SUPPLEMENTARY INFORMATION – ITEM 1: Supplementary text

1. Extended methods text

Endothelial cell isolation and culture. Commercial EC lines used: HAECs (Lonza, Barcelona, Spain; CatN°CC-2535), HCAECs (Lonza, CatN°CC-2585), HIAECs (ATCC, Barcelona, Spain; CatN° CRL-2475), HPAECs (ATCC, CatN° CRL-2598), HIVECs (ATCC, CatN° CRL-2606), and HPVECs (ATCC, CatN° CRL-2607). EC lines were cultured according to the provider's instructions. HHAECs, HHVECs, HUVECs and HUAECs were isolated at the Clinica Universidad de Navarra (after obtaining informed consent) by perfusing the corresponding vessel with collagenase type I (Invitrogen, Carlsbad, CA). Harvested cells were cultured for 24 hours, washed to discard non-attached cells, grown until 100% confluence and split 1:3 every 3-4 days. For the analysis of fresh cells or short time culture for time course analysis, ECs from umbilical arteries or veins were magnetically selected using anti-hCD34 magnetic beads (Miltenyi-Biotec, Madrid, Spain) and an AutoMACS magnetic selector (Miltenyi-Biotec) according to the manufacturer's instructions. Purity of magnetically sorted HUVEC-F or HUAEC-F was assayed by FACS. Only samples with more than 95% purity for $CD31^+CD34^+CD45^-$ cells were eligible for microarray hybridization or short time culture (Figure S1A,B). Analysis of probe set intensities of general endothelial or hematopoietic markers revealed high enrichment for the former and very low expression levels of the latter, supporting the purity of the used cell preparations (Figure S1C). To further exclude the possibility that the obtained fingerprint from the cell preparations isolated based on CD34 was biased by a small contamination of $CD34^+$ hematopoietic cells, we show that a similar enrichment of arterial or venous genes is obtained from cell preparations isolated based on another method, *i.e.*, by using $Tie2^+CD31^+CD45^-$ cell populations (Figure S1D,E).

RNA isolation, quality control and qRT-PCR. Total RNA from (sorted) cell lysates was extracted using TRIzol® reagent or RLT lysis buffer (Qiagen, Venlo, The Netherlands). The RNA integrity/quality of the samples used for microarray hybridization was determined with a Bioanalyzer-2100 (Agilent Technologies, Santa Clara, CA). mRNA was reverse transcribed using Superscript III Reverse Transcriptase (Invitrogen) and cDNA underwent 40 amplification rounds on an ABI PRISM-7700 cycler (Perkin Elmer/Applied Biosystems, Foster City, CA) for standard qRT-PCR. Primer sequences are listed in Table S1. mRNA levels were normalized using *GAPDH* as housekeeping gene, except for experiments in which cells were exposed to different oxygen levels, in which case we used *ACTB* for normalization. Data, expressed as mean ± SEM comparing two groups were analyzed by Student's *t*-test. SPSS software was used for statistical analyses and differences were considered significant when $P<0.05$.

Microarray hybridization and statistical analysis. RNA hybridization of the 38 human EC samples was done in collaboration with the Department of Hematology, Hospital Universitario de Salamanca, using the Affymetrix HG-U133 Plus2.0 GeneChip Oligonucleotide Microarray (Affymetrix, Santa Clara, CA, USA). All steps were carried out according to the manufacturer's protocol. 100 ng of RNA was amplified and 15 μg of amplified and labeled cRNA was hybridized on the array. Arrays were scanned using a GeneChip Scanner-7G. Background correction and normalization were done using the RMA (Robust Multichip Average) algorithm[1]. The *GAPDH* gene was used for normalization with coefficient of variation of < 5% between all conditions used for comparison.

The method for differential gene expression analysis was the one contained in the LIMMA Bioconductor package. To control the false discovery rate, multiple testing correction was performed and probes with a corrected *P*-value below 0.05 were selected. For the classification analysis, a filtering process was applied first to eliminate probe sets with low expression values. Applying the criterion of an expression value greater than 32 in 5 samples for each experimental condition, 32,939 probe sets were selected for statistical analysis using the LIMMA Bioconductor package. Prediction analysis for microarrays[2] was applied to classify arterial and venous samples and identify genes that were associated with each specific class. This algorithm ranked genes using a penalized *t*-statistic and identified a gene set for classification with soft thresholding. Gene number was controlled by a thresholding parameter, which was determined with a 10-fold cross-validation. This parameter was manually selected to minimize the overall error rate (t=7). The obtained classifier required 78 probes. Functional and pathway enrichment analysis was done using Ingenuity Pathway Analysis software (Ingenuity Systems, Redwood City, CA, http://www.ingenuity.com).

Time course analysis with TLDA. Taqman® Low Density Array plates with Taqman® primers for the arteriovenous fresh profile were obtained from Applied Biosystems. HUAEC-F/HUVEC-F samples and samples from HUAECs and HUVECs cultured for 24 hours, 48 hours or 6 days without passaging were run on a 7900HT fast real-time PCR system (Applied Biosystems, Lennik, Belgium) and analyzed according to the manufacturer's instructions. *GAPDH* was used as housekeeping gene with coefficient of variation of < 5% between all conditions used for comparison. Each condition was run in quadruplicate. Data, expressed as mean ± SEM comparing two groups were analyzed by Student's *t*-test. SPSS software was used for statistical analyses and differences were considered significant when $P<0.05$.

nCounter analysis. DLL4-Fc activated or BSA-treated HUAECs ($N=3$) and HUVECs transduced with Cherry, each of the 8 TFs or a combination of them ($N=4$-6) were used for nCounter analysis. Briefly, RNA, extracted using TRIzol® reagent was quantified and quality controlled with a Bioanalyzer-2100 (Agilent Technologies) and samples were processed in collaboration with the VIB Nucleomics Core Facility. 100-500 ng of total RNA was hybridized according to the manufacturer's instructions. Some of the results were confirmed by qRT-PCR and genes for which the probe intensity value was low were analyzed by qRT-PCR. Data were normalized by scaling to the *GAPDH* gene with coefficient of variation of < 5% between all conditions used for comparison. The scaled counts were base log2-transformed. Testing whether a contrast was significantly different from 0 was done by using a moderated *t*-test, as implemented in LIMMA. The resulting *P*-values were corrected for multiple testing with Benjamini-Hochberg to control the false discovery rate. $P<0.05$ and at least 50% change differences in comparison with control (BSA-treated HUAECs or Cherry-transduced HUVECs) was considered as significant. The Nanostring probe list is provided in Table S2. The global expression profiles of the samples were represented in the form of a heat map, using the gplots package of R. Hierarchical clustering was used to cluster the individual gene expression profiles based on Pearson correlation and complete linkage.

