Published in final edited form as:

Lipids. 2009 May ; 44(5): 459–464. doi:10.1007/s11745-009-3285-7.

Plasma lipid transfer enzymes in non-diabetic lean and obese men and women

Faidon Magkos^{1,2}, B. Selma Mohammed¹, and Bettina Mittendorfer^{1,*}

¹ Center for Human Nutrition, Washington University School of Medicine, St. Louis, MO, USA

² Department of Nutrition and Dietetics, Harokopio University, Athens, Greece

Abstract

There are considerable differences in the plasma lipid profile between lean and obese individuals and between men and women. Little, however, is known regarding the effects of obesity and sex on the plasma concentration of enzymes involved in intravascular lipid remodeling. Therefore, we measured the immunoreactive protein mass of lipoprotein lipase (LPL), hepatic lipase (HL), cholesterol-ester transfer protein (CETP) and lecithin-cholesterol acyl transferase (LCAT) in fasting plasma samples from 40 lean and 40 obese non-diabetic men and premenopausal women. Women, compared with men, had ~5% lower plasma LCAT ($p<0.041$), ~35% greater LPL ($p=0.001$) and ~10% greater CETP ($p=0.085$) concentrations. Obese, compared with lean individuals of both sexes, had ~30% greater plasma LCAT ($p<0.001$), ~20% greater CETP ($p<0.001$) and ~20% greater LPL ($p=0.071$) concentrations. Plasma HL concentration was not different in lean men and women. Obesity was associated with increased (by ~50%) plasma HL concentration in men ($p=0.018$) but not in women; consequently, plasma HL concentration was lower in obese women than obese men ($p=0.009$). In addition, there were direct correlations between plasma lipid transfer enzyme concentrations and lipoprotein particle concentrations and sizes. There are considerable differences in basal plasma lipid transfer enzyme concentrations between lean and obese subjects and between men and women, which may be partly responsible for respective differences in the plasma lipid profile.

Keywords

adiposity; sex differences; lipid transport; lipid profile; lipoprotein subclasses

Introduction

Obese subjects and men are at greater risk for cardiovascular disease (CVD) than lean subjects and women, probably because obese individuals and men have a more pro-atherogenic plasma lipid profile than lean subjects and women, respectively [1]. Several mechanisms, including hepatic and intestinal secretion of very low-density lipoproteins (VLDL), high-density lipoproteins (HDL), and possibly also low-density lipoproteins (LDL), intravascular delipidation and remodeling, and final catabolism and removal from the circulation, act in concert to maintain a more or less pro-atherogenic lipid profile [2]. Intravascular remodeling of lipoproteins involves the exchange of core and surface lipids and apolipoproteins, mediated largely by the action of plasma lipid transfer enzymes, i.e., lipoprotein lipase (LPL), hepatic

*Corresponding author: Bettina Mittendorfer, Ph.D, Washington University School of Medicine, Division of Geriatrics & Nutritional Science, 660 South Euclid Avenue; Campus Box 8031, St. Louis, MO 63110; USA, Phone: (314) 362 8450, Fax: (314) 362 8230, E-mail: E-mail: mittendb@wustl.edu.

lipase (HL), cholesterol-ester transfer protein (CETP), and lecithin-cholesterol acyl transferase (LCAT) [3]. Although differences between lean and obese subjects and between men and women in the plasma lipid and lipoprotein profile are well established [1,4], we know little regarding the effects of obesity and sex on the plasma concentration (i.e., the protein mass) of enzymes involved in intravascular lipid remodeling [5,6]. A better understanding of the regulation of plasma lipid transfer enzyme concentrations may be clinically important because the immunoreactive protein mass of these enzymes has recently been shown to be related to CVD risk [7–12]. The relationship between plasma lipid transfer enzyme concentrations and CVD risk could simply be a reflection of their enzymatic actions and consequent changes in plasma lipid profile but likely goes beyond that because their function is not only limited to their catalytic activity, e.g., LPL [13] and HL [14] serve as bridges/ligands that facilitate lipoprotein uptake by various cell types. The purpose of our study therefore was to examine potential differences in basal plasma lipid transfer enzyme concentrations between lean and obese men and women. We studied non-diabetic, healthy, normoglycemic and normotriglyceridemic subjects with normal oral glucose tolerance to avoid potential confounding due to obesity-related metabolic complications [15,16].

