Effects of glucose ingestion on postprandial lipemia and triglyceride clearance in humans

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Abstract To determine whether the metabolism of diet-derived triglycerides (TG) is acutely regulated by the consumption of insulinogenic carbohydrates, we measured the effects of glucose ingestion on oral and intravenous fat tolerance, and on serum triglyceride concentrations obtained during duodenal fat perfusion. Postprandial lipemia was diminished by the ingestion of 50 g (148 ± 121 mg · dl^{-1} · 7 h^{-1} vs 192 ± 124 mg · dl^{-1} · 7 h^{-1}, $P<0.05$) and 100 g (104 ± 106 mg · dl^{-1} · 7 h^{-1} vs 171 ± 104 mg · dl^{-1} · 7 h^{-1}, $P<0.05$) glucose. Peak postprandial TG concentrations occurred later after meals containing glucose and fat than after meals containing fat alone. This effect could be reproduced when an iso-osmotic quantity of urea was substituted for glucose in the test meal. Starch ingestion had no discernible effect on postprandial lipemia. Intravenous fat tolerance was similar before (4.9 ± 1.2% · min^{-1}) and 2 h (4.4 ± 1.3% · min^{-1}) and 4 h (4.8 ± 1.5% · min^{-1}) after 50 g glucose ingestion. During duodenal fat perfusion, glucose ingestion caused a progressive decrease in plasma triglyceride concentrations. ▒▒ These data suggest that glucose ingestion diminishes postprandial lipemia in a dose-dependent manner, but that this effect is not due to increased clearance of triglyceride from the circulation. The hypotriglyceridemic effects of glucose appear to reflect delayed gastric emptying and decreased hepatic secretion of triglyceride. —**Cohen, J. C., and G. M. Berger.** Effects of glucose ingestion on postprandial lipemia and triglyceride clearance in humans. *J. Lipid Res.* 1990. **31:** 597–602.

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Although it has been known for decades that serum triglyceride (TG) concentrations are increased after fat ingestion (1), the factors that determine the magnitude of postprandial lipemia remain poorly understood. In healthy humans, fat absorption is essentially complete (2, 3), and the serum TG response to a given fat load appears to depend largely on the rate of chylomicron TG clearance (4, 5). The major fraction of diet-derived triglycerides is cleared from the plasma after enzymatic hydrolysis at the vascular endothelial surfaces by lipoprotein lipase (6). Therefore the observation that rat adipose tissue lipoprotein lipase activity was increased by acute insulin administration (7) led some authors to propose

that postprandial changes in serum TG concentrations are actively damped by insulin-mediated increases in lipoprotein lipase activity (8, 9). This hypothesis was supported by the finding that adipose tissue lipoprotein lipase activity may be increased (10) and postprandial lipemia diminished or abolished (11, 12) by acute glucose ingestion.

More recently, the role of glucose and insulin in the regulation of postprandial lipemia has been questioned. In 21 normolipidemic subjects studied in our laboratory (13) postprandial lipemia was not significantly diminished by 50 g glucose administered by oral or by intravenous route, despite markedly increased postprandial serum insulin concentrations. Similar findings were reported by Nicholls and Cohen (14). Furthermore, studies using the euglycemic-hyperinsulinemic clamp technique (15) indicated that insulin-mediated increases in adipose tissue lipoprotein lipase activity were only manifest 6 h after intravenous insulin administration, and that these increases were largely abolished by acute fat feeding.

The studies described in this report were conducted to further elucidate the effects of glucose ingestion on postprandial serum triglyceride metabolism, and to examine the mechanisms whereby such effects may be mediated. Our findings indicate that acute glucose ingestion reduces postprandial lipemia in a dose-related manner, but does not increase the rate of removal of chylomicron triglycerides from the circulation.

