Hydrolysis of chylomicron arachidonate and linoleate ester bonds by lipoprotein lipase and hepatic lipase

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Abstract Chylomicrons labeled with [^{3}H]arachidonic and [^{14}C]linoleic acid were incubated with bovine milk lipoprotein lipase or rat postheparin plasma, containing both lipoprotein lipase and hepatic lipase. During incubation with bovine lipoprotein lipase, [^{3}H]arachidonic acid was released from chylomicron triacylglycerols at a slower rate than [^{14}C]linoleic acid. Only small amounts of [^{14}C]linoleic acid were found as 1,2(2,3)-diacylglycerols, whereas a transient accumulation as [^{14}C]monoacylglycerols was observed. In contrast, significantly more [^{3}H]arachidonic acid was found as 1,2(2,3)-diacylglycerols than as monacylglycerols at all time intervals investigated. The initial pattern of triacylglycerol hydrolysis by postheparin plasma was similar to that of bovine lipoprotein lipase. However, in contrast to the results obtained with bovine lipoprotein lipase, little [^{3}H]1,2(2,3)-diacylglycerol accumulated. The addition of antiserum to hepatic lipase increased the amount of ^{3}H found in 1,2(2,3)-diacylglycerols and inhibited the formation of free [^{3}H]arachidonic acid. The antiserum also caused a significant inhibition of the hydrolysis of [^{3}H)- but not of [^{14}C]triacylglycerol. With regard to chylomicron phospholipids, the rate of hydrolysis of [^{14}C]linoleoyl phosphatidylcholine with milk lipoprotein lipase was twofold higher than that of the [^{3}H]arachidonyl phosphatidylcholine. However, the hepatic lipase of postheparin plasma had similar activity towards the two phosphatidylcholine species. Postheparin plasma rapidly hydrolyzed chylomicron ^{3}H-labeled and ^{14}C-labeled phosphatidylethanolamine to the same degree, and lipoprotein lipase similarly hydrolyzed ^{3}H-labeled and ^{14}C-labeled phosphatidylethanolamine at approximately equal rates. Antiserum to hepatic lipase inhibited the postheparin plasma hydrolysis of phosphatidylethanolamine and ^{3}H-labeled phosphatidylcholine by about 60%, but the ^{14}C-labeled phosphatidylcholine by only 27%. Thus, rat chylomicron triacylglycerols, phosphatidylcholine, and 1,2(2,3)-diacylglycerols containing arachidonic acid are less efficiently hydrolyzed by lipoprotein lipase than are the linoleic acid esters of these chylomicron lipids. Hepatic lipase may participate in the hydrolysis of arachidonate-containing 1,2(2,3)-diacylglycerols, formed by the action of lipoprotein lipase on chylomicrons. —**Nilsson, Å., B. Landin, and M. C. Schotz.** Hydrolysis of chylomicron arachidonate and linoleate ester bonds by lipoprotein lipase and hepatic lipase. *J. Lipid Res.* 1987. **28:** 510–517.

Supplementary key words arachidonic acid • linoleic acid • triacylglycerol • diacylglycerol • phosphatidylcholine • phosphatidylethanolamine

Despite the crucial role of arachidonic acid (20:4) as precursor of prostaglandins and leukotrienes, relatively little attention has been paid to the pathways by which arachidonic acid, present in intestinal lipoproteins, is assimilated by the tissues. Arachidonic acid accounts for about 20% of the fatty acids located at the 2-position of rat bile phosphatidylcholine (1, 2) and significant amounts are present also in dietary (3) and mucosal phospholipids (4), as well as in triacylglycerols (TG) and phospholipids of chyle chylomicrons, VLDL, and HDL (2, 5–7).

Intestinal TG, formed during lipid absorption, contains 20:4 mainly at the *sn*-3 and *sn*-2 positions (8). Since lipoprotein lipase has a relative preference for the *sn*-1 as compared to the *sn*-3 ester bond and does not attack the *sn*-2 ester bonds of TG (9, 10), the lipolysis of chylomicron TG 20:4 might be influenced by the asymmetric distribution of this acid within the TG molecule. Morley and Kuksis (11), using artificial TG and purified milk lipoprotein lipase, found hydrolysis of the 20:4 ester bonds, when present in TG of natural cod liver oil, slower than in TG molecules with randomized rearrangement of the ester bonds. Recently, Ridgway and Dolphin (12) reported that remnants formed from VLDL in hypothyroid rats were enriched in polyunsaturated 20-carbon fatty acids, indicating that the TG ester bonds of these fatty acids, indicating that the TG ester bonds of these fatty acids were hydrolyzed at a slower rate than the esters of the predominant fatty acids in VLDL TG.

