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Regulation of Lipoprotein Lipase by Fasting in Epididymal and Mesenteric Adipocytes of Rats

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ABSTRACT : There are marked variations in the activity of lipoprotein lipase (LPL) among adipose depots. The aim of this study was to compare the mechanisms of 24 h of fasting on LPL regulation between epididymal (EPI) adipocytes and mesenteric (MES) adipocytes in rats. 1-Day fasting consistently decreased activities of heparin-releasable LPL, total extractable LPL and cellular LPL markedly in both EPI and MES fat pads. LPL activity in MES fat pads was relatively lower than in the EPI fat pads. Consistent with data on LPL activity, the levels of expression of LPL mRNA in both nutritional states were lower in MES than EPI adipose tissue and isolated adipocytes. The decreased LPL activity after 1 day of fasting in MES adipocytes was explained mainly by a 50% decrease in the relative abundance of LPL mRNA level and a parallel 50% decrease in relative rate of LPL synthesis. In contrast, fasting of 1 day in EPI adipocytes decreased total LPL activity by 47% but did not affect LPL mRNA level or relative rate of LPL synthesis. A decrease in overall protein synthesis contributed to the decreased LPL activity after 1 day fasting both in EPI and MES adipocytes. In MES adipocytes the decrease in LPL activity, LPL mRNA and LPL synthesis were comparable, but in EPI adipocytes the changes in LPL activity were substantially larger than the changes in LPL mRNA level and LPL synthesis. Therefore, fasting decreased fat cell size, LPL activity, LPL mRNA level and relative rate of LPL synthesis in rats, and these effects were more marked in the MES adipocytes. These results clearly demonstrate the regional variations in the metabolic response of adipose tissue and LPL functions to fasting. (**Key Words :** Adipose Tissue, Adipocytes, Fasting, Lipoprotein Lipase, Rat)

INTRODUCTION

Activity of the adipocyte enzyme lipoprotein lipase (LPL; EC 3.1.1.34) is an important determinant of the amount of triacylglycerol stored in adipose tissue (Eckel, 1989; Liu et al., 2006; Choi et al., 2007). LPL activity in adipose tissue increases several-fold within a few hours of ingestion of a meal (Eckel, 1989; Cruz and Williamson, 1992) and decreases progressively with fasting (Cryer, 1981; Lien and Jan, 2003; Wu et al., 2003). However, the mechanisms by which fasting decreases LPL activity are not clear.

LPL activity declines within a few hours of fasting in guinea pigs which does not appear to be due to changes in rate of synthesis of LPL protein because several days of fasting are required to reduce abundance of LPL mRNA and/or rate of synthesis of LPL in adipose tissue (Semb and Olivecrona, 1986, 1989). Measurements of LPL mRNA, protein and activity in adipose tissue from rats indicate that fasting reduces specific activity of LPL (Doolittle et al., 1990). However, other studies observe a tight correlation between abundance of LPL mRNA and activity of the enzyme in adipose tissue from rats fasted for 1 to 6 days (Ladu et al., 1991). The decrease in adipose tissue LPL activity during fasting may be due to pre- and/or post-translational mechanisms (Doolittle et al., 1990; Ladu et al., 1991; Oliver and Rogers, 1993; Bergö et al., 1996).

LPL activity varies between adipose tissue depots in rats (Lladó et al., 1999; Panarotto et al., 2000; Sponarova et al., 2005; Tchernof et al., 2006), being highest in internal depots, lowest in subcutaneous tissue and intermediate in mesenteric adipose tissue (Rebuffe-Scrive et al., 1992; Sugden et al., 1993). These regional variations in LPL activity have been correlated with sensitivity to and/or expression of receptors for insulin, glucocorticoids and estrogen (Gray and Wade, 1980; Pedersen, 1992; Rebuffe-Scrive et al., 1992).

