

# Intralipid administration induces a lipoprotein lipase-like activity in the livers of starved adult rats

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The administration of Intralipid to starved adult rats induces the appearance of lipoprotein lipase (LPL)-like activity in the liver, whereas the so-called hepatic triacylglycerol lipase is unaffected. This LPL-like activity is eluted by 1.5 M-NaCl from heparin–Sephacrose columns. This partially purified fraction is inhibited by 1.0 M-NaCl (91%) and by 1.0 mg of protamine sulphate/ml (79%), whereas it is stimulated 69-fold by the presence of 8.0 µg of apolipoprotein C-II/ml and inhibited by anti-LPL antibodies. We conclude that Intralipid administration induces the appearance of LPL activity in livers of starved adult rats. Its possible origin is discussed.

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## INTRODUCTION

Lipoprotein lipase (LPL; EC 3.1.1.34) is an extracellular enzyme that is physiologically active at the luminal surface of the capillary endothelia of most extrahepatic tissues, where it hydrolyses triacylglycerols in chylomicra and very-low-density lipoproteins, allowing the tissues to use the hydrolytic products (see Cryer, 1981). LPL requires the presence of serum or apo C-II for its maximal activity, and is inhibited by the presence of high NaCl or protamine sulphate concentrations. Typically it is eluted with high NaCl concentrations from heparin–Sephacrose columns (see Olivecrona & Bengtsson, 1984).

Liver is the main source of the endogenous triacylglycerols which are exported as very-low-density lipoproteins. In agreement with this role of liver as an exporter of these lipoproteins, it has been shown that this tissue normally lacks LPL activity, but contains the so-called hepatic triacylglycerol lipase (H-TAGL) which, like LPL, is releasable by heparin (LaRosa *et al.*, 1970; Assmann *et al.*, 1973; Ehnholm *et al.*, 1975; Waite *et al.*, 1978; Jensen & Bensadoun, 1981). Its properties are, however, quite different: it is insensitive to high salt or protamine sulphate concentrations, and it does not require apo C-II for maximal activity. It also binds to heparin–Sephacrose columns, but is eluted with lower NaCl concentrations. Both enzymes, LPL and H-TAGL, are immunologically differentiated too (see Kinnunen, 1984). The physiological role of H-TAGL has not been established yet, but several lines of evidence show that it could act by hydrolysing the excess of phospholipids and triacylglycerol on high-density and intermediate-density lipoproteins (Kuusi *et al.*, 1979; Groot *et al.*, 1981; Landin *et al.*, 1984).

In the past 8 years, however, several laboratories have reported the occurrence of a LPL-like activity in livers from chicken (Bensadoun & Koh, 1977; Jensen *et al.*, 1980), mice (Paterniti *et al.*, 1983; Masuno *et al.*, 1984; Olivecrona *et al.*, 1985), cod and rainbow trout (Black *et al.*, 1983) and Chang liver cells (Ogata & Hirasawa, 1982). We have reported the appearance of a LPL-like activity in livers from newborn rats (Llobera *et al.*, 1979; Ramírez *et al.*, 1983a). This activity is increased by star-

vation (Grinberg *et al.*, 1985). Also Chajek *et al.* (1977) described NaCl-sensitive lipolytic activity in newborn-rat livers. In adult rats Testar *et al.* (1985) have shown that starvation for 24 h induces the appearance of the same LPL-like activity in livers from 21-day-pregnant rats. Also Knobler *et al.* (1984) detected LPL-like activity in livers from cholera-toxin-treated adult rats.

Because most of those papers lack an appropriate characterization, and we have suggested that the appearance of LPL-like activity in liver could be related to substrate availability combined with a high energy demand by the liver (Ramírez *et al.*, 1983a), we have now studied and characterized the induction of LPL activity in livers by Intralipid injection in starved rats. A preliminary report has been published (Vilaró *et al.*, 1985).

## MATERIALS AND METHODS

### Materials

Glycerol trioleate, egg yolk phosphatidylcholine, bovine serum albumin (essentially fatty acid-free), Tris, protamine sulphate (salmine) and goat IgG were purchased from Sigma Chemical Co., St. Louis, MO, U.S.A. Glycerol tri[1-<sup>14</sup>C]oleate was obtained from Amersham International, Amersham, Bucks., U.K. Heparin–Sephacrose CL-6B was purchased from Pharmacia PL Biochemicals, Uppsala, Sweden. Intralipid (20%) was generously given by Kabi Vitrum, Stockholm, Sweden. Human apo C-II and C-III<sub>2</sub> were generously given by Dr. M. Huff, University of Western Ontario, London, Ont. S, Canada. Anti-(rat heart LPL) goat IgG was generously given by Dr. M. C. Schotz, V.A. Hospital Wadsworth Center, Los Angeles, CA, U.S.A. All other chemicals and reagents were of the highest quality and purity available from Merck, Darmstadt, Germany.

