Supplementary Appendix

Supplement to: Vafai SB, Täubel J, Ashdown T, et al. In vivo base editing of *PCSK9* with VERVE-102 for hypercholesterolemia. N Engl J Med 2026;395:648-59. DOI: 10.1056/NEJMoa2601283

This appendix has been provided by the authors to give readers additional information about the work.

Contents

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Study Monitoring Committee Members

Per protocol charter, voting members consist of the sponsor medical monitor and investigator(s) who have enrolled a patient in the cohort.

Description of VERVE-102

VERVE-102 comprises two drug substances: a synthetic guide RNA and a messenger RNA encoding the adenine base editor. The guide RNA sequence with modifications is provided below. The messenger RNA coding sequence for the adenine base editor protein (ABE8.8-m) and description of modifications is provided below. The full amino acid sequence of the editor has been published.[1] These drug substances are co-encapsulated in a GalNAc-lipid nanoparticle that consists of five lipid excipients: an ionizable lipid, PEG-conjugated lipid, cholesterol, 1,2-distearoyl-sn-glycero-3-phosphocholine, and a trivalent GalNAc-lipid.[2]

Guide RNA drug substance sequence:

5' -

cscscsGCACCUUGGCGCAGCGGgUUUUAGagcuaGaaauagcaaGUUaAaAuAaggcuaGUcc GUUAucAAcuuGaaaaagugGcaccgagucggugcusususu

-3'

Bases in bold target the gene sequence in *PCSK9*.

Abbreviations: A = adenosine; a = 2'-O-methyladenosine; C = cytidine; c = 2'-O methylcytidine; G = guanosine; g = 2'-O-methylguanosine; U = uridine; u = 2'-O-methyluridine; s = phosphorothioate backbone modification

Messenger RNA coding sequence for ABE8.8-m:

atgagcgaggtggagttcagccacgagtactggatgcgccacgccctgaccctggccaagcgcgcccgcgacgagcg

cgaggtgcccgtgggcgccgtgctggtgctgaacaaccgcgtgatcggcgagggctggaaccgcgccatcggcctgca

cgaccccaccgcccacgccgagatcatggccctgcgccagggcggcctggtgatgcagaactaccgcctgatcgacg

ccaccctgtacgtgaccttcgagccctgcgtgatgtgcgccggcgccatgatccacagccgcatcggccgcgtggtgttcg

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acgcccagaagaaggcccagagcagcaccgacagcggcggcagcagcggcggcagcagcggcagcgagacac

ccggcaccagcgagagcgccacccccgagagcagcggcggcagcagcggcggcagcgacaagaagtacagcat

cggcctggccatcggcaccaacagcgtgggctgggccgtgatcaccgacgagtacaaggtgcccagcaagaagttca

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ggtgcagacctacaaccagctgttcgaggagaaccccatcaacgccagcggcgtggacgccaaggccatcctgagcg

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ccgcatcccctactacgtgggcccccctggcccgcggcaacagccgcttcgcctggatgacccgcaagagcgaggaga

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gaccaaggtgaagtacgtgaccgagggcatgcgcaagcccgccttcctgagcggcgagcagaagaaggccatcgtg

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caacagcgacaagctgatcgcccgcaagaaggactgggaccccaagaagtacggcggcttcgacagccccaccgtg

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gcatcaccatcatggagcgcagcagcttcgagaagaaccccatcgacttcctggaggccaagggctacaaggaggtg

aagaaggacctgatcatcaagctgcccaagtacagcctgttcgagctggagaacggccgcaagcgcatgctggccag

cgccggcgagctgcagaagggcaacgagctggccctgcccagcaagtacgtgaacttcctgtacctggccagccacta

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cctacaacaagcaccgcgacaagcccatccgcgagcaggccgagaacatcatccacctgttcaccctgaccaacctg

ggcgccccgccgccttcaagtacttcgacaccaccatcgaccgcaagcgctacaccagcaccaaggaggtgctgga

cgccaccctgatccaccagagcatcaccggcctgtacgagacacgcatcgacctgagccagctgggcggcgacgagg

gcgccgacaagcgcaccgccgacggcagcgagttcgagagccccaagaagaagcgcaaggtgtaa

The messenger RNA is codon optimized and incorporates N[1]-methylpseudouridine triphosphate nucleotides instead of UTP during the transcription reaction.

Nonclinical Assessments of VERVE-102

Genetic consistency at the on-target site across ancestries

In a prior analysis of the consistency of the target site in *PCSK9*, the Genome Aggregation Database (gnomAD v4.0) was used to explore potential single nucleotide variants in the target site that may impact editing efficiency across major genetic ancestry categories.[3] We found that across > 700,000 individuals in the gnomAD v4.0 database, the target site was identical in > 99.9% of all individuals and in > 99.9% of individuals within each ancestry category.[4] These results support the potential for VERVE-102 to be effective broadly across ancestries.

Off-target screening of candidate sites

Our approach to off-target screening consisted of three steps: 1) nomination of candidate sites; 2) deep sequencing of candidate sites using hybrid capture in cells exposed to VERVE-102 at super-saturating doses to maximize sensitivity; 3) confirmatory targeted-amplicon sequencing.

For nomination of candidate sites, we used three complementary biochemical and bioinformatic discovery methods — oligonucleotide enrichment and sequencing (ONE-seq)[5], digested whole-genome sequencing (Digenome-seq)[6,7], and in silico analysis — to nominate DNA sequences from the human genome to carry forward into cellular editing assessments in vitro. For the in silico analysis, Cas-Designer v1.2,[8] an interface to Cas-OFFinder,[9] was used to search the reference human genome for sequences with up to 3 mismatches to the on-target site, or up to 2 mismatches to the on-target site and up to 1 DNA or 1 RNA bulge. The inclusion of this in silico analysis method was intended to ensure

that potential off-target sites that are most similar to the on-target *PCSK9* site are evaluated by experimental screening methods, even if those sites were not detected by ONE-seq or Digenome-seq biochemical discovery assays. Using these approaches, we identified 1171 sites with ONE-seq, 5222 sites with Digenome-seq, and 17 sites with the in silico method. After consolidating across methods, a total of 6081 unique candidate sites were identified.