Experimental Procedure

Subjects

Eighty subjects between the ages of 18 and 50 years participated in the study: 40 (18 men; 22 premenopausal women) were lean with a body mass index (BMI) between 18.5 and 25 kg/m² and 40 (13 men; 27 premenopausal women) were obese with a BMI between 30 and 45 kg/m². Lean and obese subjects and men and women were matched on age. All subjects were considered to be in good health after completing a medical evaluation, which included a history and physical examination and standard blood tests. Subjects were included in the study if they were free of hypertension (blood pressure < 140/90 mm Hg) and were normoglycemic (plasma glucose concentration < 5.5 mmol/l) and normotriglyceridemic (plasma triglyceride concentration < 1.69 mmol/l); in addition, all obese subjects had normal oral glucose tolerance (plasma glucose concentration 2 h after a 75 g oral glucose challenge < 7.77 mmol/l). None of the subjects satisfied the criteria for metabolic syndrome, and none were smoking or taking medications known to affect glucose or lipid metabolism. Total body fat was determined by using dual-energy X-ray absorptiometry (Delphi-W densitometer, Hologic, Waltham, MA), and total abdominal, intra-abdominal, and subcutaneous abdominal fat areas were determined by magnetic resonance imaging on a 1.5T scanner (Siemens, Iselin, NJ), as previously described [17]. Written informed consent was obtained from all subjects before their participation in the study, which was approved by the Human Studies Committee and the General Clinical Research Center (GCRC) Advisory Committee at Washington University School of Medicine in St. Louis, MO.

Sample collection and analyses

Subjects were instructed to adhere to their regular diet and to refrain from physical activity for a minimum of 3 days before they were admitted to the GCRC where they consumed a standardized meal at ~1930 h, and then fasted (except for water) and rested in bed until fasting venous blood (total volume ~20 ml) was collected in chilled tubes containing sodium EDTA between 0700 and 0800 h the next day. Plasma was separated by centrifugation (3000 rpm for 15 min at 4°C) and stored at –80°C until analyses were performed.

The concentrations of LPL, LCAT and CETP in plasma were determined with commercially available ELISA kits (Daiichi Pure Chemicals, Tokyo, Japan) [18] and plasma HL concentration was determined by using a sandwich ELISA method with monoclonal antibodies

generated against human HL [19]; the assays used for LPL and HL do not cross-react with each other or with pancreatic lipase.

Plasma glucose concentration was determined by using an automated glucose analyzer (YSI 2300 STAT plus, Yellow Spring Instrument Co., Yellow Springs, OH). Total plasma triglyceride, VLDL-triglyceride and HDL-cholesterol concentrations, and plasma concentrations of VLDL, LDL, and HDL particles and average lipoprotein particle sizes (diameter in nm) were determined (LipoScience, Raleigh, NC) by using an AVANCE INCA NMR Chemical Analyzer equipped with a Bruker BioSpin UltraShield super-conducting magnet (Bruker BioSpin, Billerica, MA), as previously described [4].

Statistical analysis

All data sets were normally distributed and are presented as means $\pm$ standard error (SEM). Two-way analysis of variance was used to evaluate the significance of differences between lean and obese subjects and men and women. In a secondary analysis, we compared the LPL, LCAT, and CETP data obtained from a subgroup of 15 men and 15 women who were matched on percent body fat to evaluate the effect of sex independently of differences in body composition between men and women, by using the Student's *t*-test for independent samples. Plasma HL concentration measurements were excluded from this secondary analysis because we detected a significant interaction between sex and obesity on plasma HL concentration. Associations between variables of interest were assessed with Pearson's linear correlation analysis. A *p*-value < 0.05 was considered statistically significant.

Results

Plasma LCAT concentration was ~5% lower and plasma LPL concentration was ~35% higher in women than in men; women also tended to have higher plasma CETP concentration than men (Table 1). Obese subjects had 20–30% higher plasma LCAT and CETP concentrations than lean subjects, irrespective of sex; they also tended to have higher plasma LPL concentration than lean subjects (Table 1).

Plasma HL concentration was not different in lean men and women. Obesity was associated with ~50% higher plasma HL concentration in men but not in women; consequently, obese women had ~30% lower plasma HL concentration than obese men (Table 1).

