SI Appendix

Cell culture

Human Aortic Endothelial Cells (HAECs) were procured from Lonza (Allendale, NJ) (CC-2535). HAEC lines that are heterozygous at rs17114036 were from aortic explants of heart transplant donors in the UCLA transplant program. For CRISPR/Cas9-mediated genome editing, we used teloHAECs (ATCC #CRL-4052) that express hTERT and large-T antigen. teloHAECs were used due to their ability to form colonies, which was necessary for generating isogenic cell lines with deletions. Cells were grown in EGM-2 medium supplemented with SingleQuots from Lonza (CC-3156 & CC-4176) and Antibiotic-Antimycotic from Gibco (Grand Island, NY) (15240062) in a 37°C incubator with 5% CO_2. HAECs were used from passages 6-10. teloHAECs, and CRISPR-edited teloHAECs were used from passages 10-15. THP-1 cells were maintained using RPMI 1640 medium (Gibco) containing 10% FBS (Biowest).

H3K27ac and H3K4me2 chromatin immuno-precipitation with whole genome sequencing (ChIP-seq)

HAECs were washed three times with warm PBS and then trypsinized. Cells were pelleted at 3000 x g for 5 min before being fixed at room temperature with 1% paraformaldehyde in PBS for 10 min, and quenched with 125 mM glycine. 1 million cells were used for each ChIP-seq. Cell lysates were sonicated using BioRuptor Pico (Diagenode, Belgium), and then immunoprecipitated using antibodies against H3K27ac (Active Motif, Carlsbad, CA, #39135) or H3K4me2 (EMD Millipore, Billerica, MA, #07–030), bound to a 2:1 mixture of Protein A Dynabeads (Invitrogen #10002D) and Protein G Dynabeads (Invitrogen #10004D). Following immunopreciptation, crosslinking was reversed and libraries were prepared using the same method described previously (1) for RNA-seq beginning with dsDNA end repair and excluding UDG. For each sample condition, an input library was also created using an aliquot of sonicated cell lysate that had not undergone immunoprecipitation. These samples were sequenced on an Illumina HiSeq 4000 and used to normalize ChIP-seq results.

Chromatin accessibility quantitative trait locus (caQTL) mapping and Allelic Imbalance

ATAC-seq was performed as previously described (2) using Tn5 transposase (Illumina, San Diego CA). Libraries were sequenced on an Illumina HiSeq 4000 according to manufacturer's specifications by the Genomics Core Facility at the University of Chicago. The reads were aligned to the UCSC hg19 genome using Bowtie2 (3).

Chromatin accessibility quantitative trait locus (caQTL) mapping was performed to test for association between genotype at rs17114036 and chromatin accessibility measured by ATAC-seq. We pulled genotypes for HAEC donors from our previous study (4) and imputed linked alleles using IMPUTE2 and SHAPEIT as we published previously (1). Association testing between ATAC-seq tags at the rs17114036 enhancer and genotype were performed using the Combined Haplotype Test in WASP (5). To perform allelic imbalance (AI) analysis that assigns next generation sequencing reads overlapping heterozygous sites to one chromosome or the other, we quantified ATAC-seq tags at the rs17114036 enhancer using HOMER's annotatePeaks function to express the log2 normalized tags in this region.

To test whether alleles at SNP rs17114036 associated with differences in chromatin accessibility, we used the Combined Haplotype Test (CHT) in the software suite WASP (5). The CHT test provides one statistic from jointly modeling two components of association between alleles and sequencing reads. The two components of the model are visualized separately in Figures 4A and C, but result in one p-value (4.21e-04). The first component of the model takes into account the allelic imbalance at phased heterozygous SNPs (Fig. 4C), whereas the second component measures the total read depth in the target region versus the diploid genotype of the individual (Fig. 4A). Therefore the p-value of 4.21e-04 reflects the CHT statistic derived from a joint test of data shown in 4A and 4C.

RNA-seq

Total RNA was isolated from HAECs using the Quick-RNA Micro Prep kit from ZymoResearch (#R1051), including optional DNase I treatment. mRNA was selected through poly-A isolation using Oligo d(T)25 beads (New England BioLabs #S1419S), fragmented, and cDNA was synthesized with the SuperScript III First-Strand Synthesis System (Invitrogen # 18080051). Second strand synthesis was performed using DNA Polymerase I (Enzymatics #P7050L). To generate sequencing libraries, DNA ends were repaired with T4 DNA Polymerase (Enzymatics #P7080L). Six-mer barcoded adapters (BIOO Scientific NEXTflex #514104) were ligated with T4 DNA Ligase (Enzymatics #L-6030-HC- L) and samples were treated with Uracil DNA Glycosylase (UDG) (Enzymatics #G5010L). Libraries were then amplified less than 14 cycles by PCR (Phusion Hot Start II #F549L) and purified (Zymo #D5205) for high-throughput sequencing at the University of Chicago.

Normalization of High-throughput Sequencing Data

Reads from ATAC-seq and ChIP-seq were mapped to the hg19 build of the human genome with Bowtie2 (3) and RNA-seq reads were mapped with STAR (6). For RNA-seq, ATAC-seq, and ChIP-seq mapped reads were normalized to 10 million tags per experiment and PCR duplicates were removed in HOMER (7). The ATAC-seq and ChIP-seq aligned files were then converted to BAM format and sorted using SAMtools (8). The files were then filtered to remove unmapped and mitochondrial reads using SAMtools (8). Peak calling was performed using MACS2 (9). The bedgraph files from MACS2 were converted into BigWig files using HOMER for visualization on the genome browser (7). The images in this manuscript had the peaks smoothed to 3 pixels for aesthetics. Using the RNA-seq datasets, PLPP3 expression was quantified using the 'analyzeRepeats' function, and is expressed as log2 normalized tag counts. Sequencing data generated in this study is available under Gene Expression Omnibus #GSE112340 (https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE112340; token: srqrygwoxhajbgx).

