SUPPLEMENTAL MATERIAL

Detailed Methods

Animals and diets

KLF14$^{flox/flox}$ mice[1] were mated with Cdh5-Cre transgenic mice (stock number: 006137, Jackson Laboratory) to generate endothelial specific KLF14 knock out mice. Six to eight weeks old male Cdh5-Cre^{+}/KLF14$^{flox/flox}$ mice were used for *in vivo* leukocyte adhesion assay.

KLF14 global knockout mice were generated by crossing KLF14$^{flox/flox}$ mice with *Ayu1*-Cre mice that express Cre ubiquitously[2, 3]. Five to six weeks old female *Ayu1*-Cre^{+}/KLF14$^{flox/flox}$ mice were used for isolation of pulmonary endothelial cells. Genomic DNA of mouse tail was extracted through phenol/chloroform extraction and ethanol precipitation. Mouse genotypes were determined by PCR. Cdh5-Cre and *Ayu1*-Cre genotyping was performed by using different primer sets: Cdh5-Cre forward, 5'-CTAGGCCACAGAATTGAAAGATCT-3'; Cdh5-Cre reverse, 5'-GTAGGTGGAAATTCTAGCATCATCC-3'. The resultant product of 324bp appeared only in Cdh5-Cre+ samples. *Ayu1*-Cre forward, 5'-GTAGGATCACCTGCCTGTGTCC-3'; *Ayu1*-Cre reverse, 5'-CTTGCGAACCTCATCACTCG-3'. Single PCR product of *Ayu1*-Cre+ mouse DNA was between 400-500bp. To detect KLF14 $^{flox/flox}$ alleles, the following primers were used: forward, 5'-TAGTGAGGAAAGGAAGAGCAGGTAGGA-3'; reverse, 5'-TCACATGAGGAAACAGACAAGCAAAAG-3'. These primers provided a 344 bp product if the KLF14 flox allele was present or a 170 bp product if a WT allele was present. Six to eight weeks old wild type male mice in the C57BL/6 background purchased from the Jackson Laboratory were used for acute and chronic inflammation stimuli and aortic immunostaining.

Mice had free access to water and rodent chow diet. Chronic inflammation stimulus was performed by feeding the mice a western diet (17.3% protein, 21.2% fat, 48.5% carbohydrate, 0.2% cholesterol by mass, and 42% calories from fat; TD.88137, Envigo) for three months. All animal procedures followed guidelines of the University of Michigan Animal Care and Use Committee.

Cell culture and stimulation

Human coronary artery endothelial cells (HCAECs) and Human umbilical vein endothelial cells (HUVECs) were purchased from Lonza. HCAECs were cultured in EGM-2MV medium (Lonza, CC-3202). HUVECs and isolated mouse primary endothelial cells were cultured in M199 medium (Gibco) supplemented with 16% fetal bovine serum (FBS) (Sigma-Aldrich), 24 mM HEPES (Gibco), 0.5 mg/ml penicillin/streptomycin (Gibco), 2500 USP units of heparin (Sagent Pharmaceuticals, 400-30) and 1 ng/ml recombinant human fibroblast growth factor (Sigma-Aldrich). Bovine aortic endothelial cells (BAECs), AD-293 cells and THP-1 cells were obtained from American Type Culture Collection). BAECs and AD-293 cells were cultured in Dulbecco's modified Eagle's medium (DMEM) (Gibco) with 10% FBS. THP-1 cells were cultured in RPMI-1640 medium (Gibco) supplemented with 10% FBS and 0.05 mM 2-mercaptoethanol (Gibco). Endothelial cells were plated on Collagen I (Corning Life Sciences, 354236)

coated dishes. No more than passage six of HCAECs, HUVECs and BAECs were used in all of the experiments. Passage one to three of isolated mouse primary endothelial cells were used in the experiments. Human endothelial cells were stimulated with 5 ng/ml recombinant human IL-1β (R&D, 201-LB) and 2 ng/ml human TNFα (R&D, 210-TA) respectively for 4 hours, unless otherwise indicated. Mouse primary endothelial cells were stimulated with 20 ng/ml recombinant mouse IL-1β (R&D, 401-ML) for 4 hours. BAECs and AD-293 cells were stimulated with 10 ng/ml human TNFα (R&D, 210-TA) for 16 hours after 24-hour plasmid transfection. HUVECs were pretreated with DMSO or perhexiline maleate salt (Sigma Aldrich, SML0120, 15 μM) for 20 hours before IL-1β administration. NF-κB inhibitors BAY11-7082 (Cayman, 19542-67-7) and ACHP (Tocris, 4547) were used to pretreat the cells at a concentration of 5 μM for 1 hour before pro-inflammatory stimuli.

RNA isolation and quantitative real-time PCR (qRT-PCR) analysis

RNA was extracted from mouse aortas and lung tissues using Trizol reagent (Ambion,15596-018) [4]. RNA from tissues and cells was purified with RNeasy kits (QIAGEN), followed by cDNA synthesis using SuperScript III Reverse Transcriptase System (Invitrogen). qRT-PCRs were run in triplicates with the use of SYBR green Supermix (Bio-Rad). 18S rRNA served as the internal control. Relative message RNA abundance was calculated utilizing the $^{\Delta\Delta}$CT method and normalized to control groups. Primers for qRT-PCR are listed in supplemental table 1.

Nuclear and cytoplasmic protein extraction

Nuclear and Cytoplasmic Extraction Reagents Kit (Thermo Scientific, 78835) were used following the manufacturer's protocol. Lamin A/C was used as the internal control for nuclear protein, while β-actin was used as the internal control for cytoplasmic protein.