METHODS

The procedures used in this study were approved by the Ethics and Research Committee of the University of Cape Town. All procedures were performed on an outpatient

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Abbreviations: TG, triglyceride; VLDL, very low density lipoproteins.
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basis on normolipidemic students aged between 19 and 25, who were following self-selected diets. None of the subjects smoked, and none were obese (BMI > 25). Subjects were requested to abstain from alcohol and vigorous exercise (for 72 h) and food (for 12 h) before each test.

Effects of glucose ingestion on the serum triglyceride responses to fat feeding (postprandial lipemia)

Postprandial lipemia was measured as described previously (5). Each subject was studied twice, with an interval of 3 days between the tests. On one occasion, a stock meal comprised of 100 ml cream (40 g fat), 300 ml water, and 3 g chocolate flavoring was consumed. For the second test, 50 g (n = 55, 40 men and 15 women) or 100 g (n = 18, 14 men and 4 women) glucose was added to the stock meal. The tests were performed in random order. Blood samples were drawn before, and at 2, 3, 4, 6, and 7 h after the meal. Serum TG concentration was determined enzymatically in each sample (Boehringer Mannheim test kit 240052, Mannheim, FRG). Postprandial lipemia was quantified from the area under the curve described by serum TG concentrations (normalized to the zero hour time point) plotted against time. This area was calculated by numerical integration using the trapezoid rule. In 10 subjects who performed all three tests, additional blood samples were drawn at half hour intervals for 2 h after each meal. The insulin concentrations of these samples were measured by radioimmunoassay (Pharmacia Diagnostics kit #805595, Piscataway, NJ). Postprandial insulinemia was calculated as described above for postprandial lipemia.

In 8 of these subjects (6 mean and 2 women), two additional tests were performed. In one test, 16.6 g urea was added to the stock meal. In the other, 100 g soluble starch was added to the stock meal.

Effects of glucose ingestion on intravenous fat tolerance

Intravenous fat tolerance was measured in 20 men as described previously (5). For this test, an intravenous bolus (0.5 ml · kg^{-1}) of 20% Intralipid (Kabi Vitrium, Stockholm, Sweden) was given, and blood samples were drawn 5, 7.5, 10, 12.5, 15, 20, 25, and 30 min thereafter. Intralipid TG concentration was measured in each sample by spectrophotometry (5). A fractional clearance coefficient (k2) was calculated from the slope of the curve described by plasma Intralipid concentration plotted against time. Each subject was tested three times, with an interval of 3 days between each test. On one occasion, intravenous fat tolerance was measured in the fasted state. On subsequent days, the test was repeated 2 h and 4 h after 50 g glucose ingestion. The tests were performed in random order. Blood samples for insulin analysis were drawn 30 min after glucose ingestion.

Effects of intravenous heparin infusion on intravenous fat tolerance

To ascertain whether the intravenous fat tolerance test can resolve small changes in plasma triglyceride clearance, the effects of low-dose heparin infusions on intravenous fat tolerance were examined. In a preliminary study of 7 subjects (5 men and 2 women), intravenous infusion of heparin (25 U · min^{-1}) reduced plasma TG concentrations by 25% within 15 min. Thereafter, plasma TG concentrations remained relatively stable for a further 30 min. Accordingly, intravenous fat tolerance was measured in the fasting state, and 15 min after the commencement of 25 U · min^{-1} (n = 6) or 12.5 U · min^{-1} (n = 6) heparin infusions.

Effects of glucose ingestion on plasma and chylomicron TG concentrations during steady-state duodenal fat perfusion

The procedure used to perform duodenal fat perfusions has been described in detail previously (5). For this test, a naso-duodenal tube was positioned in the third portion of the duodenum under fluoroscopic guidance. A fat emulsion (olive oil emulsified in water with gum acacia) was then perfused through the tube at 175 mg TG · kg body weight^{-1} · h^{-1}. Blood samples were taken before, and 5, 6, 7, 8, 9, and 10 h after commencing the perfusion. Plasma TG concentration was analyzed in each sample. Duodenal perfusion studies were performed in 20 men. Each subject was tested twice. On one occasion the perfusion contained fat only. For the second test, the subjects (n = 10) consumed 50 g glucose in 300 ml water 3 h after commencing the perfusion. Alternatively, glucose (20 g · h^{-1}) was added to the perfusate (n = 10).

