Arakawa H, Katsuyama K, Ito T, Fujii E, Hayashi S, Kato A, Suzuki M: Granulocyte colony-stimulating factor has no adverse effects on atherosclerotic lesions in high cholesterol-fed miniature Swine. J Vet Med Sci, 2008; 70: 943-950

13) Kawaguchi H, Miyoshi N, Miura N, Fujiki M, Horiuchi M, Izumi Y, Miyajima H, Nagata R, Misumi K, Takeuchi T, Tanimoto A, Yoshida H: Microminipig, a non-rodent experimental animal optimized for life science research: Novel atherosclerosis model induced by high fat and cholesterol diet. J Pharmacol Sci, 2011; 115: 115-121
14) Miyoshi N, Horiuchi M, Inokuchi Y, Miyamoto Y, Miura N, Tokunaga S, Fujiki M, Izumi Y, Miyajima H, Nagata R, Misumi K, Takeuchi T, Tanimoto A, Yasuda N, Yoshida H, Kawaguchi H: Novel microminipig model of atherosclerosis by high fat and high cholesterol diet, established in Japan. In Vivo, 2010; 24: 671-680
15) Vergnes L, Phan J, Strauss M, Tafuri S, Reue K: Cholesterol and cholate cpomponents of an atherogenic diet induce distinct stages of hepatic inflammatory gene expression. J Biol Chem, 2003; 278: 42774-42784
16) Altmann SW, Davis HR Jr, Zhu LJ, Yao X, Hoos LM, Tetzloff G, Iyer SP, Maguire M, Golovko A, Zeng M, Wang L, Murgolo N, Graziano MP: Niemann-Pick C1 Like 1 protein is critical for intestinal cholesterol absorption. Science, 2004; 303: 1201-4120
17) Temel RE, Tang W, Ma Y, Rudel LL, Willingham MC, Ioannou YA, Davies JP, Nilsson LM, Yu L: Hepatic Niemann-Pick C1-like 1 regulates biliary cholesterol concentration and is a target of ezetimibe. J Clin Invest, 2007; 117: 1968-1978
18) Kawaguchi H, Yamada T, Miura N, Takahashi Y, Yoshikawa T, Izumi H, Kawarasaki T, Miyoshi N, Tanimoto A: Reference Values of Hematological and Biochemical Parameters for the World Smallest Microminipigs. J Vet Med Sci, 2012; 74: 933-936
19) Miura N, Kawaguchi H, Nagasato T, Yamada T, Ito T, Izumi H, Shameshima H, Miyoshi N, Tanimoto A, Maruyama I: Coagulation activity and white thrombus formation in the microminipig. In Vivo, 2013; 27: 357-361
20) Lichtman AH, Clinton SK, Iiyama K, Connelly PW, Libby P, Cybulsky MI: Hyperlipidemia and atherosclerotic lesion development in LDL receptor-deficient mice fed defined semipurified diets with and without cholate. Arterioscler Thromb Vasc Biol, 1999; 19: 1938-1944
21) Gordon DJ, Rifkind BM: High-density lipoprotein--the clinical implications of recent studies. N Eng J Med, 1989; 321: 1311-1316
22) Quig DW, Zilversmit DB: High density lipoprotein metabolism in a rabbit model of hyperalphalipoproteinemia. Atherosclerosis, 1989; 76: 9-19
23) Brinton EA, Eisenberg S, Breslow JL: A low-fat diet decreases high density lipoprotein (HDL) cholesterol levels by decreasing HDL apolipoprotein transport rates. J Clin Invest, 1990; 85: 144-151
24) Hayek T, Ito Y, Azrolan N, Verdery RB, Aalto-Setala K, Walsh A, Breslow JL: Dietary fat increases high density lipoprotein (HDL) levels both by increasing the transport rates and decreasing the fractional catabolic rates of HDL cholesterol ester and apolipoprotein (Apo) A-I. J Clin Invest, 1993; 91: 1665-1671