Immunofluorescence staining and Western blot. The procedure for immunofluorescence staining was done on human umbilical cord paraffin sections as described previously[3]. Antibodies used were: goat anti-human Msx1 (R&D Systems, Abingdon, UK; AF5045), Alexa-488-labeled mouse anti-human smooth muscle α-actin (Sigma,

F3777), and rabbit anti-human Nr3c2 (SantaCruz, Santa Cruz, CA, USA; SC-11412). Images were recorded on a Zeiss Axiovert 40CFL microscope equipped with a Zeiss MRm camera and Axiovision 4.5 software (Carl Zeiss, Zaventem, Belgium). Western blot was performed as described[4]. Samples were collected in RIPA buffer (Sigma) and protein concentration was measured by the BCA assay. 40 μg of protein was used for blotting. Blot pictures were recorded with a Bio-Rad Chemidoc XRS+ molecular imager, equipped with Image Lab software (Bio-Rad laboratories, Nazareth, Belgium). Antibodies used were: rabbit anti-human Rasgrf2 (Sigma, HPA018679), rabbit anti-human A2M (Sigma, HPA002265), rabbit anti-human MAP9 (Sigma, HPA037864) and mouse anti-human α-tubulin (Sigma, T6199; used as loading control).

siRNA knockdown and Notch activity assays. siRNA knockdown was performed using *Silencer*® Select pre-designed siRNA from Applied Biosystems for *RBPJκ* (siRNA ID#: s7251 and s7253), Negative Control-1 (siRNA ID#: am4636). Briefly, 2,500 HUAECs/cm^2 were cultured overnight. The next day, cells were transfected with 5 pmol siRNA mixed with 0.5 μl of lipofectamine 2000 (Invitrogen) in 100 μl of OPTI-MEM (Invitrogen). The day after transfection, media was replaced and cells were maintained for 6 days, with an additional siRNA transfection at day 3. The canonical Notch pathway was induced in cultured HUAEC (passage 1-2) by immobilized DLL4 ligand activation. Briefly, DLL4-Fc (R&D Systems, CatN° 1389-D4) was incubated overnight at 4ºC at 1 μg/ml in 0.1% gelatin 1% BSA in PBS with gently shaking to allow its adsorption to the cell culture dish. The next day, DLL4-Fc coated plates were incubated at 37º for 1 hour. Non-attached DLL4-Fc was removed by washing and 2,500 HUAECs/cm^2 were seeded and cultured for 72 hours. The canonical Notch pathway was blocked by γ-secretase inhibitor DAPT (Calbiochem, San Diego, CA, USA; CatN° 565784; alone or in combination with immobilized DLL4-Fc) at 3 μM concentration for 24 or 72 hours, followed by RNA extraction and gene expression analysis. The corresponding DMSO volume was used as a control. In a separate set of experiments, freshly isolated HUAECs were cultured for 24 hours in medium containing DAPT (or DMSO) before RNA extraction and gene expression analysis.

Lentivirus production and overexpression. The lentiviral construct for constitutively overexpressing human Hey2 was obtained from Genecopoeia (Rockville, USA; CatN° EX-U0515-Lv114). Open reading frames (ORF) for human *MSX1*, *EMX2*, *NKX2-3*, *TOX2* and murine *Aff3* and *Prdm16* were cloned from cDNA-containing plasmids (Thermo Scientific Molecular Biology, Pittsburgh, PA, USA) or total human cDNA (reverse transcribed from human normal tissues universal RNA; Gentaur, Brussels, Belgium) after the cytomegalovirus (CMV) promoter in pRRL2-CMV-PGK-Cherry. The lentiviral construct for constitutive overexpression of human Sox17 was kindly provided by C. Verfaillie (Stem Cell Institute, KU Leuven; viral backbone: pLVX-IRES-Hyg from Clontech, CatN°672185). For lentiviral particle production, HEK293 cells were plated (5x10^6 cells/10 cm dish) and the next day transfected with the plasmid of interest together with two helper plasmids (psPax2 and PMD2G) using Fugene® transfection reagent (Roche Applied Science, Vilvoorde, Belgium). In brief, 400 μl of OPTI-MEM (Invitrogen) was mixed with 1 μg PMD2G, 3 μg psPax2 and 4 μg lentiviral construction plasmid. 24 μl of Fugene® was added and the mixture was incubated for 20 minutes at room temperature and gently applied to the cells. The next day, medium was replaced and lentiviral particle-containing supernatant was collected 36 hours later. Viruses were concentrated by centrifugation using 50,000 MWCO Vivaspin® 20 ml centrifugal

concentrators (Sartorius AG, Goettingen, Germany). Transduced cells were kept for 6 days or passaged until 28 days and collected into TRIzol® buffer. For regulable overexpression, *EMX2*, *NKX2-3* or *GFP* were cloned behind a doxycyclin-inducible minimal CMV promoter in a lentiviral vector also containing rtTA3 and Cherry under the control of a constitutively active ubiquitin promoter, the latter allowing for evaluation of transduction efficiency (Figure S2A). Transduced cells were cultured in media containing 2 μg/ml doxycyclin for 6 days, one half was harvested for RNA extraction and the other half was divided in two parts, one for continuing culture for 4 days in doxycyclin-containing media, the other for switching to media without doxycyclin for 4 days. GFP-encoding virus was used to test the doxycyclin-inducible switch (Figure S2B).

In vivo *Matrigel implantation assay.* $0.5x10^6$ HUVECs transduced with a lentivirus encoding Cherry or each of the 8 TFs were mixed with pre-cooled 0.5 ml of Growth Factor-Reduced Matrigel containing 300 ng/ml $VEGF_{165}$ and 700 ng/ml bFGF (R&D Systems) and subcutaneously injected in the back of 8-weeks-old athymic nu/nu mice (*N*=5 per group). 14 days later, mice were sacrificed by cervical dislocation and the Matrigel plug was dissected out. The Matrigel plug was divided in two equal pieces, one piece was processed for cryo sectioning and the other for paraffin sectioning. Human cells were detected by the Cherry fluorescent signal or by human-specific CD31 staining (Dako). Smooth muscle coverage was analyzed on smooth muscle α-actin (Sigma) stained sections and collagen deposition was quantified on Sirius red-stained sections (and examined under polarized light). Animal studies were approved by the Ethical Committee at KULeuven.