Statistical analysis

The mean values of each parameter measured before and after glucose administration were compared using the paired t-test (16). Where repeated measures were compared, the P value was adjusted using Bonferroni's procedure.

RESULTS

In general, the tests were well tolerated, and none of the subjects reported nausea or diarrhoea after any of the meals, or after any of the duodenal perfusions.

Effects of glucose ingestion on postprandial lipemia

Mean postprandial lipemia was significantly lower after meals containing glucose and fat than after meals containing fat alone (**Table 1**). The mean peak postprandial TG concentration was lower and occurred later after meals containing glucose and fat than after fat alone (**Fig. 1** and **Fig. 2**). The ingestion of urea diminished and

TABLE 1. Effects of glucose ingestion on postprandial lipemia in normolipidemic young men and women

	Stock Meal[a]	Stock Meal 50 g Glucose	Stock Meal	Stock Meal 100 g Glucose
n	55	55	18	18
FTG	78 ± 25	77 ± 25	80 ± 27	76 ± 31
PPL	192 ± 124	148 ± 121[b]	171 ± 104	104 ± 106[b]

Abbreviations and units: FTG, fasting triglyceride concentration $(mg \cdot dl^{-1})$; PPL, postprandial lipemia $(mg \cdot dl^{-1} \cdot 7 \ h^{-1})$.

[a]Stock meal comprised of 100 ml cream (40 g fat), 300 ml water, and 3 g chocolate flavoring.

[b]$P < 0.05$ using Student's paired t-test.

Fig. 2. Serum triglyceride concentrations in 18 normolipidemic subjects after meals containing 100 g glucose + 40 g fat (●), or 40 g fat alone (O).

delayed the postprandial TG excursion (**Fig. 3**). Starch ingestion had no effect on postprandial TG concentrations (**Fig. 4**). Mean plasma insulin concentrations measured in 10 of the subjects are shown in **Table 2**.

Effects of glucose ingestion on intravenous fat tolerance

The curve of plasma Intralipid concentration plotted against time was adequately fitted by a single exponential (r was invariably > 0.96). Glucose ingestion did not influence intravenous fat tolerance (**Fig. 5**). Mean k2 was $4.9 \pm 1.2\% \cdot min^{-1}$ in the fasted state, $4.4 \pm 1.3\% \cdot min^{-1}$ 2 h after glucose ingestion, and $4.8 \pm 1.5\% \cdot min^{-1}$ 4 h after glucose ingestion. Mean plasma insulin concentrations increased from $5 \pm 3 \ mU \cdot l^{-1}$ before glucose ingestion to $46 \pm 19 \ mU \cdot l^{-1}$ 30 min after glucose ingestion.

Effects of heparin infusion on intravenous fat tolerance

Heparin infusion increased the clearance of Intralipid in all subjects, and the data from both doses (12.5 and 25 $U \cdot min^{-1}$) were combined for the purposes of analysis.

Mean k2 was $6.3 \pm 2\% \cdot min^{-1}$ before and $12.4 \pm 5\% \cdot min^{-1}$ after heparin infusion.