25) Greeve J, altkemper I, Dieterich JH, Greten H, Windler E: Apoprotein B mRNA editing in 12 different mammalian species: hepatic expression is reflected in low concentrations of apoB-containing plasma lipoproteins. J Lipid Res, 1993; 34: 1367-1383
26) Greeve J, Jona VK, Chowdhury NR, Horwitz MS, Chowdhury JR: Hepatic gene transfer of the catalytis subunit of the apolipoprotein B mRNA editing enzyme results in a reduction of plasma LDL levels in normal and Watanabe heritable hyperlipidemic rabbits. J Lipid Res, 1996; 37: 2001-2017
27) Hardtmann DA, Pullinger CR, Hamilton RL, Kane JP, Malloy MJ: Molecular and metabolic basis for the metabolic disorder normotriglyceridemic abetalipoproteinemia. J Clin Invest, 1991; 88: 1722-1729
28) Brown MS, Goldstein JL: A receptor-mediated pathway for cholesterol homeostasis. Science, 1986; 232: 34-47
29) Goedeke L, Fernandez-Hernando C: Regulation of cholesterol homeostasis. Cell Mol Life Sci, 2012; 69: 915-930
30) Rudling M: Hepatic mRNA levels for the LDL receptor and HMG-CoA reductase show coordinate regulation in vivo. J Lipid Res, 1992; 33: 493-501
31) Rudling M, Angelin B, Stahle L, Reihner E, Sahlin S, Olivecrona H, Bjorkhem I, Einarsson C: Regulation of hepatic low-density lipoprotein receptor, 3-hydroxy-3-methylglutaryl Coenzyme A reductase, and cholesterol 7α-hydroxylase mRNAs in human liver. J Clin Endocrinol Metab, 2002; 87: 4307-4313
32) Kong WJ, Liu J, Jiang JD: Human low-density lipoprotein receptor gene and its regulation. J Mol Med, 2006; 84: 29-36
33) Lammert F, Wang DQ: New insights into the genetic regulation of intestinal cholesterol absorption. Gastroenterology, 2005; 129: 718-734
34) Redinger RN: Nuclear receptors in cholesterol catabolism: molecular biology of the enterohepatic circulation of bile salts and its role in cholesterol homeostasis. J Lab Clin Med, 2003; 142: 7-20
35) Betters JL, Yu L: NPC1L1 and cholesterol transport. FEBS Lett, 2010; 584: 2740-2747
36) Pramfalk C, Jiang ZY, Cai Q, Hu H, Zhang SD, Han TQ, Eriksson M, Parini P: HNF1α and SREBP2 are important regulators of NPC1L1 in human liver. J Lipid Res, 2010; 51: 1354-1362
37) Alrefai WA, Annaba F, Sarwar Z, Dwivedi A, Saksena S, Singla A, Dudeja PK, Gill RK: Modulation of human Niemann-Pick C1-like 1 gene expression by sterol: Role of sterol regulatory element binding protein 2. Am J Physiol Gastrointest Liver Physiol, 2007; 292: G369-G376
38) Yamashita S, Hirano K, Sakai N, Matsuzawa Y: Molecular biology and pathophysiological aspects of plasma cholesteryl ester transfer protein. Biochim Biophys Acta, 2000; 1529: 257-275
39) Rosenson RS, Brewer Jr HB, Davidson WS, Fayad ZA, Fuster V, Goldstein J, Hellerstein M, Jiang XC, Phillips MC, Rader DJ, Remaley AT, Rothblat GH, Tall AR, van-Charvet LY: Cholesterol efflux and atheroprotection:

