ELECTRONIC SUPPLEMENTARY INFORMATION (ESI)

(r)HDL in theranostics: how do we apply HDL's biology for precision medicine in atherosclerosis management?

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Supplementary Information **Methodology:** ***3-step (r)HDL purification/preparation methods****

Plasma HDL purification

1. Centrifuge the blood collected in EDTA-coated tubes for 10 min at 2,000 x *g* at 4ºC; then re-centrifuge the plasma for 30 min at 15,000 x *g* and adjust the density of the resulted chylomicron-free cell-free plasma to 1.063 g/mL with KBr.
2. Transfer the plasma to ultracentrifuge tubes, finish filling them with KBr density adjusted PBS to 1.063 g/mL and ultracentrifuge the samples for 20 h at 100,000 x *g* at 4ºC.
3. Discard VLDL/LDL top fraction and collect the HDL-containing lower fraction. Re-adjust the density of the sample to 1.21 g/mL with KBr, deposit it in a new ultracentrifuge tube and fill the tube as before with KBr density adjusted PBS to 1.21 g/mL. Run the centrifuge at 100,000 x *g* for 18 h at 4ºC and collect the top fraction of the tube.

For FPLC-based purification, the use of a Superose 6 10/300 GL column (GE Healthcare, IL, USA) mounted on an automated FPLC chromatography system is recommended. This column allows the user to separate components in a range between 5-5000 kDa (Mw of lipoproteins: >5000 kDa VLDL, 2000-5000 kDa LDL, 200-400 kDa HDL) with high resolution results.

ApoA-I purification from plasma – Cold ethanol precipitation method

1. Centrifuge the blood collected from donors in EDTA-coated tubes for 10 min ant 2,000 x *g* at 4ºC. Transfer the plasma to a new container, adjust pH to 5.85 and add ethanol to a final concentration of 19% (% Vol) at -5 ºC. Then separate the precipitate from the supernatant by filtration or centrifugation (*e.g.* 15,000 x *g* for 10 min), discard the precipitate and add ethanol to the supernatant to a final concentration of 40%, pH 5.85, at -8ºC. Save the precipitate (Kistler-Nitschmann's Precipitate IV).
2. Resuspend the last precipitate in 4 volumes (w/v) of a buffer resulting in a final concentration of 65% ethanol, 10 mM $NaCO_3$, 1 mM EDTA, pH 7.3. Filter/centrifuge the suspension and lower the pH of the filtrate to 5.5. Collect the precipitated ApoA-I by filtration or centrifugation.
3. The obtained ApoA-I is delipidated with 94% ethanol and solubilized in 3 volumes of 4 M GdnHCl, 1 mM EDTA, pH 5.2. Pasteurize purified ApoA-I for 10 h at 60ºC, adjust the pH to 7.5 and desalt the preparation to 10 mM NaCl by gel filtration.

Recombinant ApoA-I purification

1. Express the recombinant ApoA-I in transformed *E. coli* (LB medium, 0.4 mM IPTG, 3 h, 37ºC) and sonicate in ice the pelleted bacteria resuspended in buffer (20 mM Tris-HCl, 0.1% (v/v) IGEPAL® CA-630, 1 mM PMSF, protease inhibitors, pH 8.0).

2. Clarify the lysate, add equilibration buffer to a final concentration of 20 mM $NaPO_4$, 0.5 M NaCl, 3 M GdnHCl, pH 7.4, and incubate it with a preequilibrated nickel charged IMAC resin for 1 h at 4ºC.
3. Mount the pre-loaded resin on a chromatography column and, by gravity, wash extensively with equilibration buffer, washing buffer (20 mM $NaPO_4$, 0.5 M NaCl, pH 7.4) and a washing buffer supplemented with 0.1% (v/v) Triton™ X-114 (for LPS elimination). Finally, elute the protein with a buffer containing 0.5 M imidazole, pool the ApoA-I containing fractions and dialyze them overnight at 4ºC against storage buffer containing 10% glycerol.

rHDL preparation

1) Sonication

1. Dry a lipid mixture under a N_2 stream until a thin film forms. Then disperse the lipids in buffer vortexing vigorously at a temperature above the higher lipid's gel-to-liquid phase transition temperature (T_m).
2. Sonicate the sample under a stream of N_2 for 30 min at $>T_m$ with a probe sonicator at a power setting of 100 W. After that, lower the temperature to 39-42ºC, resume sonication and add the ApoA-I (approximately in a 1:60 ApoA-I:lipid mole ratio) gradually over 5-7 min. Continue the sonication for 10 min more.
3. Centrifuge the sample at 10,000 x *g* for 20 min at 4ºC to remove aggregates, large vesicles and metal particles and size-separate the populations of nanoparticles by gel filtration (*e.g.* Superdex 200 10/300 GL).