2. Legends to supplementary figures

Figure S1. ***Purity after and validation of MACS column-sorting of HUAECs and HUVECs***

(**A-B**) FACS analysis of freshly MACS column-sorted HUAECs (A) or HUVECs (B) revealing only minimal (< 1% in both cell populations) contamination with $CD45^+$ blood or hematopoietic cells and high purity (> 97% in both cell populations) for $CD31^+$ and $CD34^+$ endothelial cells. (**C**) Diagram representing average log2-transformed probe set intensities (± SEM) for endothelial cell (EC; left) and hematopoietic (right) marker genes for HUAECs (red bars) and HUVECs (blue bars) preparations used for microarray analysis (*N*=5; *: *P*<0.05 *versus* HUVEC). (**D-E**) Alternative FACS sorting strategy for endothelial cells from 3 human umbilical cords based on positive selection for Tie2 and CD31 (Dleft) and negative selection for CD45 (Dright), revealing an enrichment of arterial and venous markers in HUAECs (red bars) and HUVECs (blue bars) preparations (E), respectively, thereby validating the MACS-based method based on CD34 positive selection.

Figure S2. ***Effect of reversible overexpression of EMX2 or NKX2-3 on gene expression in HUVECs***

(**A**) The gene encoding EMX2, NKX2-3 or GFP was cloned behind a doxycyclin-responsive minimal CMV promoter in a lentiviral vector also containing rtTA3 and Cherry under the control of the constitutively active ubiquitin promoter. (B) Cells were plated at day 0, and infected with lentiviral particles 1 day later. After 6 days of exposure to doxycyclin RNA was harvested from half of the cells. The GFP encoding lentivirus was used to monitor the efficacy of the doxycyclin switch. The other half of the cells was replated and exposed to two different conditions, *i.e.*, continuation of culture in doxycyclin-containing media for 4 days (upper arm, dark green) or a switch to culture in media without doxycyclin for 4 days (lower arm, light green). Cells were lysed for

RNA extraction at day 10. (**C**) Upper left panel represents expression levels of *EMX2*, expressed as ΔC_T *versus GAPDH* (± SEM), the remaining panels show expression levels of EMX2-responsive genes relative to those of GFP overexpressing cells (± SEM) at day 6 or 10 with doxycyclin (dark green bars) or day 10 following removal of doxycyclin (light green bars; *N*=5-6; *: *P*<0.05 *versus* day 10 + doxycylin). (**D**) Upper left panel represents expression levels of *NKX2-3*, expressed as ΔC_T *versus GAPDH* (± SEM), the remaining panels show expression levels of NKX2-3-responsive genes relative to those of GFP overexpressing cells (± SEM) at day 6 or 10 with doxycyclin (dark green bars) or day 10 following removal of doxycyclin (light green bars; *N*=5-6; *: *P*<0.05 *versus* day 10 + doxycyclin).

Figure S3. ***The cell culture process assimilates arterial and venous endothelial cells***

Hierarchical clustering analysis of all 38 endothelial cell (EC) samples for all 102 probes (corresponding to 76 annotated genes of the arteriovenous fresh profile) reveals that freshly isolated cells (on the left) cluster according to their venous or arterial origin, while for cultured cell types (on the right) the clustering does not classify the sample groups correctly, suggesting that the differences in expression profile have been largely erased. Arterial cell types are represented by a red color while venous cell types are represented by a blue color in the color bar below. The color code for expression levels is displayed on top. HUAEC: human umbilical artery EC; HUVEC: human umbilical vein EC; HPVEC: human pulmonary vein EC; HHAEC: human hepatic artery EC; HHVEC: human hepatic vein EC; HIVEC: human iliac vein EC; HCAEC: human coronary artery EC; HAEC: human aortic EC; HPAEC: human pulmonary artery EC; HIAEC: human iliac artery EC; NA: not assigned.

Figure S4. ***Kinetics of the culture-induced assimilation process***

Diagrams on the left represent expression levels for all arterial genes (*N*=64) of the arteriovenous fresh profile in HUAECs. Panel A shows expression levels after 24 hours of culture (filled red diamonds) relative to those in freshly isolated HUAECs (open black triangles). Panel C represents expression levels after 48 hours of culture (open red circles) relative to those after 24 hours of culture (filled black diamonds). Panel E represents expression levels after 6 days of culture (filled red squares) relative to those after 48 hours of culture (open black circles). Diagrams on the right show expression levels for all venous (*N*=12) genes of the arteriovenous fresh profile in HUVECs. Panel B represents expression levels after 24 hours of culture (filled blue diamonds) relative to those in freshly isolated HUVECs (open black triangles). Panel D represents expression levels after 48 hours of culture (open blue circles) relative to those after 24 hours of culture (filled black diamonds). Panel F represents expression levels after 6 days of culture (filled blue squares) relative to those after 48 hours of culture (open black circles). All diagrams together reveal that loss of expression occurs within 24 hours after culturing for the majority of arterial and venous genes in HUAECs and HUVECs, respectively, an effect that further increases rapidly independent of cell passaging. The percentage of genes with expression lower than the reference (corresponding to the expression levels in freshly isolated cells indicated by a dashed line) are mentioned on the right side of each diagram.

3. Legends to supplementary tables

Table S1. ***Primer list for qRT-PCR.***

Genes are sorted alphabetically and primer sequences are listed as 5' to 3'. For the transcription factors, to make a distinction between the endogenous expression levels and those resulting from the overexpression, the former levels were determined by using primers annealing to the 3' untranslated region (UTR) which was not cloned into the lentiviral expression vectors.

Table S2. ***Nanostring probelist.***

The table lists probes corresponding to all the genes of the arteriovenous fresh profile, in addition to some established arterial or venous endothelial markers, as well as housekeeping genes. Probes for the arteriovenous fresh profile are ordered according to Table S3.

Table S3. ***Probe set intensities in HUAECs/HUVECs of genes contained within the arteriovenous fresh profile.***

The table represents the average microarray probe set intensities for all 102 differentially expressed probe sets (corresponding to 64 annotated arterial genes and 12 annotated venous genes) in freshly isolated HUAECs (column A; *N*=4) and HUVECs (column B; *N*=4) or cultured HUAECs (column C; *N*=5) or HUVECs (column D; *N*=5). Rows for both arterial and venous markers are sorted according to the degree of differential expression between freshly isolated HUVECs and HUAECs (column E). Calculated differences between freshly isolated HUAECs or HUVECs and their cultured counterparts reveal dramatic loss of expression of arterial markers in cultured HUAECs (column F) and venous markers in cultured HUVECs (column G). As a result, expression differences between cultured HUAECs and HUVECs for the majority (~73%) of the probe sets in the arteriovenous fresh profile were small (≥ -1 or ≤ 1; column H). NA: not annotated.