Advancing the concept of reverse cholesterol transport. Circulation, 2012; 125: 1905-1919
40) Guyard-Dangremont V, Desrumaux C, Gambert P, Lallemant C, Lagrost L: Phospholipid and cholesteryl ester transfer activities in plasma from 14 vertebrate species. Regulation to atherogenesis susceptibility. Comp Biochem Physiol B Biochem Mol Biol, 1998; 120: 517-525
41) Hogarth CA, Roy A, Ebert DL: Genomic evidence for the absence of a functional cholesterol ester transfer protein gene in mice and rats. Comp Biochem Physiol B Biochem Mol Biol, 2003; 135: 71-81
42) Marotti KR, Castle CK, Boyle TP, Lin AH, Murry RW, Melchior GW: Severe atherosclerosis in transgenic mice expressing simian cholesteryl ester transfer protein. Nature, 1993; 364: 73-75
43) Zuckerman SH, Evans GF, Schekm JA, Eacho PI, Sandusky G: Estrogen-mediated increases in LDL cholesterol and foam cell-containing lesions in human apo B100 x CETP transgenic mice. Arterioscler Thromb Vasc Biol, 1999; 19: 1476-1483
44) Hayek T, L Massuci-Magoulas, Jiang X, Walsh A, Rubin E, Breslow JL, Tall AR: Decreased early atherosclerotic lesions in hypertriglyceridemic mice expressing cholestery ester transfer protein transgene. J Clin Invest, 1995; 96: 2071-2074
45) Föger B, Chase M, Amar MJ, Vaisman BL, Shamburek RD, Paigen B, Fruchart-Najib J, Paiz JA, Koch CA, Hoyt RF, Brewer HB Jr, Santamarina-Fojo S: Cholesteryl ester transfer protein corrects dysfunctional high density lipoproteins and reduces aortic atherosclerosis in lecithin cholesterol acyltransferase transgenic mice. J Biol Chem, 1999; 274: 36912-36920
46) Gardner CD, Fortmann SP, Krauss RM: Association of small low density lipoprotein particles with the incidence of coronary artery disease in men and women. JAMA, 1996; 276: 875-881
47) Lamarche B, Tchernof A, Moorjani S, Cantin B, Dagenais GR, Lupien PJ, Després JP: Small, dense low-density lipoprotein particles as a predictor of the risk of ischemic heart disease in men. Prospective results from the Québec Cardiovascular Study. Circulation, 1997; 95: 69-75
48) Schaefer EJ, McNamara JR, Tayler T, Daly JA, Gleason JA, Seman LJ, Ferrari A, Rubenstein JJ: Effects of atorvastatin on fasting and postprandial lipoprotein subclasses in coronary heart disease patients versus control subjects. Am J Cardiol, 2002; 90: 689-696
49) Brousseau ME, Schaefer EJ, Wolfe ML, Bloedon LT, Digenio AG, Clark RW, Mancuso JP, Rader DJ: Effects of an inhibitor of cholesteryl ester transfer protein on HDL cholesterol. N Engl J Med, 2004; 350: 1505-1515
50) Ishigami M, Yamashita S, Sakai N, Arai T, Hirano K, Hiraoka H, Kameda-Takemura K, Matsuzawa Y: Large and cholesterylester-rich high density lipoproteins in cholesteryl ester transfer protein deficienct can not protect macrophages from cholesterol accumulation induced by acetylated low density lipoproteins. J Biochem, 1994; 116: 257-262
51) Ordovas JM, Cupples LA, Corella D, Otvos JD, Osgood D, Martinez A, Lahoz C, Coltell O, Wilson PW, Schaefer EJ: Association of cholesteryl ester transfer protein-TaqIB polymorphism with variations in lipoprotein subclasses and coronary heart disease risk: the Framingham study. Arterioscler Thromb Vasc Biol, 2000; 20: 1323-9132
52) Brousseau ME, O'Connor JJ Jr, Ordovas JM, Collins D, Otvos JD, Massov T, McNamara JR, Rubins HB, Robins SJ, Schaefer EJ: Cholesteryl ester transfer protein TaqI B2B2 genotype is associated with higher HDL cholesterol levels and lower risk of coronary heart disease end points in men with HDL deficiency: Veterans Affairs HDL Cholesterol Intervention Trial. Arterioscler Thromb Vasc Biol, 2002; 22: 1148-1154
53) Yamashita S, Hui DY, Wetterau JR, Sprecher DL, Harmony JAK, Matsuzawa Y, Tarui S: Total deficiency of plasma cholesteryl ester transfer protein in subjects homozygous and heterozygous for the intron 14 splicing defect. Biochem Biophys Res Commun, 1990; 170: 1346-1351
54) Hirano K, Yamashita S, Kuga Y, Sakai N, Nozaki S, Kihara S, Arai T, Yanagi K, Takami S, Menju M, Ishigami M, Yoshida Y, Kameda-Takemura-K, Hayashi K, Matsuzawa Y: Atherosclerotic disease in marked hyperalphalipoproteinemia. Combined reduction of cholesteryl ester transfer protein and hepatic triglyceride lipase. Arterioscler Thromb Vasc Biol, 1995; 15: 1849-5618
55) Hirano K, Yamashita S, Sakai N, Arai T, Yoshida Y, Nozaki S, Kameda-Takemura K, Matsuzawa Y: Molecular defect and atherogenicity in cholesteryl ester transfer protein deficiency. Ann N Y Acad Sci, 1995; 748: 599-602
56) Schwartz GG, Olsson AG, Abt M, Ballantyne CM, Barter PJ, Brumm J, Chaitman BR, Holme IM, Kallend D, Leiter LA, Leitersdorf E, McMurray JJ, Mundl H, Nicholls SJ, Shah PK, Tardif JC, Wright RS; dal-OUTCOMES Investigators: Effects of dalcetrapib in patients with a recent acute coronary syndrome. N Engl J Med, 2012; 367: 2089-2099
57) Peterson J, Bengtsson-Olivecrona G, Olivecrona T: Mouse preheparin plasma contains high levels of hepatic lipase with low affinity for heparin. Biochim Biophys Acta., 1986; 878: 65-70
58) Mezdour H, Jones R: Dengremont C, Castro G, Maeda N: Hepatic lipase deficiency increases plasma cholesterol but reduces susceptibility to atherosclerosis in apolipoprotein E-deficient mice. J Biol Chem, 1997; 272: 13570-13575
59) Dichek HL, Qian K, Agrawal N: Divergent effects of the catalytic and bridging functions of hepatic lipase on atherosclerosis. Arterioscler Thromb Vasc Biol, 2004; 24: 1696-1702
60) Fan J, Wang J, Bensadoun A, Lauer SJ, Dang Q: Overexpression of hepatic lipase in transgenic rabbits leads to a marked reduction of plasma high density lipoproteins and intermediate density lipoproteins. Proc Natl Acad Sci USA, 1994; 91: 8724-8728