In addition to adipocytes, adipose tissues contain large numbers of stromal-vascular cells which when isolated

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develop into preadipocytes which can be induced to differentiate into adipocytes. Both adipocytes and stromal-vascular cell-derived preadipocytes produce LPL. Preadipocytes express LPL mRNA well before they accumulate significant intracellular triacylglygerol (Vannier, 1985). Therefore, variations in LPL activity may be due to differences in the ratio of adipocytes and preadipocytes and differential regulation of expression of LPL in these two cell types. In the present study the abundance of LPL mRNA, the rate of synthesis of LPL protein and the activity of LPL were therefore measured in tissue sections and in isolated adipocytes and preadipocytes from epididymal and mesenteric adipose tissue obtained from fed and fasted rats.

MATERIALS AND METHODS

Materials

All chemicals were obtained from the Aldrich Chemical Company (St. Louis, Mo, USA), unless otherwise noted.

Animal

Male Wister rats weighing between 350 and 450 g were used in this study. Rats were housed individually in hanging cages under controlled conditions at temperatures between 21 and 23°C with a 12:12 h light-dark cycle (08:00-20:00 h light). Rats were provided unrestricted access to rat chow and water in our animal facility for at least 2 week before the experiments to minimize the effects of changes in feeding schedules and diurnal rhythms on data variability. The rats (eight per group) were divided into two groups. Rats (fasted group) were fasted for 1 day by removing rat chow at 09:00 h. Fed rats (fed group) had free access to food and water, but fasted rats had only access to water. Fed and fasted rats were weighed before and at the end of the fasting period. On the day of an experiment, rats were killed by decapitation after light anesthesia in a CO_2-saturated chamber.

Preparation of adipocytes

Isolated fat cells were prepared by minor modification of the Rodbell procedure (1964). EPI and MES adipose tissue fragments were placed in Krebs-Ringer N-2-hydroxyethylpiperazine-N-2-ethan-sulfonic acid buffer [HEPES: 30 mM HEPES, 10 mM HCO_3^-], pH 7.4, containing 4% bovine serum albumin, 5 mM glucose (KRBH-albumin), 100 mM phenylisopropyl adenosine (PIA), and 1 mg/ml collagenase (type I; Worthington Biochemiclas, NJ, USA) at 37°C with shaking at 60 cycles/min. After digestion, isolated fat cells were separated from undigested tissues by filtration through a 250 μm nylon mesh and washed with 37°C KRBH-albumin (without collagenase) four times. The stromal-vascular cells were also obtained by centrifugation of the infranatent below the floating fat cells after collagenase digestion and then washed the digestion mixture with KRBH-albumin to remove the collagenase.

Measurement of fat cell size and number

A sample of each adipose tissue was fixed with osmium, and the number and size of fat cells were determined by the method of Hirsh and Gallian (1968) and Digirolamo et al. (1971). Percentage of lipid in the tissue was extracted using chloroform:methanol according to Folch et al. (1957). Mean fat cell weight (micrograms lipid per cell) was calculated by dividing the lipid weight per gram by the number of fat cells per gram as assessed by the Coulter counter (Coulter Electronics, FL, USA).

Measurement of LPL activity

Activity of LPL releasable by heparin from tissue fragments and activity of total extractable LPL from homogenates containing 5 g/L deoxycholate were measured as previously described (Iverius and Bruzell, 1985; Fried et al., 1990). To measure LPL activity in the isolated fat cells and stromal-vascular cells, incubation medium was removed, and aliquots of cells resuspended without washing to avoid loss of cell-surface LPL in lysis buffer to assess total extractable LPL activity. One unit of LPL activity is defined as the release of 1 μmol free fatty acid in 1 h.