There were no differences in plasma LPL and CETP concentrations between men and women who were matched on percent body fat (Table 2). However, women had ~20% lower plasma LCAT concentration than men, even when matched on percent body fat (Table 2).

Total body fat (% body weight) correlated positively with plasma LCAT ($r = 0.296$, $p = 0.008$), LPL ($r = 0.473$, $p < 0.001$), and CETP ($r = 0.351$, $p = 0.002$) concentrations, but not with HL concentration ($r = 0.104$, $p = 0.362$). Total abdominal fat (in cm^2) was positively associated with plasma LCAT ($r = 0.451$, $p < 0.001$) and LPL ($r = 0.329$, $p = 0.013$) concentrations, but not with plasma CETP ($r = 0.181$, $p = 0.175$) and HL ($r = 0.201$, $p = 0.131$) concentrations. Intra-abdominal fat (% total abdominal fat) was not significantly associated with plasma lipid transfer enzyme concentrations (all *p*-values > 0.05).

Plasma LCAT concentration was inversely correlated with average HDL particle size. Plasma HL concentration correlated directly with average VLDL particle size and negatively with average LDL particle size (Table 3). There was no relationship between plasma LPL concentration and average VLDL, LDL, or HDL particle sizes. In addition, plasma LCAT concentration was directly correlated with VLDL (all), LDL (total and small), and HDL (total and small and medium) particle concentrations (Table 3). Plasma CETP concentration was directly correlated with the concentrations (total and small) of VLDL, LDL, and HDL in plasma

(Table 3). Plasma HL concentration was directly correlated with the plasma concentration of small LDL and small HDL particles and inversely related to the concentration of large HDL particles. No robust relationships were found between plasma LPL concentration and lipoprotein concentrations (Table 3).

Discussion

We measured the immunoreactive protein mass of plasma lipid transfer enzymes in non-diabetic lean and obese men and women with normal oral glucose tolerance and plasma triglyceride concentrations, and found considerable differences in plasma LPL, CETP, LCAT, and HL concentrations between sexes and between lean and obese subjects.

A few previous studies evaluated the effect of obesity on plasma CETP concentration without considering potential sex differences [5], and measured plasma LPL concentration in men and women without considering the potential role of obesity [6]. Furthermore, both of these studies included subjects with hypertriglyceridemia and of unknown glucose tolerance status, which might have confounded the results [15,16]. We found that obese individuals have higher plasma LPL, CETP, and LCAT concentrations than lean individuals and obese men (but not obese women) also have higher plasma HL concentration than lean men. LPL is the central enzyme in plasma triglyceride hydrolysis, however, LPL also functions as a molecular bridge to enhance lipoprotein uptake into cells via pathways that are independent of its catalytic activity [13]. The absence of robust relationships between the immunoreactive protein mass of LPL and plasma lipoprotein concentrations suggests that the relationship between plasma LPL concentration and CVD risk [7,12] may be related to recently discovered non-catalytic functions of the enzyme, such as tissue binding of VLDL [13] and native and oxidized LDL [20], and the facilitation of monocyte adhesion [21]. CETP primarily carries triglyceride from VLDL in exchange for cholesterol esters from other lipoproteins, especially HDL but also LDL [22,23]. Depletion of core triglyceride makes the VLDL particle smaller and denser, whereas triglyceride enrichment of LDL [24] and HDL [25] eventually leads to the generation of small and dense LDL and small HDL particles. Consistent with this metabolic cascade, we observed positive associations between plasma CETP mass and small VLDL, LDL, and HDL particles.

Differences between men and women in plasma LPL and CETP concentrations (women greater than men) seem to manifest secondary to typical male and female phenotypes (i.e., greater body fat in women than men). This is consistent with the observation that CETP and LPL mRNA is highly expressed in mammalian adipose tissue [26], as well as with the positive relationships found between percent body fat and CETP and LPL concentrations in plasma. On the other hand, sex differences in plasma LCAT and HL concentrations are independent of differences in total body fat accumulation between men and women, and might be considered true sexual dimorphism rather than a secondary phenomenon.