Protein extraction and Western blot

Cells and mouse tissues were lysed in T-PERTissue Protein Extraction Reagent (Thermo Scientific) supplemented with a protease inhibitor cocktail (Roche Applied Science). Protein extracts were resolved in 10% SDS–PAGE gels and transferred to PVDF membranes (Bio-Rad). After blocking in TBST containing 5% non-fat dry milk at room temperature for 1 hour, the PVDF membranes were immunoblotted with primary antibodies at 4°C overnight. The membranes were washed by TBST before incubated with IRDye-conjugated secondary antibodies (1:8000, LI-COR Biosciences) at room temperature for 1 hour[5]. After TBST wash, western blots were scanned and quantified using an Odyssey Infrared Imaging System (LI-COR Biosciences, version 2.1). Primary antibodies used in this study were KLF14 (Sigma-Aldrich, SAB1304202, 1:1000 working dilution was used to human overexpressed KLF14), human VCAM-1 (Santa Cruz Biotechnology, sc-13160, 1:1000 dilution), human and mouse E-selectin (Santa Cruz Biotechnology, sc-14011, 1:1000 working dilution was used to detect human E-selectin. 1:500 working dilution was used to detect mouse E-selectin), human p65 (Cell Signaling Technology, 8242S,1:1000 dilution), human Lamin A/C (Santa Cruz Biotechnology, sc-

7292,1:1000 dilution), human and mouse β-Actin (Santa Cruz Biotechnology, sc-1616,1:1000 dilution), mouse endogenous KLF14 (Antibodies were generated by Syd labs Inc., 1:2000 dilution) and mouse VCAM-1 (Cell Signaling Technology, 39036S,1:500 dilution).

Plasmid construction and transfection

NF-κB response element and desired promoter regions of VCAM-1 (-1116 to +35), E-selectin (-2107 to +109) and p65 (-408 to +92) were PCR-amplified from human genomic DNA and inserted into the pGL4.11 luciferase reporter vector (Promega)[6]. The numbers indicate the distance in nucleotides from the transcription start site (+1). The two CACCC boxes ("a" and "b") on the p65 promoter (a: -359 to -355, b: -179 to-175), which are putative KLF14 binding sites, were mutated to AGTTT by using Q5 Site-Directed Mutagenesis Kit (New England Biolabs). Human KLF14 and p65 coding regions were inserted into pcDNA3.1 mammalian expression vector (Thermo Scientific) to generate overexpression plasmids. All plasmids were verified by DNA sequencing. BAECs or AD-293 cells were co-transfected with plasmids at 70-80% confluence with Lipofectamine 2000 (Invitrogen) according to the manufacturer's protocol. *Renilla*-null (Promega) luciferase activity served as the internal control. Luciferase activity was measured using a Dual-luciferase reporter assay system (Promega).

Adenovirus construction and adenovirus-mediated overexpression

Previously described adenovirus encoding LacZ (AdLacZ), human KLF14 (AdKLF14)[1], GFP (AdGFP), human KLF2 (AdKLF2), human KLF4 (AdKLF4) and human KLF11 (AdKLF11) [6] were used in gain of function experiments in this study. Adenovirus encoding Flag-tagged KLF14 (AdKLF14-Flag) was generated by inserting the Flag tag at the C terminus of full-length human KLF14 coding region and cloning the KLF14-Flag sequence into the pCR8/GW/TOPO entry vector (Invitrogen). The KLF14-Flag sequence was verified via DNA sequencing before cloning from the entry vector to the destination vector pAd/CMV/V5-DEST (Invitrogen) by LR recombination. AdKLF14-Flag was packaged in AD293 cells. All adenoviruses used in this study were purified by $CsCl_2$ density gradient ultracentrifugation. Titrations of adenoviruses were performed using Adeno-XTM qPCR titration kit (Clontech) following the manufacturer's protocol. Adenovirus-mediated overexpression was performed by transfecting the cells with 10 multiplicity of infection (MOI) for 48 hours, unless otherwise indicated.

Isolation of mouse pulmonary endothelial cells

Mouse pulmonary endothelial cells were isolated by two rounds of cell sorting using CD31 and ICAM-2 antibody coated magnetic beads. The CD31 antibody coated beads were used for the first cell sorting and the ICAM2 antibody coated beads were used for the second cell sorting. Aliquots of Dynabeads (Invitrogen, 11035) were placed on DynaMag™- Spin Magnet (Thermo Fisher, 12320D), washed with DPBS (without Ca^{2+}/Mg^{2+}) containing 0.1% BSA for three times in accordance with manufacturer's protocol and incubated with CD31 antibody (Southern Biotech, 1625-01,1:20 dilution) and ICAM-2 antibody (Southern Biotech, 1925-01 ,1:20 dilution) respectively overnight

at 4 ° C on a rotator. The antibody-coated beads were then washed for four times with DPBS (without Ca^{2+}/Mg^{2+}) containing 0.1% BSA. Lung tissues were isolated from 3 adult mice per group and placed in cold isolation buffer (DEME supplemented with 20% heat-inactivated fetal bovine serum, 20 mM HEPES and 0.5 mg/ml penicillin/streptomycin). Bronchi were removed as much as possible from the isolated lung tissues in a sterile laminar flow hood. Lung tissues were then transferred to a dry 10cm dish, minced finely with scissors on ice and digested in 25ml DPBS (+Ca^{2+}/Mg^{2+}) containing 2mg/ml collagenase A (Sigma Aldrich, 10103586001) at 37 °C for 30 minutes. Tissues were further triturated by aspirating the collagenase A-digested suspension 12 to 15 times with a cannula-attached to a 30ml syringe. The suspension was then pipetted through a 70μm cell strainer and centrifuged at 400g, 4 °C for 8 minutes. The pellets were resuspended in 3 ml DPBS (without Ca^{2+}/Mg^{2+}), transferred to 3 1.5ml tubes (1ml/tube) and incubated rotating with CD31 antibody coated beads (15ul beads/ml suspension) at room temperature for 10 minutes. The beads and sorted endothelial cells were washed with isolation buffer for 5 to 6 times until the buffer was clear and cultured in Collagen I coated 10ml dishes until the cells grew to 90% confluence. The cells were detached with 0.25% trypsin-EDTA (Gibco) and spun down for a second round of sorting with ICAM2 antibody coated beads following equivalent steps as for the first sorting. More than 80% sorted cells were endothelial cells (verified by Ac-LDL uptake assay and vWF immunostaining). The primary endothelial cells were split at a ratio of 1:2 for passage.