RNA extraction and Northern blotting

Total RNA was extracted from adipose tissue, fat cells and stromal-vascular cells isolated from EPI or MES adipose tissue of rats by minor modification of the method of Chomzczynski and Sacchi (1987). LPL mRNA levels were determined by Northern blotting and laser scanning of autoradiographs. For Northern blotting, total RNA (10 μg) was electrophoresed on 1% agarose gel containing 6% formaldehyde and blotted onto a nylon mesh (Fried and Zechner, 1989). Blots were prehybridized for 8-24 h at 42°C and then hybridized for 24 h at 42°C in the presence of a human LPL cDNA labeled with ^{32}P-deoxycytidine by the random primer method using a kit from Amersham. Blots were washed 3×with 1% SDS and 1 ×SSC for 15 min each at 55°C. The blot was reprobed with cDNA to the 28s ribosomal subunit as a loading control. After exposure of blots to X-ray film, autoradiographs were quantified by scanning laser densitometry (LKB Produkter, Bromma, Sweden).

Analysis of LPL biosynthesis

Aliquots of the EPI or MES adipocytes were incubated for 30 min pulse period in minimal essential medium containing [^{35}S] methionine (100 μCi/ml) and [^{35}S] cysteine

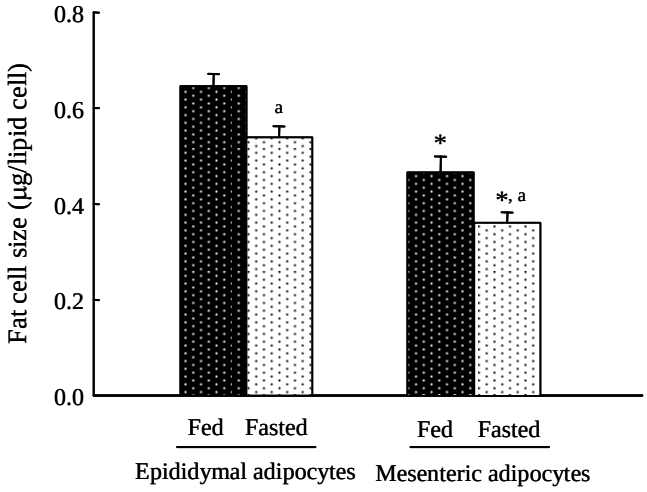

Figure 1. Effects of fasting on EPI and MES fat cell size in rats that were fed or fasted for 1 day. Data means±SEM (n = 8 per group). * Value for MES significantly less than EPI, p<0.05. [a] Value for Fasted significantly less than Fed, p<0.05.

(100 μCi/ml). After homogenization in lysis buffer, incorporation of radioactivity into TCA-precipitable protein was measured, and volumes of homogenates representing equal TCA counts were used for immunoprecipitation of LPL as the method of Freid and Zechner (1989), using a rabbit anti-human milk LPL antiserum (Zechner, 1990). Immunoprecipitated proteins were separated by SDS-PAGE (9%) and detected by fluorography. Scanning laser densitometry of fluorographys was used to quantitate the alterations in LPL biosynthesis. To quantitate relative rates of LPL synthesis (as a percentage of total protein synthesis), the LPL band or the corresponding location in a control immunoprecipitation (nonimmune chicken IgG) was cut out of the dried gels, digested in 30% H_2O_2 (v/v) for 24 h at 60°C and radioactivity was determined by scintillation counting. Rates of total protein synthesis were determined by measuring the incorporation of ^{35}S and TCA precipitable protein.

Statistical analysis

All data are presented as means±SEM where applicable. Individual means were compared by post hoc paired *t* tests or independent *t* test, when ANOVA indicated significant overall effects (p<0.05).

RESULTS

Animals and LPL activity

Rats (initially weighing 400±9.82 g, 4 months old) lost an average of 29±2.38 g after 1 day of fasting, while comparable control (fed group) gained 19±1.50 g during the same time period. Fat cell sizes in two adipose tissue regions were significantly larger in the fed states than in the fasted states as expected (Figure 1). MES, compared with EPI, adipose tissues of the fed and fasted rats exhibited smaller fat cell size.