Supplemental Text

Materials and Methods

CETP Assay

Sample Preparation and Western blotting

For lipoprotein fractionation, equal volumes of plasma samples were pooled from six MMPs and six rabbits fed normal chow. The lipoproteins were fractionated using fast protein liquid chromatography (FPLC) with a Superose 6 10/300 GL FPLC column (Amersham Biosciences, Piscataway, NJ). Diluted plasma and aliquots of the FPLC fractions were subjected to SDS-PAGE and transferred unto PVDF membranes. Immunoblotting was performed as previously described. Antibodies against cholesteryl ester (CE) transfer protein (CETP) (mouse monoclonal, designated as "antibody A," and rabbit polyclonal, designated as "antibody B") were purchased from EMD Millipore (Billerica, MA) and Abcam (Cambridge Science Park, Cambridge, UK), respectively. The proteins were visualized using a chemiluminescence method (ECL Plus Western Blotting Detection System; GE Healthcare Piscataway, NJ).

Diagnosis of CETP Deficiency and Determination of the CETP Mass

A 59-year-old man and 54-year-old woman with high HDL-C levels (172 and 191 mg/dL, respectively) were referred to the National Defense Medical College Hospital. Blood samples were obtained, and the sera were isolated with centrifugation. Genomic DNA was prepared using a commercially available kit (Qiagen, Frederick, MA). The CETP mass was measured using a sandwich ELISA with JHC1 and JHC2, two monoclonal antibodies specific to human CETP, as previously described[1]. Common CETP mutations (an intron 14 splicing defect, Int14 +1 G>A; amino acid substitution from aspartic acid to glycine at position 442, D442G) were identified using an Invader® assay[2].

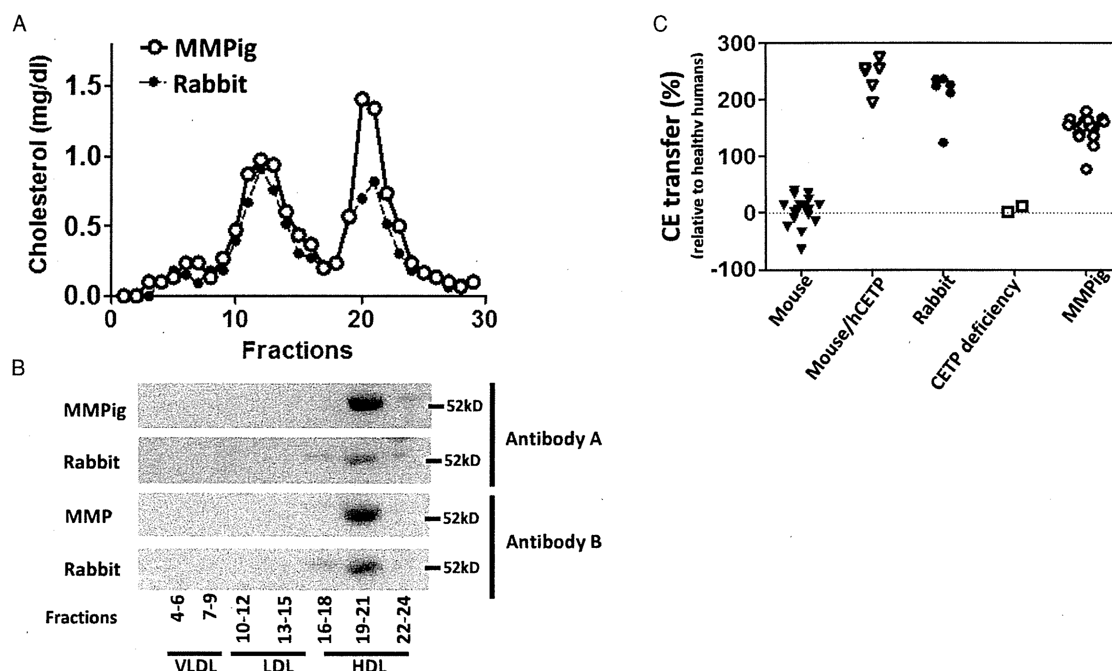
Cloning and Generation of Recombinant Adenoviruses Encoding for Human CETP