Dual luciferase assay

HAECs and teloHAECs were electroporated using the Neon system (ThermoFisher Scientific). Briefly, the cells were re-suspended in resuspension buffer to have around 40,000 cells per µl. 1.2 µg of eGFP expression plasmid (pmaxGFP) was first used to assess transfection efficiency. For dual luciferase reporter assays, 1 µg of pGL4.23 plasmids carrying firefly luciferase inserted with chr1:56962213-56963412 (T or C at rs17114036, UCSC VERSION hg19) or chr1:56911623-56912823 (A or G at rs2184104, UCSC VERSION hg19) and PLPP3 promoter or minimal promoter at 1 µg/µl

and 200 ng of pRL-TK (control reporters) at 1 µg/µl were added per 8.8 µl of cells. The cells were electroporated at 1200 V, for 1 pulse and then were immediately plated onto 12-well culture plates. Media was changed after 4-6 hours to remove dead cells. The peak expression of plasmids was shown at 24 hours post-electroporation and was chosen as the end-point for most luciferase experiments. Cells were collected by adding passive lysis buffer and then put at -80°C overnight. Luminescence was measured by Dual-Luciferase Reporter Assay System (Promega) according to manufacturer's instructions using a Cytation3 plate reader (BioTek).

CRISPR Cas9-mediated deletion of enhancer in teloHAECs

The CRISPR reagents were adapted from the Alt-R system from IDT (IDT, Coralville, IA). The guide RNAs were designed using an online tool at http://crispr.mit.edu/ to minimize off targeting effects using two guides to create a ~66 bp deletion. The guide RNAs were made by annealing the tracrRNA to the sgRNA. Cas9-guide RNA ribonucleoprotein (RNP) complex by incubating guide RNAs with recombinant S. pyogenes Cas9, followed by the delivery to cells using Lipofectamine RNAiMAX (Thermo). For each successive treatment the reagent amounts were scaled relative to the size of the destination vessel to compensate for the number of cells in the reaction. The volumes for each part of the reaction was increased 4x when treating cells from the 96-well to a 6-well, and 16x when moving from the 6-well to the T-75 flask. After four treatments with the CRISPR RNP, treated cells were trypsinized to a single cell suspension. Using a FACS AriaII flow cytometer single cells were plated onto 96 well plates containing EGM-2 media with 10% FBS for a total of 576 possible clones to screen. The cells were incubated for 20 days prior to grow to confluence. The cells were then split and half of each clone was placed onto two new plates to continue growing clones, and to collect DNA for screening. To confirm the CRISPR Cas9-mediated deletion, DNA for screening was collected using 50 µl QuickExtract solution (Epicentre, Madison WI). Mutations were detected using SYBR green (Roche, Indianapolis, IN) and CRISPR Genotyping Screening primers flanking the deletion region. Positive clones were checked with another PCR using Platinum Taq and CRISPR Genotyping TA Cloning primers flanking the deletion. The PCR products for clones were purified using Qiagen PCR clean-up kit (Qiagen, Venlo, Netherlands). The PCR products were ligated into the pGEMT-easy linear vector, cloned, and then submitted for Sanger sequencing to confirm deletions of the target.
(Guide RNA sequences: 5'-TAGTGATATCAACCATTTGACGG-3', 5'-TGACTTCAGCTCTTGCTGATAGG-3')
(CRISPR Genotyping Screening F: 5'- TCCTCCACGTTTAGTTGCCA -3', R: 5'-AAGGAATCCAGGGTGTAACCG -3')
(CRISPR Genotyping TA Cloning, F: 5'- GGACGCTGGGAATGAGTGAT-3', R: 5'-ATTGCCCATATCTGCAACCC-3')

CRISPR Interference (CRISPRi)

A fusion protein of catalytically dead Cas9 (dCas9) fused to KRAB repressor protein was expressed in HAEC using *in vitro* transcripts. In a 6-well plate, 100 ng of fusion protein and 100 ng of sgRNA were diluted in 50 µl of opti-MEM. 1.5 µl of Messenger MAX was diluted into 50 µl of opti-MEM. The *in vitro* transcripts and Messenger MAX dilutions were combined and incubated for 10 minutes at RT. The complex was then

added to the cells, which were incubated for 8 hours prior to cell lysis, RNA collection, and analysis via qPCR. Non-targeting control guide RNA was purchased from IDT. (Guide RNA sequences targeted to rs17114036: 5'-GTTGATATCACTAAGTTTTCAGG-3', 5'-CAAGAGCTGAAGTCAGGCAGTGG-3')
(Guide RNA sequence targeted to rs2184104: 5'-GGACGACTGCAAACACCAGA-3')

Leukocyte adhesion assay

Monolayers of teloHAECs were activated using lysophosphatidic acid (LPA) for 3 hours. THP-1 cells at 5-fold number of teloHAECs were pelleted and resuspended in serum-free RPMI and then labeled with 5 μM Calcein AM dye (ThermoFisher) for 30 minutes at 37 °C. The cells were then pelleted and resuspended in serum-free RPMI. Labeled THP-1 cells were then added to the teloHAECs and incubated at 37 °C for 1 hour, with gentle rocking every 30 minutes. The cells were then washed extensively 5 times with warm DPBS. Fluorescence of the Calcien AM dye was then measured on a Cytation 3 (Biotek, Winooski, VT) device in area scanning mode, with gain of 80, and excitation at 492 nm, and emission 550 nm.

Measurement of Transendothelial Electrical Resistance

Cell permeability was evaluated by measuring transendothelial electrical resistance (TER) across CRISPR/Cas9-edited clones on 8 well electrode arrays 8W10E+ (Applied Biophysics) by an electrical cell-substrate impedance sensing system Model 1600R (Applied Biophysics).

Chromatin Immunoprecipitation PCR

Two CRISPR clones, one non-deletion clone and one bi-allelic deletion clone were chosen for ChIP PCR experiments. The CRISPR clones were grown under static or subjected to 24 hours of unidirectional flow. Cell crosslinking and chromatin immunoprecipitation were performed as previously described (1). 3 µl of H3K27ac antibody (Active Motif, Carlsbad, CA, #39135) was added to chromatin and incubated for 2 hours for IP. The enhancer was detected by qPCR (F: 5'-GGAGTTCATGTTGGCTGATCT-3', R 5'-TCACAAAGGAATCCAGGGTGT-3'). A total of 4 IPs were performed for each genotype grown under static and UF conditions.