Next, the 6081 nominated candidate sites were assessed for editing by hybrid capture screening experiments in primary human hepatocytes exposed to super-saturating doses of VERVE-102. Four different PHH lots were used, two Caucasian males, one Caucasian female, and one Hispanic/Latino female. For the hybrid capture experiments, a mean dose > 40 times the required dose for 90% maximal editing at the on-target site in *PCSK9* (EC_{90}) was used to maximize sensitivity. Median total coverage per site in hybrid capture was 127,609. Successful transfection in each lot was tested by assessing editing at the on-target site in *PCSK9*. Net editing at each site was assessed by comparing VERVE-102 transfected cells to untransfected controls. In step 3, further confirmatory analysis using targeted amplicon sequencing was performed on a subset of sites from hybrid capture experiments. In confirmatory sequencing, no off-target editing was observed above the 0.2% frequency threshold at the EC_{90} (confirmed by mean editing at the on-target site of 88%).

Structural variant screening with optical genome mapping

Adenine base editors are engineered not to create double-stranded DNA breaks and therefore should be associated with low risk for large chromosomal rearrangements (i.e., structural variants). To characterize the VERVE-102 editing profile across the entire human genome, the Bionano Saphyr Optical Genome Mapping (OGM) method[10-12] was used to screen for structural variants in PHH that were transfected with VERVE-102 and then compared with untransfected controls. OGM uses fluorophores that bind with high density throughout the genome at CTTAAG motifs, giving rise to a patterned genomic map that can be compared against a reference map and across test conditions for the bioinformatic identification of all major structural variant classes.

We found no evidence for treatment-related structural variant formation following VERVE-102 treatment in two lots of PHH (Fig. S1).

Non-human primate (NHP) studies

VERVE-102 was administered to four cynomolgus monkeys (2 male and 2 female) at a dose of 3.0 mg/kg. Changes from baseline in circulating PCSK9 protein and LDL cholesterol were -83% and -68%, respectively, prior to the scheduled necropsy at month 6.

To assess the selectivity of GalNAc-LNP delivery to hepatocytes we evaluated the biodistribution of *PCSK9* editing across 26 tissue types following the month 6 necropsy (Fig. S2). VERVE-102 delivery was highly selective for liver where mean (±SD) *PCSK9* editing was 57% ±8%. Across the remaining 25 tissues, mean (±SD) *PCSK9* editing $\geq$ 1% was observed only in adrenal glands (4.6% ±1.7%) and spleen (1.4% ±0.2%). The findings of low levels of editing in adrenal glands and spleen tissue are consistent with prior reports of intravenously administered lipid nanoparticle therapies.[13,14] The potential risks of editing a small proportion of cells in adrenal glands and spleen are expected to be low based on the human genetic data showing even complete loss-of-function in *PCSK9* across all cells in the body is not associated with adverse health effects.[15,16]

Mouse vertical transmission study of germline editing

To assess the risk of editing in germline cells and subsequent transmission of the *Pcsk9* edit to offspring, we performed a vertical transmission study in C57BL/6J mice. Male and female mice aged 70-90 days were administered a bolus dose of either vehicle control (25 per sex) or a murine surrogate of VERVE-102 (VERVE-102mu) at a dose of 2 mg/kg (90 per sex) via tail injection. VERVE-102mu contains a different guide RNA to account for sequence differences in the human *PCSK9* and mouse *Pcsk9* at the target site (see below).

Treated mice were then mated with untreated mice. The mating phase began after 14 days for treated females and 42 days for treated males to account for a full cycle of spermatogenesis. Treated females (mated to untreated males) and untreated females (mated to treated males) were allowed to deliver litters and *Pcsk9* editing was assessed in the liver for all animals in the F0 and F1 generations.

In the F0 generation, mean liver editing of *Pcsk9* was 62.7% in treated females and 63.4% in treated males. The expected outcome for liver editing in the offspring of a pair of mice where one parent has experienced editing in a germline cell is 50%. No instance of *Pcsk9* editing was observed in any mice in the F1 generation. Among F1 pups from VERVE-102mu treated F0 mice, 449 of the pups were from treated females and 513 were from treated males.

VERVE-102mu guide RNA

5' -
cscscsAUACCUUGGAGCAACGGgUUUUAGagcuaGaaauagcaaGUUaAaAuAaggcuaGUccGUUAucAAcuuGaaaaagugGcaccgagucggugcusususu

-3'

Bases in bold target the gene sequence in *Pcsk9*.

Abbreviations: A = adenosine; a = 2'-O-methyladenosine; C = cytidine; c = 2'-O methylcytidine; G = guanosine; g = 2'-O-methylguanosine; U = uridine; u = 2'-O-methyluridine; s = phosphorothioate backbone modification