Table S4. ***Additional information regarding the genes contained in the arteriovenous fresh profile.***

The table lists, in an alphabetical order for arterial and venous genes, information from the literature and taken from the Gene Cards website (http://www.genecards.org/cgi-bin/cardsearch.pl) about the 76 genes contained within the arteriovenous fresh profile. For 43 of the genes, a knockout mouse has been generated and reported in the literature. For 13 of these (gene name highlighted in bold-face), a vascular phenotype has been described. For 7 genes (or a related family member) an association with cardiovascular disease or risk factors has been described (underlined text). Twenty-two genes encode a cell surface protein, 14 of the genes encode a secreted protein and 27 of the genes encode an intracellular protein, 9 of which encode a transcription factor. The remaining genes encode a protein with unknown ('?') or variable subcellular localization or are non-coding ('-'). For 16 of the genes (gene name box colored in orange) expression has been documented in arterial or venous endothelial cells and for 4 of them a role in arterial specification has been previously demonstrated (gene name underlined). Finally, 10 genes have been linked with the Notch pathway (either the gene itself or a close family member or orthologue; text highlighted in bold). Abbreviations: EC: endothelial cell; TF: transcription factor; FGF: fibroblast growth factor; VEGF: vascular endothelial growth factor; CHD: coronary heart disease; KO: knockout; E: embryonic day: P: postnatal day; SMC: smooth muscle cell; BM: basement membrane; NAAG: N-acetylaspartylglutamate; EMT: epithelial-to-mesenchymal transition; IGF: insulin-like growth factor; GC: guanylate cyclase; ER:

endoplasmatic reticulum; NMDAR: N-methyl-D-aspartate receptor; CREB: cAMP response element-binding; HIMEC: human intestinal microvascular endothelial cells; HSC: hematopoietic stem cells; ISV: intersomitic vessels; AAA: abdominal aortic aneurysm; ECM: extracellular matrix; MHV-3: murine hepatitis virus strain 3; TJ: tight junctions; IEL: internal elastic lamina; ROS: reactive oxygen species; HGF: hepatocyte growth factor; NSC: neuronal stem cells; MDS: myelodysplastic syndrome; PDGF: platelet-derived growth factor; LTP: long term potentiation; NRP: neuropilin; CNS: central nervous system; PDZ: post synaptic density protein (PSD95), Drosophila disc large tumor suppressor (Dlg1), and zonula occludens-1 protein (ZO-1).

Table S5. ***Expression of genes from the arteriovenous fresh profile in DLL4-Fc-treated HUAECs.***

The table represents relative expression levels (± SEM; *N*=4) for the 76 genes in the arteriovenous fresh profile upon exposure of cultured HUAECs to DLL4-Fc compared to bovine serum albumin (BSA)-treated HUAECs. Genes are sorted according to the same order as in Table S3. *: $P<0.05$ *versus* BSA.

Table S6. ***Expression of genes from the arteriovenous fresh profile in cultured HUVECs overexpressing TFs.***

The table represents relative expression levels (± SEM; *N*=4-6) for the 76 genes in the arteriovenous fresh profile upon lentiviral transduction of HUVECs with a single TF or a combination of all 8 TFs, compared to those in HUVECs transduced with a Cherry control lentivirus. Genes are sorted according to the same order as in Table S3. *: $P<0.05$ *versus* Cherry control. #: $x10^3$; aEC: arterial endothelial cell; vEC: venous endothelial cell; ND: not detectable.

4. References for all supplementary items

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SUPPLEMENTARY INFORMATION – ITEM 2: Table S1