2) Sodium cholate dialysis

1. Form a lipid dispersion as before and partially solubilize the formed multilamellar vesicles adding sodium cholate.
2. Add the ApoA-I to the lipid-cholate mixture (typically, for 9.6 nm rHDL formation, a 1:125 protein:lipid mole ratio is used), vortex gently and maintain the sample agitating overnight at a temperature $>T_m$.
3. Dialyze extensively the nanoparticle preparation for 48 h at 37ºC. Discard the high molecular aggregates and insolubilized vesicles by centrifugation and select the specific sized particles by SEC.

3) Thermal cycling

1. Dissolve the lipids and the peptides in glacial acetic acid (the optimum peptide:lipid mole ratio for ~10 nm nanoparticles formation is 1:7) and lyophilize them.
2. Hydrate the mixture in bicarbonate buffered saline and cycle between $>T_m$ and room temperature until the nanodiscs have formed.

3. Separate the possible peptides that have not been complexed by gel filtration chromatography.

4) Microfluidics

1. Design and fabricate a microfluidic device with three-inlet channel and one outlet so that they converge to create a single mixing zone with dimensions 2 mm wide, 400 μm high and 20 mm long.
2. Inject the organic solution containing the lipids (5 mg/mL) in the middle inlet channel at a rate of 1 mL/min using a programmable syringe pump, and inject the ApoA-I in PBS (0.2 mg/mL) in both the outer channels at a rate of 5 mL/min.
3. Collect the μHDL formed at the outlet of the device, wash them in PBS and concentrate them to desired volume.

5) High-pressure homogenization (HPH)

1. Dry a lipid mixture until a thin film forms by reduced-pressure rotary evaporation and keep drying 1 hour more under a N_2 stream. Then disperse the lipids at 37ºC in a PBS buffer containing ApoA-I using the rotary evaporator.
2. Homogenate the crude suspension by high-pressure microfluidics homogenization (8-12 cycles, 120 psi, refrigerate the solution each cycle with an ice-water bath). Leave overnight the resulting solution at room temperature and next morning centrifuge the solution at 4,000 rpm for 1 h at 4ºC.
3. Filter, concentrate and wash twice the supernatant with PBS. Leave overnight the preparation at 4ºC, re-centrifuge at 4,000 rpm for 1 h at 4ºC and re-filter under clean, sterile conditions.

* **The references to the described methodologies are cited in the main text and included in the *Bibliography* section.**

Supplementary Information ***Table 1***. Pre-clinical studies with ApoA-I mimetic peptides

Peptide	Animal model of atherosclerosis [1]	Treatment	Dose	Effect (% inhibition atherosclerosis lesion) [2]	Ref.
2F	mice on HFD	?	?	0%	[1]
3F-2	10-week old female *apoE*$^{-/-}$ mice on CD	6 weeks treatment; daily IP administration	20 µg/mouse	20% $	[2]
L-4F	9-month old female New Zealand White rabbits on 1% cholesterol diet	1 month treatment; daily SQ administration	10 mg/kg	45% *	[3]
	15.5-month old female *apoE*$^{-/-}$ mice on CD	6 months treatment (regression); daily oral administration in diet (L-4F + 2 mg/mouse niclosamide + 50 µg/mouse pravastatin)	200 µg/mouse	20% *	[4]
	14/28-month female *apoE*$^{-/-}$ mice on CD	4/8 weeks treatment; every other day IP administration	~2.5 mg/kg (4F) / ~5 mg/kg (4F-Pro-4F)	4F= 79% (4 weeks) / ~25% (*ns*; 8 weeks); 4F-Pro-4F= 0% (4 weeks) / ~15% (*ns*; 8 weeks) &	[5]
	30-week old female *apoE*$^{-/-}$ mice on CD	16 weeks treatment; daily IP administration	100 µg/mouse	55% (4F, [K4,15>R]4F) / 20% ([K9,13>R]4F) *	[6]
	30-week old female *apoE*$^{-/-}$ mice	6-8 weeks treatment; daily IP (8 weeks) or RO 3 times week (6 weeks) administration	100 µg/mouse	35% (IP) / 25% (RO) *	-[7]
	~26-week old male *apoE*$^{-/-}$ mice on HFD	16 weeks treatment; daily IP administration (L-4F ± 5 mg/kg simvastatin administered gastrically)	1 mg/kg	50% (L-4F) / 72% (L-4F + simvastatin) *	[8]
	22-week old male *LDLR*$^{-/-}$ mice on HFHSC diet	8 weeks treatment; daily SQ administration	100 µg/mouse	~10% (*ns*) *	[9]
D-4F	10-week old female *LDLR*$^{-/-}$ mice on WD and *apoE*$^{-/-}$ mice on CD	6 weeks treatment; twice daily oral gavage administration (*LDLR*$^{-/-}$) or daily oral administration in drinking water (*apoE*$^{-/-}$)	2.5 mg/mouse (*LDLR*$^{-/-}$) or 0.125-5 mg/mouse (*apoE*$^{-/-}$)	79% (*LDLR*$^{-/-}$) / ~75% (*apoE*$^{-/-}$) $	[10]
	20-week old *apoE*$^{-/-}$ mice on HFHC	4 weeks treatment; daily oral administration in drinking water or IP administration	~750 µg/mouse (oral) or 50 µg/mouse (IP)	evolving lesion= 42-43% (oral, IP); stablished lesion= 0% (oral, IP) $	[11]
	21-week/12-month-old female *apoE*$^{-/-}$ mice on CD	17 weeks (inhibition) or 6 months treatment (regression); daily oral administration in diet (D-4F + 50 µg/mouse pravastatin)	12.5 µg/mouse (inhibition) / 50 µg/mouse (regression)	65% (inhibition) / 29% (regression) $	[12]
	9-month old female New Zealand White rabbits on 1% cholesterol diet	1 month treatment; daily SQ administration	10 mg/kg	55% *	[3]
	15-week old female *apoE*$^{-/-}$ diabetic mice on CD [3]	8 weeks treatment; daily oral administration in drinking water	1 mg/mouse	50% [4], $	[13]
	10-11-month-old female *apoE*$^{-/-}$ mice on WD	8-9 weeks treatment; daily SQ or oral administration in diet	45 mg/kg	~50% (SQ, oral) *	[14]
	18-week old female *LDLR*$^{-/-}$ mice on WD	4-10 weeks treatment; daily IP administration	1 mg/kg	30 (4 weeks) - 65% (10 weeks) *	[15]