TE-LPL activities in EPI and MES adipose tissues after 1 day fast decreased to 45% and 40%, and HR-LPL activities decreased to 20% and 18% of the values in fed controls, respectively (Table 1). Irrespective of the nutritional status of the rats (fed or 24 h-fast), activities of HR-LPL and TE-LPL in EPI adipose tissue depot were somewhat higher than those of in the MES adipose tissue depot. To study the distribution of LPL within the adipose tissue, we also used collagenase to digest the extracellular matrix. The adipocytes were then isolated by floatation. LPL activity in EPI and MES adipocytes after 1 day fasting decreased by about 56% and 45% compared with the fed rats, respectively. LPL activity in MES adipocytes was also lower than the activity measured in EPI adipocytes. However, there was no effect of fasting on LPL activities in stromal-vascular cells isolated from both EPI and MES adipose tissues. LPL activity was similar in both stromal-

Table 1. Effects of fasting on LPL activities in EPI and MES adipose tissue, isolated fat cells and stromal-vascular cells from fed and 1 day fasted rats

	EPI		MES	
	Fed	Fasted	Fed	Fasted
	μmol FFA released/(h·10^6 cells)			
Adipose tissues				
TE-LPL	19.49±1.92	8.75±1.02[a]	9.62±1.92*	3.85±0.81*, [a]
HR-LPL	3.45±0.92	0.69±0.08[a]	2.09±0.90*	0.38±0.04*, [a]
Isolated fat cells				
LPL	0.91±0.09	0.51±0.05[a]	0.52±0.05*	0.23±0.04*, [a]
Stromal-vascular cells				
LPL	0.028±0.008	0.023±0.007	0.029±0.009	0.023±0.004

Values are means±SEM (n = 8 per group). Total extractable (TE-) LPL activity was measured in deoxycholate extracts of EPI and MES adipose tissues of rats. Heparin-releasable (HR-) LPL activity was measured in media samples after incubation of both adipose tissue fragments with 5×10^3 units/L heparin for 45 min at 24°C. LPL activities in cellular and stromal-vascular cells were measured in deoxycholate extracts of isolated adipocytes and preadipocytes by collagenase digestion for 1 h at 37°C.

* Value for MES significantly less than EPI, p<0.05. [a] Value for Fasted significantly less than Fed, p<0.05.

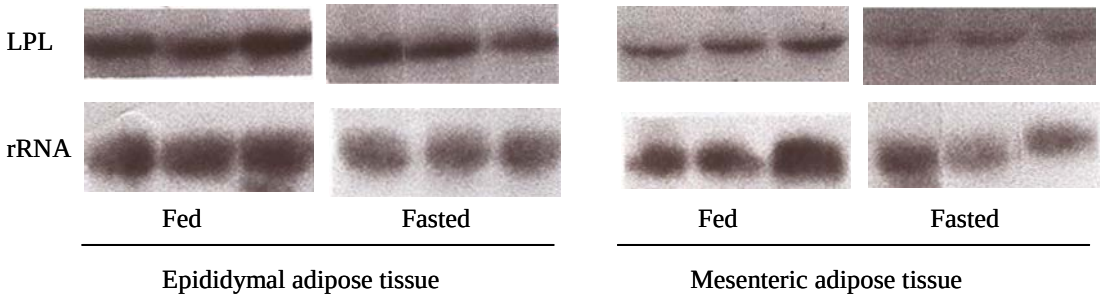

Figure 2. The Levels of LPL mRNA in EPI and MES adipose tissues extracts from fed rats and rats fasted for 1 day. After RNA extraction and electrophoresis, blots were hybridized with cDNA for human LPL (hLPL) and 28s rRNA as a loading control. Each lane represents adipose tissues RNA from an individual rat.