Plasma LCAT concentration was ~30% greater in obese than lean subjects and slightly (~5%) but significantly lower in women than men. Since women have more body fat than men, the difference in plasma LCAT concentration is unlikely to be due to differences in body composition between men and women. Indeed, in our subset of men and women who were matched on percent body fat, plasma LCAT concentration was ~20% lower in women than in men. LCAT is a lipoprotein-associated enzyme responsible for esterifying free cholesterol to cholesterol esters, primarily on the surface of HDL; hydrophobic cholesterol esters then move to the core of HDL thereby maintaining a free-cholesterol gradient from cells to HDL, which is essential for reverse cholesterol transport [9]. Thus, our results are in agreement with the lower high-density lipoprotein (HDL)-cholesterol concentration and smaller HDL particle size in obese compared with lean subjects and in men compared with women [1,4], because LCAT in plasma is mainly associated with HDL particles and large, cholesterol-rich HDL particles

(in lean subjects and women) contain little or no LCAT [27]. Corroborating these observations, we found that plasma LCAT concentration correlated positively with small HDL particles and negatively with average HDL size.

In agreement with an earlier study that measured post-heparin plasma HL activity [5], we found that obesity was associated with increased plasma HL concentration in men but not in women. Although HL in non-heparinized plasma is catalytically mostly inactive [28], the difference in HL protein mass may be important for our understanding of the regulation of lipid metabolism because HL has multiple roles in lipoprotein metabolism and cellular lipid uptake. It hydrolyzes triglycerides and phospholipids present in circulating plasma lipoproteins, including intermediate-density lipoproteins and lipoprotein remnants, LDL, and HDL, resulting in the generation of smaller LDL and HDL particles [14,29], consistent with the observed relationships between plasma HL concentration and small LDL and HDL particles and average particle sizes. Besides its function as lipase, HL serves as a ligand that facilitates lipoprotein uptake by various cell types [14].

In summary, there are considerable differences in the plasma lipid transfer enzyme concentrations between lean and obese subjects and between men and women, which may be partly responsible for respective differences in the plasma lipid profile and CVD risk.

Acknowledgements

This publication was made possible by Grant Number UL1 RR024992 from the National Center for Research Resources (NCRR), a component of the National Institutes of Health (NIH), and NIH Roadmap for Medical Research, by National Institutes of Health grants AR 49869, HD 057796, DK 56341 (Clinical Nutrition Research Unit), RR 00036 (General Clinical Research Center), and grants from the American Heart Association (0365436Z and 0510015Z).

We wish to thank Megan Steward for help in subject recruitment and the study subjects for their participation.

Abbreviations used

VLDL	very low-density lipoprotein
LDL	low-density lipoprotein
HDL	high-density lipoprotein
LPL	lipoprotein lipase
HL	hepatic lipase
CETP	cholesterol-ester transfer protein
LCAT	lecithin-cholesterol acyl transferase
CVD	cardiovascular disease