Effects of glucose ingestion on plasma TG concentrations during duodenal fat perfusion

Results from subjects given glucose by mouth were similar to those obtained from subjects given glucose by duodenum, therefore data from the two groups were combined for statistical analysis. Duodenal fat perfusion increased plasma TG concentrations in all subjects (**Fig. 6**). On average, the extent of the increment measured 2 h after glucose ingestion ($77 \pm 36 \ mg \cdot dl^{-1}$) was similar to that achieved at the corresponding point during the baseline perfusion ($85 \pm 42 \ mg \cdot dl^{-1}$, $P > 0.2$). Five hours after glucose ingestion, however, the mean plasma TG increment ($53 \pm 21 \ mg \cdot dl^{-1}$) was significantly lower ($P < 0.05$) than the corresponding value measured during the baseline perfusion ($91 \pm 49 \ mg \cdot dl^{-1}$).
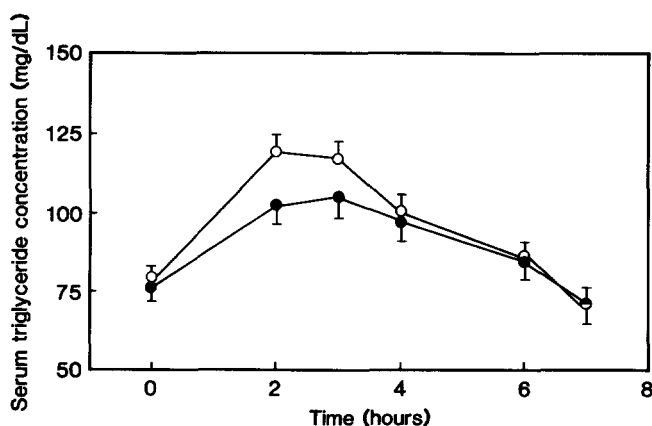

Fig. 1. Serum triglyceride concentrations in 55 normolipidemic subjects after meals containing 50 g glucose + 40 g fat (●), or 40 g fat alone (O).
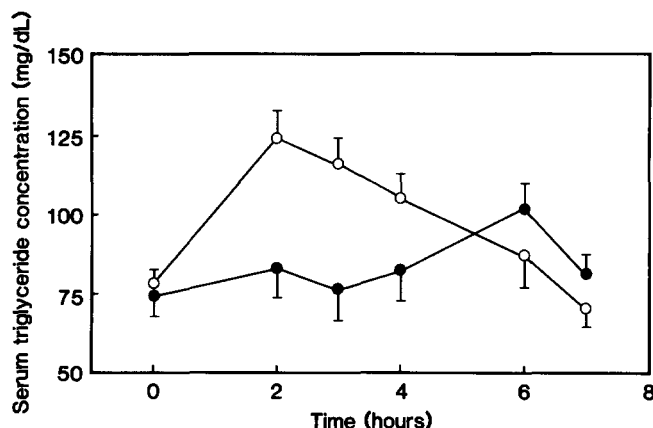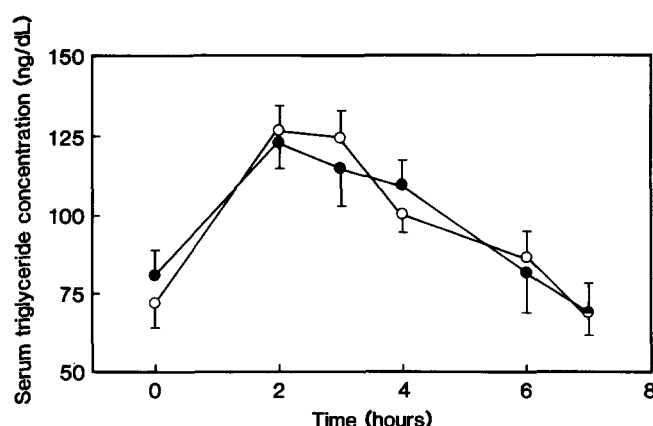

