to 6-h plasma apoB-48 and insulin postprandial responses (Fig. 5). Conversely, as shown in Fig. 6, VLDL apoB-100 did not change noticeably during the four postprandial periods, and thus no relationship was found with insulin postprandial responses.

TG concentrations in chylomicron fractions transiently and significantly \( (P < 0.05) \) rose over baseline after the four test meals (3-h peak) and returned to baseline after 6 h. The amplitudes of the chylomicron TG responses were not markedly different after the four test meals (incremental 0- to 6-h TG AUC range: 1.26–1.90 mmol · h\(^{-1} \) · l\(^{-1} \)), nor were any differences found for 0- to 3-h or 3- to 6-h AUCs for chylomicron TGs. The VLDL TG concentrations were comparable after the four test meals (incremental 0- to 6-h TG AUC range: 0.30–0.59 mmol · h\(^{-1} \) · l\(^{-1} \)).

LPL and HL activities determined on postheparin plasma 6 h after food intake did not show marked differences after the four test meals (3.8–5.2 vs. 4.6–5.1 µmol · h\(^{-1} \) · ml\(^{-1} \) for LPL and HL activity, respectively).
FIG. 5. Regression analysis for plasma apoB-48 for 0- to 6-h incremental AUCs and plasma insulin 0- to 6-h incremental AUCs. $P = 0.0001; r^2 = 0.70$.

**Study 2: Postprandial lipemia responses to CF test meal during a euglycemic insulin clamp.** The test procedures were well tolerated by the subjects. Glycemia was maintained at fasting level. As expected, plasma insulin increased to 460–620 pmol/l (550 ± 145) 20 min after intravenous injection of insulin until the end of the infusion (Fig. 7A). At the time of food intake (30 min after beginning insulin infusion), insulinemia had a mean value of 491 ± 61 pmol/l. The stop in insulin infusion at time 2 h 30 min resulted in a drop in insulinemia to values close to fasting values at 3–6 h.

The postprandial changes in serum TG concentrations elicited by intake of the CF test meal without or with insulin (EIC test meal) are shown in Fig. 7B. Serum TGs peaked 4 h after the EIC test meal, whereas they peaked 3 h after the CF test meal. The serum TG responses (incremental 3- to 6-h or 0- to 6-h AUCs) after the EIC test meal were significantly higher than after the CF test meal. Conversely, no differences were found among serum TG incremental 0- to 3-h AUCs (Table 1).

Postprandial changes in plasma apoB-48 concentrations are shown in Fig. 7C. ApoB-48 peaked 4 h after the EIC test meal but 3 h after the CF test meal. Furthermore, the apoB-48 level 2 h after the EIC test meal was significantly lower than the corresponding value after the CF test meal. The incremental 0- to 3-h AUCs after the EIC test meal were significantly lower than after the CF test meal (Table 1), whereas no differences were found for the incremental 3- to 6-h AUC values.

Postprandial VLDL apoB-100 concentration variations (µg/ml) are shown in Fig. 7D. Postprandial values were not significantly different from baseline during the postprandial period after the CF and EIC test meals. There were no differences in apoB-100 levels under both conditions.

Figure 8 shows the postprandial changes in TG concentrations in chylomicrons (Fig. 8A) and VLDL (Fig. 8B). As for plasma TGs and apoB-48, TGs in chylomicrons peaked 4 h after the EIC test meal and 3 h after the CF test meal. TGs in VLDL rose moderately 5 h after the EIC test meal and 4 h after the CF test meal. For both plasma and chylomicrons after the EIC test meal, TG rises tended to be lower (Table 1) during insulin infusion (~30 min to 2 h 30 min) and peaked after the stop in insulin infusion. As shown in Table 1, the TG incremental 3- to 6-h AUCs were significantly higher for chylomicron TGs after the EIC test meal than after the CF test meal, whereas VLDL fraction TGs showed a transient and moderate elevation during the same period.

**DISCUSSION**

In the vast majority of studies already conducted in the field of postprandial lipid metabolism, both insulin resistance and related hyperinsulinism took part in the abnormal metabolic syndrome exhibited by the subjects (5,12,14,15). Thus, although key data showing a positive association between insulin resistance/hyperinsulinism and exaggerated postprandial lipemia have been obtained, it has not yet been possible to discriminate between the respective roles of insulin resistance or hyperinsulinism on postprandial lipid metabolism. The present work was designed specifically to test the role of hyperinsulinism. Particular emphasis was given to the changes in apoB-48 TG-rich lipoproteins from intestinal origin (chylomicrons) and apoB-100–containing TRLs (VLDL) secreted by the liver, as both kinds of particles are involved in postprandial hyperlipemia (5,12,16) as well as atherogenesis (1,26–28).