The preferential incorporation of 20:4 into the *sn*-3 and *sn*-2 positions of chylomicron TG and phospholipids, as

Abbreviations: 20:4, arachidonic acid; 18:2, linoleic acid; TG, triacylglycerols; DG, diacylglycerols; MG, monoacylglycerols; PC, phosphatidylcholine; PE, phosphatidylethanolamine; VLDL, very low density lipoproteins; LDL, low density lipoproteins; IDL, intermediate density lipoproteins; HDL, high density lipoproteins, HEPES, 4-(2-hydroxyethel)-1-piperazine-ethanesulfonic acid; FFA, free fatty acids.

well as the substrate and positional specificities of lipoprotein lipase and hepatic lipase, could contribute to differences between the metabolism of 20:4 and other fatty acids of intestinal lipoproteins. In the present study the action of bovine milk lipoprotein lipase and postheparin plasma lipoprotein lipase and hepatic lipase on chyle and chylomicrons labeled with [^{3}H]arachidonic acid (20:4) and [^{14}C]linoleic acid (18:2) was examined.

MATERIALS AND METHODS

5,6,8,11,12,14,15-[^{3}H]20:4 (83.8 Ci/mmol) and [1-^{14}C]18:2 (52.6 mCi/mmol) were purchased from New England Nuclear Corporation, Boston, MA. Unlabeled 20:4 and 18:2 were obtained from Nu-Chek-Prep, Elysian, MN. Intralipid and heparin (5000 IE/ml) were obtained from Kabi-Vitrum AB, Stockholm, Sweden.

The thoracic ducts of male white Sprague-Dawley rats, weighing about 250 g, were cannulated and the rats were treated postoperatively as described earlier (13). Twenty to 24 hr after surgery, fat containing the radioactive fatty acids was fed through a gastric fistula. Fifty to 100 μCi of [^{3}H]20:4 and 10–20 μCi of [^{14}C]18:2 were dispersed with 0.33 mg of egg phosphatidylcholine in 1 ml of 0.9% NaCl and added to either 0.5 ml of cream (40% fat) plus 0.5 ml of 0.9% NaCl or 1 ml of 20% Intralipid.

After feeding cream, chyle was collected at room temperature and used within 6 hr to avoid structural changes during cooling due to the content of saturated fat (13). Chyle obtained after feeding Intralipid was collected on ice in the presence of Na_2EDTA (final conc, 2 mM). The chyle was stored at 4°C for less than 7 days. After the chyle had been diluted with 1.1% NaCl containing 2 mM Na_2EDTA, chylomicrons were isolated by ultracentrifugation at 25,000 rpm for 2 hr at 20–24°C using a Beckman SW 41 swinging bucket rotor. Postheparin plasma was obtained from rats by aortic puncture 2 min after intravenous injection of 100 units of heparin. Antiserum against rat hepatic lipase, prepared as described previously, has been shown not to react with lipoprotein lipase (14). The pure bovine milk lipoprotein lipase was prepared and characterized as described by Bengtsson and Olivecrona (15).

Unless stated otherwise, the incubation medium consisted of 2 ml of Hanks solution, buffered with 40 mM HEPES, pH 7.4, 2 ml of 10% bovine serum albumin, and 1 ml of serum or postheparin plasma. In the experiments with postheparin plasma, 50 μl of antiserum to hepatic lipase (anti-hepatic lipase) or 50 μl of control rabbit serum was added. Incubations were performed at 37°C. The activity of the added milk lipoprotein lipase was 490 μmol of FFA/min per ml.

Aliquots (0.5–1 ml) of the incubation mixture were taken after 5, 10, 20, and 30 min. Lipids were extracted with 8 volumes of chloroform–methanol 1:1 (v/v) containing 0.005% butylated hydroxytoluene as an antioxidant. After removal of the protein precipitate and washing of the extract, the neutral lipids were separated by thin-layer chromatography on silica gel G in low boiling petroleum ether–diethyl ether–methanol–acetic acid 80:20:2:1 (v/v). Phospholipids were separated by thin-layer chromatography on silica gel G plates developed in chloroform–methanol–acetic acid–water 65:25:4:4 (v/v). The lipids were visualized by staining with iodine. The different fractions were scraped into vials. One ml of methanol–water 1:1 and 10 ml of Instagel–toluene 1:1 (v/v) were added, and the samples were counted in a Packard 460 CD liquid scintillation system, using the computerized automatic external standard for quench correction. In the presentation of the TG data the cholesteryl ester radioactivity, accounting for less than 0.3% of the ^{3}H and less than 0.8% of the ^{14}C radioactivity, is included in the TG fraction.