18A peptide family

	Rev-D-4F	10-week old female *apoE*$^{-/-}$ mice on CD	6 weeks treatment; daily oral administration in drinking water	1.6 mg/mouse	46% $	[16]
	5F	24-week old female *wt* mice on modified Thomas-Hartroft diet	16 weeks treatment; daily IP administration	20 µg/mouse	40% $	[17]
	L-6F	6-8-month old female *apoE*$^{-/-}$ mice on WD	7 weeks treatment; daily oral administration in diet	60 mg/kg	40-50% *	[18]
	Tg6F tomatoes	7-9-month old female *LDLR*$^{-/-}$ mice on WD	13 weeks treatment; daily oral administration in diet	45 mg/kg		
37pA derivative 5A peptide	***5A***	12-week old male *apoE*$^{-/-}$ mice on HFD	4 weeks treatment; IP administration 3 times a week	30 mg/kg	85% #	[19]
	5A-CH1				40% #	
	5A-C1	12/20-weeks old male *apoE*$^{-/-}$ mice on HFD	4/12 weeks treatment; IP administration 3 times a week		47% (4 weeks) / 30% (12 weeks) #	
	5A-POPC	6-7/10-11-month old female *apoE*$^{-/-}$ mice on CD	13 weeks treatment; IV administration 3 times a week	30 mg/kg	29% (6-7-week old) / 30% (10-11-week old) *	[20]
	5A-SM	28-week old male *apoE*$^{-/-}$ mice on HFHC	6 weeks treatment; IP administration 3 times a week	50 mg/kg	10% (*trend, ns*) *	[21]
					28% *	
	5A-SM:DPPC (1:3.5:3.5)	6-7/10-11-month old female *apoE*$^{-/-}$ mice on CD	13 weeks treatment; IV administration 3 times a week	30 mg/kg	54% (6-7-week old) *	[20]
	ESP24218 (22A) peptide	28-week old male *apoE*$^{-/-}$ mice on HFHC diet	6 weeks treatment; IP administration 3 times a week (± 1.5 mg/kg T1317)	30 mg/kg	28% (*ns*; rHDL) / 40.8% (rHDL-T1317) $	[22]
ETC-642	***ETC-642 preparation (22A-DPPC:SM) (1:3.75:3.75)***	10-14-month old WHHL-MI rabbits on CD	12 weeks treatment; IV administration 2 times a week	15 or 50 mg/kg	0% (15 mg/kg) / 25% (50 mg/kg) €	[23]
	ETC-642 preparation (1:1:1)	20-22-week old male *apoE*$^{-/-}$ mice on HFHC diet	6 weeks treatment; IP administration 3 times a week (± 1.5 mg/kg T1317)	30 mg/kg	28% (*ns*; rHDL or T1317) / 32% (rHDL+T1317) $	[24]
A10	***monomeric-A10***	20-week old female *LDLR*$^{-/-}$ mice on HFD	10 weeks treatment; daily IP administration (40 mg/kg) or oral administration in drinking water	7.5 or 75 mg/kg	IP= 50%; oral= 74% $	[25]
	trimeric-A10				IP= 61%; oral= 50% (7.5 mg/kg) - 64% (75 mg/kg) $	
FAM	***FAMP5***	22-week old male *apoE*$^{-/-}$ mice on HFD	16 weeks treatment; IP administration 3 times a week	10 or 50 mg/kg	20% (10 mg/kg) / 50% (50 mg/kg) *	[26]

	i-FAMP-D1	22-week old male *apoE*$^{-/-}$ mice on HFD	16 weeks treatment; IP administration 2 times a week	50 mg/kg	50% *	[27]
ELK peptides d	***ELK-2A2K2E***	14/26-week old male *apoE*$^{-/-}$ mice on HFD	4/16 weeks treatment; IP administration 3 times a week	30 mg/kg	63% (4 weeks) / 31-38% (16 weeks) $^{\#}$	[28]
	ELK-2A2K2E	12/20-week old male *apoE*$^{-/-}$ mice on HFD	4/12 weeks treatment; IP administration 3 times a week	30 mg/kg	50% (4 weeks) $^{\#}$	[19]
	ELKA-CH1				45% (4 weeks) / <10% (12 weeks)$^{\#}$	
	ELK-2A				75% (4 weeks) $^{\#}$	