A recombinant adenovirus expressing human CETP (Ad-hCETP) was produced using the ViraPower Adenoviral Expression System (Invitrogen, Carlsbad, CA, USA), according to the manufacturer's instructions. Briefly, an entry clone of the Gateway system (Invitrogen) was generated by cloning the open reading frame into a pENTR/D-TOPO vector (Invitrogen) using first-strand complementary DNA derived from human liver RNA (Clontech, Pao Alto, CA) as a template. The specific primers were as follows: forward: 5'- CAC CAT GCT GGC TGC CAC AGT -3'; reverse: 5'- CTA GCT CAA GCT CTG GAG GAA A -3'. An expression clone for the adenoviral vector was then generated via an LR recombination reaction between the entry clone and a pAd/CMV/V5-DEST (Invitrogen), according to the manufacturer's protocol.

Supplemental Table 1. Genes investigated and primers used for PCR

Gene	Assay ID	Custom made primers	RefSeq	GenBank
LDLr	Ss03374441_u1		NM_001206354.1	AF065990.1
SR-BI	Ss03391104_m1		NM_213967.1	AF467889.2
HMGCR	Ss03390147_m1		NM_001122988.1	DQ432054.1
SREBP-2	Ss03376492_u1			AY493571.1
APOBEC-1		Forward: CCATGGTGTGACCATCCAGAT Reverse: AGTTGACAAAATTCCTCCAGCAGTA Probe: TGGGAGCCCCAGAGTA	XM_003126519.1	
NPC1L1		Forward: CCTGTTCGGAGCGAGTCTCTA Reverse: GAAAGAGGAAATAGTCGAGCAGGTA Probe: CTGCCCAAGGACTC	XM_003134893.1	
GAPDH	Ss03375435_u1		NM_001206359.1	AY307771.1

The assay IDs are listed for the predesigned gene expression assay (ABI). The primers and probes for the APOBEC-1 and NPC1L1 analyses were created based on swine sequences.



Supplemental Fig. 1. MMPig sera activate CE transfer from HDL to LDL

(A) The cholesterol levels in the MMPig and rabbit lipoprotein fractions (pooled from six animals each) isolated using FPLC. (B) A Western blot analysis using two antibodies to detect the CETP expression in the MMPig and rabbit lipoprotein fractions, as described in the Methods. (C) The cholesteryl ester (CE) transfer activity in the sera obtained from C57BL/6 mice ($n=18$), C57BL/6 mice injected with Ad-hCETP ($n=6$), rabbits ($n=6$), subjects with CETP deficiency ($n=2$) and MMPigs ($n=18$).

The recombinant adenoviral plasmid was purified and then transfected into 293A cells. The adenovirus was purified using the Adeno-X Virus Purification Kit (Clontech), after a sufficient cytopathic effect was observed in the cells. The multiplicity of infection (MOI) was defined as the ratio of the total number of plaque-forming units to the total number of cells infected.

Injection of the Adenoviral Vector and Blood Sampling

C57BL/6 mice were obtained from Clea Japan (Tokyo, Japan) and handled according to the guidelines of the National Defense Medical College Institutional Animal Care and Use Committee. The mice were injected with Ad-hCETP (5×10^8 pfu) via the tail vein. Five days after injection, the mice were exsanguinated, and blood samples were obtained.