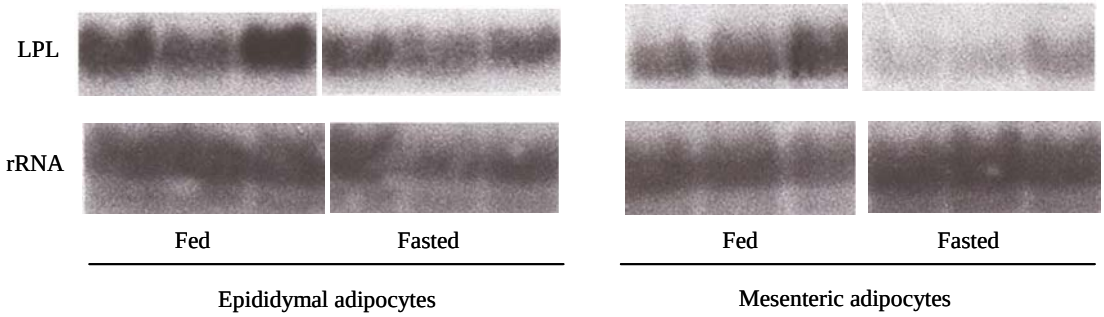

Figure 3. The Levels of LPL mRNA in isolated EPI and MES adipocytes from fed rats and rats fasted for 1 day. Adipocytes were prepared from EPI or MES fat pads. After RNA extraction and electrophoresis, blots were hybridized with cDNA for hLPL and 28s rRNA as a loading control. Each lane represents adipocyte RNA from an individual rat.

Table 2. Northern-blot analysis of the relative abundance of LPL mRNA in adipose tissue, isolated fat cells and stromal-vascular cells from rats fed or fasted for 1 day

	EPI		MES	
	Fed	Fasted	Fed	Fasted
	Arbitary densitometry units			
Adipose tissues				
LPL/rRNA	1.95±0.92	1.88±0.22	0.96±0.12*	0.39±0.11*, [a]
Isolated fat cells				
LPL/ rRNA	1.44±0.31	1.21±0.09	0.89±0.08*	0.29±0.07*, [a]
Stromal-vascular cells				
LPL/ rRNA	0.46±0.05	0.49±0.07	0.43±0.09	0.42±0.04

Values in arbitary units derived from laser densitometry of autoradiograms of Northern blots. Data are means±SEM (n = 8 per group). Northern blots were verified to have equal loading of total RNA (10 μg) per lane by visual inspection of photos of ethidium bromide stained gels.
* Value for MES significantly less than EPI, $p<0.05$. [a] Value for Fasted significantly less than Fed, $p<0.05$.

vascular cells. Furthermore, LPL activity was detected in stromal-vascular cells in a low activity (Table 1).

Effects of fasting on LPL mRNA levels

To determine whether decreased LPL activity in both EPI and MES fat pads after 1 day fasting in rats was associated with changes in levels of LPL mRNA, Northern analyses of RNA were performed (Figures 2 and 3). LPL mRNA levels were also expressed as the ratio LPL/28s ribosomal RNA (rRNA) peak in adipose tissue, isolated fat cells and stromal-vascular cells from autoradiographys (Table 2). In extracts of total MES adipose tissue, the levels of LPL mRNA were decreased by 47% after 1 day of fasting (Figure 2). However, in EPI adipose tissue, fasting did not affect LPL mRNA levels. Levels of LPL mRNA in the fed and fasted rats were slightly higher in EPI than in MES adipose tissue. In EPI adipocytes LPL mRNA levels were also not affected by fasting, whereas it was decreased in MES adipocytes (Figure 3). There was no significant difference in LPL mRNA levels between EPI and MES adipocytes in fed states. However, LPL mRNA levels were significantly higher in EPI compared with MES adipocytes of fasted rats. In addition, densitometry of Northern blots also showed a significant depot differences in rats (Table 2).