Significance of differences was estimated by Student's paired *t*-test.

RESULTS

Hydrolysis of triacylglycerols

Chylomicrons labeled with [^{3}H]20:4 and [^{14}C]18:2, obtained from rats fed cream or Intralipid, were used in incubations with lipoprotein lipase and postheparin plasma. The preparations contained different proportions of radioactivity in TG and phospholipids. In chylomicrons from the cream-fed rats, 29% of the ^{3}H and 16% of the ^{14}C was in phospholipids. In chylomicrons from the Intralipid-fed rats the phospholipids contained 15–18% of the ^{3}H and 8–10% of the ^{14}C. The decline in the proportion of radioactivity found in the total phospholipid fraction including the lyso compounds was, however, small or undetectable during the incubations with bovine lipoprotein lipase and postheparin plasma. The released radioactive fatty acids and the partial glycerides formed during the incubation must thus have been derived from hydrolysis of TG.

The time course for TG hydrolysis of cream and Intralipid chylomicrons with bovine milk lipoprotein lipase is shown in **Fig. 1A and B.** With both types of chylomicrons the disappearance of [^{14}C]18:2 from TG exceeded that of [^{3}H]20:4. The ^{3}H/^{14}C ratio of the remaining TG increased with time, and thus [^{3}H]20:4 accumulated in the chylomicron remnants that were formed. The proportion of [^{14}C]18:2 free fatty acid formed exceeded that of [^{3}H]20:4 by more than would be expected from the different rates of disappearance of ^{14}C-labeled and ^{3}H-labeled TG. This could be explained by the finding that the 1,2(2,3)-diacylglycerols (DG) formed contained significantly more ^{3}H than ^{14}C. The ratio of the monoacylglycerol (MG) fraction was consequently significantly lower than that of the added and remaining TG.