$^{-/-}$= null or KO genotype; *ns*= non-significant; IP= intraperitoneal; IV= intravenous; SQ= subcutaneous; RO=retro-orbital; CD= Chow diet; WD= Western diet; HFD= High-fat diet; HFHC= high-fat high-cholesterol; HFHSC= High-fat high-sucrose with added cholesterol; DPPC= 1,2-dipalmitoyl-sn-glycero-3-phosphocholine; SM= sphingomyelin.

[1] ages of the animals have been calculated for the end of the treatment; **[2]** compared to control or non-treated group; data extracted from the text or from graphics; **[3]** diabetes was induced by IP injections of streptozotocin at a dosage of 65 mg/kg daily for 5 consecutive days; **[4]** compared to untreated diabetic *apoE*$^{-/-}$ mice. * data related to inhibition measured in *en face* preparation; $^{\&}$ data related to inhibition measured in the innominate artery; $^{\#}$ data related to inhibition measured in the aortic arch; $^{\$}$ data related to inhibition measured in the aortic root; $^{€}$ data related to inhibition measured as changes in plaque volume % after treatment.

Supplementary Information ***Table 2***. Pre-clinical studies with rHDLs

	rHDL formulation [1, 2]	Animal model of atherosclerosis [3]	Treatment	Dose	Main results	Ref
SRC-rHDL / CSL-111 /	ApoA-I + PC (1:55)	3-4-month old female CHB rabbits on CD	One single IV administration	75 mg/kg	CSL112 infusion rapidly (1 h) raised ApoA-I and HDL levels in plasma, promoted *in vitro* cholesterol efflux and LCAT activation and had strong anti-inflammatory effects	[29]
ETC-216 / MDCO-216	r-ApoA-I Milano + PC	30-week old *apoE*$^{-/-}$ mice on HFHC diet	5 weeks treatment (18 infusions); every other day IV administration	40 and 80 mg/kg	100% inhibition of progression (40 and 80 mg/kg) / 6% and 70% promotion of regression (40 and 80 mg/kg, respectively)	[30]
	r-ApoA-I Milano + DPPC (1:100)	26-week old *apoE*$^{-/-}$ mice on HFHC diet	One single IV administration	400 mg/kg	Treatment promoted HDL-C mobilization after 1 h and reduced plaque lipid and macrophage content 48h after IV infusion	[31]
	r-ApoA-I Milano + DPPC	12-13-week old male New Zealand White rabbits with perivascular injury on cholesterol-rich diet	One single IV administration	250-1000 mg	A single infusion elevated ApoA-I Milano levels in plasma dose-dependently for about 2 days, promoted HDL-C mobilization and plaque area regression of 20-30% at higher doses	[32]
	r-ApoA-I Milano + POPC (1:40)	15-16-week old male New Zealand White rabbits with perivascular injury on cholesterol-rich diet	20 days treatment; IV administration every 4 days	5-150 mg/kg	ETC-216 treatment reduced (5-10 mg/kg doses) and regressed (20-150 mg/kg) total atheroma volume measured by IVUS and MRI	[33]
		9-month old male New Zealand White rabbits with 2 aortic denudations on cholesterol-rich diet	5 days treatment; IV administration on Day 0 and Day 5	75 mg/kg	Treatment resulted in 5.1% regression of the plaque size from baseline and reduction of plaque vulnerability markers	[34]
					Both rHDL (4.1% ApoA-I Milano; 2.6% ApoA-I wt) produced aortic plaque regression compared to baseline; ETC-216 exerts superior anti-inflammatory and plaque stabilizing effects than human wt HDL	[35]
CER-001	r-ApoA-I + SM:DPPG (1:103:3)	9-10-week old male C57BL/6J mice on CD; 10-11-week old *LDLR*$^{-/-}$ mice on high-cholesterol diet	One single RO administration (C57BL7/6J); 15-30 days treatment, IV administration every second day (*LDLR*$^{-/-}$)	10 mg/kg	CER-001 infusion in C57BL/6J mice raised ApoA-I and total cholesterol in plasma and stimulated cholesterol efflux *in vitro*; in *LDLR*$^{-/-}$ mice, reduced plaque size (17-23%, 5-10 dose) and inflammation state and increased cholesterol clearance by the liver into the feces, primarily in the form of unesterified cholesterol	[36]
		11-12-week old *apoE*$^{-/-}$ mice with carotid artery ligation on high-cholesterol diet	2 weeks treatment; RO administration every second day	2-50 mg/kg	CER-001 treatment induced U-shaped dose-response curve in ligatured carotids lipid content (2-10 mg/kg mobilized lipids; >20 mg/kg inhibited) and reduced ABCA1 expression dose-dependently	[37]
		New Zealand White rabbits on CD; healthy volunteers	One single IV administration (rabbits and healthy patients)	2.5-20 mg/kg rabbits; 0.25-45 mg/kg healthy	Rabbits: CER-001 treatment increased plasma ApoA-I and PL, and mobilized cholesterol in a dose-dependent manner at all doses; healthy patients: CER-001 treatment caused ApoA-I, SM and TG elevation in plasma and cholesterol mobilization; all dose were safe and well tolerated	[38]