Determination of the CETP Activity

The serum CETP activity was determined according to the method of Cheung *et al*[3]. with a minor modification. In brief, the d>1.125 fraction isolated via ultracentrifugation from pooled healthy human plasma ($n=6$, fasting) was incubated with ^{14}C-cholesteryl oleate (Perkin-Elmer) at 37℃ for 16 hours, after which the 1.125<d<1.21 fraction was isolated via ultracentrifugation. Following dialysis, the HDL fractions were incubated with human LDL isolated from pooled human plasma ($n=6$, fasting) in the presence or absence of serum obtained from MMPigs, rabbits, mice or humans. Ten hours after incubation, LDL was precipitated by adding heparin (500 U/mL) and manganese chloride (0.2 μmol/L) to the samples. The radiotracer counts in the supernatants were determined using a scintillation counter. The percentage cholesteryl ester transfer was calculated by dividing the differences between the values obtained in the absence and presence of sera by the values obtained in the absence of sera. The data are expressed as the CE transfer relative to the mean value of sera obtained from eight healthy humans.

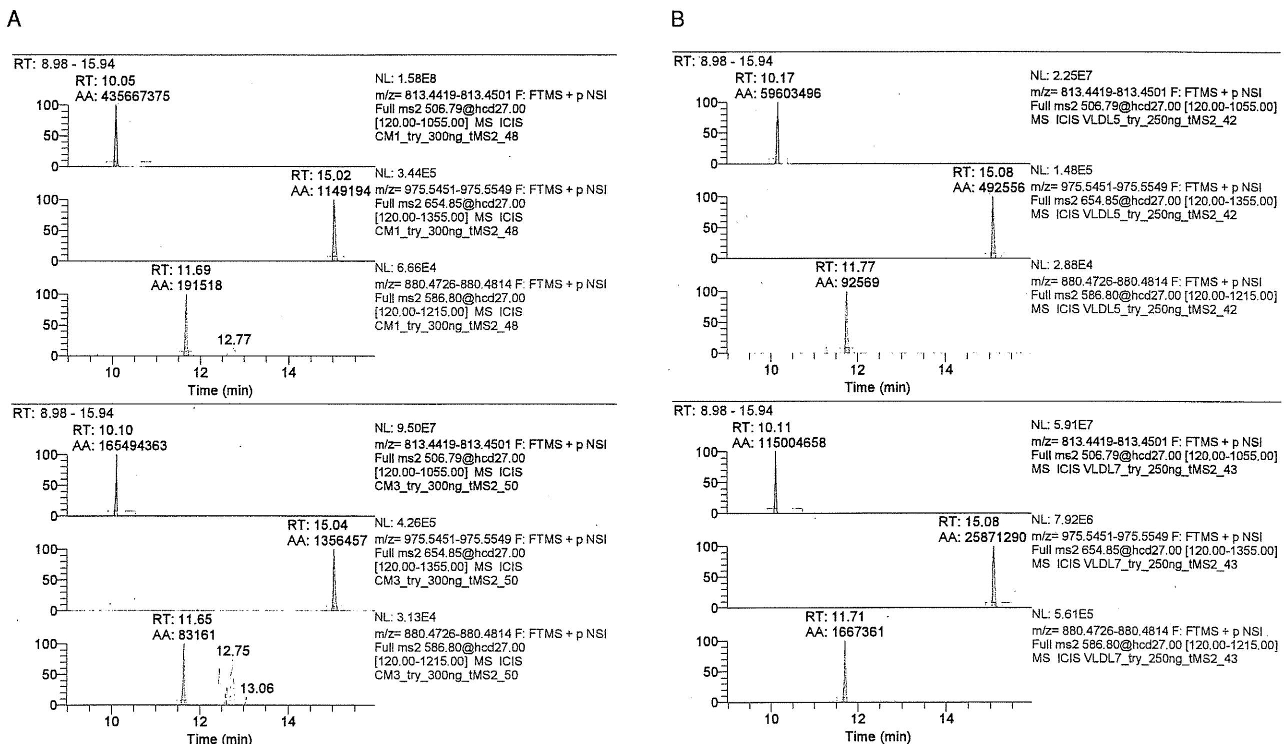
MS/MS Analysis of Apolipoprotein A1 and B

To detect apolipoprotein (apo)A1, B48 and B100 in the plasma, we utilized a HPLC/MS/MS-based method that can be applied across species. A multiple reaction monitoring (MRM) method was developed for apoA1 and apoB48/100[4]. First, the chylomicron

Supplemental Table 2. MRM transitions for the targeted peptides

	m/z (precursor, z=2)	m/z (product)
Apolipoprotein A1		
AKPALEDLR	506.7929	813.446
Apolipoprotein B48/100		
GFEPTLEALFGK	654.8453	975.55
YENYELTLK	586.7953	880.477

Thermo tube lens = 120 for all precursor ions. Z = 2 for all precursor ions



Supplemental Fig. 2.