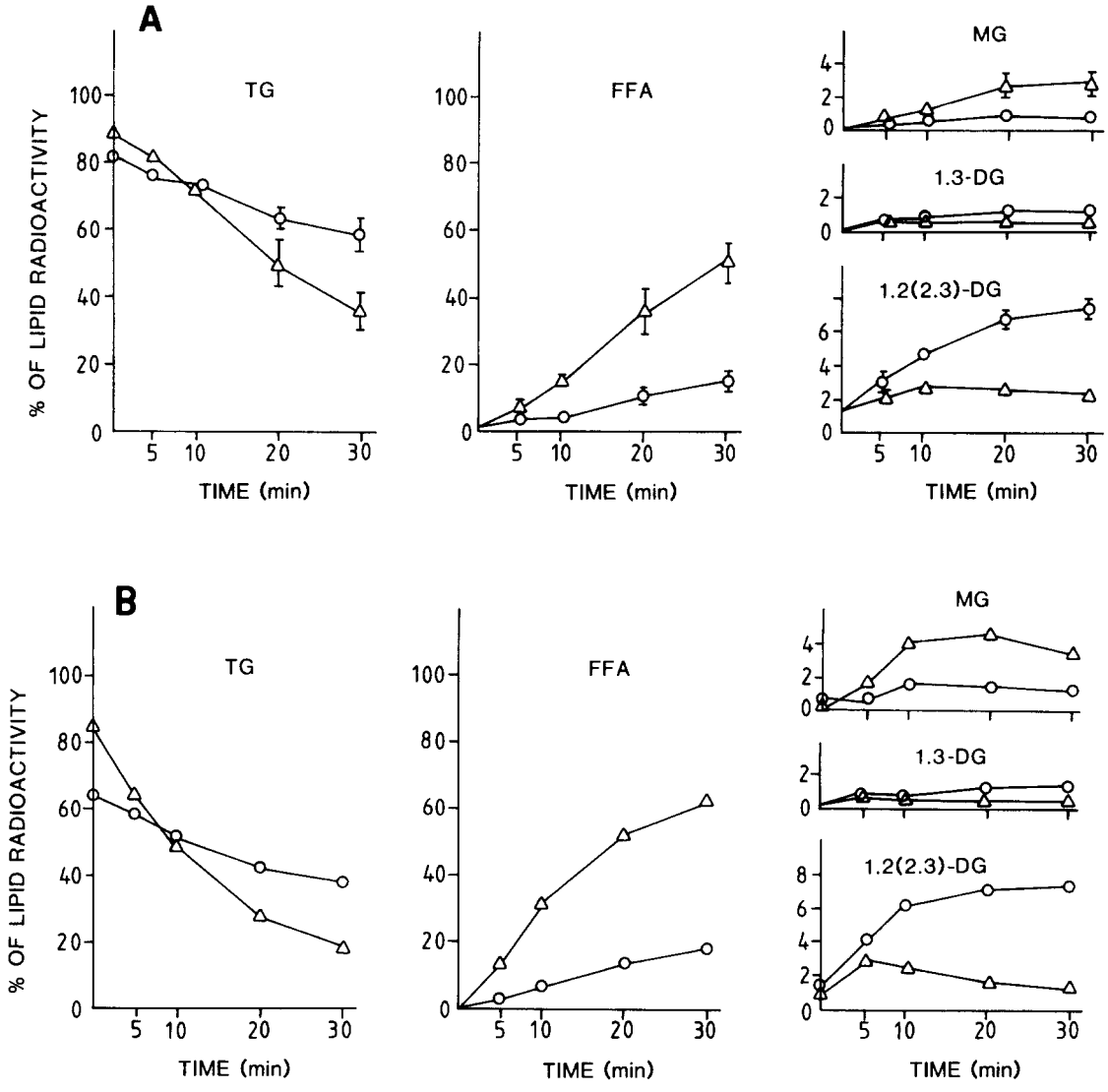

Fig. 1. Hydrolysis of Intralipid and cream chylomicron triacylglycerols with bovine milk lipoprotein lipase. A: Time course for the hydrolysis of Intralipid chylomicron TG. Incubations were performed at pH 7.4 in the presence of 20% rat serum and 4% albumin. Other conditions are given in the Methods section. The added chylomicrons contained 148 μg of TG, 8.5 × 10^5 dpm as ^{3}H-labeled TG and 4.2 × 10^5 dpm as ^{14}C-labeled TG. Two μl of bovine milk lipoprotein lipase was added. Values indicate percent of total lipid radioactivity and are means ± SEM of three observations with chylomicrons prepared from the same chyle. When not indicated, the SEM was less than the size of the symbols. B: An identical experiment with cream chylomicrons. In this case the added chylomicrons contained 658 μg of TG, 3.2 × 10^5 dpm as ^{3}H-labeled TG and 6 × 10^5 dpm as ^{14}C-labeled TG. Values are means of three observations with the same batch of chylomicrons. SEM was less than the size of the symbols and is not shown in the figure; (○—○) ^{3}H; (△—△) ^{14}C.

In order to compare the sole effect of lipoprotein lipase with the integrated effects of lipoprotein lipase and hepatic lipase, the time course of postheparin plasma lipolysis was examined with and without the addition of antiserum to hepatic lipase. The time course for the disappearance of chylomicron TG was similar to that with bovine lipoprotein lipase, i.e., the [^{14}C]18:2 disappeared faster than [^{3}H]20:4 (**Fig. 2A and 2B**). With the addition of anti-hepatic lipase there was a significant inhibition of the hydrolysis of ^{3}H-labeled TG at 30 min (**Table 1**). However, the effects of anti-hepatic lipase on the disappearance of ^{14}C-labeled TG varied between experiments. With cream chylomicrons the hydrolysis of ^{14}C-labeled TG in the presence of anti-hepatic lipase even exceeded that in the control incubations (Fig. 2B, Table 1). With Intralipid chylomicrons anti-hepatic lipase addition had no significant effect (Fig. 2A). Initially, the accumulation of ^{3}H-labeled 1,2(2,3)-DG was small, but after addition of the antiserum it increased to levels seen in the incubations with bovine lipoprotein lipase. The antiserum had no