In addition to adipocytes, adipose tissue contains other cell types, including preadipocytes. Therefore, LPL mRNA

Table 3. Effects of fasting on EPI and MES adipocytes total protein synthesis and LPL synthesis in rats that were fed or fasted for 1 day

	EPI		MES	
	Total protein synthesis ($mBq/10^8$ cells. 15 min)	Relative LPL synthesis (arbitrary units)	Total protein synthesis ($mBq/10^8$ cells. 15 min)	Relative LPL synthesis (arbitrary units)
Fed	175±32	952±250	143±46*	868±254*
Fasted	99±29[a]	914±263	51±14*, [a]	235±97*, [a]

Values are means±SEM (n = 3 per group). Prepared adipocytes were pulse-labeled with 3.7 GBq/L ^{35}S-methionine and cysteine (Expre^{35}S label, Dupont NEW), and incorporation into total proteins was measured by trichloroacetate (TCA) precipitation. LPL was immunoprecipitated from adipocytes containing 10^6 CPM of TCA-precipitable protein. Relative rates of LPL synthesis were determined by densitometric scanning of fluorographs of SDS-PAGE gels of LPL immunoprecipitation.

* Value for MES significantly less than EPI, $p<0.05$. [a] Value for Fasted significantly less than Fed, $p<0.05$.

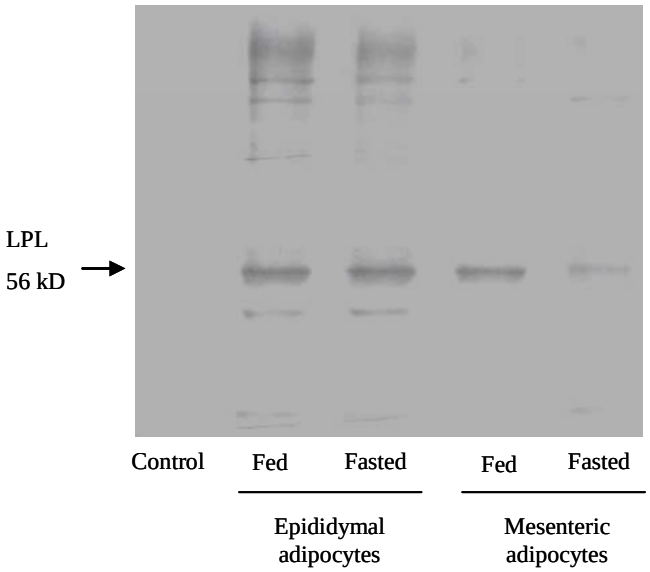

Figure 4. Effects of fasting on EPI and MES adipocytes total protein synthesis and LPL synthesis in rats that were fed or fasted for 1 day. EPI and MES adipocytes were isolated from 4-mo-old (-450 g) rats that had been fasted overnight (1 d), or fed rats that had free access to food until killing at 11:00 h. After 30 min preincubation with 3.5 nmol/L insulin, cells were pulse-labeled with ^{35}S-expression (^{35}S-methionine and cysteine) label for 30 min and immediately homogenized. Equal TCA-precipitable counts were used for immunoprecipitation of cellular LPL from fed and fasted rat adipocytes, followed by SDS-PAGE. The figure shows a fluorography of one experiment responsive of the four performed. Control (immunoprecipitation performed with nonimmune immunoglobulin G (IgG)).

expression in RNA extracts of the stromal-vascular cell fraction was also assessed. LPL mRNA was present in the stromal-vascular cells, but its expression did not change with fasting. LPL mRNA levels were not difference between EPI and MES stromal-vascular cell fractions.