Fig. 3. Serum triglyceride concentrations in eight normolipidemic subjects after meals containing 100 g starch + 40 g fat (●), or 40 g fat alone (O).
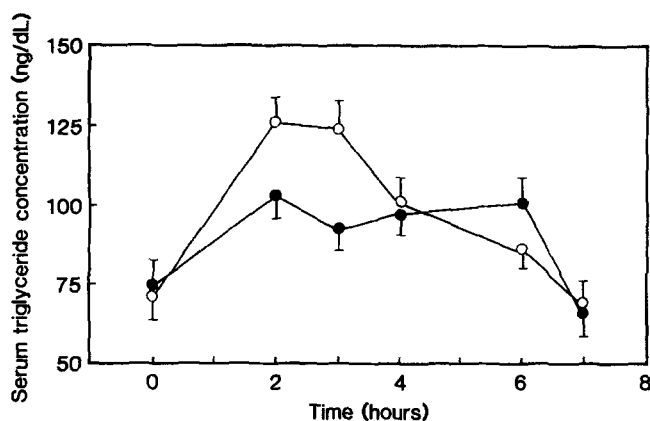
Fig. 4. Serum triglyceride concentrations in eight normolipidemic subjects after meals containing 16.6 g urea + 40 g fat (●), or 40 g fat alone (○).

Fig. 5. Plasma Intralipid concentrations in 20 normolipidemic men receiving intravenous Intralipid (0.5 ml · kg⁻¹ of 20% solution), before (●) and after (○) 50 g glucose ingestion.

DISCUSSION

The results of this study indicate that the ingestion of 50 g glucose causes a small (25%) but significant decrease in the serum TG response to a fatty meal. In 38 of the 56 people in this experiment (68%), postprandial lipemia was lower after meals containing 50 g glucose and 40 g fat than after 40 g fat alone. The hypotriglyceridemic effect of glucose was greater (39%) and more consistent (78% of subjects studied) when larger doses of glucose (100 g) were used.

These findings may explain the conflicting results obtained in previous studies of the effects of glucose ingestion on postprandial serum TG concentrations. When low (50 g) doses of glucose are used, the reduction in postprandial lipemia (about 25% on average) is similar in magnitude to the mean intra-individual variation in oral fat tolerance (about 20%) (17). Accordingly, "true" hypotriglyceridemic effects of glucose will tend to be concealed when insufficient numbers of subjects are studied. The reduction of postprandial lipemia effected by larger doses (100 g) of glucose is appreciably greater than the intra-individual variation (40% vs 20%). Attenuation of postprandial lipemia is therefore more consistent at these
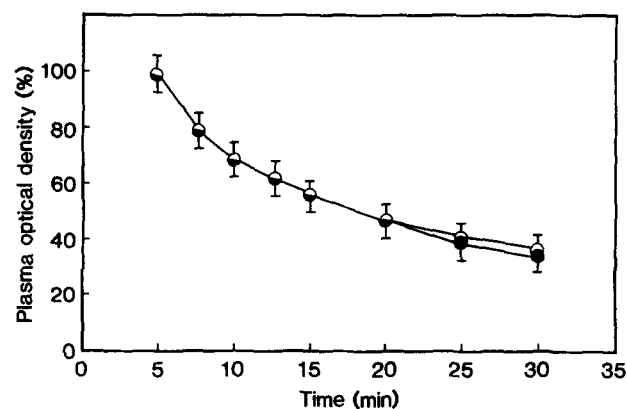
dosages, and is more likely to be recognized. Thus studies reporting no effect of glucose have tended to use low (50 g) doses (13, 14) while those reporting a hypotriglyceridemic effect of glucose have tended to use much larger (>100 g) doses (11, 12).

The hypotriglyceridemic effect of glucose is widely considered to reflect increased TG clearance secondary to insulin-mediated increases in adipose tissue lipoprotein lipase acitivity (18). In the present study, this hypothesis was examined using the intravenous fat tolerance test. Since the effects of glucose ingestion on postprandial lipemia are most evident between 2 and 4 h after the meal, the clearance of an artificial fat emulsion was measured 2 and 4 h after a 50 g glucose load. At these times, the average rate of disappearance of the fat emulsion was slightly lower after glucose ingestion, a finding that is not consistent with increased TG clearance. The interpretation of these data is subject to two caveats.