References

1. National Cholesterol Education Program. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Final Report. *Circulation* 2002;106:3143–3421. [PubMed: 12485966]
2. Betteridge, DJ.; Illingworth, DR.; Shepherd, J. Lipoproteins in Health and Disease. Arnold Publishers; London: 1999.
3. Havel, RJ.; Goldstein, JL.; Brown, MS. Lipoproteins and lipid transport. In: Bondy, PK.; Rosenberg, LE., editors. *Metabolic Control and Disease*. Vol. 8. W. B. Saunders Company; Philadelphia, PA: 1980. p. 393-494.
4. Magkos F, Mohammed BS, Mittendorfer B. Effect of obesity on the plasma lipoprotein subclass profile in normoglycemic and normolipidemic men and women. *Int J Obes (Lond)* 2008;32:1655–1664. [PubMed: 18779822]
5. Arai T, Yamashita S, Hirano K, Sakai N, Kotani K, Fujioka S, Nozaki S, Keno Y, Yamane M, Shinohara E, et al. Increased plasma cholesteryl ester transfer protein in obese subjects. A possible mechanism for the reduction of serum HDL cholesterol levels in obesity. *Arterioscler Thromb* 1994;14:1129–1136. [PubMed: 8018669]
6. Watanabe H, Miyashita Y, Murano T, Hiroh Y, Itoh Y, Shirai K. Preheparin serum lipoprotein lipase mass level: the effects of age, gender, and types of hyperlipidemias. *Atherosclerosis* 1999;145:45–50. [PubMed: 10428294]
7. Rip J, Nierman MC, Wareham NJ, Luben R, Bingham SA, Day NE, van Miert JN, Hutten BA, Kastelein JJ, Kuivenhoven JA, Khaw KT, Boekholdt SM. Serum lipoprotein lipase concentration and risk for future coronary artery disease: the EPIC-Norfolk prospective population study. *Arterioscler Thromb Vasc Biol* 2006;26:637–642. [PubMed: 16373616]
8. Cuchel M, Rader DJ. Is the cholesteryl ester transfer protein proatherogenic or antiatherogenic in humans? *J Am Coll Cardiol* 2007;50:1956–1958. [PubMed: 17996560]
9. Movva R, Rader DJ. Laboratory assessment of HDL heterogeneity and function. *Clin Chem* 2008;54:788–800. [PubMed: 18375481]
10. Boekholdt SM, Kuivenhoven JA, Wareham NJ, Peters RJ, Jukema JW, Luben R, Bingham SA, Day NE, Kastelein JJ, Khaw KT. Plasma levels of cholesteryl ester transfer protein and the risk of future coronary artery disease in apparently healthy men and women: the prospective EPIC (European Prospective Investigation into Cancer and nutrition)-Norfolk population study. *Circulation* 2004;110:1418–1423. [PubMed: 15337694]
11. Borggreve SE, Hillege HL, Dallinga-Thie GM, de Jong PE, Wolffenbuttel BH, Grobbee DE, van Tol A, Dullaart RP. High plasma cholesteryl ester transfer protein levels may favour reduced incidence of cardiovascular events in men with low triglycerides. *Eur Heart J* 2007;28:1012–1018. [PubMed: 17409111]
12. Hitsumoto T, Ohsawa H, Uchi T, Noike H, Kanai M, Yoshinuma M, Miyashita Y, Watanabe H, Shirai K. Preheparin serum lipoprotein lipase mass is negatively related to coronary atherosclerosis. *Atherosclerosis* 2000;153:391–396. [PubMed: 11164428]
13. Merkel M, Kako Y, Radner H, Cho IS, Ramasamy R, Brunzell JD, Goldberg IJ, Breslow JL. Catalytically inactive lipoprotein lipase expression in muscle of transgenic mice increases very low density lipoprotein uptake: direct evidence that lipoprotein lipase bridging occurs in vivo. *Proc Natl Acad Sci U S A* 1998;95:13841–13846. [PubMed: 9811888]
14. Santamarina-Fojo S, Gonzalez-Navarro H, Freeman L, Wagner E, Nong Z. Hepatic lipase, lipoprotein metabolism, and atherogenesis. *Arterioscler Thromb Vasc Biol* 2004;24:1750–1754. [PubMed: 15284087]
15. Hanyu O, Miida T, Obayashi K, Ikarashi T, Soda S, Kaneko S, Hirayama S, Suzuki K, Nakamura Y, Yamatani K, Aizawa Y. Lipoprotein lipase (LPL) mass in preheparin serum reflects insulin sensitivity. *Atherosclerosis* 2004;174:385–390. [PubMed: 15136071]
16. Sandhofer A, Kaser S, Ritsch A, Laimer M, Engl J, Paulweber B, Patsch JR, Ebenbichler CF. Cholesteryl ester transfer protein in metabolic syndrome. *Obesity (Silver Spring)* 2006;14:812–818. [PubMed: 16855190]