LPL synthesis

To confirm that changes in LPL mRNA were paralleled by changes in LPL synthesis, biosynthetic labeling and immunoprecipitation were carried out in two different kinds of adipocytes. As shown in Figure 4 and Table 3 fasting for 1 day did not affect rates of LPL synthesis relative to rates of total protein synthesis in EPI adipocytes, but significantly decreased total protein synthesis of EPI adipocytes to 57% of control values. Therefore, the decrease in TE-LPL activity to 45% of control values after 1 day of fasting in EPI adipocytes appeared to be mainly accounted for by the decrease in total protein synthesis. However, in EPI fat pads, the decrease in HR-LPL activity (to 20% of control) was proportionately greater than the decrease in TE-LPL activity and could not be explained at the level of LPL synthesis.

Fasting for 1 day decreased relative rates of LPL synthesis in MES adipocytes to 27% of control (n = 3, paired comparisons to control-fed levels, Table 3). Total MES adipocytes protein synthesis after 1 day of fasting also decreased to 36% (n = 3) of fed controls. Thus, combined effects of decreased total protein synthesis and relative rates of LPL synthesis in MES adipocytes appeared to largely explain the decrease in TE- and HR-LPL activities to 40 and 18% of control values, respectively.

There were no significant differences in relative LPL synthesis and total protein synthesis between EPI and MES adipocytes in fed states. However, relative LPL synthesis and total protein synthesis were significantly higher in EPI than MES adipocytes of fasted groups.

DISCUSSION

Fasting decreases adipose tissue LPL activity, but reports are conflicting on whether this is related to a decline in LPL mRNA level or the rate of LPL synthesis (Semb and Olivecrona, 1986, 1989; Doolittle et al., 1990; Ladu et al., 1991; Oliver and Rogers, 1993; Bergö et al., 1996). Previous studies have analyzed mRNA levels in extracts of total adipose tissue and have not taken into consideration the fact that up to one third of the total RNA is representing preadipocytes. Thus, in this study, the effects of fasting on LPL regulation were examined using rat isolated adipocytes and stromal-vascular cell fractions as well as adipose tissue fragments.

Regional variations in adipose tissue LPL activity have been demonstrated, and the differences closely parallel regional variations in fat cell size (Fried and Kral, 1987; Rebuffe-Scrive et al., 1992). Variations in fat cell size and LPL activity among different anatomical fat depots have

been well documented in the literature. For instance, fat cells in the EPI depots remain larger and have higher LPL activity than MES fat cells (Rebuffe-Scrive et al., 1992). The present results provide a possible molecular basis for these differences by demonstrating variations in the expression of LPL mRNA in EPI and MES adipose tissues and adipocytes of rats.

Numerous previous studies have measured the response of LPL activity to change in nutritional states (Eckel, 1989). In both rats and humans, LPL activity was higher in adipose tissue in the fed state than in the fasted or hypocaloric state (Lee et al., 1998; Sugden et al., 1999; Wu et al., 2003). After 1 day fast, in the current study, TE-LPL activities in EPI and MES adipose tissues decreased, and HR-LPL activity decreased compared with the fed control. In addition, cellular LPL activities in both isolated adipocytes were also significantly higher in the fed states than in the fasted states. However, no changes in LPL activity were observed in either EPI or MES stromal-vascular cells after fasting. The activities of HR-LPL, TE-LPL and cellular LPL were somewhat higher in EPI than MES fat pads. In contrast, LPL activity in stromal-vascular cells did not differ between the two adipose tissue regions. The lower activity of LPL in MES adipose tissue and isolated adipocytes of fed and fasted rats must be considered relative to the smaller size of the fat cells in this depot.