HAECs heterozygous at rs17114036 were transfected with *in vitro* transcripts of KLF2 with HA tag (10) for 6 hours before cross-linking, digestion, and immunoprecipitation according to the Pierce Agarose ChIP Kit. Briefly, cells were cross-linked with 1% formaldehyde in growth media (EGM2). After a 10-minute incubation, glycine was added to 125 mM final concentration and, after 5 minutes, the solution was aspirated. The cells were washed with ice-cold PBS twice, before being scraped in 1mL PBS and 10 µL protease inhibitors (Halt Cocktail, Pierce). The cells were pelleted at 3000 x g for 5 min before undergoing MNase digestion at 10 U/µL for 15 min. After recovery of digested chromatin, the solution was immunoprecipitated with anti-HA antibody at 3 µg/ 2 million cells, and with normal rabbit IgG as control overnight. After binding to agarose beads, the immunoprecipitate was eluted before DNA clean up and then purified DNA was detected by qPCR with PLPP3 enhancer (F: 5'-AGACTAAGACGACGCTCTCC-3', R: 5'- GTGGCACCTACATCATGTTGT -3') and PLPP3 promoter primers (F: 5'-TTGCTAACCCTCACAGAGCA-3', R: 5'-ATCCTGTGACTCTGTGCCTC-3') as well as negative control primers (F: 5'-ATGTGGCCAGAGTGAAACCA-3', R: 5'-TCTACACCCAACAGCCTTCT-3').

mRNA quantitative real-time PCR

Total RNA was isolated using Direct-zol RNA MiniPrep (Zymo Research). mRNA was reverse-transcribed into cDNA using High Capacity cDNA Reverse Transcription kit (Life Technologies). cDNA quantification was performed on LightCycler 480 II (Roche) using SYBR Green I Master for mRNA. Absolute quantification of each gene of interest was normalized to the geometric mean of beta-actin, ubiquitin B, and GAPDH. The following primer sequences were used (IDT, Coralville, IA):

PPAP2B, 5'-CAGCGCCATGCAAAACTACA-3' 5'-AAAACCCTCGGTGGTAAGGC-3'

SELE, 5'-CCGAGCGAGGCTACATGAAT-3' 5'-GCCACATTGGAGCCTTTTGG-3'

GAPDH 5'- TGCACCACCAACTGCTTAGC-3' 5'- GGCATGGACTGTGGTCATGAG-3'

ACTB 5'- TCCCTGGAGAAGAGCTACGA-3' 5'- AGGAAGGAAGGCTGGAAGAG-3'

UBB 5'- ATTTAGGGGCGGTTGGCTTT-3' 5'- TGCATTTTGACCTGTTAGCGG-3'

Reagents and antibodies

Anti-HA antibody was from Abcam (ab9110)**.** Lysophosphatidic acid (LPA) was obtained from Santa Cruz Biotechnology (Cat# 22556-62-3). High molecular weight dextran was obtained from Sigma (Cat# 31392-50G). H3K27ac antibody was purchased from Active Motif (Cat# 39135).

Application of athero-relevant flows

Hemodynamic forces were applied to cultured HAECs in a 6 well plate using a 1° tapered stainless steel cone held by a computerized stepper motor UMD-17 (Arcus Technology, Livermore CA). The cone rotation recapitulated the shear stress waveform mimicking the disturbed flow at the athero-susceptible human carotid artery or the shear stress profile mimicking the unidirectional flow at the athero-resistant distal internal carotid artery (11). The flow device was placed in a 37°C incubator with 5% CO2. HAECs at 100% confluence, maintained in EGM2- medium containing 4% dextran (Sigma-Aldrich, St. Louis, MO, 31392) in 6-well plates, were subjected to unidirectional flow or disturbed flow for 24 hours before being processed.

References for Supplemental Methods

1. Hogan NT*, et al.* (2017) Transcriptional networks specifying homeostatic and inflammatory programs of gene expression in human aortic endothelial cells. *Elife* 6.
2. Buenrostro JD, Giresi PG, Zaba LC, Chang HY, & Greenleaf WJ (2013) Transposition of native chromatin for fast and sensitive epigenomic profiling of open chromatin, DNA-binding proteins and nucleosome position. *Nat Methods* 10(12):1213-1218.
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4. Romanoski CE*, et al.* (2010) Systems genetics analysis of gene-by-environment interactions in human cells. *Am J Hum Genet* 86(3):399-410.

5. van de Geijn B, McVicker G, Gilad Y, & Pritchard JK (2015) WASP: allele-specific software for robust molecular quantitative trait locus discovery. *Nat Methods* 12(11):1061-1063.
6. Dobin A*, et al.* (2013) STAR: ultrafast universal RNA-seq aligner. *Bioinformatics* 29(1):15-21.
7. Heinz S*, et al.* (2010) Simple combinations of lineage-determining transcription factors prime cis-regulatory elements required for macrophage and B cell identities. *Mol Cell* 38(4):576-589.
8. Li H*, et al.* (2009) The Sequence Alignment/Map format and SAMtools. *Bioinformatics* 25(16):2078-2079.
9. Zhang Y*, et al.* (2008) Model-based analysis of ChIP-Seq (MACS). *Genome Biol* 9(9):R137.
10. Huang RT*, et al.* (2017) Experimental Lung Injury Reduces Kruppel-like Factor 2 to Increase Endothelial Permeability via Regulation of RAPGEF3-Rac1 Signaling. *Am J Respir Crit Care Med* 195(5):639-651.
11. Dai G*, et al.* (2004) Distinct endothelial phenotypes evoked by arterial waveforms derived from atherosclerosis-susceptible and -resistant regions of human vasculature. *Proc Natl Acad Sci U S A* 101(41):14871-14876.

Table S1. Results from the bioinformatic screening for predicted, causal variants. The results of each test are either "pass" or "fail" highlighted in green or red respectively for each SNP.

SNP ID	Conditional and Joint Association	Bayesian Statistical Approach
rs4926709	Pass	Fail
rs778125	Pass	Fail
rs6666317	Fail	Fail
rs11206809	Pass	Fail
rs2184104	Pass	Pass
rs4912172	Fail	Fail
rs12122628	Fail	Fail
rs12137498	Fail	Fail
rs1889145	Fail	Pass
rs6691109	Fail	Fail
rs6698602	Fail	Pass
rs11206815	Fail	Pass
rs6421497	Fail	Pass
rs4912286	Fail	Pass
rs2780857	Fail	Fail
rs7368386	Fail	Pass
rs7365290	Fail	Pass
rs7527084	Fail	Pass
rs7513178	Fail	Pass
rs7535319	Fail	Pass
rs1261410	Fail	Pass
rs10888971	Fail	Fail
rs6588634	Fail	Fail
rs1815487	Fail	Fail
rs10789024	Fail	Fail
rs11811433	Pass	Fail
rs10888975	Fail	Fail
rs17114036	Pass	Pass
rs9970807	Fail	Pass
rs17114046	Fail	Pass
rs4912314	Fail	Fail
rs7528118	Fail	Fail
rs7515808	Fail	Fail
rs6688009	Fail	Fail
rs953857	Fail	Fail
rs953856	Fail	Fail
rs10888977	Fail	Fail
rs1930760	Fail	Fail
rs2404715	Fail	Fail
rs12134109	Fail	Fail
rs11206838	Fail	Fail
rs1041602	Fail	Fail
rs17416285	Pass	Fail
rs11579321	Fail	Fail
rs736109	Fail	Fail