Measurement of Pharmacodynamic Parameters

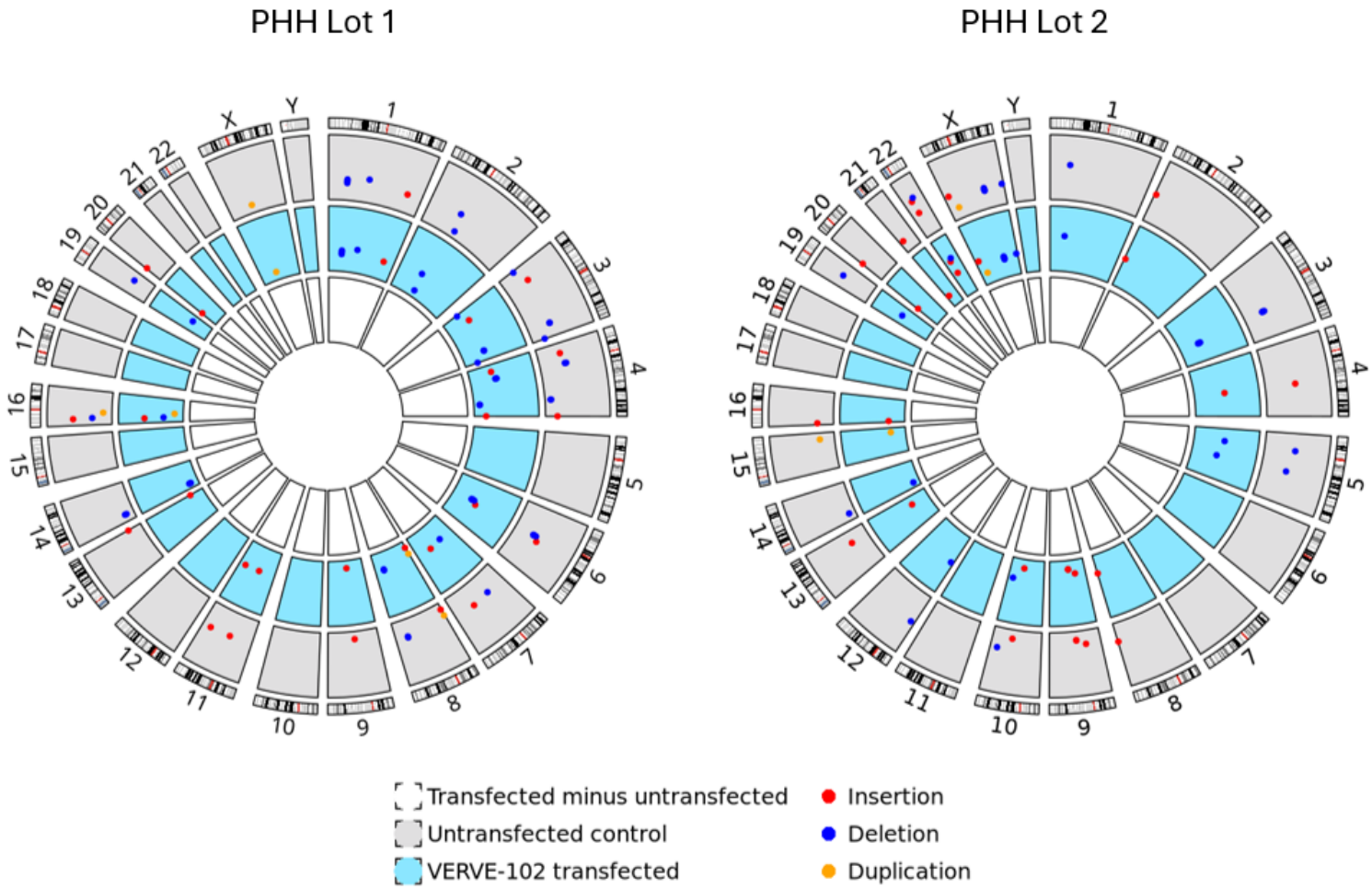
Plasma PCSK9 concentrations were measured using a validated quantitative sandwich enzyme-linked immunosorbent assay (Human Proprotein Convertase 9/PCSK9 Quantikine ELISA Kit, R&D Systems, Minneapolis, MN). Fasting LDL-C was calculated using the Friedewald formula (PPD Laboratories). In cases where the calculated LDL-C was < 40 mg/dL or missing, or triglycerides were ≥ 400 mg/dL values were imputed with direct LDL-C measurements by beta quantification (Mayo Clinic Laboratories, Rochester, MN, USA). In cases where LDL-C results by beta quantification were not available, a direct homogenous LDL-C measurement using an enzymatic method (Cobas® analyzer Roche Diagnostics, PPD Laboratories) was used.

Narrative of One Participant's Serious Adverse Event of Aspiration Pneumonitis

One participant experienced a serious adverse event of Grade 3 aspiration pneumonitis in the 0.45 mg/kg cohort. The participant is a 40-year-old man with a diagnosis of premature coronary artery disease who is taking atorvastatin 80 mg daily and ezetimibe 10 mg daily. He also has a history of gastroesophageal reflux disease and sliding hiatal hernia.

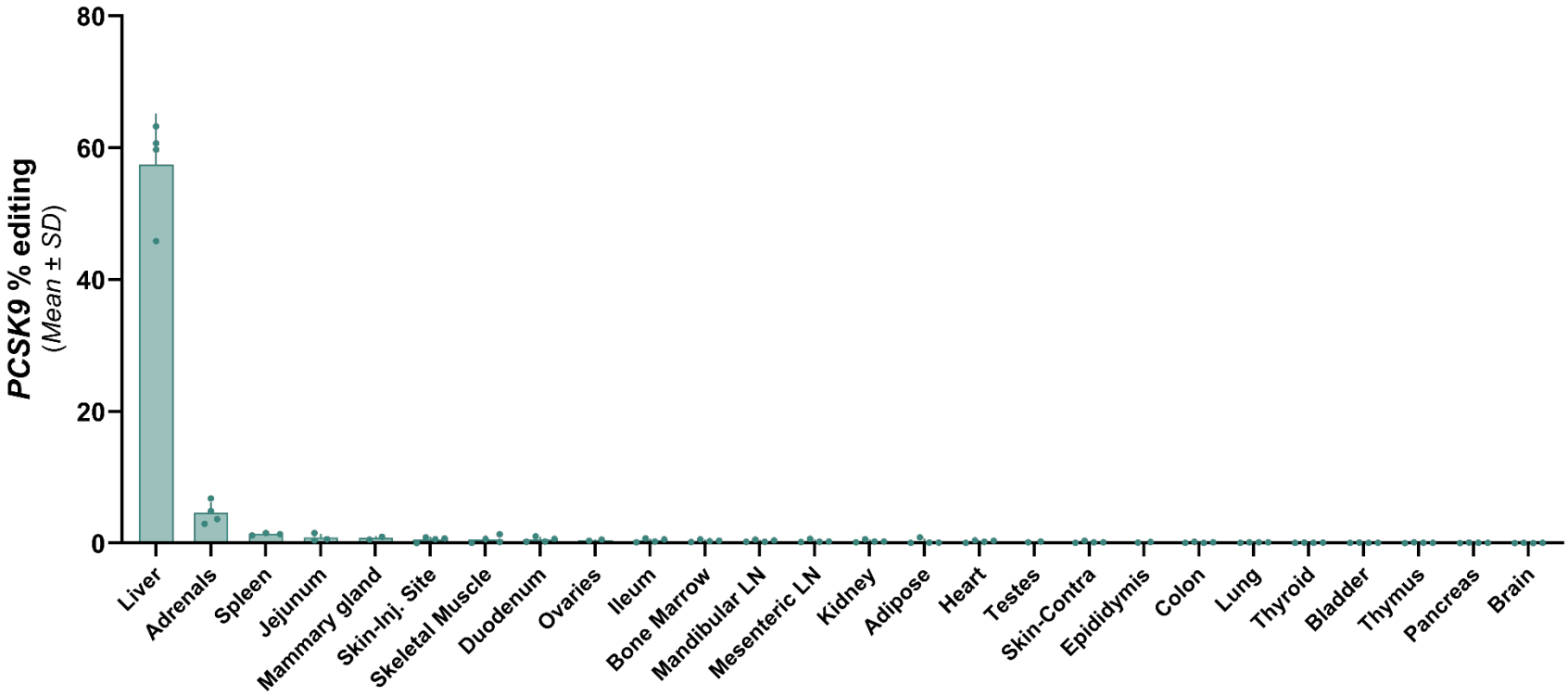
He was enrolled in the 0.45 mg/kg cohort and received a dose of 42 mg total RNA. On Day 1, during the infusion, he experienced an unrelated grade 1 adverse event of headache, and on Day 12, following the infusion, he experienced an unrelated non-serious grade 2 adverse event of respiratory tract infection. On Day 16, the participant awoke with choking and became acutely short of breath and was noted to be pale, sweaty, and bluish around the lips. The participant presented to the emergency room, where he denied chest pain, abdominal pain, fevers, urinary symptoms, or bowel symptoms. Chest x-ray revealed opacification in the right lung, and a computed tomography pulmonary angiogram was negative for pulmonary embolus. Treatment of the event included salbutamol, prophylactic anticoagulant, glucocorticoid, an unspecified antibiotic, IV fluids, oxygen, amoxicillin, and omeprazole. He was admitted to the hospital overnight for observation. The participant's condition improved rapidly, and the participant was discharged from the hospital the next day on a course of oral antibiotics. The outcome of the event was reported as recovered/resolved with sequelae of grade 1 aspiration pneumonitis with resolution on Day 40. The Investigator considered the event to be not related to VERVE-102.