We assessed whether decreases in LPL activity after fasting was also accompanied by changes in proportionate LPL mRNA expression. LPL mRNA levels, relative to rRNA, in total MES adipose RNA was decreased by only 53% after 1day fasting. In addition, collagenase-isolated MES adipocytes LPL mRNA levels were also decreased to 42% after 1day of fed control values. In contrast, the LPL mRNA levels in EPI adipose tissue and adipocytes were not changed by fasting. LPL mRNA is expressed predominantly in adipocytes but also in the stromal-vascular cells, within preadipocytes. To facilitate comparisons of rates of adipocytes LPL synthesis with adipocytes LPL mRNA levels, we assessed mRNA levels out of total RNA extracted from isolated adipocytes and stromal-vascular cell fractions as well as total adipose tissue. Unlike in adipocytes, levels of LPL mRNA were similar in both states of stromal-vascular cells. However, the quantitative contribution of the stromal-vascular cells LPL mRNA to tissue expression is probably minor. Thus measurement of total adipose tissue LPL mRNA levels closely reflects the levels in adipocytes and is adequate for most studies. Adipocytes LPL mRNA levels decreased more markedly than total adipose tissue LPL mRNA levels. This was because stromal-vascular mRNA did not change with fasting. Therefore, the extents of these changes in LPL mRNA levels were underestimated by probing extracts of total adipose tissue RNA.

Because MES adipocytes exhibited smaller fat cell and a less marked response to LPL activity in this study, we also compared the levels of LPL mRNA and rates of LPL synthesis between EPI and MES adipocytes of rats. MES, compared with EPI, adipocytes of 1 day fasted rats exhibited lower levels of LPL activity and LPL mRNA expression. The relative rates of LPL synthesis and total protein synthesis were also significantly lower in MES than in EPI adipocytes of fed and fasted rats. Changes in the rate of LPL synthesis per fat cell during fasting can be due to nonspecific effects on total protein synthesis as well as to alterations in relative rates of LPL synthesis (as a percentage of total protein synthesis). In agreement with earlier studies (Semb and Olivecrona, 1989), we found that total protein synthesis in the two fat depots decreased by fasting and contributed to the decrease in LPL activity. In MES adipocytes, alterations in LPL mRNA expression contributed importantly to the marked decreases in TE-LPL and HR-LPL activities. There was a decline in MES adipocytes LPL mRNA expression and LPL synthesis with 1 day fasting that closely paralleled the decline in LPL activity. This indicates that pre-translational mechanism contributes to the regulation of LPL in MES fat pads after 1 day fasting.

On the other hand, in EPI adipocytes LPL mRNA levels and relative rates of LPL synthesis did not change, and the decline in HR-LPL activity was partially accounted for a decrease in total protein synthesis. The changes of LPL mRNA levels in EPI adipocytes occurred more slowly than the changes in LPL activity. In contrast to these results, Oliver and Rogers (1993) found a 50% decrease in relative LPL synthesis as well as a 19% decrease in total protein synthesis in the EPI adipose tissue of rats fasting for 1 day compared to those that were fed. The use of intact fragments of adipose tissue in that study, compared with the isolated adipocytes in this study, may explain this discrepancy. However, Doolittle et al. (1990) have shown that after a short fast of 12 h, LPL activity was decreased but LPL synthesis was actually increased. This was apparently explained by an increase in LPL degradation. It is expected that short term adaptation of fasting in EPI fat pads will involve mainly post-translational mechanisms. Therefore, the changes of LPL degradation and LPL secretion in EPI adipocytes after 1 day fasting will need further investigation.

In conclusion, we found a differential response of LPL regulation in EPI and MES fat pads in response to two different nutritional states: fed and 24 h of fasting. After 1 day fasting, the decrease in LPL activity of MES adipocytes results from a combination of a specific decrease in LPL mRNA level and LPL synthesis, and a decrease in overall protein synthesis. MES and EPI fat pads from rats differ in responsiveness to effects of fasting on LPL gene expression,

LPL synthesis and LPL activity. These differences could arise by the depot-specific expression of "tissue-specific" regulatory factors, in a manner analogous to differences in LPL regulation in adipose tissue and muscle (Eckel, 1989). Therefore, these data show that the relative importance of pre- and post-translational mechanisms in regulatory adipose tissue LPL activity depends on adipose depots of rats. In addition, these differences in the nutritional regulation of LPL mass play a role in modulating regional deposition of fat in rats.

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