### Animals and treatment

Male Wistar rats (160–180 g) from our own colony were used throughout the study under controlled conditions. Intralipid (20%); 1.8, 3.5 or 7.0 ml/kg body wt.) was injected into the tail vein of 48 h-starved rats

between 09:00 and 11:00 h. After 10, 30 or 60 min, five animals for each time and dose were killed by decapitation, and blood was collected into receptacles containing EDTA. Plasma was obtained and used for triacylglycerol determination (Ramírez *et al.*, 1983b). Liver and epididymal-adipose-tissue samples were rapidly excised and placed in liquid N<sub>2</sub>. As controls, untreated animals were used and processed simultaneously.

#### Preparation of acetone/diethyl ether-dried powder

Approx. 200 mg of either frozen liver or epididymal adipose tissue was homogenized with 2.0 ml of 0.2 M-Tris/HCl, pH 8.2 for adipose tissue and 8.5 for liver, in a Potter/Elvehjem homogenizer with 20 strokes of a tight-fitting pestle. Acetone/diethyl ether-dried powders of the homogenates were prepared by consecutive precipitation with chilled acetone and ether (Garfinkel & Schotz, 1972). Dried powders were resuspended in 2.0 ml of 5 mM-barbital buffer, pH 7.2 (buffer A), within 1 week of its preparation, and used immediately.

#### LPL assay

A stable emulsion of glycerol tri[1-<sup>14</sup>C]oleate and phosphatidylcholine in glycerol was prepared as described by Nilsson-Ehle & Schotz (1976). This emulsion was used as substrate in the LPL activity assay for both acetone/ether-dried powders and partially purified fractions from heparin-Sephacel CL-6B columns. The assay mixture contained: 2.1 mM-glycerol tri[1-<sup>14</sup>C]oleate (0.16 Ci/mol), 88 µg of phosphatidylcholine/ml, 1.5 M-glycerol, 20 mM-NaCl, 0.12 mM-albumin, 6% (v/v) rat plasma (pre-heated 10 min at 60 °C), 27 mM-Tris/HCl, pH 8.2 and 0.2–2.0 mg of sample protein in a final volume of 0.3 ml. The incubation was carried out at 37 °C for 30 min with constant shaking. The reaction was terminated by addition of 3.5 ml of methanol/chloroform/heptane (141/125/100, by vol.) (Belfrage & Vaughan, 1969). Oleate was extracted by the addition of 1.0 ml of 0.1 M-borate/carbonate buffer, pH 10.5, and 1.0 ml samples of the supernatant were counted for radioactivity. Samples and blanks were always assayed in duplicates. Proper corrections were made for the counting efficiency, and the activity was finally expressed as µmol of oleate released/h per g of tissue or nmol of oleate released/h per ml for the column fractions.

When indicated, protamine sulphate, NaCl or apo C-III<sub>2</sub> were included in the assay mixture at the concentrations shown. The final volume and the concentrations of other components were held constant. Also, when the effect of the presence of plasma or the effect of different amounts of plasma or apo C-II were tested, the final protein concentration was adjusted with 7% albumin in 0.2 M-Tris/HCl, pH 8.2.

#### H-TAGL assay

For H-TAGL assay, the substrate emulsion of glycerol tri[1-<sup>14</sup>C]oleate in gum arabic was prepared fresh every day by the procedure described by Ehnholm *et al.* (1975). The incubation mixture contained 4.0 mM-glycerol tri[1-<sup>14</sup>C]oleate (0.15 Ci/mol), 1.25 mg of gum arabic/ml, 0.75 M-NaCl, 0.45 mM-albumin, 50 mM-Tris/HCl, pH 8.5, and 0.05–0.5 mg of sample protein, in a final volume of 0.2 ml. The incubation was carried out at 37 °C with constant shaking. The reaction was stopped and the oleate produced was extracted and counted for radioactivity as for LPL. Blanks and samples were always

assayed in duplicate, and the activity is expressed in the same units as LPL.