Chromatin immunoprecipitation assay (ChIP assay)

HUVECs were infected with 10 MOI Ad-KLF14-Flag for 48 hours to overexpress KLF14. KLF14 protein was pulled down using anti-Flag antibody (Cell Signaling Technology, 14793S). ChIP assay was performed with EZ-ChIP Kit (Millipore) following the manufacturer's protocol. Purified DNA was used as template for qRT-PCR. DNA fragments pulled down and containing the two putative binding boxes on p65 promoter were detected using the following qRT-PCR primers: box a, F: ctagattggggtgggtagga, R: gagcaaagccagggctact; box b, F: cccagagtgacatcaccaaa, R: ctcgccgaggacgaagag.

In vitro leukocyte endothelial adhesion assay

HUVECs were infected with 10 MOI AdLacZ or AdKLF14 for 48 hours before stimulated with proinflammatory cytokines for 6 hours. Simultaneously, human monocytes, THP-1 cells, were incubated with fluorescence-labelled LeukoTracker (Cell Biolabs, CytoSelect™ Leukocyte- Endothelium Adhesion Assay kit, CBA-210) for 1 hour at 37 °C and washed according to the manufacturer's protocol. Fluorescence-labelled THP-1 cells were co-incubated with control or activated HUVECs for 30 min. Unbound THP-1 cells were removed by washing. The number of adhered cells was counted under a fluorescence microscope.

Intravital microscopy

Intravital microscopy analysis was performed as previously described[5, 6]. There were two experimental approaches in this study. In the first experiment, 6-8 weeks old male

EC-specific KLF14 knock out mice and littermate wild type mice were injected intraperitoneally with LPS (Sigma Aldrich, L4391, 5 mg/Kg). In the second experiment, 6-8 weeks old male wild type mice on C57BL/6 background and EC-specific KLF14 knock out mice were injected intraperitoneally with DMSO or perhexiline maleate salt (Sigma Aldrich, SML0120, 10 mg/Kg/d) every other day at least three times before LPS injection (10 mg/Kg). Four hours after LPS administration, mice were anesthetized and injected with rhodamine 6G chloride (Thermo Scientific, 0.3 mg/kg) to stain leukocytes for 20 minutes. The cremaster muscle was dissected from surrounding tissues, kept flat by fixing the edges of the tissue *via* silk suture and superfused with 37°C saline for moisture. Vessel wall-leukocyte interaction in post-capillary venules were observed using an intravital microscope. Leukocytes that remain stationary for at least 30s were counted to be adherent to vascular endothelial cells. All animal procedures followed guidelines of the University of Michigan Animal Care and Use Committee.

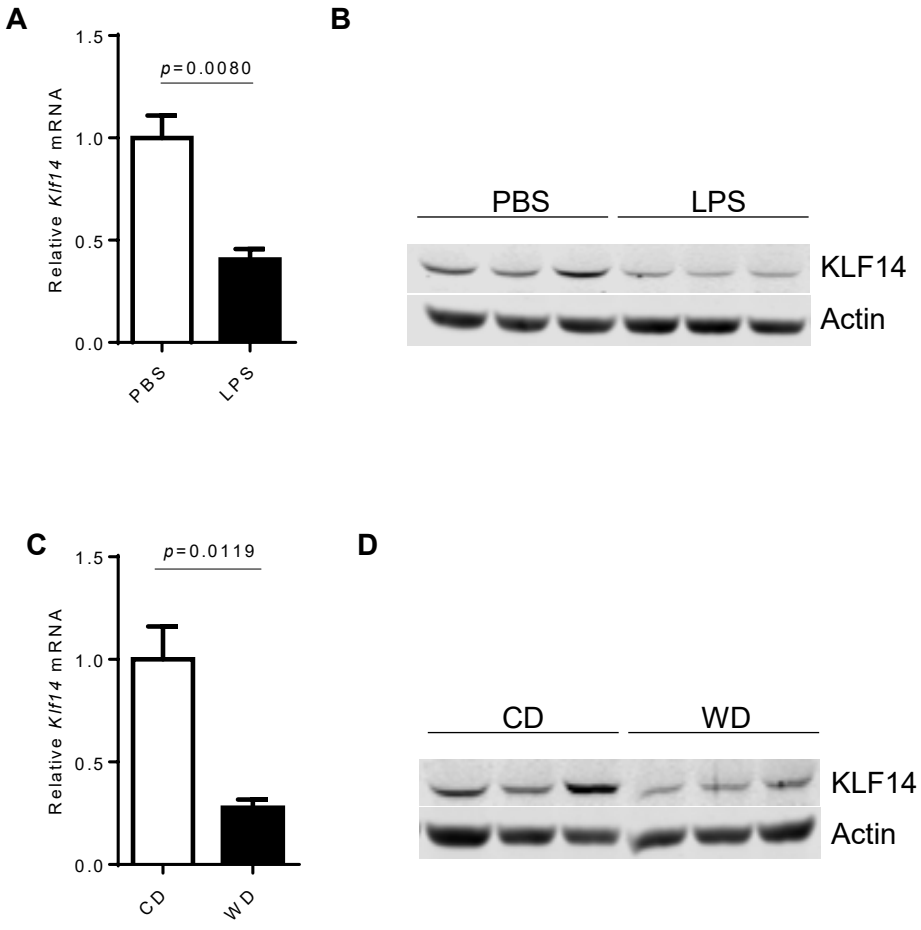
Immunostaining and confocal images

Mouse aorta was harvested and fixed in formalin. After dehydration with 20% and 30% sucrose solution, the samples were embedded in Optimal Cutting Temperature compound (Thermo Scientific) and cut in a Leica cryostat. The frozen sections were blocked in 4% goat serum for 1 hour at room temperature and then incubated with primary antibodies at 4°C overnight. Primary antibodies were used to detect mouse CD31 (Dianova, 1:50 dilution), KLF14 (Syd labs Inc 1:100 dilution) and E-selectin (Novus, NBP1-45545,1:25 dilution). After PBS washing, the samples were incubated with Alexa Fluor-labeled secondary antibody (Jackson ImmunoResearch Laboratories,1:1000 dilution) at room temperature for 1 hour. The sections were then washed and mounted before image collection. Images were obtained using a Nikon A1 confocal microscope. Background correction was performed using the appropriate IgG negative controls. The fluorescent intensity was quantified using Image J software.