Table S1. *Primer list for qRT-PCR.*

Gene symbol	Forward primer	Reverse primer	Gene symbol	Forward primer	Reverse primer
A2M	CGCCTCAGTCTCTGGAAAAC	GCCTCCAGGTCAGTGAAGAG	*MFAP5*	ATACCCCTGGGGGCTAAATAG	CGTCGTAAACTGGTGAAGCA
ACE2	ATACTGTGACCCCGCATCTC	ATGCTAGGGTCCAGGGTTCT	*MGST1*	GACCTCACCCAGGTAATGGA	TACAGGAGGCCAATTCCAAG
ADRB1	CGAGACCCTGTGTGTCATTG	AGCACTTGGGGTCGTTGTAG	*MPP7*	AAAAAGCCTGCATTCATTGG	AGGCATAGGAGGCAACACTG
AFF3	TTTCAGTCATCAGCCAGCAG	AAGTGTTCTGGATCCGGTTG	*MPPED2*	GGTTTAATGGATGGGGCTTT	CATGGATTCCACCAAACACA
AFF3-UTR	GCCGCCTGTGTATGTGTGTA	ACGGTTTAGCACTGGAATGG	*MSX1*	ACTCCTCAAGCTGCCAGAAGA	AGCTCTGCCTCTTGTAGTCTCTTT
APM2	TGCTCTTGACGACTCCACAG	TCAGAGGCCTGGTTAGCAGT	*NAV1*	CCCAAAGAACTTCGGATCAA	GGAGGGTGAAGCAGTCTCTG
ARL15	AAGAACTTGGAGGGGCTGAT	CTGCTGGCTTGTCTTGATGA	*NKX2-3*	GAGCCCAAGGAACATGGAGA	CTGGGCTTGCGAGAAGAG
ACTB	TGGCACCACACCTTCTACAATG	TAGCAACGTACATGGCTGGG	*NKX2-3-UTR*	ATTTCGTTTCGCAGCTGTCT	AAAGGAAACCCGGTAACACC
CCDC3	TTGCCTCACGGAGTCAATTT	CGAGGAGCACATGAGCCTA	*NOS1*	CCCTTCAGTGGCTGGTACAT	CGGAGTGATGGTCAACAATG
CDH5	GTTCACGCATCGGTTGTTC	TCTGCATCCACTGCTGTCA	*NOTCH1*	CCACGGGCGACGTCACCC	TCCACTCTGGCGGGCACG
CMBL	GCAGTTGCCCAATACCAGAT	TCTGGGCATGACACTGTTGT	*NOTCH4*	ATGTCTCAATGGCGGCTCC	GGAGAAGGTGCCAGGCCT
CNTN3	CCAGCAGCTAAAGGTTCGAC	GTAAGTGAGACGCCCTCTGG	*NPR3*	GGAGACCGATATGGGGATTT	CACTGCCGATTCTTCTAGGC
COL4A1	CTGGCCAGAAAGGAGAGATG	TCATTGCCTTGCACGTAGAG	*NR2F2*	CGCCTCAAAAAGTGCCTCA	GCATCCTGCCCCTCTGC
COL4A2	CACCTTCCACCCAGATCAGT	CTCTGGCACCTTTTGCTAGG	*NR3C2*	AACAGGTAGACGGCGAGAGA	TTTGGAATAGCACCGGAAAC
CUBN	ACTGTGAAGGGGGTTCTGTG	GACAGGCCCCACAGTAGAAA	*NRCAM*	TCACCATTGTGGACCAAAGA	CTCTGGGAGGACATTGGAAA
CYSLTR1	TGAGAACAAACGCAAAAGGA	CTTGATTGCGGAAGTCATCA	*NRP2*	GCGAGTGGATTGTTTACGCC	CAGTCTTTGCCCAGGAGGTC
DLL4	ATGACCACTTCGGCCACTATG	GCCCGAAAGACAGATAGGCTG	*ODAM*	CAGGCCAAGTTGATCCCTTA	TGAGGTTGTTCCCAGGGTAG
EFNB1	GTTCTCGACCCCAACGTGTT	CAGGCTTCCATTGGATGTTGA	*OLFML3*	AACGCCGACTAGCTGCTTTA	GCTCCAGACGATCCACTCTC
EFNB2	CTGCTGGATCAACCAGGAAT	TCCGGTACTTCAGCAAGAGG	*PAPLN*	GGCAAGAGGGATGTCTGTGT	AGCTCGGCTGAGGTCATTAG
EMX2	ACCTTCTACCCCTGGCTCAT	AGCCTTCTTCCTCCAGCTTC	*PDE4D*	CCAGTCTGCGAACTGTACGA	TGCCTGGTCTGTAGGGTCTC
ENG	TGCCACTGGACACAGGATAA	CCTTCGAGACCTGGCTAGTG	*PECAM1*	TCTGCACTGCAGGTATTGACAA	CTGATCGATTCGCAACGGA
EPHB4	GAGCTGTGTGGCAATCAAGA	GAACTGTCCGTCGTTTAGCC	*PRDM16*	CAAACGCTTCGAATGTGAAA	CGTGTAGGACTTGTGGCAGA
FAM19A5	CGCAGTTCCTCAAAGAAGGT	GACACGGAAGCATGTCACAC	*PRDM16-UTR*	CTGCAAAACACTTGCCTGAA	GACCAGGAGCAGCTATGTCC
FAP	CCAGGAGATCCACCTTTTCA	ACGCAGGGTAAGTGGTATCG	*PSMAL*	TTGGAATCTTCCTGGAGGTG	CTGCTATCTGGTGGTGCTGA
FAT1	GTGGAAAAGGGGACAGTGAA	CCTGATCGGTTGCAAAGACT	*PTPRR*	TTGTCTGCCAGCTTCGTATG	GGGCTTCAGAGCTTCTCCTT
FGL2	TTGGATGGCAAATGTTCAAA	CCATGGTCTCCATGTCACAG	*RASGRF2*	ACCTTGCCATCGAAAGAATG	ACAGAACCCCACCTGTCTTG
FLRT3	ACAGTGTATCCTGCCCAAGG	CTGCGTTCCCCTGTTACAAT	*RBPJ*	GGCAGTGGATGGAAGAAAA	CTTTTATCCGCTTGCTGAGG
FOLH1	TTGGAATCTTCCTGGAGGTG	CTGCTATCTGGTGGTGCTGA	*RUNDC3B*	GTATCTGCAGCATTGAAAATATGG	CAATAGCATTGAGTCCTAGAAGCA
FREM1	ATGGATGTAGTGGGGCAGAC	GTCAGCTGAAGGAGGTCAGG	*RYR3*	AAATTGCTGGCTGCTCTCAT	TTAAGGCTTCTGGGCTTTCA
GALNTL4	CGACAAGAAGCTGGAGGAAG	GGTAGGCGTTGTAGCCGTAG	*SCARA3*	CGCTGCCAGAAGAACCTATC	CAGCTCCTCCTGCAGTTTTC
GAPHD	TGGTATCGTGGAAGGACTCATGAC	ATGCCAGTGAGCTTCCCGTTCAGC	*SEMA3C*	TCATTCCATGATTGCTCGAA	CCTCGGGTTATCAGTTTCCA
GLIPR2	GCTCTGCAAGAACCTCAACC	ATACCATGGCCGTGAAGTGT	*SEMA3G*	ACGGAGCACAATAGCACCTT	GACCACAGTCTGGGAGAAGC
GRB14	AAATCCCACTGAAGCCCTTT	CCCGTACCAAGAAAACTCCA	*SLC2A1*	CCTGCAGGAGATGAAGGAAG	ACAGCGACACGACAGTGAAG
H19	GAGCTCTCAGGAGGGAGGAT	CCAGCCTAAGGTGTTCAGGA	*SLC2A3*	TGGTTATTGGCCTCTTCTGC	AAGGGCTGCACTTTGTAGGA
HECW2	TCCAGCATTCCCTATGAAGG	GCTTCTGAAACTGCCTGTCC	*SNTB2*	GCAAAGATACAGCCACAGCA	TGTCCACGGCATACAGTCAT
HEY1	GAGAAGGCTGGTACCCAGTG	GCTCAGATAACGCGCAACTT	*SOX17*	CAGCAGAATCCAGACCTGCA	CAGCGCCTTCCACGACTT
HEY2	CCCCTGCGAGGAGACGA	ATCTAATCACAGAGCTAGTACTTTGCCC	*SOX17-UTR*	TGACTCCGGTGTGAATCTCC	GCAACAACAAAAACCCAGGA
HEY2-UTR	TTCAAGGCAGCTCGGTAACT	CAGGCACTTACGAAACACGA	*SOX18*	AGAACCCGGACCTGCACA	CAGCTCCTTCCACGCTTTG
HSF2BP	TCGTTAGACGGTGATGTCCA	CCCAGAAGCTGCAATATGGT	*SOX7*	GGCGGCCCATGAACG	TCCACGTACGGCCTCTTCTG
JAG1	CCAATGACTGCAGCCCTCAT	GCTCCAAAGGCACAAGGTGA	*SYTL2*	TGTTTTTGTGGCCCAGTGTA	AGTTCCACCTCCCCTAGGAA
JAG2	TCATCCCCTTCCAGTTCGC	AGGCTCTTCCAGCGGTCCT	*TFPI2*	GTCGATTCTGCTGCTTTTCC	CACTGGTCGTCCACACTCAC
KDR	TGGCATCGCGAAAGTGTATC	AAAGGGAGGCGAGCATCTC	*TEK*	ACACCTGCCTCATGCTCAGC	AGCAGTACAGAGATGGTTGCATTC
KLHL6	TTTGAGACCGTGATGAGCTG	CTCATTGCCAGAAAGGTGGT	*TMEM200A*	GAGCAGCATTTGCATTCTGA	ACCAGTGTACATGCCGTTCA
LAMA2	AGGTGAATGTGGAAGGCATC	GGGGTAGAATGGTCTGCTCA	*TMEM200C*	CAGTAGCAGTGGCAGCAAAA	CCGAAGACCTTGAGCTTGTC
LMCD1	CCAAGAGGACCACTGCCTAA	GTAAAAGGCACCCTCTGTGC	*TOX2*	TCAGGAAGAGGAGTCGGAAG	CACGATTTTGGACACGTCAC
LOC401022	TCAGGTCCCTGGATAAGGTG	GTGAGAGCTTTCCCTGCAAC	*TOX2-UTR*	ATCTCTGAGTTCCCCAGCAG	TTTTCTGCTGTCAGCCCTTC
LRCH1	GAGCATACAAGCATGCCAGA	TGGCACATTGAGGCTAACAG	*VWF*	TGCTGGTATGGAGTATAGGCAGTG	CCGGAATGCACGCAGG
MAP9	ACATGGAGGAGAAGGATGGA	CAGATGCGTTTCCCTCAGAT	*XG*	AGCTGGGAGACCAGAAGTCA	GGCATCTGCCAAATCAAAGT