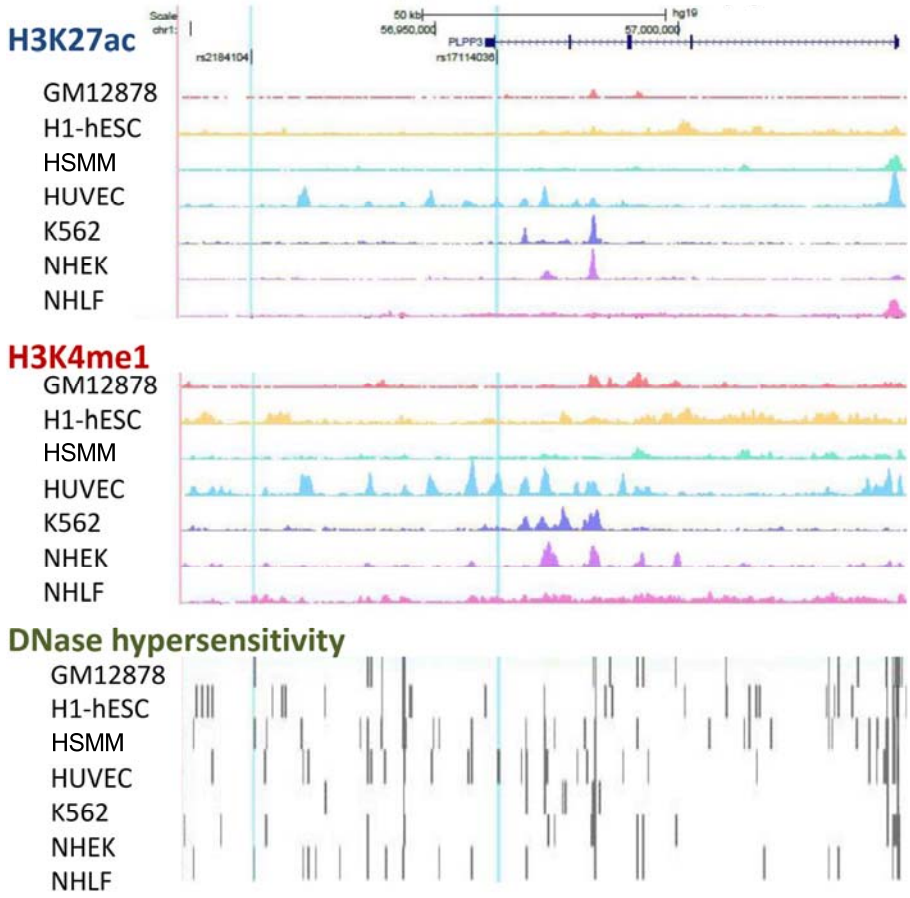

Figure S1. Encyclopedia of DNA Elements (ENCODE) studies indicate rs17114036 is located in an enhancer-like element in Human Umbilical Vein Endothelial Cells (HUVEC). H3K27ac ChIP-seq, H3K4me1 ChIP-seq, DNase hypersensitivity data sets curated by the ENCODE project in GM12878, h1-ESC, HSMM, HUVEC, K562, NHEK, and NHLF are plotted at the PLPP3 locus alongside the predicted causative CAD SNPs rs17114036 and rs2184104.

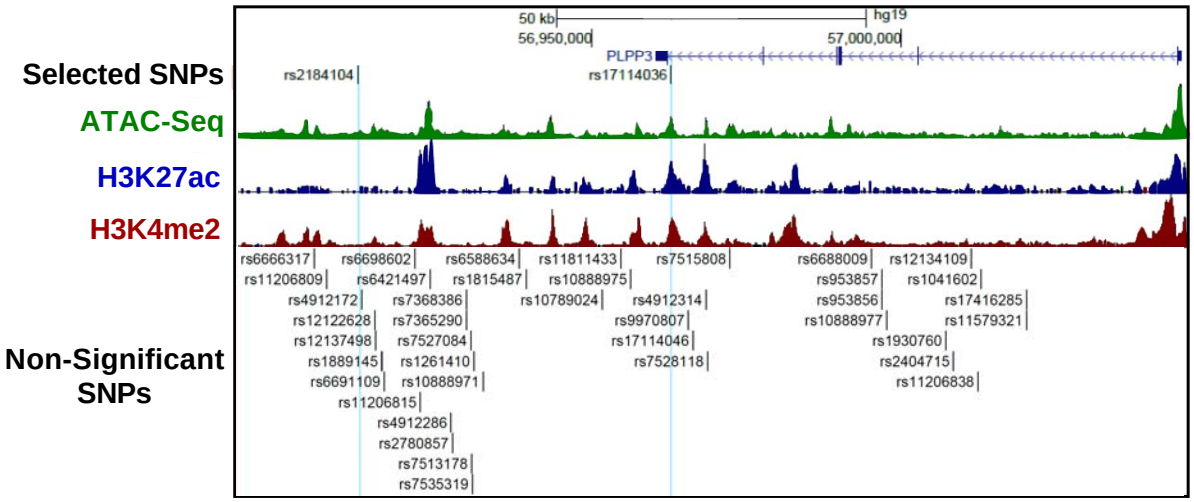

Figure S2. Chromatin accessibility and canonical enhancer marks along with common SNPs in CAD locus 1p32.2 in human aortic endothelial cells. All common SNPs at CAD locus 1p32.2 tested for predicted causality are plotted next to the ATAC-seq, H3K27ac ChIP-seq, and H3K4me2 ChIP-seq data performed in HAEC.