Figure S1. Circos Plot of Structural Variant Screen using OGM



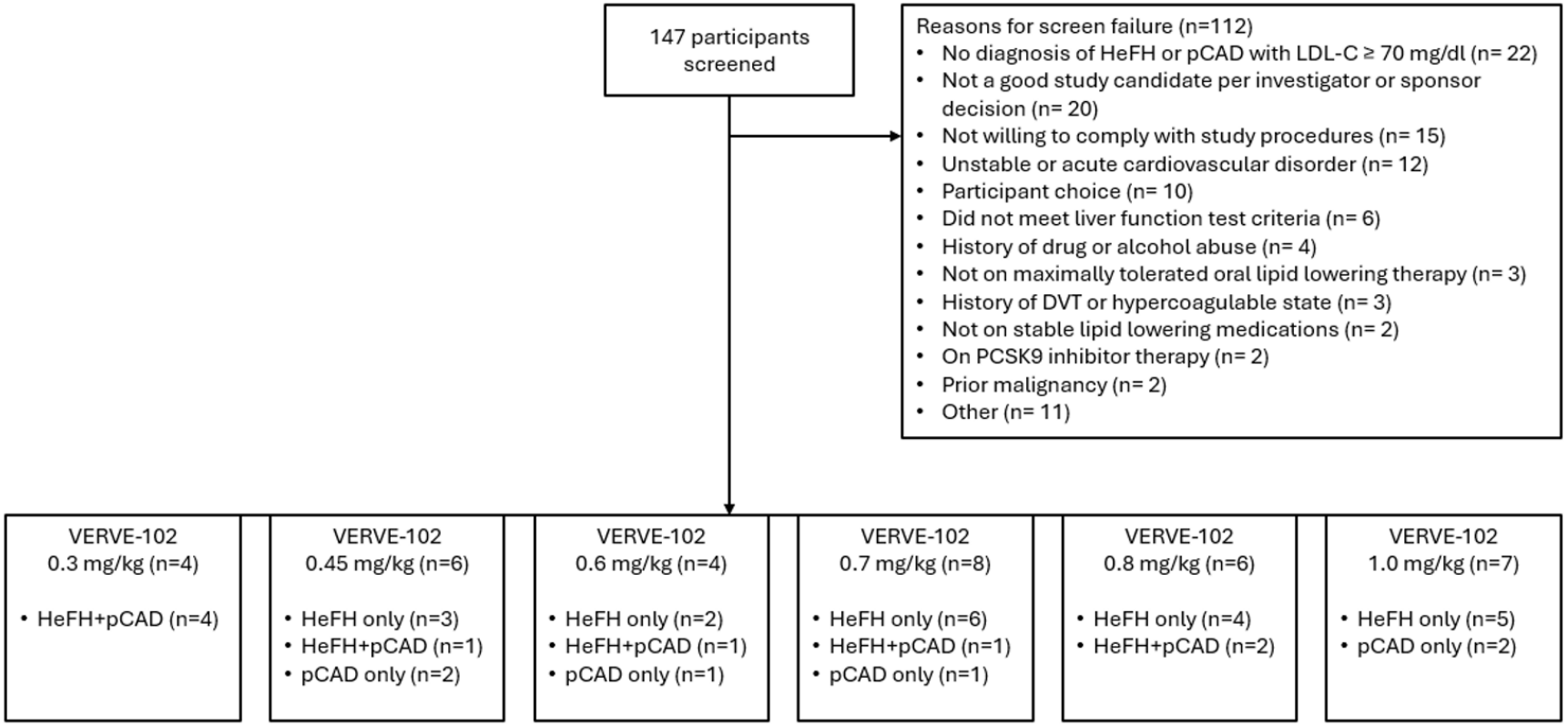
Circos plots comparing structural variants detected using Optical Genome Mapping in untransfected controls (grey boxes) and VERVE-102 transfected samples (blue boxes). The inner ring (white boxes) subtracting untransfected controls from the VERVE-102 transfected cells shows no new structural variants were observed after VERVE-102 treatment.

Figure S2. Biodistribution of *PCSK9* Editing Across NHP Tissues



Percent editing at the intended site in PCSK9 is shown across 26 tissue types in NHPs (two male and two female) treated with a VERVE-102 dose of 3 mg/kg. High selectivity for liver was observed with low levels of editing in adrenal glands and spleen. 'LN' denotes lymph nodes.