SUPPLEMENTARY INFORMATION – ITEM 3: Table S2

Table S2. *Nanostring probelist.*

Probe set ID	Gene	Targeted region	Target sequence
Arterial genes from the arteriovenous fresh profile			
NM_003480.2	*MFAP5*	55-155	CTCATCTCATTGTTTCAGCGGAGGCCAAATCTGAAGTCCTTTCCAGGGAGTGGCTCTGTTCATCTTATTCGCCAGCCAAAGTAGGAACAGCGTAAGAGGA
NM_015381.3	*FAM19A5*	320-420	GCCGGCACCACGAGAGCCCGGCCCGCCTGTGTGGACGCAAGAATCATCAAGACCAAGCAGTGGTGTGACATGCTTCCGTGTCTGGAGGGGGAAGGCTGCG
NM_130846.1	*PTPRR*	670-770	GTCCAACGTATCTCTTACATTGGACATGAGTAGCTTGGGGAACATTGAACCCTTTGTGTCTATACCAACACCACGGGAGAAGGTAGCAATGGAGTATCTG
NR_002196.1	*H19*	1592-1692	GGCCTTTGAATCCGGACACAAAACCCTCTAGCTTGGAAATGAATATGCTGCACTTTACAACCACTGCACTACCTGACTCAGGAATCGGCTCTGGAAGGTG
NM_006682.2	*FGL2*	250-350	CAATTCAGCAGGATCGAGGAGGTGTTCAAAGAAGTCCAAAACCTCAAGGAAATCGTAAATAGTCTAAAGAAATCTTGCCAAGACTGCAAGCTGCAGGCTG
NM_000908.2	*NPR3*	35-135	TTGCAGAGAAGGACGCTTCCTCTCTATCTTTTGGCGCATTAGTGAAGGGGGTATTCTATTTTGTTAAAGCGCCCAAGGGGGCGCAGGGACCTTGGAGAGA
NM_020872.1	*CNTN3*	3030-3130	CCAGTAGTGAACAGATCAGGATTCCACGAATAACCAGTATGGATGCAAGAGGATCCACTTCAGCCATCTCGAATGTCCACCCTATGTCAAGTTATATGCC
NM_006909.1	*RASGRF2*	2675-2775	GAGTCCACCAGGCTTTAACAACACCGAGAGAACATGTGATAAAGAGTTTATTATACGGAGAACGGCTACCAATCGAGTTCTGAACGTCCTCCGTCACTGG
NM_020163.1	*SEMA3G*	2591-2691	CCCAAGGGATCTGCCATTTGTTCTCAGAGATGGCCTGGCTTCCGCAACACATTTCCGGGTGTGCCCAGAGGCAAGAGGGTTGGGTGGTTCTTTCCCAGCC
NM_199454.2	*PRDM16*	4455-4555	AACCCGCACTGTGCCTGGGTGTATTCTTTATACTGTAGATAATGGAGAAATTTTCTATCTCTGTCCCTATTTGTATAAGCCAAGGTGATGCTGGGTGCCC
NM_001584.2	*MPPED2*	550-650	TGAAACCAGAGGACTTTGACAATGTTCAGTCCCTCCTGACAAACAGTATTTACTTACAAGATTCGGAGGTAACAGTGAAGGGATTCAGGATATACGGTGC
NM_000014.4	*A2M*	1685-1785	TCCATCTCAATCCCTGTGAAGTCAGACATTGCTCCTGTCGCTCGGTTGCTCATCTATGCTGTTTTACCTACCGGGGACGTGATTGGGGATTCTGCAAAAT
NM_014059.2	*RGC-32*	765-865	TCCGAGGACCTGCTAAAATCAGCTACTAGAATCTGCTGCCAGAGGGGACAAAGACGTGCACTCAACCTTCTACCAGGCCACTCTCAGGCTCACCTTAAAA
NM_000426.3	*LAMA2*	3855-3955	GGCAAACTCAAGTATGCAATCTATTTCGAGGCTCGGGAAGAAACAGGTTTCTCTACATATAATCCTCAAGTGATCATTCGAGGTGGGACACCTACTCATG
NM_052913.2	*TMEM200A*	3355-3455	CCTTGTGGTTCCTTTGCCCAACACCAGTGAATCCTTCCAGCCCGTCAGCACAGTGCTACCAAGGAATAATTCCATTGGGGAGTCGTTGTCGAGTCAGTAC
NM_000684.1	*ADRB1*	795-895	TGTGCATCATGGCCTTCGTGTACCTGCGGGTGTTCCGCGAGGCCCAGAAGCAGGTGAAGAAGATCGACAGCTGCGAGCGCCGTTTCCTCGGCGGCCCAGC
NM_006528.2	*TFPI2*	600-700	TTTAATCCAAGATACAGAACCTGTGATGCTTTCACCTATACTGGCTGTGGAGGGAATGACAATAACTTTGTTAGCAGGGAGGATTGCAAACGTGCATGTG
NM_022454.3	*SOX17*	1374-1474	AGCCACGGGGCCATTTCCTCGGTGGTGTCCGACGCCAGCTCCGCGGTATATTACTGCAACTATCCTGACGTGTGACAGGTCCCTGATCCGCCCCAGCCTG
NM_006931.2	*SLC2A3*	35-135	GGCTGGGGGCTTGTCGCCCTTTCAGGCTCCACCCTTTGCGGAGATTATAAATAGTCATGATCCCAGCGAGACCCAGAGATGCTGTAATGGTAAGACTTTG
NM_032379.3	*SYTL2*	1760-1860	CATCATGTGTTTCACACCACAATTCTGTGCCACAGTTAAGAGGGTCTGGTACCCTTGCAGGACCTTTGTAGGTTGTGGGAAAAAGTCGCAGAAAGATACT
NM_130446.2	*KLHL6*	3365-3465	CCGGAACTGTCATTGCCAGACAGAATTGTACCTTTAGCACATTCTGGTGCCCTGGCCTCTTAGTGTTCACCAGTTGTACTGCAATATAGACAAGATGTCT
NM_145285.2	*NKX2-3*	1646-1746	CTCCGCGAAGCGTTGGCGGGGAGCCCAAGGACATAACAAATTAAAAGCATGAAGGAGAGAAAAATGGGGGTCGTGGCTTGAGAAATTCCAGGCCCTACCG
NM_020760.1	*HECW2*	3895-3995	GAAAAGACCTGCAGAGAAATAAGCTATATGTCACCTTCGTTGGGGAGGAAGGGCTGGATTACAGTGGGCCTTCTAGAGAGTTTTTCTTCCTGGTATCCAG
