Supplementary Table 1. Subject-specific baseline characteristics

Sex (F/M)	Age (yrs.)	Genetic diagnosis	CVD	Lipid-lowering therapy	HDL-c (mg/dl)	ApoA-I (mg/dl)
M*	46	Homozygosity *ApoA-I* (Q(-2)X)	CABG	Atorvastatin 80mg, ezetimibe 10mg, nicotinic acid 500mg	1.80	0.22
M*	55	Heterozygosity *ABCA1* (6401+T2)	MIs (3)	Rosuvastatin 15mg	19.69	28.67
M*	49	Heterozygosity *ApoA-I* (391delAAG exon 4) Heterozygosity *ABCA1* (C1477R)	MIs (2) PCI (2)	Rosuvastatin 10mg	6.21	16.49
M	51	Heterozygosity *LCAT* (T147I)	-	Ezetimibe 10mg/simvastatin 40mg, fish oil capsule	29.06	59.14
M*	68	Heterozygosity *ABCA1* (N1800H)	- Hypertension	-	13.82	51.56
F	51	Heterozygosity *ApoA-I* (L202P)	-	-	37.40	70.24
F	47	Homozygosity *ABCA1* (L996P)	Angina Pectoris PCI	Rosuvastatin 10mg, ezetimibe 10mg	0.60	7.90

This table represents individual subject characteristics at baseline, including genetic mutations. *Subject participated in fecal sterol excretion experiment

Abbreviations: ApoA-I = apolipoprotein A-I; ABCA1 = ATP-binding cassette transporter A1; CABG = coronary artery bypass graft; CVD = cardiovascular disease; HDL-c = HDL-cholesterol; LCAT = lecithin:cholesterol acyl transferase; MI = myocardial infarction; PCI = percutaneous coronary intervention

Supplementary figure legends

Supplementary figure 1. *Lipoprotein profile changes and apoA-I kinetics after six months of treatment*

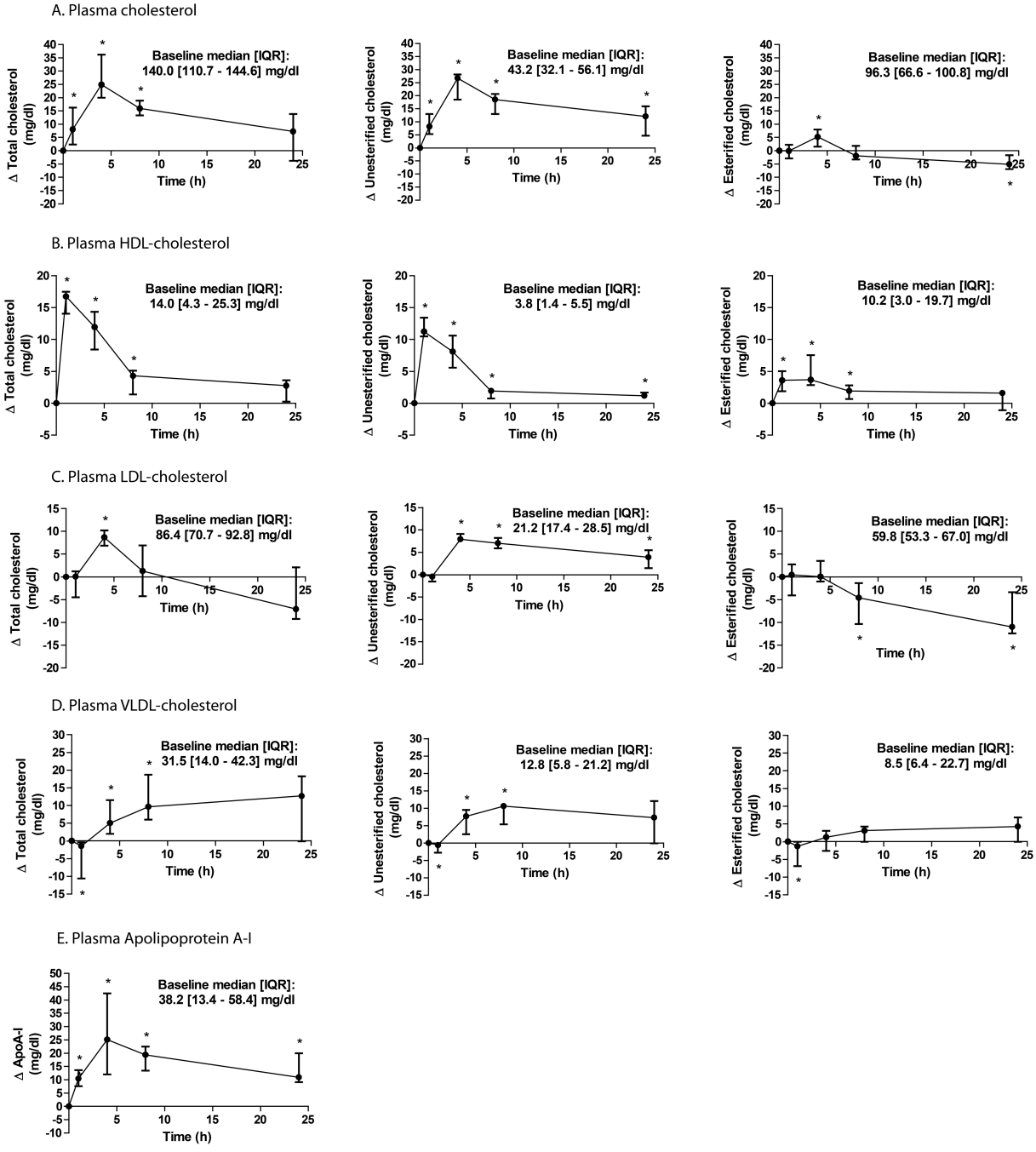
Plasma was obtained at baseline and 1, 4, 8 and 24 hours after start of the 20th infusion. Row A depicts changes in plasma cholesterol levels, row B in HDL-cholesterol, row C in LDL-cholesterol, row D in VLDL-cholesterol and row E in apoA-I. Data represent baseline-corrected medians with interquartile ranges. Values at every time point were compared to baseline. A p-value < 0.05 was considered statistically significant and is depicted with an asterisk.

Abbreviation: apoA-I = apolipoprotein A-I; IQR = interquartile range

Supplementary figure 2. ***Fecal sterol excretion***

Shown is the fecal sterol excretion (specified into neutrol sterols and bile acids) within 8 days prior to and after the first infusion of CER-001 in a subpopulation of four subjects. Subject 1 was homozygous for an ApoA-1 mutation; subject 2 heterozygous for an ABCA1 mutation; subject 3 heterozygous for an ApoA-I and ABCA1 mutation; and subject 4 heterozygous for an ABCA1 mutation.

Supplementary Figure 1. Lipoprotein profile changes and apoA-I kinetics after six months of treatment



Supplementary figure 2.