#### Heparin-Sephacel affinity chromatography

Approx. 4 g of acetone/ether-dried powder was prepared as indicated above, from liver of rats injected with 7.0 ml of Intralipid (20%)/kg body wt. 60 min before being killed, and resuspended in buffer A containing 0.5 M-NaCl to a final concentration of 50 mg of powder/ml. It was centrifuged for 30 min at 30000 g at 4 °C, and the clear supernatant was applied to a heparin-Sephacel CL-6B column (1 cm × 4 cm) equilibrated with buffer A/0.5 M-NaCl. The column was then washed with 50 ml of the equilibration buffer. Bound material was eluted first with 38 ml of buffer A/0.9 M-NaCl and then with 35 ml of buffer A/1.5 M-NaCl. Eluted fractions (1.5 ml) were collected, and albumin was added to a final concentration of 1.0 mg/ml to a sample of each. These fractions were kept at –70 °C until they were assayed for lipolytic activities. All steps were performed at 4 °C, and the whole process did not take longer than 6 h. Another sample from each fraction was used for protein determination (Zak & Cohen, 1961).

#### Immunotitration

The effect of anti-LPL IgG was studied on the activities eluted from heparin-Sephacel CL-6B columns. Before the experiments, either control or anti-LPL IgG was dialysed against 5 mM-phosphate/15 mM-NaCl, pH 7.5, and then centrifuged (1700 g, 10 min) to remove any insoluble material. In a final volume of 0.1 ml, 0.05 ml samples were incubated with increasing amounts of control or anti-LPL IgG for 10 min at 30 °C. Then assays were initiated by the addition of 0.1 ml of buffer A and 0.1 ml of the substrate for LPL determination. The concentrations of the assay components were the same as indicated above, except that the glycerol tri[1-<sup>14</sup>C]oleate specific radioactivity was increased to 0.35 Ci/mol.

## RESULTS

### Plasma triacylglycerol concentrations

Intravenous Intralipid administration produced a tremendous increase in circulating triacylglycerol (Table 1), which was proportional to the dose administered. The

**Table 1. Plasma triacylglycerol concentrations after Intralipid administration to starved adult rats**

At 10, 30 or 60 min after administration of 1.8, 3.5 or 7.0 ml of Intralipid/kg to 48 h-starved adult rats, plasma triacylglycerol concentrations were determined. Each value represents the mean ± S.E.M. for five animals. Statistical comparisons (by unpaired *t* test) with untreated-animal values (0.9 ± 0.2 mM, *n* = 5): \**P* < 0.05; \*\*\**P* < 0.001.

Intralipid dose (ml/kg)	Time (min)...	Plasma triacylglycerol (mM)		
		10	30	60
1.8		19.5 ± 2.8***	6.6 ± 0.9***	0.8 ± 0.2
3.5		23.4 ± 1.8***	15.7 ± 0.7***	7.1 ± 2.4*
7.0		39.7 ± 1.9***	34.0 ± 2.1***	14.9 ± 2.0***

**Table 2. Lipolytic activities in tissues after Intralipid administration to starved adult rats**

The same animals as in Table 1 were used to determine LPL activity in livers and adipose tissue and H-TAGL in liver. Each value represents the mean  $\pm$  S.E.M. for five animals. Statistical comparisons (by unpaired *t* test) with untreated-animal values (adipose tissue LPL,  $1.78 \pm 0.13$   $\mu$ mol/h per g; liver H-TAGL,  $2.58 \pm 0.36$   $\mu$ mol/h per g; liver LPL,  $1.32 \pm 0.21$   $\mu$ mol/h per g; *n* = 5): \**P* < 0.05; \*\**P* < 0.01.

Intralipid dose (ml/kg)	Enzyme activities ( $\mu$ mol/h per g of tissue)								
	Adipose tissue			Liver					
	LPL			H-TAGL			LPL-like		
Time (min)...	10	30	60	10	30	60	10	30	60
1.8	$1.93 \pm 0.44$	$1.88 \pm 0.39$	$2.22 \pm 0.39$	$3.37 \pm 0.48$	$6.79 \pm 2.25$	$3.55 \pm 0.82$	$1.28 \pm 0.35$	$3.35 \pm 0.58^{**}$	$2.35 \pm 0.23$
3.5	$1.49 \pm 0.20$	$1.70 \pm 0.21$	$2.10 \pm 0.44$	$4.02 \pm 2.25$	$3.90 \pm 0.78$	$4.51 \pm 0.75$	$1.65 \pm 0.16$	$2.72 \pm 0.32^*$	$2.74 \pm 0.60^*$
7.0	$2.53 \pm 0.47$	$2.30 \pm 0.45$	$2.97 \pm 0.64$	$7.10 \pm 1.82$	$2.36 \pm 0.40$	$3.62 \pm 0.49$	$2.77 \pm 0.64^*$	$2.61 \pm 0.21^*$	$3.04 \pm 0.25^{**}$