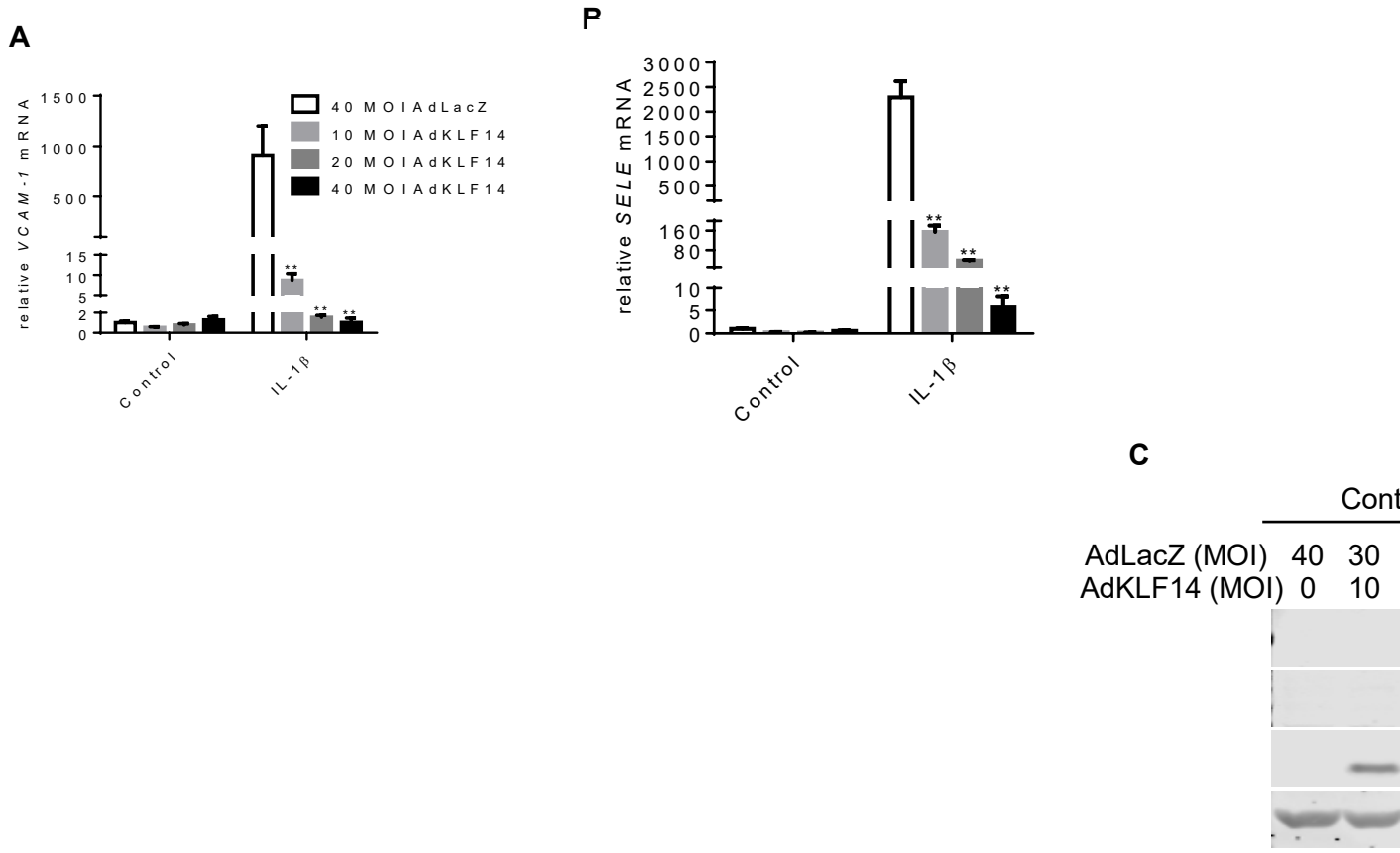
Statistics

Data are reported as mean ± SEM. Statistical analyses were performed using GraphPad Prism 6 software (GraphPad Software). Statistical comparisons between 2 groups were performed using unpaired Student's t test. Comparisons among 3 groups or more were analyzed with two-way ANOVA. $p < 0.05$ was considered statistically significant. All results were representative from at least three independent experiments.