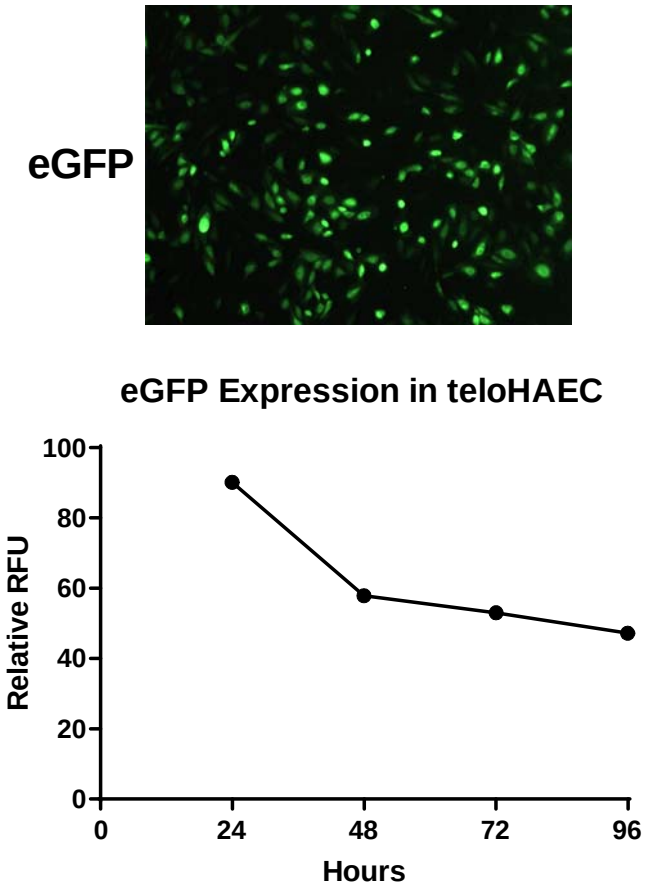

Figure S3. Electroporation results in high transfection efficiency of plasmids in teloHAEC. teloHAEC were transfected with 1.2 µ of eGFP plasmid via electroporation via the Neon® Transfection system (Thermofisher). The cells were visualized under a fluorescent microscope 24 hours post electroporation. GFP measurements were recorded at 24, 48, 72, and 96 hours post-transfection.

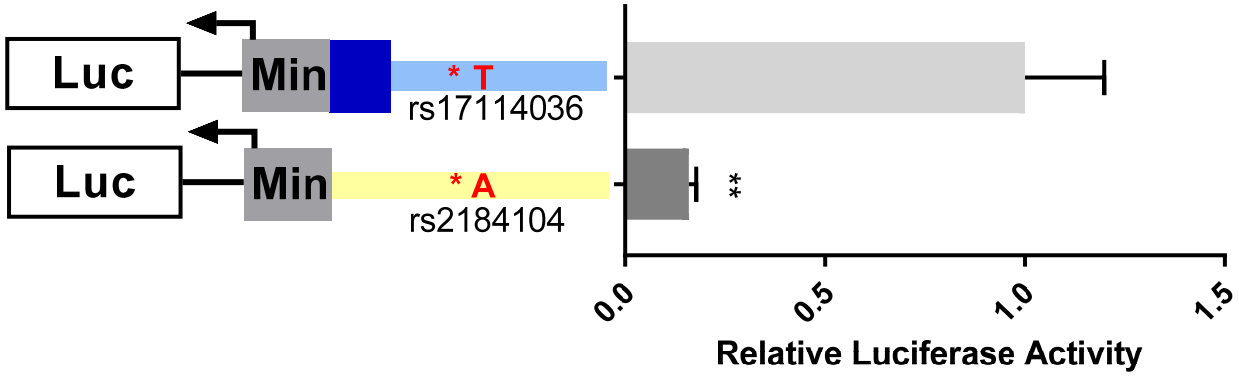

Figure S4. Dual luciferase reporter assays show a 6.3-fold difference in luciferase reporter activity in 1.2 kb regions surrounding predicted, causative SNPs rs17114036 and rs2184104. Equal sized genomic regions with either rs17114036 or rs2184104 in the middle were cloned into pGL4.23 luciferase reporter vectors, electroporated into teloHAEC along with pRLTK internal control, and incubated for 24 hours prior to lysate collection and luciferase activity measurement. **p<0.005 as determined by Student's t-test. Data represent mean ± SEM.

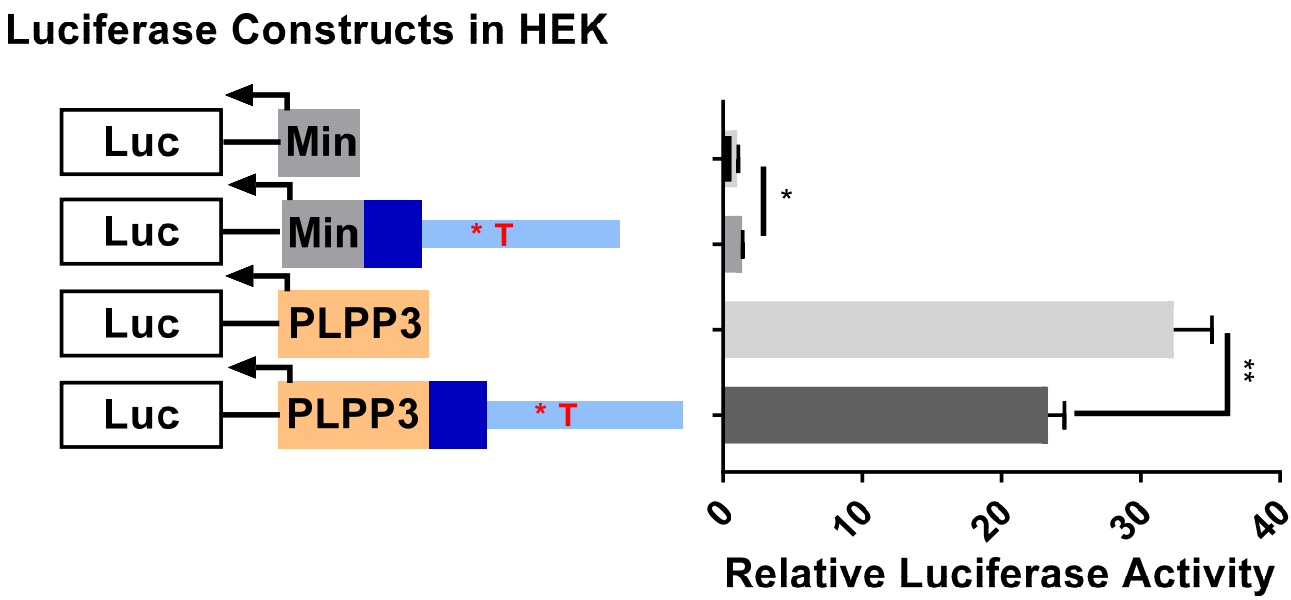

Figure S5. Dual luciferase reporter assays demonstrate minimal enhancer activity of chr1:56962213-56963412 in embryonic kidney cells 293 (HEK-293). Luciferase reporters constructs with and without the enhancer as well as with minimal promoter or human PLPP3 promoter were transfected into HEK-293 using lipofectamine and incubated 24 hours before measuring luciferase activity. n=5. *p<0.05, **p<0.005 as determined by Student's t-test. Data represent mean ± SEM.