In the nutritional approach, we observed graded physiological insulin responses in the increasing order predicted by the GIs of carbohydrates used and not used (29,30), whereas postprandial plasma TG responses were not significantly different after the four meals. This first finding indicates that, in healthy subjects, physiological ranges of postprandial hyperinsulinemia as generated by starchy foods do not induce any noticeable alterations in the overall postprandial TG response. No marked changes in hepatically derived apoB-100–containing VLDL were observed either. Variable effects of dietary carbohydrates have already been reported, with the addition of 50 and 100 g glucose (31) or dietary fiber sources (32) reducing postprandial triglyceridemia generated by a fat meal, and the addition of sucrose (33) or fructose (34) increasing postprandial triglyceridemia. In contrast, we observed two different patterns of postprandial responses in plasma apoB-48 (a specific component of intestinally derived TRLs) after meals. With the lowest GI meals (KB and CF), apoB-48 peaked 1 h earlier than with the two highest GI meals (B and S), meaning that postprandial intestinal TRL accumulated later after high-GI meals than after low-GI meals.
meals. Indeed, the apoB-48 3- to 6-h AUCs were two times higher after high-GI meals than after the CF meal. Some timing differences between carbohydrate-rich and -poor meals (31) or diets (35) have already been observed for plasma TGs. These different responses lead to the observation of a strong positive correlation between the 0- to 6-h plasma apoB-48 levels and the 0- to 6-h insulin response. This relationship has been suggested by other studies during which insulin resistance and hyperinsulinemia were found to be associated with elevated intestinal TRL or large TRL levels (5,9,12,15,36). Because an accumulation of intestinal-TRL remnants in the circulation is associated with the progression of coronary heart disease (37), low-GI foods and meals could be recommended to patients at cardiovascular risk.

In the second approach we aimed to directly test the hypothesis that hyperinsulinemia, independent from insulin resistance and hyperglycemia, is involved in the observed alteration in postprandial lipid metabolism. Thus the response to the carbohydrate-free, fat-containing meal (CF test meal) was compared to the one generated by the same meal superimposed with a 0- to 3-h hyperinsulinemic euglycemic clamp (EIC test meal). Hyperinsulinemia was maintained through the clamp for 3 h to mimic the insulin kinetics observed under high-GI meals. This experimental protocol, however, differed from high-GI meals by a higher insulin level and route of insulin delivery (systemic versus portal). Nevertheless, the need for portal insulin delivery to achieve normal lipoprotein clearance has been widely discussed in the literature as well as in reports of transplantation data on insulin infusions in animals and patients (38).

The clamped hyperinsulinemic state and CF meal induced a 1-h delay in the occurrence of the postprandial plasma TG peak as compared with the CF meal alone, which maintained baseline insulinemia. During the 0- to 3-h period under insulin infusion, plasma apoB-48 concentrations remained paradoxically much lower than after the CF meal alone. Conversely, plasma apoB-100 levels did not change noticeably. This observation suggests that postprandial hyperinsulinemia acutely and specifically slows down the accumulation of apoB-48–containing intestinally derived TRLs. This effect most likely is not attributable to an alteration in LPL activity, as VLDL TGs and apoB-100 levels were not affected during this period. A somewhat reduced apoB-48 rise has already been observed during the postprandial period in type 2 diabetic patients, who exhibit a known postprandial hyperinsulinemia (39).

In contrast, stopping the superimposed hyperinsulinemic clamp after 3 h led to a fast return to baseline insulin levels and generated exacerbated 3- to 6-h TRL responses as compared with the control CF meal without any previous clamp. Indeed, significantly higher responses for plasma TGs, chy-

![FIG. 7. Plasma insulin (A), TGs (B), apoB-48 (C), and apoB-100 (D) concentrations for 6 h after the CF (△) or EIC test meal (■). *P < 0.05 between meals. Data represent means ± SE; n = 10.](image)

**TABLE 1**
Metabolic variables (AUC) after EIC and CF test meals

<table>
<thead>
<tr>
<th></th>
<th>Change at 0- to 3-h AUCs</th>
<th>Change at 3- to 6-h AUCs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>EIC</td>
<td>CF</td>
</tr>
<tr>
<td>Plasma TG</td>
<td>0.61 ± 0.14</td>
<td>0.89 ± 0.19</td>
</tr>
<tr>
<td>Plasma apoB-48</td>
<td>0.05 ± 0.01*</td>
<td>0.10 ± 0.02</td>
</tr>
<tr>
<td>Chylomicron TG</td>
<td>0.51 ± 0.17</td>
<td>0.694 ± 0.09</td>
</tr>
<tr>
<td>VLDL TG</td>
<td>0.18 ± 0.09</td>
<td>0.10 ± 0.17</td>
</tr>
</tbody>
</table>