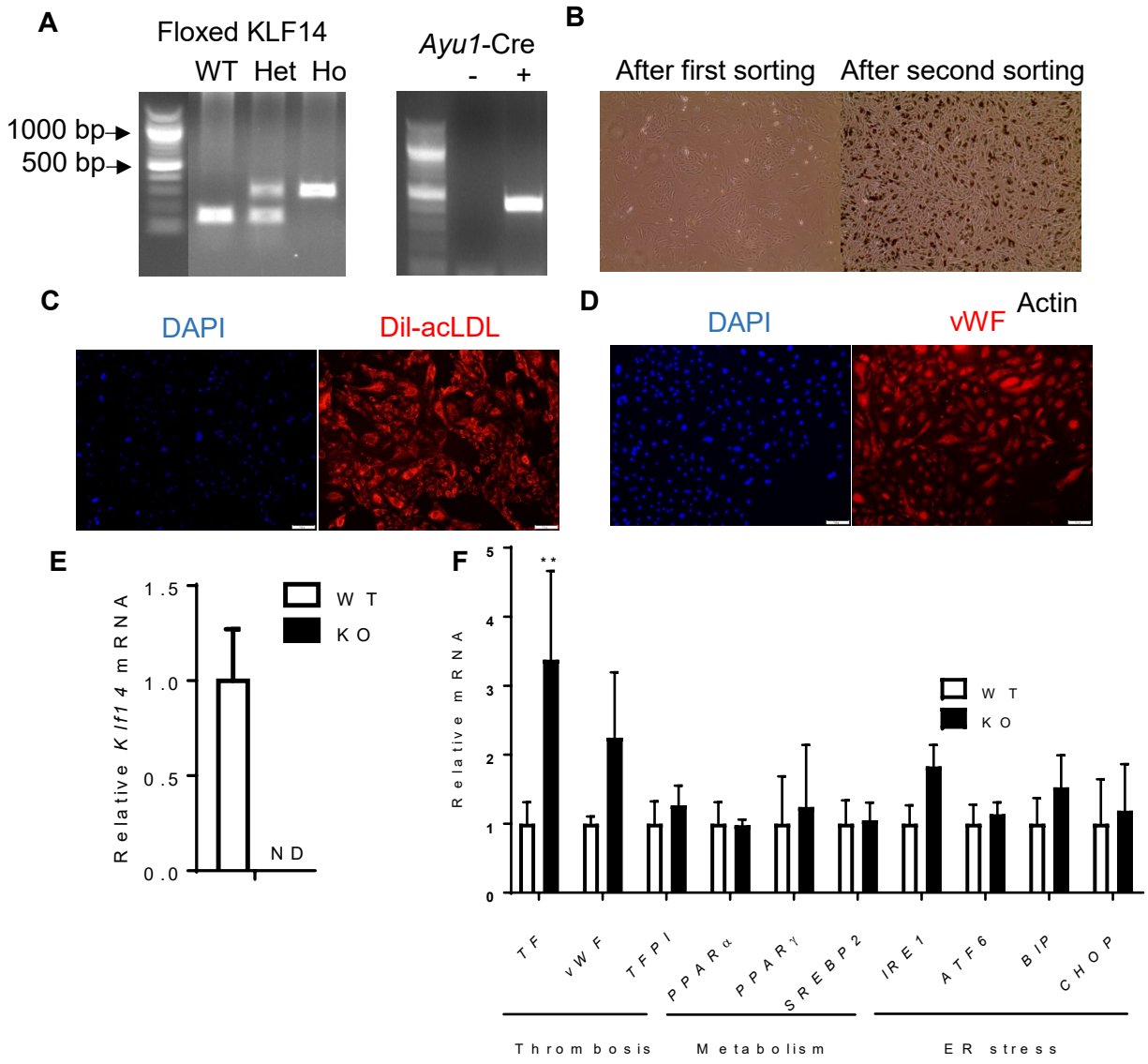
Supplementary Figures



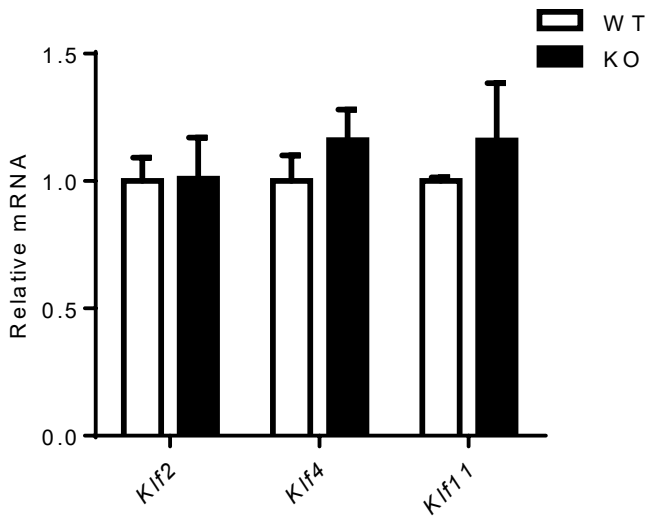
Supplement Fig. 1 Klf14 is down-regulated in mouse lung tissue by both acute and chronic inflammation. (A-B) 6-8 weeks old male wild type C57BL/6 were intraperitoneally-injected with phosphate-buffered saline (PBS) or 20 mg/kg lipopolysaccharide (LPS). After 6 hours, the mice were sacrificed to collect lung tissues. (C-D) 6-8 weeks old male wild type C57BL/6 mice were fed rodent chow diet (CD) or western diet (WD) for three months before they were sacrificed to collect lung tissues. *Klf14* mRNA levels in mouse lung tissue were determined by qRT-PCR (A and C), while Klf14 protein abundance was determined by western blot (B and D).