highest values detected, 10 min after the administration of 7.0 ml of Intralipid/kg, were 40 times basal values. The increase was followed by a rapid decrease, and basal values were reached again 60 min after administration of the lower dose, but not after the 3.5 or 7.0 ml/kg doses.

#### Tissue lipolytic activities

The changes in plasma triacylglycerol concentrations did not affect epididymal-adipose-tissue LPL or H-TAGL activities throughout the period of time studied (Table 2). The same samples of liver used for H-TAGL activity were assayed, in the presence of serum, for lipolytic activity with a stable glycerol-based emulsion normally used for LPL activity determination (Nilsson-Ehle & Schotz, 1976) and used for the LPL assay in adipose tissue. This hepatic-LPL-like activity was transitory, but increased significantly after 30 min administration of the lower dose (1.8 ml/kg). At the intermediate dose (3.5 ml/kg), LPL-like activity remained high at 60 min after injection. When 7.0 ml of Intralipid/kg was given, for 10, 30 or 60 min, the LPL-like activity was significantly higher than in controls.

Livers with the highest activity values were pooled and the inhibitory characteristics were determined. The presence of 1.0 M-NaCl produced  $75 \pm 2\%$  (*n* = 4) inhibition of the LPL-like activity, 1.5 mg of protamine sulphate/ml caused a  $65 \pm 2\%$  (*n* = 4) inhibition, and the absence of plasma resulted in a fall of  $52 \pm 0.3\%$  (*n* = 4) in the LPL-like activity.

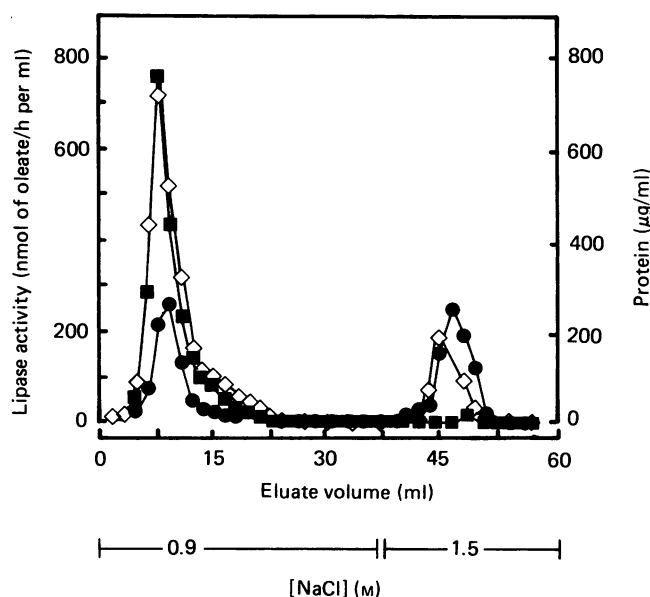
The possible appearance of LPL activity in plasma acetone/ether-dried powders from Intralipid-treated animals was studied in those rats with highest LPL-like activity in liver (*n* = 10), and compared with control animals. In both groups, the activity was completely undetectable (results not shown).

#### Heparin-Sepharose CL-6B affinity chromatography

To characterize further this LPL-like activity that appeared in livers on Intralipid administration to starved rats, its elution profile from heparin-Sepharose CL-6B columns was studied.