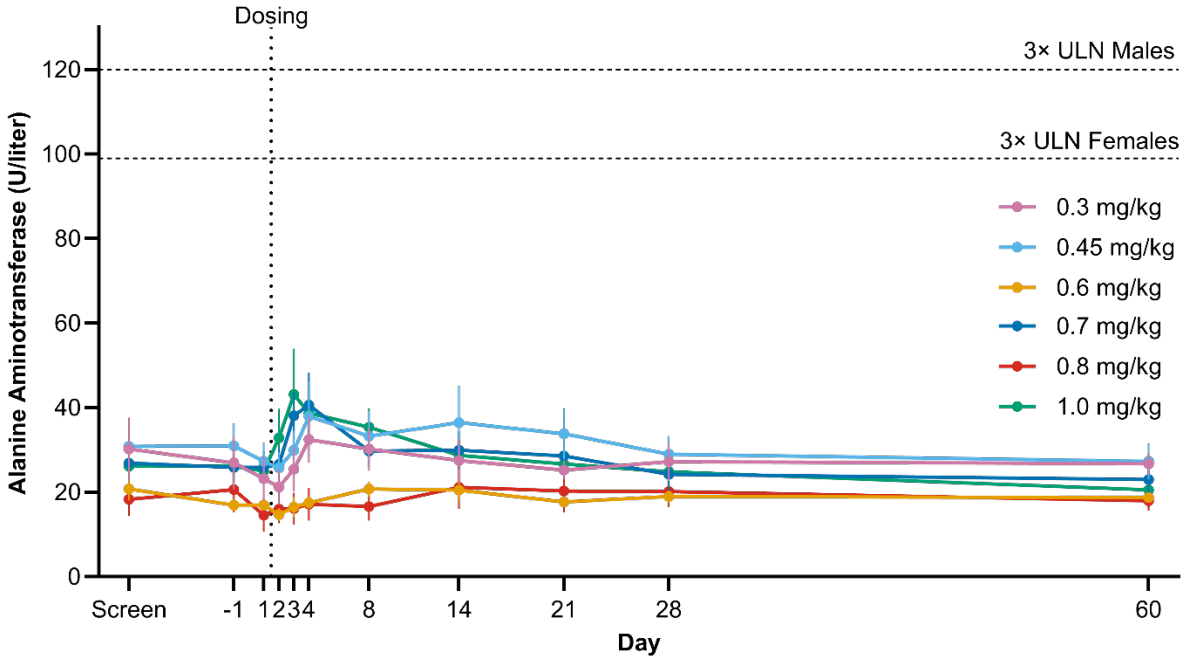
Figure S3. Participants Screened and Treated in Heart-2



From April 17, 2024, through January 28, 2026, 147 participants were screened and 35 received VERVE-102.

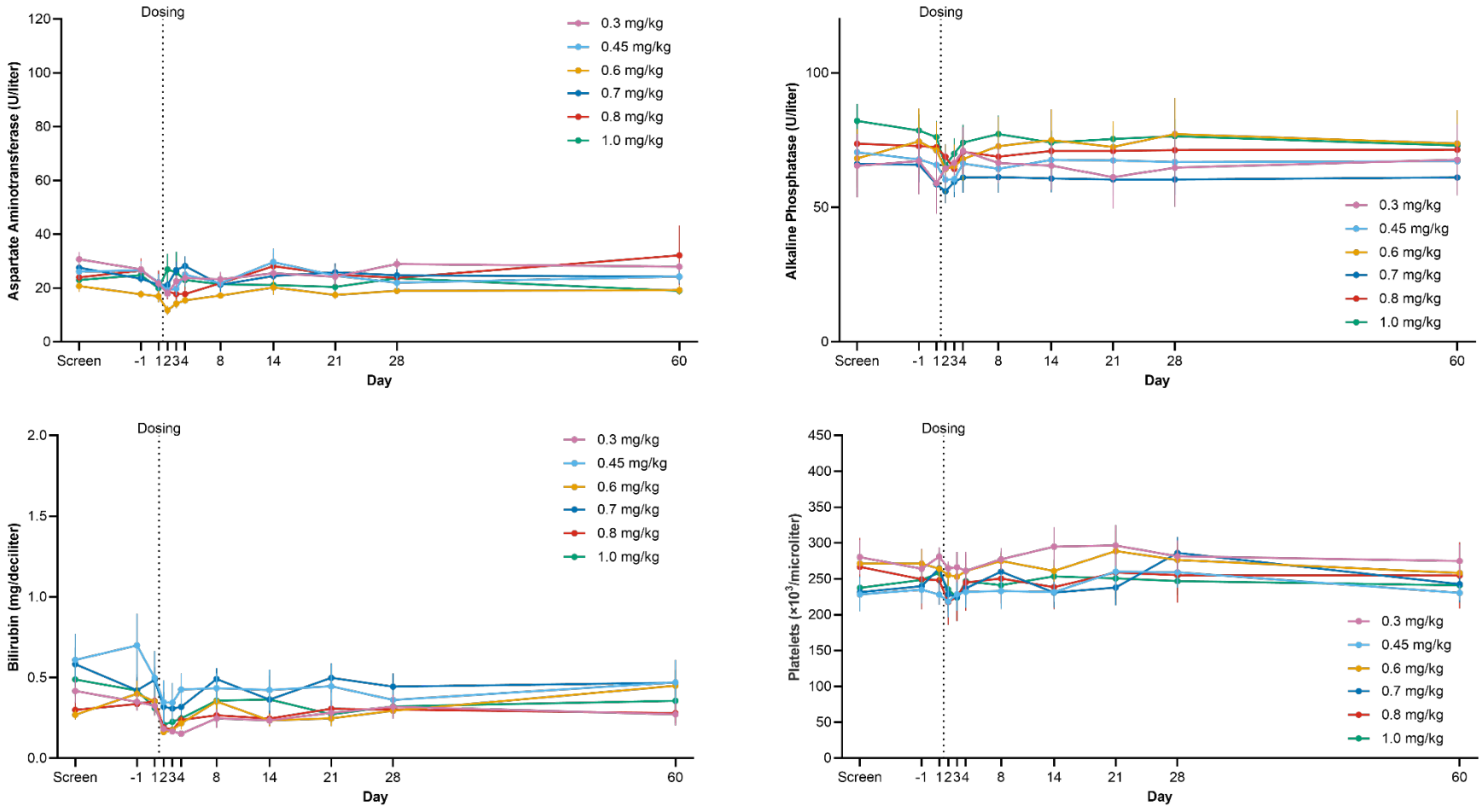
DVT, deep vein thrombosis; HeFH, heterozygous familial hypercholesterolemia; pCAD, premature coronary artery disease; PCSK9, proprotein convertase subtilisin/kexin type 9