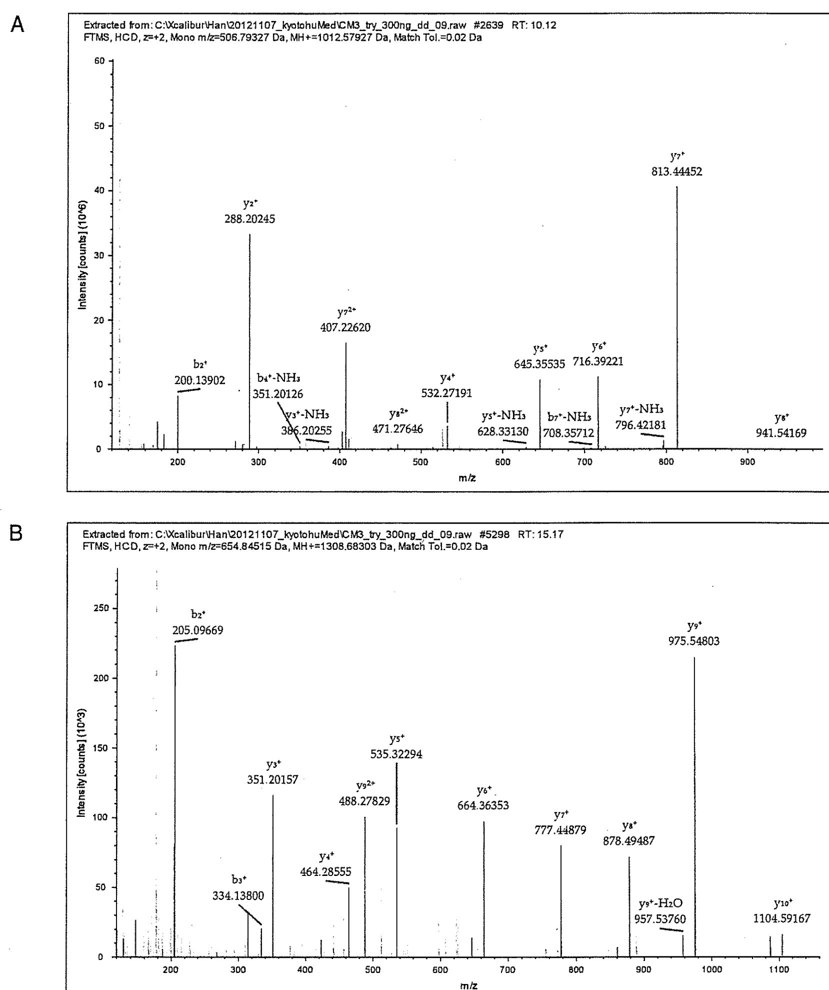
Extracted MRM chromatograms for ApoA1 (AKPALEDLR) and ApoB48/100 (GFEPTLEALFGK, YENYELTLK) from a nanoHPLC-MS/MS analysis of digested human (A) and MMPig (B) plasma fractions, chylomicron (CM) and very-low-density lipoprotein (VLDL).

(CM) and very-low-density lipoprotein (VLDL) fractions were separated from human and MMPig plasma using the Himac Centrifuge (CP70MX, Himac Centrifuge, Hitachi Medical Systems), according to the manufacturer's protocol. The samples were alkylated and digested overnight with trypsin and analyzed on a nano HPLC system (Easy-nLC, ThermoScientific) coupled with a triple quadrupole mass spectrometer (Orbitrap Q Exactive, ThermoScientific). The trap column was the EASY column, C18, 0.1 × 2 cm, 5 μm (ThermoScientific), and the analytical column was the Tip column, ODS, 0.075 × 120 mm (Nikkyo Technos). The flow rate was 0.3 mL/min.

Results

CETP Assay

Supplemental Fig. 1A shows the cholesterol distribution in the lipoprotein subfractions isolated using FPLC. The lipoprotein profiles of the MMPs were similar to those of rabbits, except for the higher HDL cholesterol levels. We performed a Western blot analysis using the FPLC subfractions to investigate whether the CETP expression was evident in the MMP. Two antibodies against CETP enabled the visualization of bands of similar molecular size in the HDL fractions obtained from both MMPigs and rabbits, as previ-



Supplemental Fig. 3.