Acetone/ether-dried powders of livers from starved rats that had received 7.0 ml of Intralipid/kg 1 h before death were resuspended and centrifuged at 30000 *g* for 30 min. The clear supernatant, containing 66% and 50% of the initial H-TAGL and LPL-like activities respectively,

was applied to the column. Over 98% of the applied protein was recovered in the unbound fraction (Table 3). Also a substantial amount of H-TAGL activity was recovered in that fraction. The column was then washed with buffer A containing 0.9 M-NaCl to eluate the H-TAGL activity. We have used this NaCl concentration instead of the usual 0.75 M because we previously found



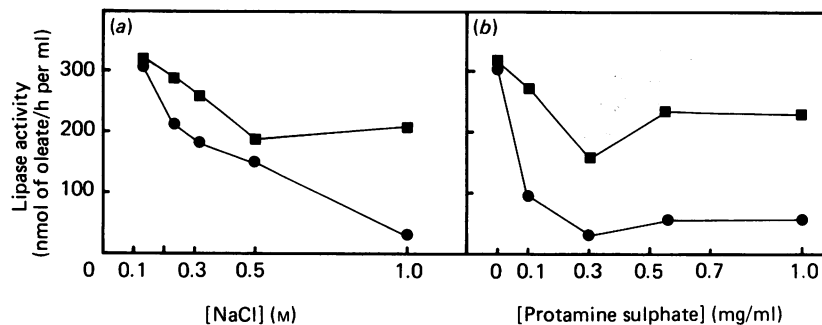
**Fig. 1. Heparin-Sepharose affinity chromatography of soluble liver acetone/ether-dried powders from Intralipid-treated animals**

Liver acetone/ether-dried powders (1.5 g) were prepared from rats which had received 7.0 ml of Intralipid/kg 60 min before death. Resuspended powders were centrifuged, and 28 ml of the supernatant was applied to the column. Then it was washed with 57 ml of the equilibrating buffer and later eluted first with 0.9 M-NaCl and afterwards with 1.5 M-NaCl at a constant flow rate (0.41 ml/min). Fractions (1.5 ml) were collected and processed as described in the Materials and methods section. In each fraction, protein ( $\diamond$ ), H-TAGL activity ( $\blacksquare$ ) and LPL activity ( $\bullet$ ) were determined.

**Table 3. Recovery and purification of lipolytic activities after heparin–Sepharose CL-6B chromatography**

Results are calculated from data shown in Fig. 1.

	H-TAGL						LPL-like			
	Volume (ml)	Protein (mg)	Total activity (nmol of oleate/h)	Specific activity (nmol of oleate/h per mg of protein)	Purification (fold)	Yield (%)	Total activity (nmol of oleate/h)	Specific activity (nmol of oleate/h per mg of protein)	Purification (fold)	Yield (%)
Extract	28.0	350	7014	20.0	(1.0)	(100)	5087	14.5	(1.0)	(100)
Unbound fraction	85.0	344	2892	8.4	0.4	41	895	2.6	0.2	18
0.9 M-NaCl	37.5	3.8	3494	902.7	45.1	50	1240	320.6	22.1	24
1.5 M-NaCl	30.0	0.8	0	0.0	0.0	0	1018	1219.9	84.0	20

**Fig. 2. Effects of NaCl and protamine sulphate on lipolytic activities eluted with 0.9 M- and 1.5 M-NaCl from heparin–Sepharose columns**

Fractions 6–10 (peak I) (■) and 27–33 (peak II) (●) were pooled. By using the LPL assay system, the effects of increasing NaCl (a) or protamine sulphate (b) concentrations were studied.

that the elution of H-TAGL at 0.75 M-NaCl is not complete under our conditions, and part of the activity could be eluted afterwards with 1.5 M-NaCl. However, when the first elution step was done with 0.9 M-NaCl, no further H-TAGL activity was recovered (Reina *et al.*, 1985). By this modification we eluted almost 50% of the applied H-TAGL activity (Table 3). No further activity was eluted with higher NaCl concentrations (Fig. 1). The overall recovery was 91%, similar to what we found in livers from control rats (Reina *et al.*, 1985). Also 24% of the activity measurable by the LPL method was released from the columns with 0.9 M-NaCl (Table 3). It most probably represents residual H-TAGL activity which is measurable by the LPL method. In fact, we have found that, in heat-inactivated (10 min at 60 °C) control liver homogenates to which partially purified H-TAGL was added, 25–30% of the total H-TAGL was measurable by the LPL method (results not shown). However, when control livers were assayed for both H-TAGL and LPL activity, the cross-reactivity value was more variable (see Table 2). For that reason, LPL values were never corrected for this cross-reactivity factor.