Data are means ± SE; n = 5. *P < 0.05 between meals.
lomicron TGs, and, to a lesser extent, VLDL TGs were observed during the 3- to 6-h postprandial period. The fact that the increase in apoB-48 was only moderate could have resulted from the dramatic drop observed just before the end of the clamp. Such a late accumulation in plasma TGs postprandially has been observed during several studies in subjects with insulin resistance and hyperinsulinism (5,16,39–41). Another new finding is that transient hyperinsulinemia is responsible for this late accumulation in plasma of TG-rich lipoproteins of intestinal origin and, to a lesser extent, hepatic origin. This concept fits well with the relationship already observed between fasting insulinemia (and thus related postprandial hyperinsulinemia) and postprandial TG response (5,14,15). It is also noteworthy to emphasize that low-fat, highly digestible carbohydrate diets are known to induce hyperinsulinemia as well as fasting and postprandial hyperlipidemia (41,42). It has been confirmed that this kind of diet induces a significant decrease in TRL clearance in normal subjects, with the presence of apoB-48–containing TRLs in the fasting state illustrating a delay in the clearance of these particles (43), in line with other data (5,9). Taken together, these data and the present ones suggest that exogenous TRLs are particularly susceptible to a hyperinsulinism-induced clearance delay.

Insulin resistance and hyperinsulinism are associated with fasting and postprandial hypertriglyceridemia, resulting on the one hand from an increase in hepatic VLDL secretion (44) and on the other hand from a decrease in LPL-mediated clearance of TG-rich lipoproteins. Nevertheless, as previously suggested in healthy (45) or type 2 diabetic patients (46), hyperinsulinism induces a decrease in hepatic VLDL production whereas insulin resistance induces an increase in VLDL production (44). In the nutritional part of our study, VLDL apoB-100 levels were not markedly affected by carbohydrates, a finding in line with the observation in healthy subjects that apoB-100 and VLDL TG synthesis are not affected by a low-fat, high-carbohydrate diet (43). The absence of a decrease in apoB-100 levels during the EIC test meal in the present study differs from the lowering of VLDL TGs observed in clamp studies performed on fasting patients (45,46). This difference is likely attributable to the existence of the fat assimilation process. In conclusion, elevated postprandial insulin levels as elicited by these meals did not noticeably modify the overall level of LPL and HL activities. Also, blockade of intestinal secretion by octreotide, a somatostatin analogue, was shown to be unable to suppress postheparin LPL activity in plasma (47). During clamps, despite the fact that postheparin LPL activity was not measured, the absence of a decrease in VLDL apoB-100 made drastic improvement in LPL activity unlikely. However, insulin is known to acutely upregulate adipose tissue LPL (48,49) and have the opposite effect on muscle (49,50). Accordingly, we cannot exclude any subtle tissue-specific alterations of LPL activity under high-insulin levels.

Observations obtained after test meals and hyperinsulinemic clamp also showed the existence of a late accumulation of apoB-48 TRLs as a consequence of transient hyperinsulinemia, suggesting that insulin delays the secretion of apoB-48–containing TRLs by the small intestine during the postprandial period. Such a mechanism has already been proposed as a regulatory pathway in the liver (51), and an abnormality in intestinal secretion has been previously suggested in diabetic patients (40). Although very scarce, some experimental evidence for this possible mechanism has been obtained from cultured fetal small intestine (52) and using cultured differentiated human CaCo2 intestinal cells (A.H., D.L., unpublished data). This leads us to suggest that marked insulin elevation would depress chylomicron secretion from enterocytes into the lymph, despite efficient apical uptake of fatty acids and insulin-activated lipogenesis (53). The return to basal insulin levels observed in this study under both experimental conditions would then permit maximum chylomicron secretion, late occurrence, and accumulation in the circulation. It is not known whether insulin and/or glucose alter the process of lipid absorption by the intestine mucosa.

In conclusion, elevated postprandial insulin levels as observed in healthy subjects without insulin-resistance syndrome could alter some postprandial lipid parameters, especially intestinally derived TG-rich lipoproteins. This may indicate that hyperinsulinemia, at the time of fat assimilation, is involved more than insulin resistance in the alteration of postprandial intestinal TRL metabolism, whereas insulin resistance could be involved more than hyperinsulinism in the alteration of hepatic TRL metabolism. These two joined phenomena can induce fasting- and postprandial-exacerbated hypertriglyceridemia as observed in patients affected with diabetes.
by the polymetabolic syndrome. High-GI food ingested as part of a mixed meal with fats can generate, via induced insulin secretion, an accumulation of intestinal apoB-48 TRLs in plasma at the last stage of the postprandial phase. Although further experimental evidence should be given, our results support the close link between meals and diet composition, insulin response, postprandial lipoprotein metabolism, and potential arteriosclerosis risk.

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