Figure S4. Changes In Alanine Aminotransferase After Dosing with VERVE-102



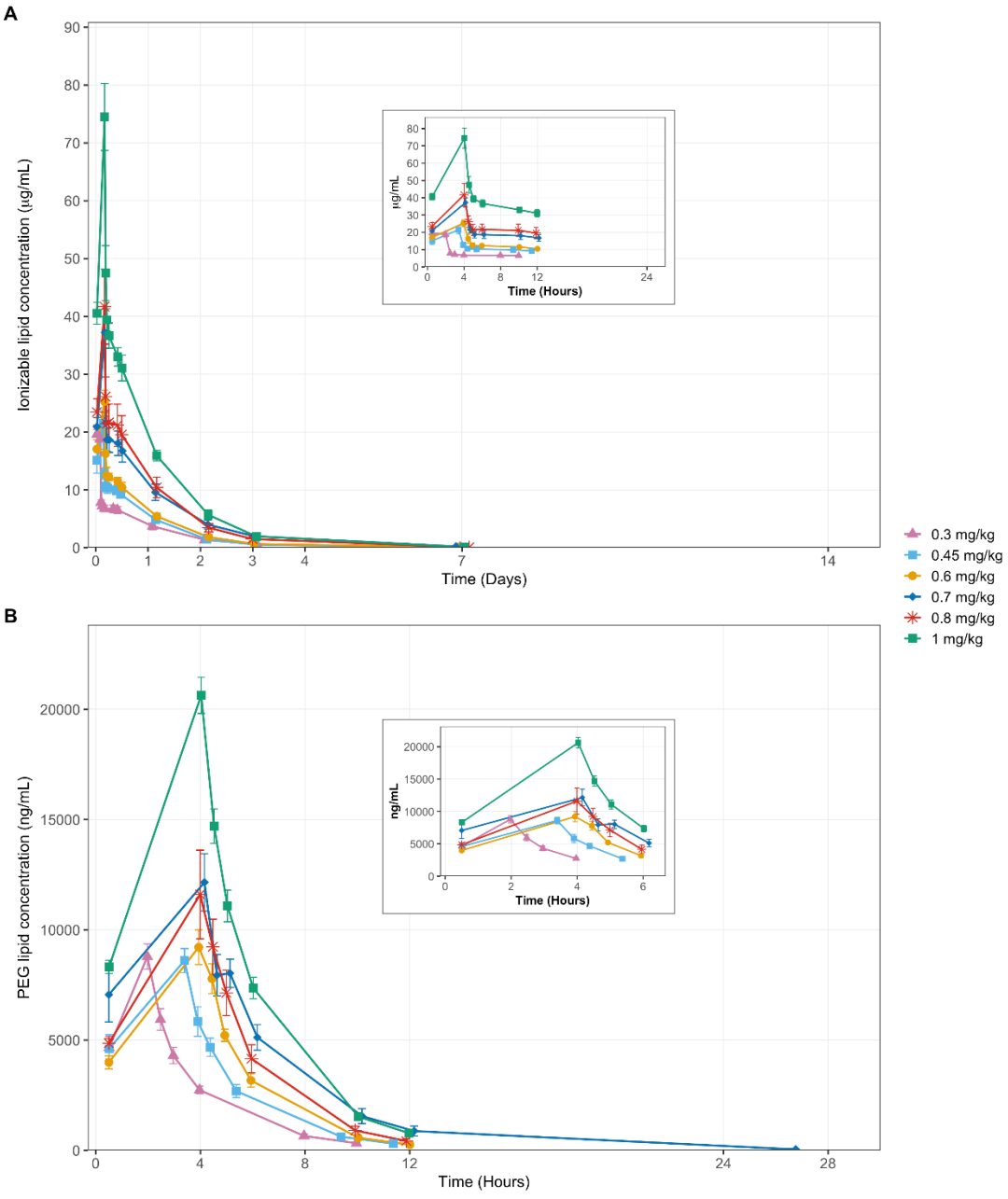
Lines show dose cohort mean values (±SEM) for alanine aminotransferase in U per liter from screening through day 60. Dosing is indicated with a vertical line. Alanine aminotransferase was assessed prior to infusion of VERVE-102 on study day 1. Horizontal lines indicate three times the upper limit of normal for males (120 U per liter) and females (99 U per liter).

Figure S5. Changes In Laboratory Parameters After Dosing with VERVE-102



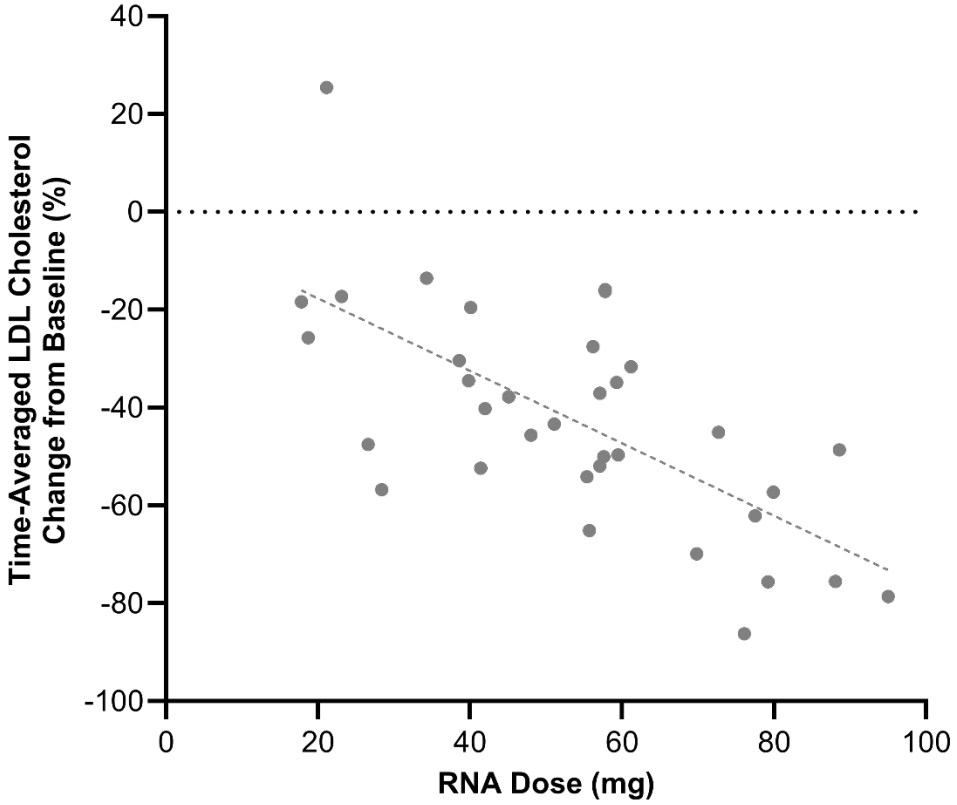
Mean laboratory parameters (±SEM) over time by VERVE-102 dose cohort are shown. The vertical line represents the infusion and day 1 results are from prior to VERVE-102 infusion.