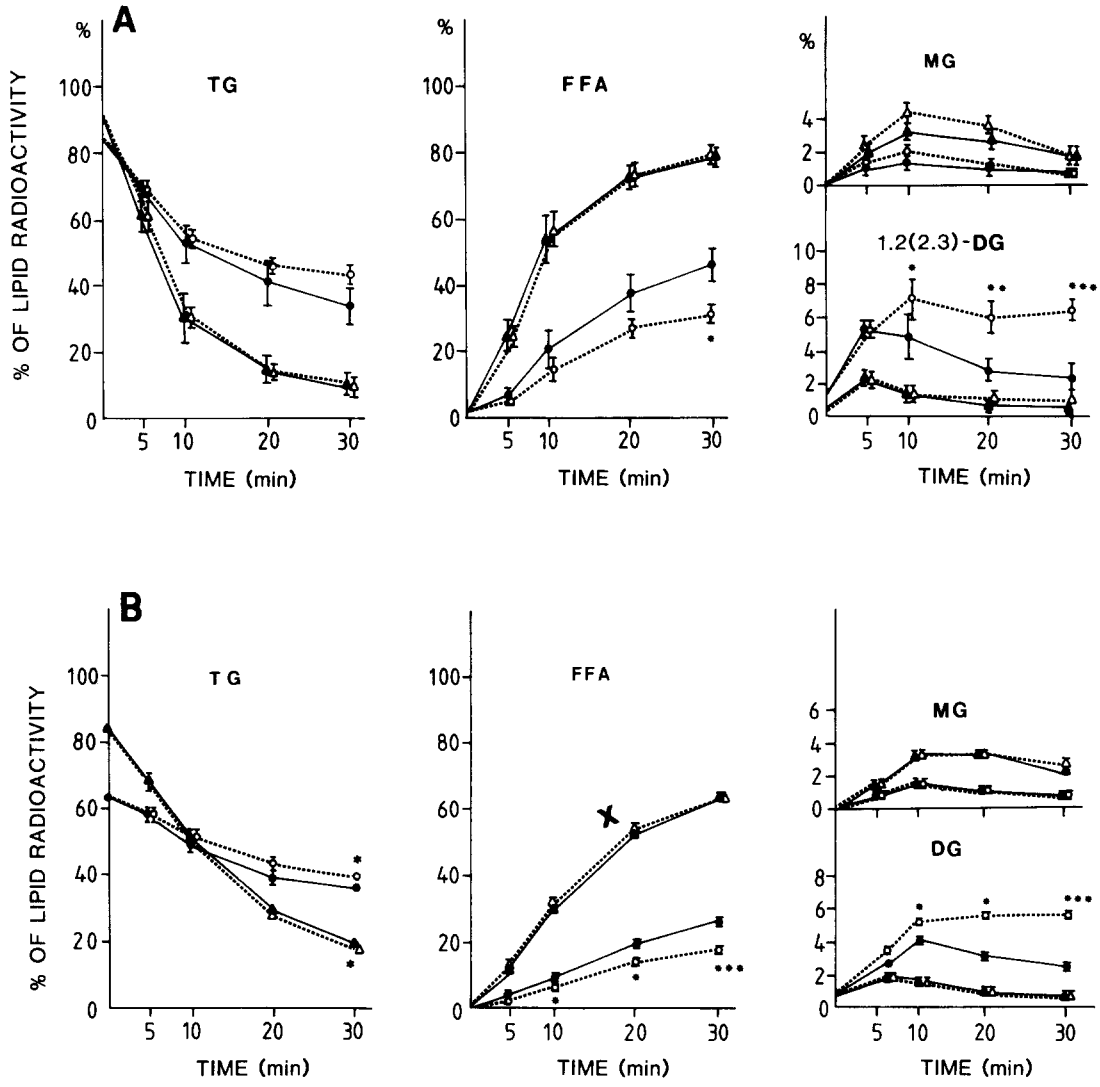

Fig. 2. Effect of anti-hepatic lipase on the hydrolysis of chylomicron triacylglycerols with postheparin plasma. Incubations were performed at pH 7.4 with 20% postheparin plasma plus 50 μl of anti-hepatic lipase or control rabbit serum present. Other details are given in Methods. A: Time course for the hydrolysis of Intralipid chylomicron TG; added chylomicrons obtained after feeding Intralipid were the same as in Fig. 1A. B: Time course of hydrolysis of cream chylomicron TG; added cream chylomicrons were the same as in Fig. 1B. Values indicate percent of total lipid radioactivity and are means ± SEM of three observations with chylomicrons isolated from the same chyle (A) or from three observations with the same batch of chylomicrons (B); (●—●) ^{3}H, control serum; (○- -○) ^{3}H, anti-hepatic lipase; (▲—▲) ^{14}C, control serum; (△- -△) ^{14}C, antihepatic lipase. Significance of differences between incubations with and without antihepatic lipase present is indicated as follows: $^{*}P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$; other differences were not significant.

effect on the proportion of ^{3}H and ^{14}C found in MG (Fig. 2A and B).

Consistent with the earlier findings that lipoprotein lipase attacks primarily the *sn*-1 and *sn*-3 positions of TG (9, 10), only small amounts of ^{3}H-labeled and ^{14}C-labeled 1,3-DG were formed during incubation with milk lipoprotein lipase, although the ^{3}H/^{14}C ratio increased with time in a manner similar to that of 1,2(2,3)-DG. However, the proportion of ^{3}H found in 1,3-DG did not exceed 2% at any time interval (Fig. 1A and 1B). It is thus possible that the labeled 1,3-DG was formed by isomerization of 1,2(2,3)-DG during incubation. In the experiments with postheparin plasma, the amount of ^{3}H-labeled and ^{14}C-labeled 1,3-DG accumulating was unaffected by the addition of anti-hepatic lipase (Table 1).