				patients		
Other preparations	rabbit ApoA-I + PLPC or DPPC (1:200)	29-week old male New Zealand White rabbits with balloon denudation of the abdominal aorta on HCD	5 days treatment; IV administration on Day 1 and Day 3	8 mg/kg	The treatment with ApoA-I:PLPC rHDL reduced plaque lesion area by 36%, as native HDLs did, while treatment with ApoA-I:DPPC rHDL did not lead to significantly change; both rHDL preparations stabilized the atheroma plaque	[39]
	trimeric ApoA-I + DMPC (1:125)	9-10-week old male *LDLR*$^{-/-}$ mice on WD	24 days treatment; IP administration, 2 times a week	25 or 100 mg/kg	Trimeric ApoA-I exhibited prolonged retention time in plasma compared with WT ApoA-1; trimeric-apoA-1 rHDL treatment did not significantly improve the total area of lesions, but improved their severity	[40]
	ApoA-I (V156E, V156K or R173C) + POPC:Chol (1:100:5)	18- (V156E) or 21- (V156K, R173C) week old male *apoE*$^{-/-}$ mice on WD	One single IV administration	120 mg/kg (V156K, R173C) or 150 mg/kg (V156E)	V156E rHDL was more potent than wt rHDL on plaque lesion reduction (40% *vs.* 26%); V156K and R173C rHDL were also more potent than wt rHDL on plaque reduction (50-55% *vs.* 30%); all variants had better anti-inflammatory profile than wt rHDL	[41, 42]
	apoA-1 (N74C) + soyPC (1:120)	20-21-week old male *apoE*$^{-/-}$ mice with carotid collar on HFHC diet	30 days treatment (3 infusions); IV administration, one every 10 days	40 mg/kg	N74C rHDL treatment reduced atherosclerotic lesion by 58%, 20% more than wt rHDL	[43]
	PEG-rHDL (*CSL-111*)	At least 6-week old *apoE*$^{-/-}$ mice on WD	2 weeks treatment; IV administration, once a week	40 mg/kg	PEGylation increased the half-life in plasma of rHDL by 7-fold; PEGylated-rHDL induced 30-40% more plaque reduction than non-PEGylated rHDL at the same dose and stabilized more the plaque	[44]
	TA-d-rHDL (*ApoA-I + soyPC:cholesterol + TA*) TA-s-rHDL (*ApoA-I + soyPC:Chol:CE:TG + TA*)	16-week old male New Zealand White rabbits on HFD and 3 injections of 100 mg/kg BSA at the beginning	8 weeks treatment; IV administration, every other day	60-180 μg/kg TA	TA-d-rHDL and TA-s-rHDL improved Tanshinone II PK and PD *in vivo*; TA-s-rHDL had more potent anti-atherogenic effect	[45]
	[S]-rHDL (*ApoA-I + MHPC:DMPC + simvastatin*)	32/34-week old male *apoE*$^{-/-}$ mice on HCD	1 or 12 weeks treatment; IV administration 2 times (12 weeks) or 4 times a week (1 week)	10 mg/kg and 40 mg/kg	[S]-rHDLs accumulated at the atherosclerotic lesion and reduced significantly both the total plaque and the macrophage-rich area when administrated for 12-week (10 mg/kg) or 1-week (40 mg/mL); this action was more potent than of bare rHDLs	[46]
	HA-LT-rHDL (*ApoA-I + PC:OL:Chol:CE:TG + LT + HA*) AA-LT-d-rHDL (*ApoA-I + soyPC:Chol + LT + AA*) ST-HA-(C)-PLGA-rHDL (*ApoA-I + soyPC:DPPE:OL +*	16-week old male New Zealand White rabbits on HFD and 3 injections of 100 mg/kg BSA at the beginning	8 weeks treatment; IV administration, every other day	0.4 mg/kg LT	Both HA and AA decorated LT-rHDLs had diminished plasma elimination rate, improved biodistribution toward plaque, lowered liver accumulation and significantly improved atheroprotective efficacy; HA directs rHDL toward CD44 up-regulated inflammatory cells of the atherosclerotic plaque, AA probably inhibits LCAT and subsequent drug release and PLGA improves PK characteristics	[47-49]