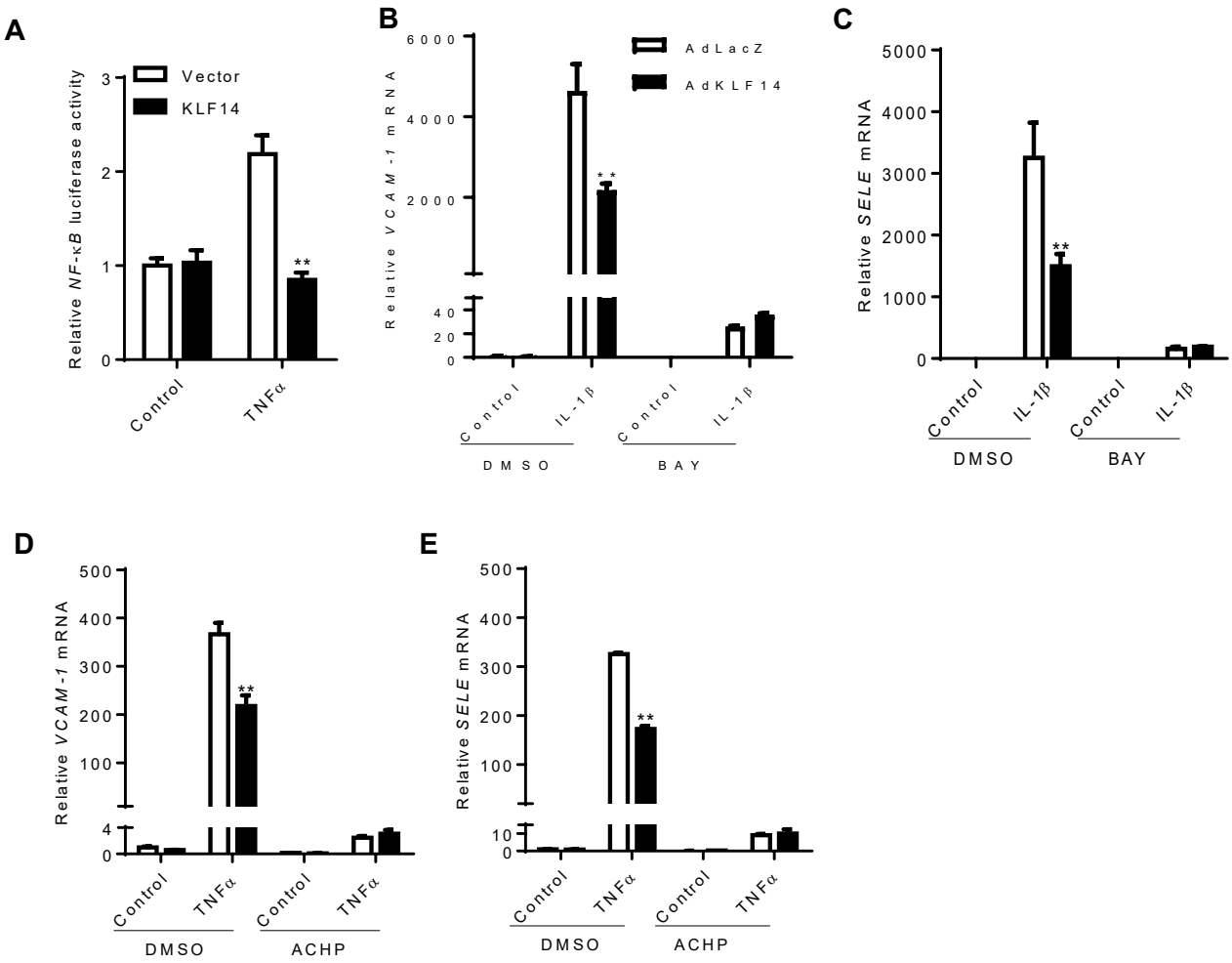
Supplement Fig. 2 Overexpression of KLF14 inhibited endothelial cell activation in a dose-dependent manner. HUVECs were infected with the indicated dose of adenovirus for 48 hours and then stimulated with 5 ng/ml IL-1β for 4 hours. VCAM-1 and SELE mRNA were determined by qRT-PCR (A and B). E-selectin and VCAM-1 protein were determined by western blot (C). (*$p < 0.05$; **$p < 0.01$, compared to AdLacZ group)



Supplement Fig. 3 Isolated mouse pulmonary endothelial cells. (A) Mice were genotyped by PCR amplification of mouse tail DNA and agarose gel electrophoresis. *Klf14* global KO mice were floxed-*Klf14* homozygotes and *Ayu1-Cre* positive (WT: wild type; Het: heterozygote; Ho: homozygote). (B) Mouse pulmonary endothelial cells were isolated by two rounds of cell sorting with antibody coated magnetic beads. The sorted cells were observed under a microscope (black dots represented beads). Scale bar=100μm (C and D) After the second sorting, primary endothelial cells were identified by DiI-labelled acetylated low-density lipoprotein (DiI-acLDL) uptake assay (C) and Von Willebrand factor (vWF) immunostaining (D). scale bar=25μm (E) *Klf14* expression of isolated mouse primary endothelial cells was determined by qRT-PCR. (ND: not detectable, Cq>40 cycles in qRT-PCR). (E) Endothelial function related genes were assessed by qRT-PCR. Tissue factor (TF), Von Willebrand factor (vWF), Tissue factor pathway inhibitor (TFPI), Peroxisome proliferator-activated receptor alpha (PPARα), Peroxisome proliferator-activated receptor gamma (PPARγ), Sterol regulatory element binding transcription factor 2 (SREBP2), Inositol-requiring enzyme 1(IRE1), Activating transcription factor 6 (ATF6), Heat shock 70 kDa protein 5 (BIP), C/EBP homologous protein (CHOP). (**$p < 0.01$, compared to WT group)

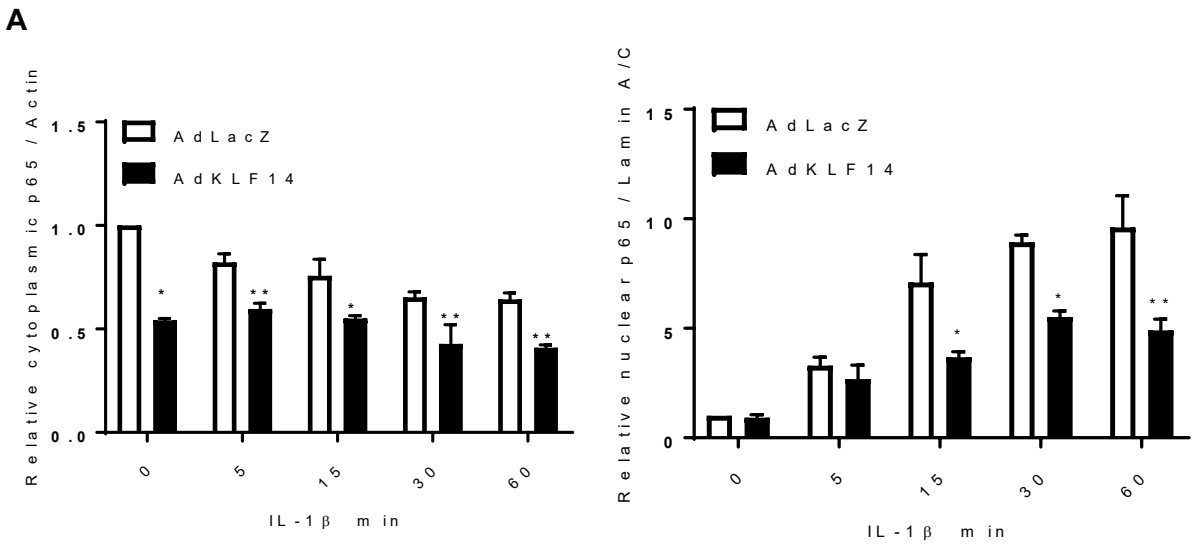


Supplement Fig. 4 *Klf14* deficiency does not affect the expression of *Klf2*, *Klf4* or *Klf11* in primary pulmonary endothelial cells. Mouse pulmonary endothelial cells were isolated from *Klf14* global KO mice and their littermate controls. *Klf2*, *Klf4* and *Klf11* expression were determined by qRT-PCR.