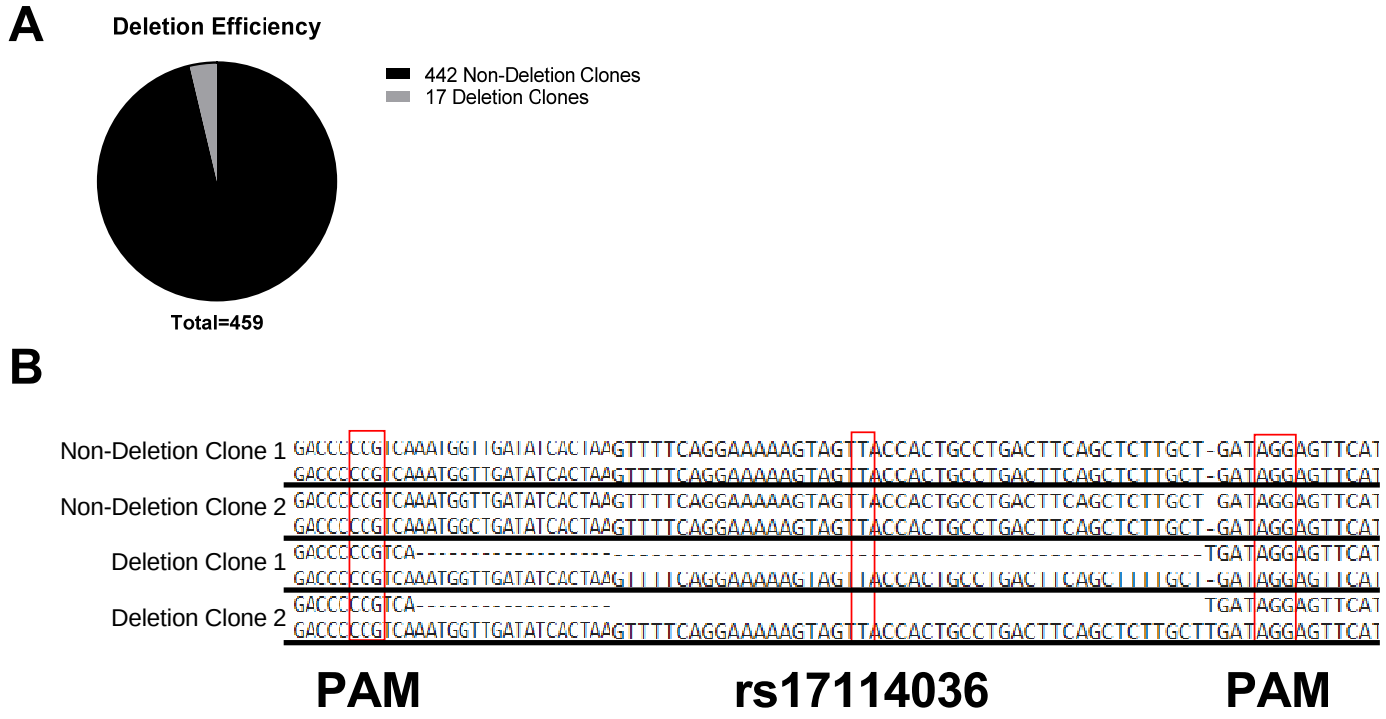

Figure S6. Bacterial CRISPR-associated protein-9 nuclease (CRISPR/Cas9) system results in successful genomic deletion at site of interest in teloHAEC. (A) The total numbers of teloHAEC clones that were grown and genotyped are reported. (B) The PCR products were cloned (TA cloning) and sequenced to confirm deletion of the targeted region surrounding rs17114036. Protospacer adjacent motif (PAM) sequences, which are recognized and cleaved by Cas9, and rs17114036 are highlighted for reference.

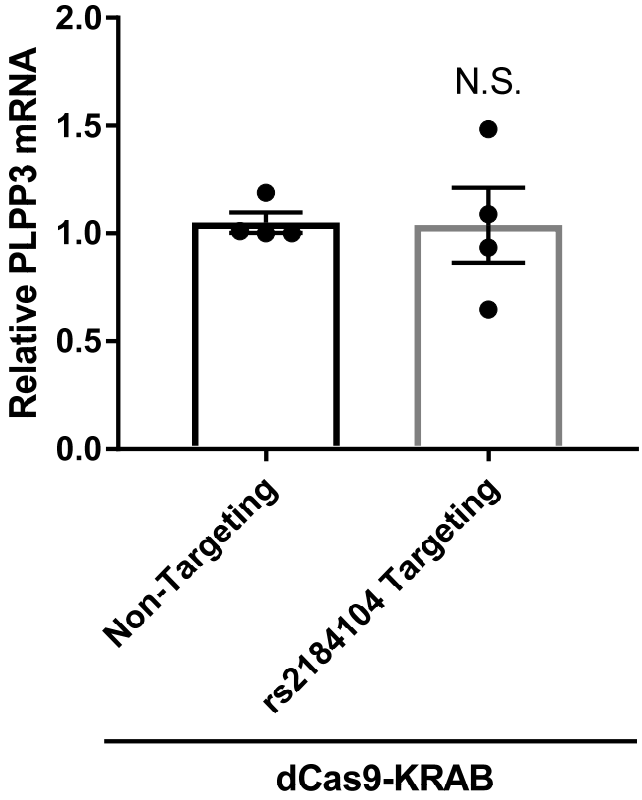

Figure S7. CRISPR interference (CRISPRi) using a sgRNA to guide a catalytically dead Cas9 (dCas9) fused to KRAB transcriptional repressor was targeted to the genomic sequence near rs2184104. Dead Cas9/KRAB transcripts and sgRNAs were transfected into HAEC and incubated for 8 hours prior to cell lysis, sample collection, and analysis via qPCR. N.S. $p>0.05$ as determined by Student's t-test. Data represent mean ± SEM.