Figure S6. Pharmacokinetics of VERVE-102 Ionizable and PEG Lipids



Data are presented as mean (±SEM) by dose cohort for the ionizable lipid (Panel A) and PEG lipid (Panel B).

Figure S7. Percent Change from Baseline in LDL Cholesterol by Total RNA Dose of VERVE-102



Percent change from baseline in time-averaged LDL cholesterol is shown for each participant based on the total RNA dose of VERVE-102 received (Pearson r = -0.68).

Table S1: Representativeness of the Study Participants to the HeFH and Premature CAD Population

Population	**Heterozygous familial hypercholesterolemia (HeFH) & Premature coronary artery disease (CAD)**
Sex and Gender	Male (69%) and female participants were included in the study. Initially, women of childbearing potential were excluded from participation which may have contributed to a higher proportion of male participants. HeFH is equally common in males and females but there are disparities in treatment and outcomes. Females with HeFH are less intensively treated but males are more likely to experience cardiovascular events.[17] Premature CAD is defined in Heart-2 as disease occurring at or before age 55 in men or age 65 in women, reflecting that onset of disease occurs earlier in males.[18]
Age	Eligible participants for Heart-2 were between 18 and 70 years of age. Participants treated in this interim analysis were between 27 and 66 with a mean age of 52. HeFH is prevalent in younger people but is often undiagnosed,[19] and therefore difficult to address in clinical trials. Age is incorporated into the definition of premature CAD.
Geography	Participants in Heart-2 were mostly white (86%) with 17% and 3% reporting Asian or Black ancestry, respectively. These numbers reflect the countries where clinical trial sites were established and an enrollment of 35 participants. HeFH and premature CAD are global diseases. HeFH is concentrated in specific areas of higher prevalence due to genetic ancestry founder effects.[20]
Overall Representativeness of this Trial	Participants in the trial reflect the inclusion/exclusion criteria for participants with HeFH or premature CAD who require additional lipid lowering. Lack of representativeness across age and genetic ancestry reflect limited diagnosis of HeFH, the countries included in the trial, and the number of participants enrolled.

Table S2: Participants Experiencing Adverse Events Related to VERVE-102

	0.3 mg/kg n=4	**0.45 mg/kg n=6**	**0.6 mg/kg n=4**	**0.7 mg/kg n=8**	**0.8 mg/kg n=6**	**1.0 mg/kg n=7**	**Total n=35**
Injury, poisoning and procedural complications							
Infusion-related reaction	0	0	1	3	1	2	7
Nervous system disorders							
Dizziness	1	0	0	0	0	0	1
Headache	0	0	0	0	0	1	1
Tension headache	0	0	0	0	0	1	1
General disorders and administration site conditions							
Fatigue	0	0	1	1	0	0	2
Gastrointestinal disorders							
Abdominal pain	0	0	0	1	0	0	1
Eructation	0	0	0	1	0	0	1
Nausea	0	0	0	1	0	0	1
Salivary hypersecretion	0	0	0	1	0	0	1
Musculoskeletal and connective tissue disorders							
Arthralgia	0	0	0	1	0	0	1
Respiratory, thoracic and mediastinal disorders							
Hiccups	0	0	0	0	1	0	1
Skin and subcutaneous tissue disorders							
Rash maculo-papular	0	0	1	0	0	0	1

The table includes adverse events with an onset after VERVE-102 treatment. Adverse events were coded using the preferred term in the Medical Dictionary for Regulatory Activities (MedDRA), version 26.1.

Table S3: Changes in Fasting Lipid Laboratory Measures and Remnant Cholesterol by VERVE-102 Dose

	0.3 mg/kg n=4	**0.45 mg/kg n=6**	**0.6 mg/kg n=4**	**0.7 mg/kg n=8**	**0.8 mg/kg n=6**	**1.0 mg/kg n=7**
Mean baseline total cholesterol (range), mg/dl	233.58 (146.3 to 289.7)	230.78 (181.7 to 288.7)	200.75 (177.7 to 226.7)	181.46 (135.3 to 223.7)	211.39 (151.3 to 254.0)	200.05 (126.3 to 318.0)
Mean total cholesterol at day 28 (range), mg/dl	191.5 (120 to 234)	163.5 (127 to 210)	134.8 (108 to 168)	134.1 (76 to 208)	142.7 (80 to 206)	118.9 (72 to 213)
Mean percent change from baseline in total cholesterol (range), %	-18.00 (-24.7 to -8.4)	-29.00 (-36.3 to -23.8)	-33.08 (-41.0 to -25.9)	-26.70 (-59.4 to -7.0)	-31.73 (-68.4 to -16.7)	-40.77 (-62.1 to -27.3)
Mean baseline triglycerides (range), mg/dl	89.00 (54.3 to 141.7)	202.00 (80.0 to 476.7)	159.58 (54.3 to 340.0)	94.29 (46.7 to 202.7)	99.39 (28.3 to 179.0)	141.57 (68.7 to 221.0)
Mean triglycerides at day 28 (range), mg/dl	66.8 (58 to 84)	173.4 (55 to 374)	126.5 (53 to 273)	68.3 (43 to 115)	79.5 (43 to 122)	104.3 (70 to 132)
Mean percent change from baseline in triglycerides (range), %	-19.15 (-40.7 to 10.4)	-16.83 (-34.7 to 32.1)	-13.85 (-35.9 to 2.7)	-18.37 (-45.6 to 12.7)	-10.73 (-32.7 to 51.8)	-18.30 (-47.1 to 25.7)
Mean baseline high-density cholesterol (range), mg/dl	61.42 (45.3 to 74.0)	47.50 (33.3 to 62.3)	48.58 (32.3 to 56.7)	57.92 (30.7 to 103.0)	53.39 (32.7 to 78.3)	43.52 (31.0 to 52.0)
Mean high-density cholesterol at day 28 (range), mg/dl	64.0 (49 to 75)	46.8 (33 to 58)	48.3 (32 to 60)	57.5 (33 to 102)	54.7 (35 to 95)	49.3 (38 to 66)
Mean percent change from baseline in high-density cholesterol (range), %	5.71 (-12.6 to 30.1)	-0.97 (-9.2 to 16.3)	-0.84 (-9.8 to 10.4)	0.42 (-16.4 to 15.6)	0.79 (-11.6 to 21.3)	13.61 (-4.2 to 28.6)