In three experiments in which linoleate-rich whole chyle, labeled with [^{3}H]20:4 and [^{14}C]18:2, was incubated for 5–30 min with milk lipoprotein lipase, or with postheparin plasma with or without anti-hepatic lipase present, the results were similar to those obtained with the Intralipid chylomicrons. Similarly the results from one experiment with doubly labeled cream whole chyle was similar to those obtained with cream chylomicrons (data not shown).

TABLE 1. Effects of anti-hepatic lipase on the hydrolysis of chyle and chylomicron triacylglycerol [^{3}H]arachidonate and [^{14}C]linoleate ester bonds

Incubation Products	Percent of Lipid Radioactivity			
	^{3}H		^{14}C	
	Control	AHL	Control	AHL
TG	34.7 ± 2.6	41.6 ± 1.5**	15.5 ± 2.4	13.0 ± 2.6
1,2 (2,3) DG	2.5 ± 0.4	6.0 ± 0.4***	0.6 ± 0.1	0.9 ± 0.1
1,3 DG	1.7 ± 0.3	1.6 ± 0.1	0.3 ± 0.0	0.4 ± 0.0
MG	0.7 ± 0.1	0.8 ± 0.1	1.6 ± 0.3	2.4 ± 0.2
FFA	36.2 ± 5.0	24.8 ± 3.1**	71.6 ± 3.7	72.2 ± 3.99

The table shows percent of total lipid radioactivity in glycerides and FFA after incubation of [^{3}H]20:4- and [^{14}C]18:2-labeled chylomicrons (n = 6) for 30 min with postheparin plasma with and without anti-hepatic lipase (AHL) present. The three observations with Intralipid and the three observations with cream chylomicrons presented in Fig. 2 are grouped together. $^{**}P < 0.01$; $^{***}P < 0.001$, according to Student's paired *t*-test.

Hydrolysis of phospholipids

Hydrolysis of phospholipids was studied in the experiments with chylomicrons from rats fed cream or Intralipid. As predicted from the specificity of lipoprotein lipase for the *sn*-1 ester bond of chylomicron phospholipids (16–18), the formation of both ^{3}H-labeled and ^{14}C-labeled lysoPC and PE increased with time. About twice as much ^{14}C-labeled PC as ^{3}H-labeled PC was converted to lysoPC during incubation with lipoprotein lipase, indicating that the enzyme hydrolyzed 18:2-PC faster than 20:4-PC (**Fig. 3**). In contrast, there was little difference in the hydrolysis of ^{3}H-labeled and ^{14}C-labeled PE except at the 30-min time interval. However, the hydrolysis of PE by lipoprotein lipase was more than twice that of PC (Fig. 3).

In incubations with postheparin plasma, hydrolysis of PE was more extensive than with lipoprotein lipase and could be inhibited to about 60% by the addition of anti-hepatic lipase (**Fig. 4**). The difference in hydrolysis of ^{3}H-labeled and of ^{14}C-labeled PC was less than with milk lipoprotein lipase. For instance, 16% of the ^{14}C-labeled PC and 20% of the ^{3}H-labeled PC was converted to lysoPC after 30 min. Addition of anti-hepatic lipase inhibited the formation of ^{3}H-labeled lysoPC by 58% and the formation of ^{14}C-labeled lysoPC by 27%. Similar results were obtained in experiments with Intralipid chylomicrons. The data thus indicated that the hepatic lipase contributed relatively more to the hydrolysis of 20:4-PC than to that of 18:2-PC.

DISCUSSION

The present study demonstrates that arachidonic acid ester bonds of chylomicron TG are hydrolyzed by milk lipoprotein lipase and postheparin plasma lipoprotein lipase less efficiently than are the linoleic acid ester bonds. Accordingly, 20:4 is enriched in the chylomicron remnants formed. If this occurs also in vivo, a larger proportion of absorbed 20:4 than of the other chylomicron fatty acids would thus be expected to be distributed to the liver with the remnant particles. The findings are consistent with the observations of Ridgway and Dolphin (12), who found an enrichment of polyunsaturated C_{20} and C_{22} fatty acids in IDL and LDL formed by the action of lipoprotein lipase on VLDL.