TABLE 2. Effects of meals containing glucose and fat on plasma insulin concentrations

	Stock Meal	Stock Meal + 50 g Glucose	Stock Meal + 100 g Glucose
n	10	10	10
F.INS	6 ± 3	7 ± 4	6 ± 4
P.INS	15 ± 7	227 ± 141	463 ± 239
M.INS	11 ± 9	56 ± 17	91 ± 34

Abbreviations and units: F.INS, fasting plasma insulin concentrations (mU · l⁻¹); P.INS, postprandial insulinemia (mU · l⁻¹ · 2 h⁻¹); M.INS, maximum postprandial insulin concentration (mU · l⁻¹).
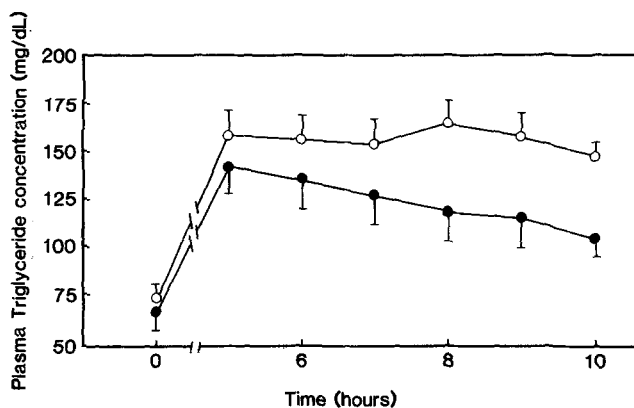

Fig. 6. Plasma triglyceride concentrations in 20 normolipidemic men during duodenal perfusion with an olive oil emulsion (175 mg/kg/h) alone (○) and after 50 g glucose administration (●)

First, the decay kinetics of Intralipid may not reflect those of chylomicrons. This contention seems unlikely, however, as several studies have indicated that Intralipid and chylomicrons exhibit similar decay kinetics after intravenous administration in humans (19) and other animals (20).

Second, the intravenous fat tolerance test may be insensitive to small changes in TG clearing activity. To examine this possibility, we measured the rate of clearance of Intralipid before and after low-dose heparin infusion, a procedure known to increase intravascular TG clearing activity (21). The doses of heparin used in the study were sufficient to lower plasma TG concentrations by 25%, a decrease similar to that achieved by 50 g oral glucose. In each subject tested, heparin infusion increased Intralipid clearance. Therefore the failure of glucose ingestion to increase fat clearance does not appear to be due to insensitivity of the testing procedure.

Given these considerations, it seems unlikely that the effects of glucose ingestion on the early phase (2–4 h) of postprandial lipemia reflect increased TG clearance.

Since glucose ingestion does not appear to increase the rate of chylomicron-TG clearance during the early postprandial phase, the effects of glucose ingestion on the serum TG concentrations measured during this time presumably reflect a decrease in the rate of entry of TG into the circulation. Although the possible effects of glucose ingestion on the rate of fat absorption were largely discounted in early studies (9, 11), glucose ingestion was subsequently found to delay the rate of gastric emptying of mixed meals (22). In the present study, the maximum increase in serum TG concentrations occurred later after meals containing glucose and fat than after fat alone. This effect was particularly marked when larger doses of glucose were given (see Fig. 2). These observations are consistent with a delay in fat absorption after glucose ingestion. To determine whether the effects of glucose ingestion on the magnitude and timing of the postprandial TG excursion reflect delayed gastric emptying, plasma TG concentrations were measured during duodenal perfusion with fat, or fat and glucose. This procedure, which circumvents the stomach entirely, largely eliminated the effects of glucose ingestion on serum TG concentrations measured 2 h after glucose ingestion. This finding suggests that the early effects of glucose ingestion on postprandial lipemia are due primarily to glucose-induced retardation of the rate of gastric emptying.