	TPGS:PLGA + LT + HA)					
	TRAF6i-rHDL (*ApoA-I + MHPC:DMPC + SMI 6877002*)	18-week old male *apoE*$^{-/-}$ mice on CD (therapeutic study); 27-week old female *apoE*$^{-/-}$ mice on HCD and non-human primates (efficacy/safety study)	6 weeks treatment; IV administration 2 times a week (therapeutic study) / 7 days treatment; IV administration 4 times a week (efficacy/safety study)	10 µmol/kg (therapeutic study) 5 mg/kg (efficacy/safety study)	TRAF6i-rHDL treatment reduced plaque volume, macrophage content and inflammation due to impaired monocyte recruitment; treatment had favorable toxicity profile in both mice and non-human primates	[50, 51]
	HA-NP (*ApoA-I + PC:DOPE:Chol + AT + LOX-1 siRNA + PLGA + HA-DOPE*)	33-week old male *apoE*$^{-/-}$ mice on HCD	12 weeks treatment; IV administration, 2 times a week	7 mg/kg AT	HA200-NP treatment reduced plaque size by 39%, lipid accumulation by 63% and pro-inflammatory macrophage content by 68%	[52]
	LT-GM1-rHDL (*ApoA-I + eggPC:OL:Chol:CE :TG:GM1 + LT*)	46-week old male *apoE*$^{-/-}$ mice on HCD	16 weeks treatment; IV administration, 3 times a week	0.4 mg/kg LT	GM1 modified LT-rHDL had prolonged circulation time, improved biodistribution and enhanced inhibitory effect in atherosclerosis	[53]
	LOV-s-rHDL (*ApoA-I + soyPC:Chol:CE:TG + LT*)	5-6-moth old male *apoE*$^{-/-}$ mice on HFD	8 weeks treatment; IV administration, 2 times a week	7 mg/kg LT	Optimized LOV-s-rHDL treatment (10:1 Dm_{LOV}:Dm_{rHDL}) had potent plaque targeting efficacy and reduced plaque size by 25%; LOV-s-rHDL exhibited a favorable safety profile	[54]
	apoA-1/PS-NP (*ApoA-I + PC:PS:Chol + SR-A siRNA + catalase + pitavastatin*)	33-week old male *apoE*$^{-/-}$ mice on HCD	12 weeks treatment; IV administration, 2 times a week	0.5mg/kg siRNA, 1.4 mg/kg PT, and 90 µg/kg CA	ApoA-I/PS-NP had improved targeting, accumulation and therapeutic efficacies	[55]

$^{-/-}$= Null or KO genotype; ApoA-I Milano= R173C ApoA-I; rHDL= Recombinant HDL; d-rHDL= Discoidal rHDL; s-rHDL= Spherical rHDL; HeFH= Heterozygous Familial Hypercholesterolemia; IV= Intravenous; RO= Retro-orbital; CD= Chow diet; HFD= High-fat diet; HCD= high-cholesterol diet; HFHC= high-fat high-cholesterol; WD= Western-diet; ACS= Acute coronary syndrome; IVUS= Intravascular ultrasound ; QCA= Quantitative coronary angiography; CAD= Coronary artery disease; SAD= Single-ascending dose; Chol= Cholesterol; FC= Free cholesterol; UC= Unesterified cholesterol; CE= Cholesterol ester; HDL-C= High-density lipoprotein cholesterol; VLDL= Very-low-density lipoprotein; LDL= Low-density lipoprotein; T2D= Type-2 diabetes; r-pro-apoA-1 / r-apoA-1= Recombinant version of pro-apoA-1 or apoA-1; PC= Phosphatidylcholine; soyPC= Soybean PC; DMPC= 1,2-dimyristoyl-sn-glycero-3-phosphocholine; MHPC= 1-myristoyl-2-hydroxy-sn-glycero-3-phosphocholine; DPPC= 1,2-dipalmitoyl-sn-glycero-3-phosphocholine; POPC= 1-palmitoyl-2-oleoyl-glycero-3-phosphocholine; PLPC= 1-palmitoyl-2-linoleoyl-sn-glycero-3-phosphocholine; DPPG= 1,2-dipalmitoyl-sn-glycero-3-phospho-(1'-rac-glycerol); DPPE= 1,2-dipalmitoyl-sn-glycero-3-phosphoethanolamine; SM= Sphingomyelin; TG= Triglyceride; OL= Octadecylamine; PLGA= poly(lactic-co-glycolic acid); TPGS= D-α-Tocopherol polyethylene glycol 1000 succinate; TA= Tanshinone IIA; LT= Lovastatin; AT= Atorvastatin; HA= Hyaluronic acid; AA= Arachidonic acid.

[1] apoA-1 sequence is of human origin, if not otherwise stated; [2] protein:lipid relation in molar ratio (mol:mol); [3] age of the animals have been calculated for the end of the treatment.