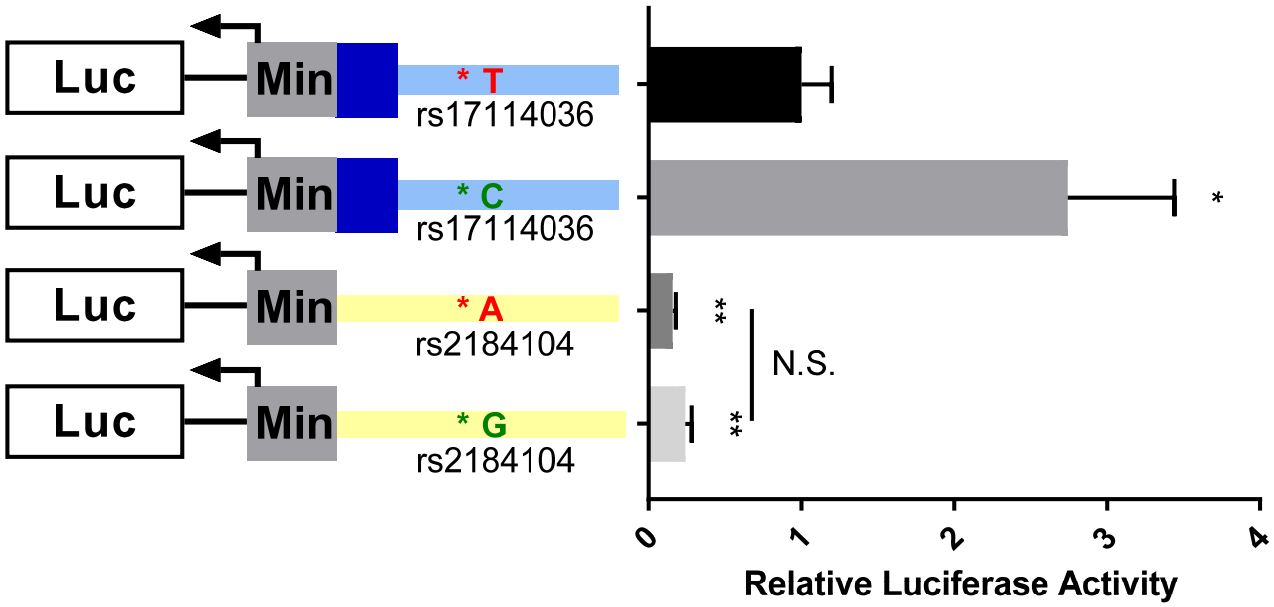

Figure S8. Comparison of luciferase reporter activity of predicted causative SNPs rs17114036 and rs2184104 reference and alternative alleles. Genomic regions around rs17114036 and rs2184104 were cloned and confirmed to harbor the reference alleles. Site-directed mutagenesis changed the reference to alternative alleles at these SNPs. Luciferase plasmids were electroporated into teloHAEC along with pRLTK internal control, and incubated for 24 hours prior to lysate collection and luciferase activity measurement. *p<0.05, **p<0.005 as determined by Student's t-test. Data represent mean ± SEM.

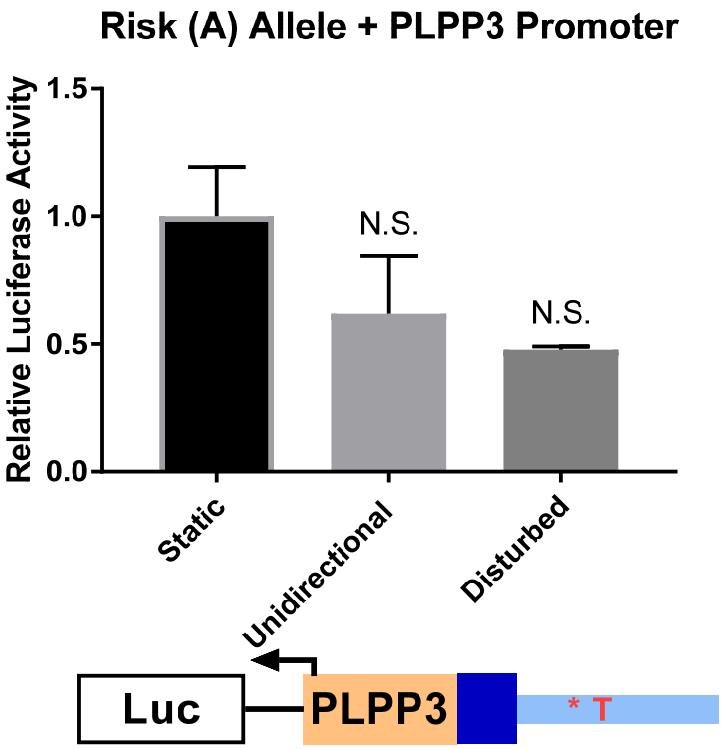

Figure S9. Cells electroporated with luciferase reporter construct harboring the PLPP3 promoter and the chr1:56962213-56963412 region (with T allele at rs17114036) under static, unidirectional flow, disturbed flow. Cells were electroporated and then left to settle for 6 hours prior to 18 hours of static, UF, or DF conditions. 24 hours post-transfection the samples were collected. Experiment was performed in biological triplicate and technical triplicate. N.S. $p>0.05$, as determined by two-way ANOVA. Data represent mean ± SEM.