Sequencing of the tandem mass spectrum of (C) a peptide with 506.8 m/z obtained from the tryptic digest of MMPig CM proteins and (D) a peptide with 654.8 m/z obtained from the tryptic digest of MMPig CM proteins.

ously demonstrated in humans[5)] and hamsters[6)] (**Supplemental Fig. 1B**). We next assessed whether sera obtained from MMPigs activated CE transfer from HDL to LDL. We confirmed that the sera obtained from C57BL/6 wild-type mice do not possess a CE transfer activity and that the intravenous injection of Ad-hCETP results in a 2.4-fold increase in the activity compared with that observed in human sera (**Supplemental Fig. 1C**). To further validate the reliability of our CETP activity assay, we measured the CE transfer activity in the sera obtained from the subjects with CETP deficiency. The CETP mass in the sera of the 59-year-old man (HDL-C, 172 mg/dL) and 51-year-old woman (191 mg/dL) was <0.1 and 0.4 μg/mL, respectively (normal range, 1.04-3.55 μg/mL). A genetic analysis revealed that the man carried the homozygous intronic mutation Int14 +1 G>A, while the woman carried the Int14 +1 G>A/D442G mutation as a compound heterozygote. We also confirmed the absence of the CE transfer activity in the sera of both subjects. Finally, we observed a robust CE transfer activity in the MMPig sera, 1.5-fold higher than that observed in the humans and 0.7-fold lower than that observed in the rabbits, supporting the possible expression of CETP.

Identification of ApoA1- and ApoB48/100-Specific Peptides and MRM Transitions

Supplemental Table 2 shows the identified peptide sequence along with the corresponding experimentally determined MRM transitions. **Supplemental Fig. 2A** and **2B** show MRM chromatograms extracted from both the human and pig plasma fractions; CM and VLDL. The amino acid sequences for apoA1 and B48/100 in the humans and pigs were obtained with Proteome Discoverer software program using a Swiss-Prot-TrEMBL database. The sequencing of the tandem mass spectrum of peptides with 506.8 and 654.8 m/z from the tryptic digest of porcine CM proteins was AKPALEDLR and GFEPTLEALFGK, corresponding to peptides of porcine apoA1 (**Supplemental Fig. 3A**) and apoB48/100 (**Supplemental Fig. 3B**), respectively.

References

1) Nagano M, Yamashita S, Hirano K, Kujiraoka T, Ito M, Sagehashi Y, Hattori H, Nakajima N, Maruyama T, Sakai N, Egashira T, Matsuzawa Y: Point mutation (-69 G--> A) in the promoter region of cholesteryl ester transfer protein gene in Japanese hyperalphalipoproteinemic subjects. Arterioscler Thromb Vasc Biol, 2001; 21: 985-990
2) Nagano M, Yamashita S, Hirano K, Ito M, Maruyama T, Ishihara M, Sagehashi Y, Oka T, Kujiraoka T, Hattori H, Nakajima N, Egashira T, Kondo M, Sakai N, Matsuzawa Y: Two novel missense mutations in the CETP gene in Japanese hyperalphalipoproteinemic subjects: high-throughput assay by Invader assay. J Lipid Res, 2002; 43: 1011-1018
3) Cheung MC, Wolf AC, Lum KD, Tollefson JH, Albers JJ: Distribution and localization of lecithin:cholesterol acyltransferase and cholesteryl ester transfer activity in A-I-containing lipoproteins. J Lipid Res, 1986; 27: 1135-1144
4) Lassman ME, McLaughlin TM, Somers EP, Stefanni AC, Chen Z, Castro-Perez JM, Hubbard BK, and Roddy TP: A rapid method for cross-spieces quantitation of apolipoproteins A1, B48 and B100 in plasma by ultra-performance liquid chromatography.tandem mass spectrometry. Rapid Commun. Mass Spectrom, 2012; 26: 101-108
5) Clark RW, Moberly JB, Bamberger MJ: Low level quantification of cholesteryl ester transfer protein in plasma subfractions and cell culture media by monoclonal antibody-based immunoassay. J Lipid Res, 1995; 36: 876-889
6) Evans GF, Bensch WR, Apelgren LD, Bailey D, Kauffman RF, Bumol TF, Zuckerman SH: Inhibition of cholesteryl ester transfer protein in normocholesterolemic and hypercholesterolemic hamsters: effects on HDL subspecies, quantity, and apolipoprotein distribution. J Lipid Res, 1994; 35: 1634-1645