NR_023391.1	*CYP4F30P*	1740-1840	GAGGTCAGACCTTGGACAGTAGTCTTGACTTCCTGCTATAGAACACATTGTTAACACTGAAAAAGATGATCTGTTCTAGGGGAATGGTGAAAGCTGACTC
NM_144966.5	*FREM1*	1942-2042	GCCATTCTGAGAGGAGACATGATGAGGTAGAATTGGAGGTATACGACTTCTTCTTTGAAAGGAGTGCACCTATGACAGTCCACATCTCCATCAGAACAGC
NM_001846.2	*COL4A2*	1150-1250	GGCATTTCCTTGAAGGGAGAAGAAGGAATCATGGGCTTTCCTGGACTGAGGGGTTACCCTGGCTTGAGTGGTGAAAAAGGATCACCAGGACAGAAGGGAA
NM_012259.2	*HEY2*	1575-1675	GAGAGAGAGTGAGAGAGTCGTGTTTCGTAAGTGCCTGAGCTTAGGAAGTTTTCTTCTGGATATATAACATTGCACAAGGGAAGACGAGTGTGGAGGATAG
NM_005010.4	*NRCAM*	875-975	CATTGTTGTCCGCCCATCCAGATCACCATTGTGGACCAAAGAAAAACTTGAACCAATCACACTTCAAAGTGGTCAGTCTTTAGTACTTCCCTGCAGACCC
NM_001845.4	*COL4A1*	780-880	TGGGCTTAAGTTTTCAAGGACCAAAAGGTGACAAGGGTGACCAAGGGGTCAGTGGGCCTCCAGGAGTACCAGGACAAGCTCAAGTTCAAGAAAAAGGAGA
NM_006379.2	*SEMA3C*	945-1045	TGTCTGTGGGAGTGGCGCTTTCAGTCCTGTCTGTACTTACTTGAACAGAGGGAGGAGATCAGAGGACCAAGTTTTCATGATTGACTCCAAGTGTGAATCT
NM_004460.2	*FAP*	1490-1590	GCATTGGAAGCTATCCTCCAAGCAAGAAGTGTGTTACTTGCCATCTAAGGAAAGAAAGGTGCCAATATTACACAGCAAGTTTCAGCGACTACGCCAAGTA
NM_001098798.1	*TOX2*	784-884	GAAGCCTGTGTCGGCCTACGCACTCTTCTTCAGAGACACTCAGGCCGCCATCAAGGGTCAGAACCCCAGTGCCACTTTCGGTGACGTGTCCAAAATCGTG
NM_002253.2	*KDR*	1420-1520	CAATCACACAATTAAAGCGGGGCATGTACTGACGATTATGGAAGTGAGTGAAAGAGACACAGGAAATTACACTGTCATCCTTACCAATCCCATTTCAAAG
NM_175569.2	*XG*	27-127	AGCTGTGGAGTTTGGGATCTGAGCTTGGAGCCCATTTGTTTCTGGCAGTTCCGCTCATATTTTCCACTTGAAGACATCGCCTCCCTTCCTTCCAAGCTGG
NM_005245.3	*FAT1*	780-880	ACCCAACCAGTGGTGTGATAGTGTTAACTGGTAGACTTGATTACCTAGAGACCAAGCTCTATGAGATGGAAATCCTCGCTGCGGACCGTGGCATGAAGTT
NM_001014986.1	*FOLH1*	1530-1630	GAAGAATTTGGTCTTCTTGGTTCTACTGAGTGGGCAGAGGAGAATTCAAGACTCCTTCAAGAGCGTGGCGTGGCTTATATTAATGCTGACTCATCTATAG
NM_198516.1	*GALNTL4*	995-1095	ACAGCAGTAACGAGGAACTGAAGGAGAAGCTGACCGAATATGTGGACAAGGTGAACAGCCAGAAGCCAGGCTTCATCAAAGTCGTGCGTCACAGCAAGCA
NM_001036.3	*RYR3*	3655-3755	TCTCAGATCGGCCGCATGAATCTCGGGACAGATGCCAGTACCTTCAAGTTTTATACCATGTGCGGTCTCCAAGAGGGCTTTGAGCCTTTTGCTGTCAACA
NM_001025108.1	*AFF3*	1174-1274	AAGCACCTGGCAAAGTGGAACCAACCAAATTTCCATTTCCAAATAAGGACTCTCAGCTTGTATCCTCTGGACACAATAATCCAAAGAAAGGTGATGCAGA
NM_004557.3	*NOTCH4*	6420-6520	ACCCCTAAGTTGGAACCAAGAATTGCAGGCATATGGGATGTAAGATGTTCTTTCCTATATATGGTTTCCAAAGGGTGCCCCTATGATCCATTGTCCCCAC
NM_004098.3	*EMX2*	2505-2605	ACATTCCCTTTCCTAACATCCTGAGGCTTAAAACCCTGATGCAAACTTCTCCTTTCAGTGGTTGGAGAAATTGGCCGAGTTCAACCATTCACTGCAATGC
NM_001167738.1	*NAV1*	294-394	CTCAGTTCAGAAGAATTCAATGCCAGCTCCTCACTCAACTCCCTCCCAAGTACTCCCACTGCTTCTCGCAGGAACTCAACAATAGTGCTACGCACAGACT
NM_019087.2	*ARL15*	75-175	GGATGCTTTAGGCTGCCGGATGTCTGATCTCCGAATAACTGAGGCGTTTCTGTACATGGATTATCTGTGTTTTAGAGCACTTTGCTGCAAGGGACCACCA
NM_001039580.1	*MAP9*	547-647	ATAAATCAAACGGTAACATAACCAAAGATGAGCCAGTGTGTGCCATCAAAAATGAAGAGGAAATGGCACCTGATGGGTGTGAAGACATTGTTGTAAAATC
NM_022343.2	*GLIPR2*	216-316	CCAGTGTGGGGAGAACCTTGCATGGGCATCCTATGATCAGACAGGAAAGGAGGTGGCTGATAGATGGTACAGTGAAATCAAGAACTATAACTTCCAGCAG
NM_001193329.1	*APO*	1375-1475	AGACCCTTCTGCGGCTGATCCCTCCTTGCCTCTCAGCAGCACATTCTGTTCTGGGAGCACACCCGTTCTCTCGGCTGGATGTTCTCATCGTCCCTGCCAA
NM_015116.2	*LRCH1*	715-815	CTGCCTGTGTGGTCTGCCTCTCAAAGTCTTAATCGCAAGTAACAACAAACTTGGATCATTACCAGAAGAGATAGGTCAGCTCAAACAGTTAATGGAGCTG
NM_002448.3	*MSX1*	1225-1325	GGCACCGCCAGCCGCCTTCCCTTTAACCCTCACACTGCTCCAGTTTCACCTCTTTGCTCCCTGAGTTCACTCTCCGAAGTCTGATCCCTGCCAAAAAGTG