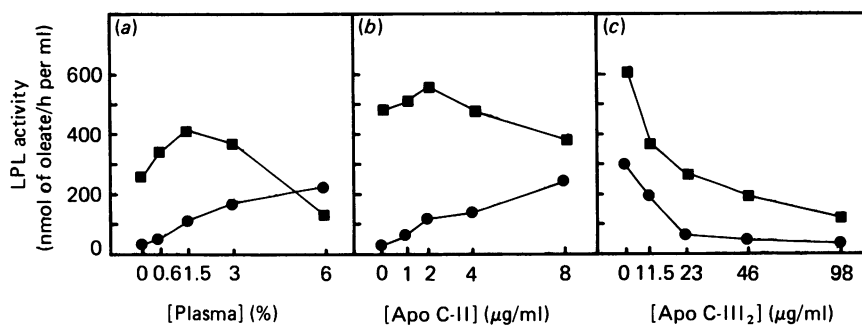
Contrarily to what happens with liver from control animals (Reina *et al.*, 1985), increasing the NaCl concentration to 1.5 M in the elution buffer resolves a second peak of lipolytic activity measurable by the LPL method but not by the H-TAGL method (Fig. 1,

Table 3). It represents 20% of the initially applied activity.

#### Characterization of lipolytic activities eluted from heparin–Sepharose CL-6B

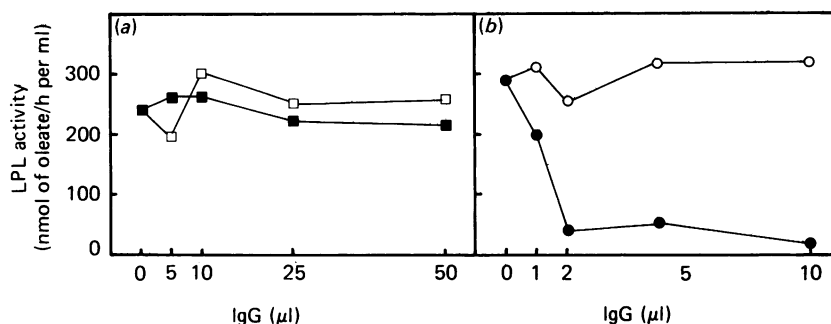
To characterize the lipolytic activities eluted from heparin–Sepharose CL-6B columns, fractions 6–10 (eluted with 0.9 M-NaCl) were pooled (peak I), and fractions 27–33 (eluted with 1.5 M-NaCl) were also pooled (peak II). Activity in peak II was concentrated by passing it through a smaller heparin–Sepharose CL-6B column eluted by the protocol described in the Materials and methods section. The effect of NaCl, protamine sulphate, plasma, apolipoproteins and anti-LPL IgG were studied on material from peaks I and II, both assayed by the LPL method. Peak I was diluted so as to give the same final NaCl concentration (0.12 M) and activity as for peak II.

Fig. 2 shows the effect of increasing concentrations of NaCl (panel a) or protamine sulphate (panel b) in the incubation mixture on peak-I and -II activities. Whereas peak I was relatively insensitive to either NaCl (35% inhibition at 1.0 M) or protamine sulphate (27% inhibition at 1.0 mg/ml), peak II was very sensitive to both agents (91% and 79% inhibition respectively). Fig. 3 shows the plasma (panel a) or apo C-II (panel b) requirements and the effect of increasing concentrations of apo C-III<sub>2</sub> (panel c). Lipolytic activity in peak II



**Fig. 3.** Effects of plasma and apolipoproteins C-II and C-III<sub>2</sub> on lipolytic activities eluted with 0.9 M- and 1.5 M-NaCl from heparin-Sephadex columns

The same peak-I (■) and peak-II (●) fractions as in Fig. 2 were used to study the effect of increasing concentrations of plasma (a), apo C-II (b) or apo C-III<sub>2</sub> (c). The effect of apo C-II was determined in the absence of plasma and the effect of apo C-III<sub>2</sub> in its presence.



**Fig. 4.** Effect of anti-LPL antibodies on lipolytic activities eluted with 0.9 M- and 1.5 M-NaCl from heparin-Sephadex columns

The same pooled fractions as in Figs. 2 and 3 were used to study the effect of increasing amounts of anti-LPL IgG. Samples of peak I (a) or peak II (b) were incubated with the indicated amounts of control (□, ○) or anti-LPL (■, ●) IgG (5.6 mg/ml) for 10 min at 30 °C and immediately assayed by the LPL method.

requires the presence of either plasma or apo C-II for its maximal activity, and is stimulated 69-fold by the presence of 8 µg of apo C-II/ml. Contrarily, peak I was insensitive to the presence of apo C-II and was inhibited 50% by 6% plasma. Both peak-I and -II activities were inhibited by the presence of apo C-III<sub>2</sub> (Fig. 3c).