17. Magkos F, Patterson BW, Mohammed BS, Klein S, Mittendorfer B. Women produce fewer but triglyceride-rich very low density lipoproteins than men. *J Clin Endocrinol Metab* 2007;92:1311–1318. [PubMed: 17264179]
18. Magkos F, Wright DC, Patterson BW, Mohammed BS, Mittendorfer B. Lipid metabolism response to a single, prolonged bout of endurance exercise in healthy young men. *Am J Physiol Endocrinol Metab* 2006;290:E355–362. [PubMed: 16219668]
19. Bensadoun A. Sandwich immunoassay for measurement of human hepatic lipase. *Methods Enzymol* 1996;263:333–338. [PubMed: 8749020]
20. Olin KL, Potter-Perigo S, Barrett PH, Wight TN, Chait A. Lipoprotein lipase enhances the binding of native and oxidized low density lipoproteins to versican and biglycan synthesized by cultured arterial smooth muscle cells. *J Biol Chem* 1999;274:34629–34636. [PubMed: 10574927]
21. Obunike JC, Paka S, Pillarisetti S, Goldberg IJ. Lipoprotein lipase can function as a monocyte adhesion protein. *Arterioscler Thromb Vasc Biol* 1997;17:1414–1420. [PubMed: 9261275]
22. Morton RE, Zilversmit DB. Inter-relationship of lipids transferred by the lipid-transfer protein isolated from human lipoprotein-deficient plasma. *J Biol Chem* 1983;258:11751–11757. [PubMed: 6619141]
23. Yen FT, Deckelbaum RJ, Mann CJ, Marcel YL, Milne RW, Tall AR. Inhibition of cholesteryl ester transfer protein activity by monoclonal antibody. Effects on cholesteryl ester formation and neutral lipid mass transfer in human plasma. *J Clin Invest* 1989;83:2018–2024. [PubMed: 2723070]
24. Berneis KK, Krauss RM. Metabolic origins and clinical significance of LDL heterogeneity. *J Lipid Res* 2002;43:1363–1379. [PubMed: 12235168]
25. Lamarche B, Rashid S, Lewis GF. HDL metabolism in hypertriglyceridemic states: an overview. *Clin Chim Acta* 1999;286:145–161. [PubMed: 10511289]
26. Jiang XC, Moulin P, Quinet E, Goldberg IJ, Yacoub LK, Agellon LB, Compton D, Schnitzer-Polokoff R, Tall AR. Mammalian adipose tissue and muscle are major sources of lipid transfer protein mRNA. *J Biol Chem* 1991;266:4631–4639. [PubMed: 1999438]
27. Borggreve SE, De Vries R, Dullaart RP. Alterations in high-density lipoprotein metabolism and reverse cholesterol transport in insulin resistance and type 2 diabetes mellitus: role of lipolytic enzymes, lecithin:cholesterol acyltransferase and lipid transfer proteins. *Eur J Clin Invest* 2003;33:1051–1069. [PubMed: 14636288]
28. Nishimura M, Ohkaru Y, Ishii H, Sunahara N, Takagi A, Ikeda Y. Development and evaluation of a direct sandwich-enzyme-linked immunosorbent assay for the quantification of human hepatic triglyceride lipase mass in human plasma. *J Immunol Methods* 2000;235:41–51. [PubMed: 10675756]
29. Zambon A, Bertocco S, Vitturi N, Polentarutti V, Vianello D, Crepaldi G. Relevance of hepatic lipase to the metabolism of triacylglycerol-rich lipoproteins. *Biochem Soc Trans* 2003;31:1070–1074. [PubMed: 14505482]

Table 1
Plasma lipid transfer enzyme concentrations in lean and obese men and women

	Lean (<i>n</i> = 40)		Obese (<i>n</i> = 40)		Two-way ANOVA <i>p</i> -values		
	Men	Women	Men	Women	obesity	sex	interaction
<i>n</i>	18	22	13	27			
Body mass index (kg/m ²)	22.6 ± 0.4	22.4 ± 0.3	34.5 ± 1.2	35.1 ± 0.7	< 0.001	0.793	0.547
Body fat (% body weight)	14.1 ± 1.3	28.2 ± 0.8	33.9 ± 2.2	48.2 ± 1.1	< 0.001	< 0.001	0.929
Abdominal fat (cm ²)	133 ± 28	166 ± 18	625 ± 66	561 ± 36	< 0.001	0.692	0.221
Intra-abdominal fat (% total)	39 ± 6	23 ± 3	46 ± 3	25 ± 2	0.291	0.000	0.478
Plasma glucose (mmol/l)	4.98 ± 0.09	4.86 ± 0.04	5.25 ± 0.09	4.95 ± 0.06	0.012	0.003	0.202
Plasma triglyceride (mmol/l)	0.80 ± 0.04	0.67 ± 0.04	1.21 ± 0.06	1.14 ± 0.06	< 0.001	0.072	0.596
VLDL-triglyceride (mmol/l)	0.54 ± 0.03	0.40 ± 0.03	0.89 ± 0.06	0.78 ± 0.05	< 0.001	0.012	0.730
HDL-cholesterol (mmol/l)	0.91 ± 0.05	1.16 ± 0.05	0.79 ± 0.05	0.90 ± 0.04	< 0.001	0.001	0.198
LCAT (μg/ml)	548 ± 20	513 ± 20	730 ± 39	654 ± 26	< 0.001	0.041	0.446
CETP (μg/ml)	170 ± 7	175 ± 6	192 ± 8	215 ± 8	< 0.001	0.085	0.248
LPL (ng/ml)	31 ± 2	40 ± 3	35 ± 3	48 ± 3	0.071	0.001	0.436
HL (ng/ml)	6.6 ± 0.8	6.8 ± 0.9	10.0 ± 1.1 ^{*†}	6.8 ± 0.6	-	-	0.048