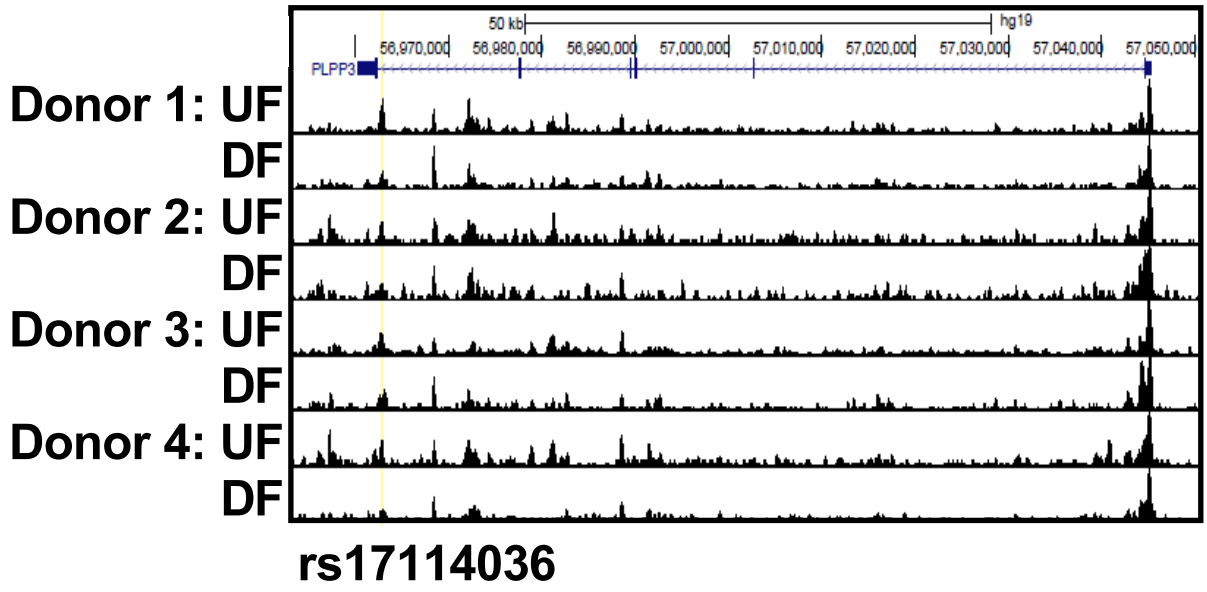

Figure S10. Unidirectional flow (UF) increases chromatin accessibility in PLPP3 intron 5 in HAECs when compared with cells under disturbed flow (DF). ATAC-seq was conducted in HAEC lines from 4 individual donors heterozygous (T/C) at rs17114036 under 24-hr athero-protective UF and athero-susceptible DF.

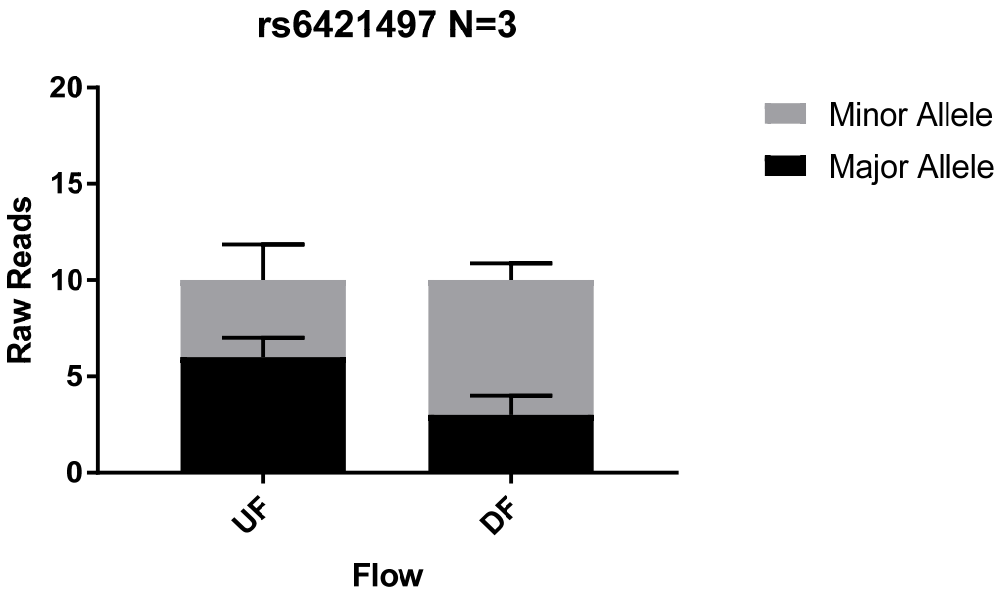

Figure S11. Allelic imbalance analyses show unidirectional flow does not increase chromatin accessibility at locus related to the minor allele at rs6421497 in human aortic endothelium. SNP rs6421497 was not predicted to be causal but resides within a peak from ATAC-seq data set in at least 3 donors. The number of ATAC-seq reads detected under unidirectional and disturbed flow are reported and the proportion harboring either the major or minor allele.

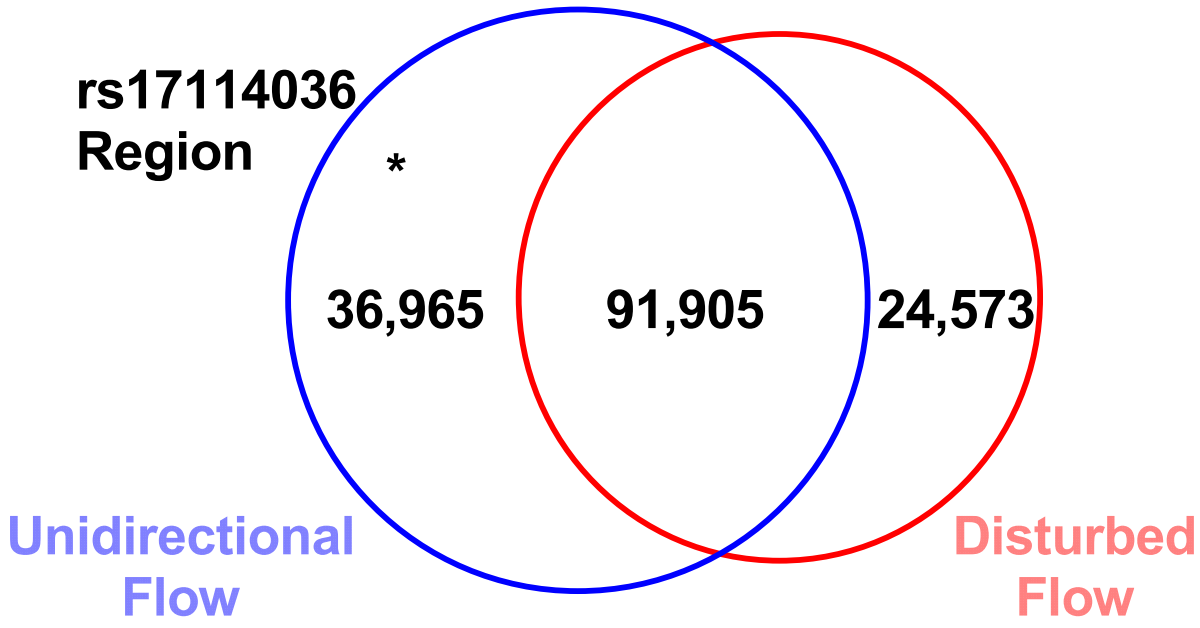

Figure S12. Numbers of open chromatin loci in HAEC regulated by athero-protect unidirectional flow or athero-susceptible disturbed flow. Model-based Analysis of ChIP-seq (MACS2) and HOMER differential analysis were conducted using ATAC-seq results from a total of 5 HAEC lines subjected to 24 hours athero-relevant flows to identify hemodynamics-regulated open chromatin sites.