Supplementary Information ***Table 3***. Clinical trials with rHDLs

	CT number [1] /Phase	Treatment	Dose	# patients	Condition or disease	Main results	Ref.
Proapo A-I liposom	Phase 1	One single IV administration	40-50 mg/kg	4	HeFH	ProapoA-I liposomes treatment elevated ApoA-I and HDL-C levels in plasma and enhanced body cholesterol excretion; no safety-related abnormalities were observed	[56]
SRC rHDL / CSL-111 / CSL112	Phase 1	One single IV administration	25 and 40 mg/kg	7	Healthy volunteers	SRC-rHDL treatment increased dose-dependently ApoA-I and PC levels during the infusion; pre-β1-HDL particles increased concomitantly as well as the UC in HDL fraction and the TG in non-HDL fraction; no evidence of an acute-phase response or other clinical effects were observed	[57]
	Phase 1	One single IV administration	80 mg/kg	7	Hypercholesterolemic patients	SRC-rHDL infusion significantly increased plasma C-HDL levels, but not of TGs	[58]
	NCT00225719 / Phase 2	4 weeks treatment; IV administration, once a week	40/80 mg/kg	183	ACS	ERASE trial: CSL-111 treatment at 40 mg/kg did not reduce significantly the PAV compared with placebo; 80 mg/kg dose was discontinued early because of liver function test abnormalities	[59]
	Phase 1	One single IV administration	80 mg/kg	20	CAD patients	CSL-111 treatment caused a significant increase of total cholesterol, LDL-C, HDL-C and TG in plasma after infusion; treatment also stimulated cholesterol efflux *in vitro*, reduced lipids and macrophage size (both by 60%) in the atheroma plaque and improved anti-inflammatory markers	[60]
	Phase 1	One single IV administration	80 mg/kg	13	T2D patients	CSL-111 treatment increased levels of ApoA-1 and HDL-C in plasma and improved anti-inflammatory properties; CSL-111 actively promoted both *in vitro* and *in vivo* cholesterol efflux from tissues and required apo-B containing lipoproteins; unmodified rHDLs accounted for only a proportion of the increment in cholesterol efflux capacity	[61, 62]
	NCT01129661 / Phase 1	One single IV administration	5-135 mg/kg	57	Healthy volunteers	CSL112 treatment caused an immediate elevation of ApoA-I and a rapid rise in all measured parameters of cholesterol transport: increased cholesterol efflux capacity, increased cholesterol mobilization from tissues and increased cholesterol esterification; TG levels were not altered after infusion at any time of the study	[63]
	NCT01281774 / Phase 1	4 weeks treatment; IV administration, once or twice a week	3.4 g (1-2 weekly) or 6.8 g (1 weekly)	36	Healthy volunteers	CSL112 treatment raised ApoA-1 levels in plasma in a dose-proportional manner and maintained above baseline approximately 3 days; MAD was safe and well-tolerated	[63, 64]
	NCT01499420 / Phase 2a	One single IV administration	1.7 g, 3.4 g or 6.8 g	45	Stable atherothrombotic disease	CSL112 treatment increased approximately in 2 hours ApoA-1 levels in plasma by 182 mg/dL (6.8 g), maintained raised for 2 days and mobilized cholesterol as HDL-C; SAD was well tolerated and there was no significantly increased bleeding risk when administered with antiplatelet therapies; PK, formation of pre-β1-HDL and body cholesterol mobilization were similar to that observed in healthy subjects after CSL112 infusion	[~~63,~~ 65-67]
	NCT02427035 / Phase 1	One single IV administration	2 g or 6 g	32	Moderate RI	PK profiles in plasma of ApoA-I and PC were similar between moderate RI and normal renal function groups and comparable to previous data; moderate renal impairment did not impact the ability of CSL112 to enhance cholesterol efflux capacity; no treatment-related SAEs or	[68, 69]