Values are means ± SEM.

^{*} Significantly different from corresponding value in lean men (*p* = 0.018).

[†] Significantly different from corresponding value in obese women (*p* = 0.009).

VLDL, very-low density lipoprotein; HDL, high-density lipoprotein; LCAT, lecithin-cholesterol acyl transferase; LPL, lipoprotein lipase; CETP, cholesterol-ester transfer protein; HL, hepatic lipase.

Table 2

Plasma lipid transfer enzyme concentrations in men and women matched on percent body fat

	Men	Women	<i>p</i> -value
<i>n</i>	15	15	
Body fat (% body weight)	32.5 ± 2.1	33.0 ± 2.3	0.874
Intra-abdominal fat (% total)	44 ± 3	28 ± 5	0.024
Plasma glucose (mmol/l)	5.17 ± 0.09	5.05 ± 0.07	0.359
Plasma triglyceride (mmol/l)	1.15 ± 0.07	0.85 ± 0.10	0.016
VLDL-triglyceride (mmol/l)	0.84 ± 0.06	0.54 ± 0.08	0.005
HDL-cholesterol (mmol/l)	0.78 ± 0.04	1.05 ± 0.07	0.002
LCAT (μg/ml)	694 ± 42	570 ± 40	0.040
CETP (μg/ml)	186 ± 8	186 ± 9	0.993
LPL (ng/ml)	34 ± 3	36 ± 3	0.660

Values are means ± SEM.

VLDL, very-low density lipoprotein; HDL, high-density lipoprotein; LCAT, lecithin-cholesterol acyl transferase; LPL, lipoprotein lipase; CETP, cholesterol-ester transfer protein.

Table 3

Relationships between plasma lipid transfer enzyme concentrations and lipoprotein profile

	LCAT	LPL	CETP	HL
Total VLDL particle concentration	0.414 [*]	0.128	0.299 [*]	0.003
Large VLDL particles	0.315 [*]	0.114	0.143	0.162
Medium VLDL particles	0.457 [*]	−0.067	0.074	−0.099
Small VLDL particles	0.287 [*]	0.203	0.370 [*]	0.047
Average VLDL particle size	0.167	0.044	−0.001	0.237 [*]
VLDL-triglyceride concentration	0.445 [*]	0.137	0.183	0.109
Total LDL particle concentration	0.333 [*]	0.232 [*]	0.459 [*]	0.184
Large LDL particles	0.031	0.099	0.169	−0.124
Small LDL particles	0.327 [*]	0.198	0.401 [*]	0.235 [*]
Average LDL particle size	−0.157	−0.125	−0.198	−0.227 [*]
Total HDL particle concentration	0.595 [*]	−0.034	0.287 [*]	0.021
Large HDL particles	−0.114	−0.182	−0.070	−0.269 [*]
Medium HDL particles	0.245 [*]	0.232 [*]	0.105	−0.206
Small HDL particles	0.477 [*]	−0.045	0.249 [*]	0.338 [*]
Average HDL particle size	−0.389 [*]	−0.053	−0.193	−0.189
HDL-cholesterol concentration	0.062	−0.104	0.030	−0.185

Pearson's simple correlation coefficients are shown;

^{*} $p < 0.05$.

LCAT, lecithin-cholesterol acyl transferase; LPL, lipoprotein lipase; CETP, cholesterol-ester transfer protein; HL, hepatic lipase; VLDL, very-low density lipoprotein; LDL, low-density lipoprotein; HDL, high-density lipoprotein.