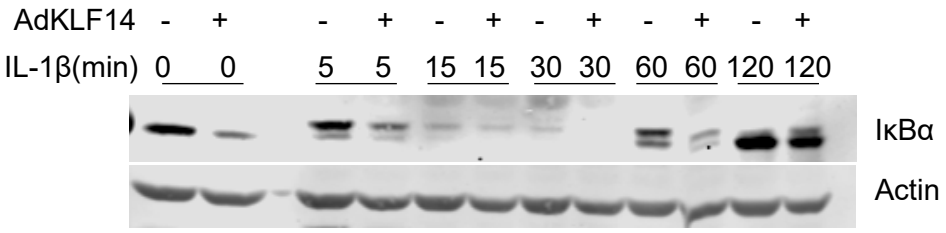


Supplement Fig. 5 KLF14 ameliorates endothelial inflammation by inhibiting NF-κB signaling pathway. (A) Bovine aortic endothelial cells (BAECs) were co-transfected with a reporter plasmid carrying the *NF-κB* response element driving the luciferase reporter and with pcDNA3.1 (vector) or KLF14 (KLF14) plasmids. After 24 hours, the transfected cells were stimulated with 10 ng/ml TNFα for 16 hours before lysis. *Renilla*-null luciferase activity served as the internal control. (B-E) HUVECs were infected with 10 MOI AdLacZ or AdKLF14 for 48 hours. The cells were then pretreated with NF-κB signaling pathway inhibitors BAY (5μM) (B and C) or ACHP (5μM) (D and E) for one hour before stimulated with IL-1β (5ng/ml) or TNFα (2ng/ml) for 4 hours. *VCAM-1* (B and D) and *SELE* (C and E) mRNA abundance was determined by qRT-PCR. (*$p < 0.05$; **$p < 0.01$, compare with Vector or AdLacZ group)

A

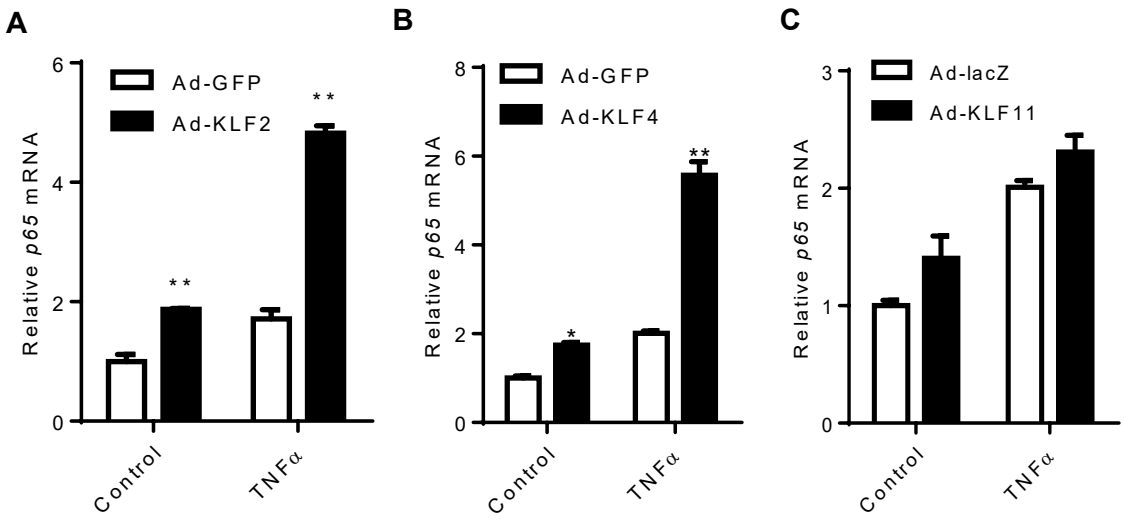


B

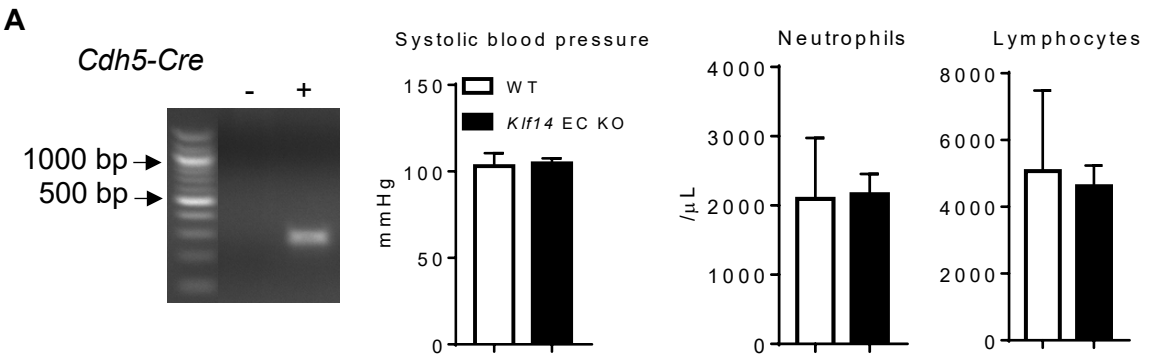


Supplement Fig. 6 KLF14 inhibits p65 expression but does not inhibit IκBα degradation. Human umbilical vein endothelial cells (HUVECs) were infected with 10 MOI of AdLacZ or AdKLF14 for 48 hours and then stimulated with 5 ng/ml Interleukin 1β (IL-1β) for the indicated time. (A) Quantification of Fig.3A. (B) NF-κB inhibitor alpha (IκBα) protein level was determined by western blot. Images shown are representative of 3 independent experiments. (*$p < 0.05$; **$p < 0.01$, compared to AdLacZ group)