Finally, we studied the effect of goat IgG raised against rat heart LPL. Whereas peak I was insensitive to both control and anti-LPL IgG (Fig. 4a), peak II was specifically inhibited by antibodies to LPL (94% inhibition by 56 µg of IgG; Fig. 4b).

## DISCUSSION

In this work we have found that intravenous administration of Intralipid to starved rats promotes the appearance of a lipolytic activity in liver which is measurable by the LPL method, and is inhibited by NaCl and protamine sulphate and is plasma-dependent. All of these are well-known characteristics of extrahepatic LPL (Korn, 1962; LaRosa *et al.*, 1970; Assmann *et al.*, 1973; Fielding & Fielding, 1976; Chung & Scanu, 1977; Vainio *et al.*, 1983). In fact, we had previously found that in different physiological situations a LPL-like activity with similar properties appears in rat liver (Llobera *et al.*, 1979; Ramirez *et al.*, 1983a; Grinberg *et al.*, 1985; Testar *et al.*, 1985).

To characterize this activity properly as LPL, samples passed through heparin-Sephadex affinity columns were eluted at different NaCl concentrations, and two activity peaks were clearly resolved. The first was eluted at 0.9 M-NaCl (Fig. 1) and showed the classical properties of the so-called H-TAGL (Kinnunen, 1984): serum inhibition (Fig. 3), insensitivity to NaCl and protamine sulphate (Fig. 2), and no inhibition by antibodies to LPL (Fig. 4). This activity eluted with 0.9 M-NaCl is inhibited by increasing concentrations of apo C-III<sub>2</sub>, as has been shown for the H-TAGL purified from human post-heparin plasma (Kinnunen & Ehnholm, 1976; Jahn *et al.*, 1982). The second activity peak fulfilled the requirements to be called 'LPL': elution at high NaCl concentration from heparin-Sephadex columns (Fig. 1), dependence on serum or apo C-II (Fig. 3), inhibition by NaCl and protamine sulphate (Fig. 2) and inhibition by antibodies to rat heart LPL (Fig. 4). Also, as shown in other studies (Twu *et al.*, 1975, 1976; Chung & Scanu, 1977; Holdsworth *et al.*, 1985), this activity is inhibited by apo C-III<sub>2</sub> (Fig. 3). So we conclude that Intralipid administration to 48 h-starved rats induces the rapid appearance of LPL activity in liver. Previously, LPL activity in normal adult chicken has also been characterized (Bensadoun & Koh, 1977).

The origin of this hepatic LPL activity is unclear. It has been shown by Wallinder *et al.* (1979, 1984) that liver

readily takes up intravenously administered  $^{125}\text{I}$ -labelled LPL and, because degradation is quite slow (half-life near 1 h), its activity could be detected in liver samples even 1 h after the bolus administration. Also, Knobler *et al.* (1984) found LPL activity in livers from cholera-toxin-treated rats. They discussed the possibility that the extrahepatic tissues could be the origin, because they also found LPL activity in plasma from these animals. We think that in our conditions an extrahepatic origin for LPL activity in liver is unlikely, because we have not found LPL activity in plasma acetone/ether-dried powders from Intralipid-treated rats. On the other hand, a hepatic origin of this LPL activity in liver is also reasonable. Jensen *et al.* (1980) demonstrated that cultured chicken hepatocytes synthesize and secrete LPL. Also, the presence of LPL-like material, immunoprecipitable by antibodies to LPL, in livers of *cld/cld* and normal mice has been demonstrated by Olivecrona *et al.* (1985). The presence of LPL-like material in livers of *cld/cld* mice strongly suggests a hepatic origin of this material because there is extremely low vascular LPL activity in those mutants (Paterniti *et al.*, 1983).

The presence of LPL activity in livers from Intralipid-treated rats could allow this organ, independently of the tissue origin of this activity, to take up circulating triacylglycerols directly. Preliminary results from our laboratory show that, shortly after injection of Intralipid into starved rats, the liver accumulates up to 25% of the administered triacylglycerols. Further experiments are needed to establish clearly both the role of this LPL activity in the observed triacylglycerol accumulation in the liver and the relevance of this phenomenon in situations such as re-feeding with a high-fat diet after starvation.

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