						clinically significant renal or hepatic safety changes were observed	
	NCT02108262 / Phase 2b	4 weeks treatment; IV administration, once a week	2 g or 6 g	1258	Acute MI	AEGIS-I trial: CSL112 treatment was well tolerated with no associated alterations in either liver or kidney function or other safety concerns; the risk for the composite of MACE was not improved with CSL112 administration compared to placebo; infusion of CSL112 caused a dose-dependent elevation of ApoA-I and favored cholesterol efflux capacity	[70, 71]
	NCT02742103 / Phase 2	4 weeks treatment; IV administration, once a week	6 g	83	Acute MI and moderate RI	CSL112-2001 trial: CSL112 treatment did not increase renal SAE or AKI events; treatment caused elevation of ApoA-I and cholesterol efflux capacity improvement similarly between acute MI patients and subjects with normal renal function	[72]
	NCT03473223 / Phase 3			17400	ACS	AEGIS-II trial: *Current primary outcome= Time to first occurrence of any component of composite of MACE*	
ETC-216 / MDCO-216	Phase 1	IV administration, SAD	5-100 mg/kg	28	Healthy volunteers	ETC-216 treatment induced an initial HDL-C elevation but an afterwards transient HDL-C diminution and mild triglyceridemia; SAD was well tolerated up to 50-75 mg/kg	[73]
	Phase 2	5 weeks treatment; IV administration, once a week	15 or 45 mg/kg	57	ACS	The ApoA-1 Milano Trial: In the combined group, ETC-216 treatment reduced from baseline by 2.7% the PAV, by 5.3% the total atheroma volume and by 9.8% the atheroma volume in the most severely diseased 10-mm subsegment; treatment also reduced by 4.8% the EEM volume from baseline and this reduction correlated with decreased atheroma volume	[74, 75]
	Phase 1	IV administration, SAD	5-40 mg/kg	48	Healthy volunteers and stable CAD patients	MDCO-216 treatment induced rapid (2h) and dose-dependent elevation of plasma ApoA-I and pre-β1, α-1, and α-2 HDL levels and decreased α-3 HDL in healthy subjects and stable CAD patients; pre-β1-HDL and α-1 HDL increments correlated strongly with cholesterol efflux; MDCO-216 treatment also increased dose-dependently plasma FC (peak at 8 h), firstly associated to HDL (bigger in size) and VLDL and later to LDL; doses >20 mg/kg also increased TGs, primarily in VLDL, but in both cohorts at 8 h; MDCO-216 was safe, well-tolerated and did not induce adverse immunostimulation effects seen before with ETC-216 in both study populations	[76-79]
	NCT02678923 / Phase 1-2	5 weeks treatment; IV administration, once a week (+ statins)	20 mg/kg	122	ACS	MILANO PILOT trial: Compared to placebo, MDCO-216 treatment did not reduced PAV not even in the most plaque burdened 10-mm segment; the treatment stimulated *in vitro* cholesterol efflux; infusions were well tolerated	[80]
	2014-005462-30 / Phase 2			600	ACS	MILANO DRIVE trial: Prematurely discontinued due to lack of efficacy	
CER-001	NCT01515241 / Phase 2			10	HeFH	EXPRESS trial	
	NCT01201837 / Phase 2	6 weeks treatment; IV administration, once a week	3-12 mg/kg	507	ACS	CHI SQUARE trial: Infusions of CER-001 at 3-12 mg/kg dosage did not reduce coronary atherosclerosis on IVUS and QCA when compared with placebo; no statistically significant differences in MACE were found btween groups; the treatment was well tolerated; a *post hoc* analysis concluded that the greatest reduction occurred in those ACS patients who presented with higher baseline PAV and were treated with 3 mg/kg CER-001 dosage	[81, 82]

	NCT01412034 / Phase 2	22 weeks treatment; IV administration, once every 2 weeks	8 mg/kg	23	HoFH	MODE trial: CER-001 treatment elevated ApoA-I levels and TG in plasma 1 h after infusion and reduced carotid artery wall thickness at the end of 12 weeks treatment	[83]
	NCT02484378 / Phase 2	10 weeks treatment; IV administration, once a week (+statins)	3 mg/kg	293	ACS	CARAT trial: CER-001 treatment in the presence of statins did not reduce the PAV; infusions were well tolerated, with no differences in clinical and laboratory adverse events observed between groups	[84, 85]
	2011-006188-23 / Phase 2	26 days treatment (28 infusions); IV administration: 1 infusion day 0, 3 infusions once weekly between days 8-29, and 11 infusions 2 weekly between weeks 6-26	8 mg/kg	7	FHA	SAMBA trial: 1-month treatment (9 infusions) increased ApoA-I and HDL-C levels directly after infusions, stimulated *in vitro* cellular cholesterol efflux and presented a trend toward increased FSE; treatment favored carotid mean vessel wall area thinning compared with baseline; CER-001 infusions were generally well-tolerated and study-drug-related AEs were mild	[86]
	NCT02697136 / Phase 3	48 weeks treatment (29 infusions) + optional 24 weeks treatment (12 infusions); IV administration, once a week (first 9 infusions) and twice a week (until 29th infusion)		30	FHA	TANGO trial: Unfavorable	

IV= Intravenous; ACS= Acute coronary syndrome; SAD= single-ascending dose; MAD= multiple-ascending dose; RI= Renal impairment (estimated glomerular filtration rate [eGFR] ≥30 and <60 mL/min/1.73 m^2); PK= Pharmacokinetics; SAE= Serious adverse event; MI= Myocardial infarction; AKI= Acute kidney injury; HeFH= Heterozygous Familial Hypercholesterolemia; HoFH= Homozygous Familial Hypercholesterolemia; IVUS= Intravascular ultrasound- ; QCA= Quantitative coronary angiography; FHA= Familial Hypoalphalipoproteinimia; FSE= Fecal sterol excretion; LDL-C= Low-density lipoprotein cholesterol; HDL-C= High-density lipoprotein cholesterol; TG= Triglycerides; MACE= Major adverse cardiac event; EEM= External elastic membrane; PAV= Percentage of plaque atheroma volume. [1] Data obtained from *https://clinicaltrials.gov/* and *https://www.clinicaltrialsregister.eu/* .

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