AdLacZ + - + - + - + - + - + -



Supplement Fig. 7 KLF2, KLF4, KLF11 do not inhibit *p65* transcription. HUVECs were infected with 10 MOI of adenovirus encoding KLF2 (A), KLF4 (B), KLF11 (C) with their appropriate control adenovirus for 48 hours and then stimulated with 2 ng/ml tumor necrosis factorα (TNFα) for 4 hours. *p65* mRNA abundance was determined by qRT-PCR. (*$p < 0.05$; **$p < 0.01$, compare with Ad-GFP or Ad-lacZ group)



Supplement Fig. 8 Characterization of KLF14 EC KO mouse. (A) Mice were genotyped by PCR amplification of mouse tail DNA and agarose gel electrophoresis. (B) Systolic blood pressure was measured by tail-cuff method. (C) Circulating neutrophils and lymphocytes were quantified with an automated hematology cell counter.

Supplement Table 1. qRT PCR primers.

Gene	Primer sequence (from 5' to 3')
hKLF14	Forward: ctgcgacaagaagtttacgc
	Reverse: gtccccggtactcgatcata
hVCAM1	Forward: cgaacccaaacaaaggcaga
	Reverse: acaggattttcggagcagga
hSELE	Forward: actttctgctgctggactct
	Reverse: tagttccccagatgcacctg
hp65	Forward: gcgagaggagcacagatacc
	Reverse: ctgatagcctgctccaggtc
hKLF2	Forward: acaccaagagttcgcatctg
	Reverse: acatgtgccgtttcatgtg
hKLF4	Forward: gccacccacacttgtgatta
	Reverse: cccgtgtgtttacggtagtg
hKLF11	Forward: cttaaggcccatcttcgcac
	Reverse: agcctgggatcttcttggtc
mKLF14	Forward: cctcaagtcacaccagcgta
	Reverse: cgacctcggtactcgatcat
mVCAM1	Forward: attttctggggcaggaagtt
	Reverse: acgtcagaacaaccgaatcc
mSELE	Forward: agctacccatggaacacgac
	Reverse: acgcaagttctccagctgtt
mp65	Forward: ggcctcatccacatgaactt
	Reverse: cactgtcacctggaagcaga
mKLF2	Forward: accaagagctcgcacctaaa
	Reverse: gtggcactgaaagggtctgt
mKLF4	Forward: ctgaacagcagggactgtca
	Reverse: gtgtgggtggctgttctttt
mKLF11	Forward: ggggagaagccttttacctg
	Reverse: agcctgggatcttcttggtt
hm18S	Forward: ggaagggcaccaccaggagt
	Reverse: tgcagccccggacatctaag
mTF	Forward: aaagggaagaacaccccgtc
	Reverse: atcagagctctccgcaacag
mvWF	Forward: ttgaagcccctggacaactt
	Reverse: gagataactgcagccccctg
mTFPI	Forward: ttttgggccactgtgtgtct
	Reverse: tcccttcacatcccccgtat
mPPARα	Forward: acaaggtcaaggcccgggtcatact
	Reverse: atcagcatcccgtctttgttcatca
mPPARγ	Forward: ttttcaagggtgccagtttc
	Reverse: aatccttggccctctgagat
mSREBP2	Forward: cacctgtggagcagtctcaa
	Reverse: tggtaggtctcacccaggag

mIRE1	Forward: cccaaatgtgatccgctact
	Reverse: ttgagagaatgcaggtgtgc
mATF6	Forward: ggccagactgttttgctctc
	Reverse: cccatacttctggtggcact
mBIP	Forward: agtggtggccactaatggag
	Reverse: caatccttgcttgatgctga
mCHOP	Forward: gcatgaaggagaaggagcag
	Reverse: cttccggagagacagacagg

h indicates human and m indicates mouse.

Supplemental References

1. Guo Y, Fan Y, Zhang J, Lomberk GA, Zhou Z, Sun L, Mathison AJ, Garcia-Barrio MT, Zhang J, Zeng L, Li L, Pennathur S, Willer CJ, Rader DJ, Urrutia R and Chen YE. Perhexiline activates KLF14 and reduces atherosclerosis by modulating ApoA-I production. *The Journal of clinical investigation*. 2015;125:3819-30.
2. Niwa H, Araki K, Kimura S, Taniguchi S, Wakasugi S and Yamamura K. An efficient gene-trap method using poly A trap vectors and characterization of gene-trap events. *Journal of biochemistry*. 1993;113:343-9.
3. Morita M, Ohneda O, Yamashita T, Takahashi S, Suzuki N, Nakajima O, Kawauchi S, Ema M, Shibahara S, Udono T, Tomita K, Tamai M, Sogawa K, Yamamoto M and Fujii-Kuriyama Y. HLF/HIF-2alpha is a key factor in retinopathy of prematurity in association with erythropoietin. *The EMBO journal*. 2003;22:1134-46.
4. Fan Y, Lu H, Guo Y, Zhu T, Garcia-Barrio MT, Jiang Z, Willer CJ, Zhang J and Chen YE. Hepatic Transmembrane 6 Superfamily Member 2 Regulates Cholesterol Metabolism in Mice. *Gastroenterology*. 150:1208-1218.
5. Lu H, Fan Y, Qiao C, Liang W, Hu W, Zhu T, Zhang J and Chen YE. TFEB inhibits endothelial cell inflammation and reduces atherosclerosis. *Science Signaling*. 2017;10.
6. Fan Y, Guo Y, Zhang J, Subramaniam M, Song CZ, Urrutia R and Chen YE. Kruppel-like factor-11, a transcription factor involved in diabetes mellitus, suppresses endothelial cell activation via the nuclear factor-kappaB signaling pathway. *Arteriosclerosis, thrombosis, and vascular biology*. 2012;32:2981-8.