	0.3 mg/kg n=4	**0.45 mg/kg n=6**	**0.6 mg/kg n=4**	**0.7 mg/kg n=8**	**0.8 mg/kg n=6**	**1.0 mg/kg n=7**
Mean baseline non high-density cholesterol (range), mg/dl	172.17 (77.7 to 215.7)	183.28 (120.7 to 247.0)	152.17 (123.3 to 170.0)	123.54 (104.7 to 148.0)	158.00 (88.0 to 221.3)	156.52 (95.3 to 266.7)
Mean non high-density cholesterol at day 28 (range), mg/dl	127.5 (60 to 159)	117.4 (69 to 153)	86.5 (67 to 113)	76.6 (29 to 106)	88.0 (41 to 171)	69.6 (34 to 147)
Mean percent change from baseline in non high-density cholesterol (range), %	-25.43 (-32.2 to -19.6)	-36.16 (-42.8 to -29.4)	-43.53 (-49.6 to -33.5)	-36.77 (-80.2 to -12.2)	-42.48 (-80.5 to -20.5)	-56.26 (-77.2 to -39.6)
Mean baseline very low-density lipoprotein cholesterol (range), mg/dl	17.83 (11.0 to 28.3)	37.22 (16.0 to 76.0)	28.46 (10.7 to 54.5)	18.83 (9.3 to 40.3)	19.94 (5.7 to 35.7)	28.24 (13.7 to 44.0)
Mean very low-density lipoprotein cholesterol at day 28 (range), mg/dl	13.5 (12 to 17)	34.8 (11 to 75)	25.5 (11 to 55)	13.6 (9 to 23)	16.0 (9 to 24)	20.7 (14 to 26)
Mean percent change from baseline in very low-density lipoprotein cholesterol (range), %	-18.68 (-40.0 to 9.1)	-12.76 (-34.9 to 31.8)	-6.81 (-36.7 to 5.4)	-18.33 (-46.7 to 11.4)	-9.44 (-32.7 to 58.8)	-18.66 (-47.7 to 23.8)
Mean baseline apolipoprotein B (range), mg/dl	117.25 (69.3 to 137.7)	125.28 (90.0 to 156.3)	114.33 (98.3 to 128.0)	95.38 (79.3 to 109.0)	112.93 (67.0 to 146.7)	110.48 (76.7 to 179.0)
Mean apolipoprotein B at day 28 (range), mg/dl	90.8 (57 to 114)	86.2 (61 to 110)	69.3 (60 to 88)	61.8 (27 to 79)	72.2 (39 to 135)	53.3 (33 to 101)
Mean percent change from baseline in apolipoprotein B (range), %	-22.05 (-30.2 to -17.2)	-31.07 (-36.5 to -22.3)	-39.67 (-45.5 to -31.3)	-34.01 (-75.2 to -9.9)	-34.55 (-73.4 to -7.7)	-52.39 (-69.8 to -36.5)

	0.3 mg/kg n=4	**0.45 mg/kg n=6**	**0.6 mg/kg n=4**	**0.7 mg/kg n=8**	**0.8 mg/kg n=6**	**1.0 mg/kg n=7**
Mean baseline apolipoprotein A1 (range), mg/dl	151.33 (131.7 to 168.7)	146.33 (108.0 to 188.3)	153.46 (114.5 to 177.0)	148.94 (95.7 to 221.0)	149.39 (110.7 to 198.0)	132.48 (101.3 to 152.0)
Mean apolipoprotein A1 at day 28 (range), mg/dl	156.8 (131 to 175)	140.8 (109 to 174)	151.8 (103 to 187)	142.6 (99 to 220)	154.0 (119 to 221)	147.4 (120 to 163)
Mean percent change from baseline in apolipoprotein A1 (range), %	3.81 (-5.7 to 14.4)	-2.49 (-18.8 to 21.1)	-1.76 (-10.0 to 10.0)	-3.79 (-17.1 to 6.2)	2.69 (-9.2 to 18.6)	11.97 (-4.6 to 21.0)
Mean baseline lipoprotein(a) (range), mg/dl	147.92 (112.3 to 204.7)	52.61 (12.0 to 129.3)	20.29 (8.0 to 50.5)	79.25 (6.0 to 240.0)	56.92 (6.0 to 218.0)	83.00 (6.0 to 232.7)
Mean lipoprotein(a) at day 28 (range), mg/dl	138.8 (86 to 211)	45.0 (9 to 112)	17.0 (6 to 42)	74.1 (6 to 227)	60.8 (6 to 240)	66.4 (6 to 183)
Mean change from baseline in lipoprotein(a) (range), %	-8.04 (-23.4 to 3.1)	-13.70 (-25.0 to 12.9)	-16.71 (-25.0 to 0.0)	-7.73 (-21.7 to 1.6)	-8.00 (-48.4 to 16.0)	-24.54 (-50.0 to 0.0)
Mean baseline remnant cholesterol (range), mg/dl	17.83 (11.0 to 28.3)	43.11 (16.0 to 111.3)	27.92 (10.7 to 52.3)	18.83 (9.3 to 40.3)	18.96 (5.7 to 35.7)	28.24 (13.7 to 44.0)
Mean remnant cholesterol at day 28 (range), mg/dl	13.5 (12.0 to 17.0)	34.8 (11.0 to 75.0)	19.5 (11.0 to 31.0)	11.88 (9.0 to 16.0)	15.5 (8.0 to 24.0)	18.14 (12.0 to 26.0)
Mean change from baseline in remnant cholesterol (range), %	-18.68 (-40.0 to 9.1)	-19.03 (-34.9 to 31.8)	-17.23 (-40.8 to 5.4)	-22.67 (-77.7 to 11.4)	-5.98 (-32.7 to 58.8)	-25.72 (-72.7 to 23.8)

Remnant cholesterol was calculated as total cholesterol minus HDL cholesterol minus LDL cholesterol.

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