The time course for lipolysis of chylomicron [^{14}C]18:2-labeled TG with lipoprotein lipase was similar to that found by Scow and Olivecrona (19), using chylomicrons labeled with oleic acid and glycerol and comparable albumin concentrations and by Wang, Hartsuch, and Weiser (20) using human VLDL as substrate. Thus, a rapid release of FFA and a transient appearance of significant amounts of ^{14}C-labeled MG, but very little ^{14}C-labeled 1,2(2,3)-DG was seen. The time course for the appearance of ^{3}H-labeled partial glycerides was different. In all experiments and at all time intervals investigated the proportion of ^{3}H found as 1,2(2,3)-DG exceeded that found as MG. Therefore, not only the initial lipolytic activity against TG, but also the further lipolysis of 1,2(2,3)-DG formed, is thus influenced by the presence of 20:4 in the molecule.

In rat intestine, 20:4 is esterified mainly to the *sn*-3 and to some extent to the *sn*-2 positions of TG during fat absorption (8). In view of the relative preference of lipoprotein lipase for position 1, 20:4 would then be less susceptible to the initial attack of this lipase. Although 18:2 is preferentially esterified at the *sn*-2 rather than the *sn*-1 position of chylomicron TG (21), more of the 18:2 than of the 20:4 may be located at the *sn*-1 position. This does not, however, immediately explain the different rates of hydrolysis observed with 18:2 and 20:4 TG. Rather our data, as well as those of Morley and Kuksis (11) and of Ridgway and Dolphin (12), would be compatible with the idea that the presence of 20:4 at position 3 or 2 in the TG molecule

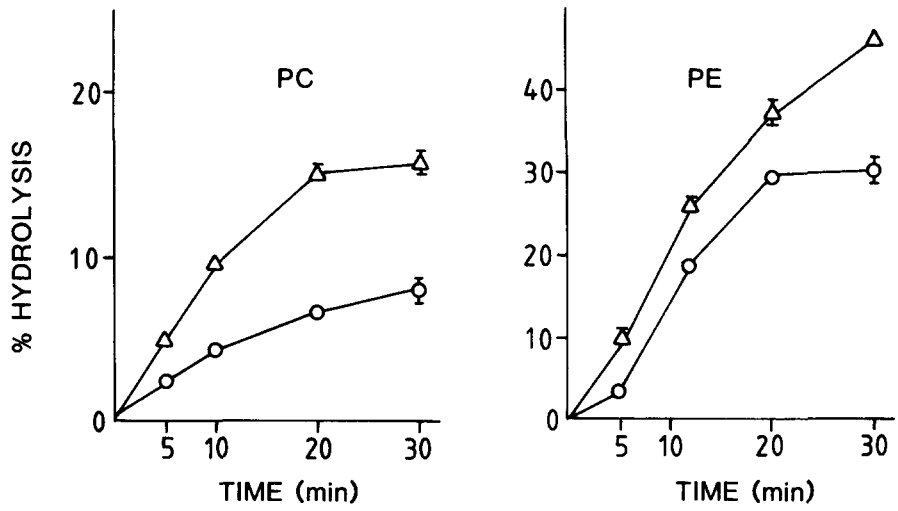

Fig. 3. Hydrolysis of chylomicron phospholipids with bovine milk lipoprotein lipase. Time course for the hydrolysis of PC (left) and PE (right) during incubation of cream chylomicrons with milk lipoprotein lipase in the presence of 20% serum at pH 7.4. Data are from the same experiment as Fig. 1B. Values are expressed as percent hydrolysis and are means ± SEM from three observations with the same batch of chylomicrons; (○—○) [^{3}H]20:4; (△—△) [^{14}C]18:2.

reduces the rate of hydrolysis of the preferred *sn*-1 or *sn*-3 acyl group. In addition, the data suggest that the presence of 20:4 in the 1,2(2,3)-DG formed may have a still more pronounced effect on the further hydrolysis of this substrate, which is very rapid in case of C_{16} and C_{18} DG containing saturated, mono- or polyunsaturated fatty acids (22). Whether the low rate of hydrolysis of TG and 1,2(2,3)-DG containing 20:4 by lipoprotein lipase is due to acyl specificity, to the physical state of these acylglycerols at the surface of the lipoprotein particle (23, 24), or to an unfavorable distribution of these molecular species between the interior and the surface of the particles remains to be established.