If the effect of glucose ingestion on postprandial lipemia is indeed due to delayed gastric emptying, then long-chain polymers of glucose, which have less effect than glucose on gastric emptying (23), would be expected to have less effect on postprandial lipemia. In the present study, ingestion of 100 g of a glucose polymer (soluble starch) had no discernible effect on the magnitude or time course of the postprandial serum TG excursion (see Fig. 4). This observation provides further support for the contention that glucose ingestion delays the gastric emptying of fat.

Since the rate of gastric emptying appears to be inversely related to the osmolarity of the duodenal contents (24), the effects of glucose ingestion on gastric emptying may be related (at least in part) to increased osmolarity of the test meal. To test this hypothesis, an iso-osmotic quantity of metabolically inert material (urea) was substituted for glucose in the test meal. Urea delayed and diminished the postprandial excursion of serum TG to a similar extent as did glucose, indicating that glucose may delay postprandial lipemia simply by increasing the osmolarity of the test meal.

On the basis of these studies, it seems reasonable to conclude that the effects of acute glucose ingestion on serum TG concentrations measured 2 h after mixed meals reflect delayed fat absorption, and that the delay in fat absorption after glucose ingestion may be secondary to increased osmolarity of the test meal.

Delayed fat absorption per se should not diminish total postprandial lipemia, however, since the fat which is absorbed during the later phase of the test would give rise to higher serum TG concentrations at this time, as was evident after urea ingestion. In addition, the duodenal perfusion studies revealed a progressive decrease in plasma TG concentrations for several hours after glucose administration, which was clearly unrelated to delayed gastric emptying. Thus an alternative hypothesis is required to explain the hypotriglyceridemic effect of acute glucose administration. One possibility is that glucose ingestion reduces the rate of secretion of triglycerides by the liver. In previous studies (25, 26) the administration of glucose in the fasted state reduced serum TG concentrations. These observations indicate that glucose reduces the plasma concentration of liver-derived TG. Several lines of evidence suggest that this effect is due to reduced hepatic TG secretion, rather than to increased TG clearance.

First, during euglycemic clamp studies in which plasma insulin concentrations were comparable with those measured in the present study after 100 g glucose ingestion, Yki-Järvinen et al. (27) found that VLDL-TG concentrations were decreased more than 50% while adipose tissue lipoprotein lipase activity was unchanged. Similarly, Sadur and Eckel (28) found that the insulin-induced decrease in plasma TG concentrations preceded changes in adipose tissue lipoprotein lipase activity.

Second, Vogelberg, Gries, and Moschinski (29) reported that intraportal insulin administration decreased hepatic TG secretion in normolipidemic men.

Finally, Patsch, Gotto, and Patsch (30) reported that insulin inhibited TG release by rat hepatocytes. These latter authors (30) proposed that inhibition of hepatic TG secretion during meal absorption would facilitate the clearance of intestinal lipoproteins, which share a common clear-

ance path with hepatic TG-rich lipoproteins (31). While a direct validation of this hypothesis is beyond the scope of the present study, our findings are consistent with the concept that glucose diminishes total postprandial lipemia by insulin-mediated reduction of hepatic VLDL secretion.

In summary, our data support the following conclusions. *1*) Glucose ingestion decreases postprandial lipemia in a dose-dependent manner. *2*) The early reduction in postprandial lipemia is largely attributable to delayed gastric emptying, possibly as a consequence of increased osmolarity of the test meal. *3*) Glucose ingestion does not increase the clearance of intestine-derived TG-rich lipoproteins for up to 4 h after ingestion of mixed meals, the period during which the bulk of dietary fat is absorbed. *4*) Glucose does diminish postprandial serum TG concentrations by a mechanism independent of its effect on gastric emptying, but these effects only become apparent during the later postprandial period. ∎

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