Hepatic lipase has a broad specificity and hydrolyzes TG, DG, MG, and the *sn*-1 ester bond of DG and phospholipids (25–29). It also has stereospecific properties similar to those of lipoprotein lipase (30). The role of the enzyme in the metabolism of various substrates in vivo is,

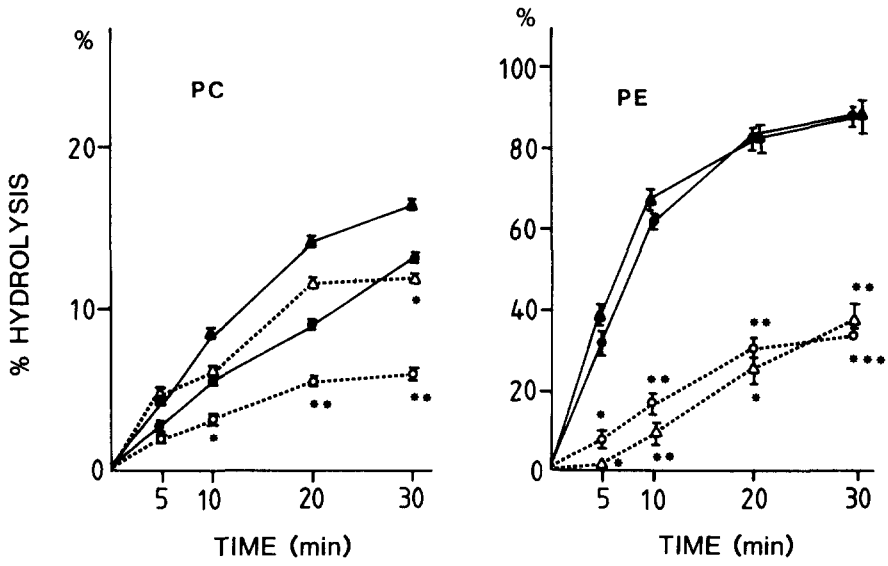

Fig. 4. Effect of anti-hepatic lipase on the hydrolysis of chylomicron phospholipids with postheparin plasma. Values are expressed as percent hydrolysis of PC and are means ± SEM of three observations with the same batch of chylomicrons. The chyle was obtained from the experiments shown in Fig. 2B where the animals were fed cream (PC left, PE right); (●—●) ^{3}H, control serum; (▲—▲) ^{14}C, control serum; (○---○) ^{3}H, anti-hepatic lipase; (△---△) ^{14}C, anti-hepatic lipase. The statistical significance of the inhibition with anti-hepatic lipase is indicated as follows: $^*P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$.

however, still uncertain. Jansen, van Tol, and Hülsmann (31) and Kuusi, Kinnunen, and Nikkilä (32) observed an increase in rat plasma HDL phospholipids after blocking the enzyme in vivo with specific antiserum, indicating that HDL_2 phospholipids may be a physiological substrate for the enzyme. PE is a preferred substrate for the enzyme in vitro (28, 29) and occurs in significant amounts in chylomicrons (5, 33). The rapid clearance of chylomicron and HDL PE was found to be blocked by anti-hepatic lipase in vivo (33), indicating that PE is also a physiological substrate for the enzyme. In addition, hepatic lipase may hydrolyze MG present at the surface of chylomicron remnants during the active clearance of chylomicron TG by lipoprotein lipase (34–36), although this role has so far not been demonstrated in the intact animal. In vitro, chylomicron TG and chylomicron remnant TG are poor substrates for hepatic lipase. Yet, some authors have been able to demonstrate a rise in the levels of IDL and VLDL levels in rat (37, 38) and monkey (39) after the administration of antiserum to hepatic lipase, whereas others found no increase in TG (31, 32) or delayed clearance of chylomicron TG (33). In humans deficient in hepatic lipase, increases of LDL and HDL TG levels have been observed (40).

The finding that a part of the hydrolysis of ^{3}H-labeled TG with postheparin plasma containing both lipoprotein lipase and hepatic lipase could be blocked by anti-hepatic lipase suggests that hepatic lipase might have a role in the metabolism of remnant TG species containing polyunsaturated C_{20} fatty acids. The data also support the possibility that 20:4-containing 1,2(2,3)-DG is a substrate for hepatic lipase since it accumulated in experiments with postheparin plasma when anti-hepatic lipase was added. It is, however, important to stress that the experiments with postheparin plasma were designed to test the integrated function of the two lipases. The effects of hepatic lipase inhibition may thus be secondary, i.e., the removal of phospholipids and partial glycerides from the remnant surface might make the 20:4-containing TG more susceptible to lipoprotein lipase. Further studies of the action of pure hepatic lipase on remnants containing [^{3}H]20:4 are therefore necessary to clarify whether the lipase hydrolyzes 20:4-containing remnant TG and 1,2(2,3)-DG, or only facilitates the action of lipoprotein lipase on these substrates.

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