NM_006516.2	*SLC2A1*	2500-2600	AGGCTCCATTAGGATTTGCCCCTTCCCATCTCTTCCTACCCAACCACTCAAATTAATCTTTCTTTACCTGAGACCAGTTGGGAGCACTGGAGTGCAGGGA
NM_004490.2	*GRB14*	980-1080	ACAGCTGGACCCTTTTTGAGCACCTGCCTCACATAGGTGTAGAAAGAACAATAGAAGACCACGAACTGGTGATTGAAGTGCTATCCAACTGGGGGATAGA
NM_017855.3	*ODAM*	445-545	TCAATACTATCCAGTTTACATGGTCCTACCCTGGGAACAACCTCAGCAAACAGTTCCAAGGTCACCTCAACAAACAAGACAGCAACAGTATGAGGAGCAG
NM_031455.3	*CCDC3*	470-570	ACCGTGGTCCAGGACTACTCCTATTTCTTCTTCCTCAGGATGGATGAAAATTATAACCTCTTGCCTCACGGAGTCAATTTCCAAGATGCCATCTTCCCAG
NM_001081.3	*CUBN*	8930-9030	TGACTTTTGTGTCCTTCCACTTAGAAGCTCGTTCCGCTGTGACGGGAAGCTGTGTCAACGATGGCGTGCACATTATCAGAGGTTACAGCGTCATGTCCAC
NM_001197221.1	*PDE4D*	2120-2220	ATTGTTTCCAAAGTGCATGTCACATGCCACAACCACGGTCACACCTCACTGTCATCTGCCAGGACGTTTGTTGAACAAAACTGACCTTGACTACTCAGTC
NM_001134406.1	*RUNDC3B*	929-1029	GCTTCTAGGACTCAATGCTATTGATTTCAGTTTCTGCCTAAAGGGAGAGGGGCTGGATGGCAGTTTTCCTGCTGTAATAGACTATACACCATATTTGAAG
NM_006639.2	*CYSLTR1*	90-190	AAAGAAGCTTGCCTATAGAGCAGGCACTCTGTGAATGGACTGTGCTTTTACGACCCTACAGGGTATCAAGATACTGTGCAGCTCGCCAACAAGGATTAAT
NM_021804.2	*ACE2*	1825-1925	GGCCCTCTGCACAAATGTGACATCTCAAACTCTACAGAAGCTGGACAGAAACTGTTCAATATGCTGAGGCTTGGAAAATCAGAACCCTGGACCCTAGCAT
NM_001080392.1	*LCHN*	4440-4540	CCAGGTGGGAGTGGAATTCAGGTTTGGGGCTCGTTGGTATCCATGCAAAATATGACAAAGGCCTGTTCAAGAGGGCATTTTCAATTCTGTAGGCTCAGCA
NM_014583.2	*LMCD1*	1470-1570	TTGTTTCCCAGCTACAACCAACTAAAGACACAAATGGCGTTCTGCAAGGGGACTCTGGGAGGAGTTTTCCAGAATGCAATTCCGAGTGAGCAAATCGCAT
NM_007031.1	*HSF2BP*	800-900	ACTGGTCAAACAATGGAGAGTTTTGTGAAGTCGTTAGACGGTGATGTCCAGGAGCTGGATTCGGATGAAAGTCAGTTTGTTTTCGCTCTGGCTGGAATTG
NM_001135032.1	*FAM176A*	510-610	CGTGGAGATGGCTTTGCTCAGCAACATCCTAGCGGCCTATTCCTTTGTCTCAGAAAATCCTGAGCGAGCAGCTCTGTACTTTGTTTCTGGCGTGTGCATC
Venous genes from the arteriovenous fresh profile			
NM_000901.3	*NR3C2*	430-530	CCTACAGAGAGGACCGATGAGAATAACTACATGGAGATTGTCAACGTAAGCTGTGTTTCCGGTGCTATTCCAAACAACAGTACTCAAGGAAGCAGCAAAG
NM_016240.2	*SCARA3*	280-380	AGAACCTATCTTTGCACACATCGGTGCGGATTCTTTACCTCTTCCTGGCCCTGCTCCTGGTGGCCGTGGCTGTGTTGGCCTCTCTGGTTTTCAGAAAAGT
NM_173462.3	*PAPLN*	424-524	CCTACTACAGCGCCCCAAACAAGTGTGAACTGAACTGCATTCCCAAGGGGGAGAACTTCTACTACAAGCACAGGGAGGCTGTGGTTGATGGGACGCCCTG
NM_006750.3	*SNTB2*	2645-2745	GTTTGAACAAGATAAATCCTAAAGAGCCAGCCTGCTTGAGGAGTAGACTTGGTGGGTGAAGCCAGCAATTCCGCACAAACGTCATGTTGAATTGTTTTGG
NM_001042521.1	*MGC13057*	550-650	ATCTTGGAATATGCACACCGCCTGTCTCAGGATATCTTGTGTGATGCCTTGCAGCAATGGGCATGCAATAACATCAAGTACCATGACATTCCATACATTG
NM_013281.2	*FLRT3*	1145-1245	GACAATATAACACAACTGATTCTTCGCAACAATCCCTGGTATTGCGGGTGCAAGATGAAATGGGTACGTGACTGGTTACAATCACTACCTGTGAAGGTCA
NM_006829.2	*APM2*	8-108	CCCTGGGGCGCCTTAAAAACCGGAGCTGGCGCTTGGCATCGCCACTCTGGGCAGGATCCAACGTCGCTCCAGCTGCTCTTGACGACTCCACAGATACCCC
NM_138809.3	*CMBL*	2425-2525	TTGTCTCCTAAGCAGTGGATTTCCAAGCACTGGCATTTTTGGGTCCCAGGGCCTGAGGCTACCTCAGAAATATCTCCTGTGTTTCTGGTGGTGGCGTAAA
NM_020190.2	*OLFML3*	1525-1625	GCCCGAAGAGTCAAAACCCTCAATGTTCCCTCCTGCTCTCCTGCCCCATGTCAACAAATTTCAGGCTAAGGATGCCCCAGACCCAGGGCTCTAACCTTGT
NM_145764.1	*MGST1*	330-430	TCTTGGAATTGGCCTCCTGTATTCCTTGAGTGGTCCCGACCCCTCTACAGCCATCCTGCACTTCAGACTATTTGTCGGAGCACGGATCTACCACACCATT
NM_173496.3	*MPP7*	573-673	TACACTACTATGAGAAGCAGAGTCCGGTGCCCATTCTCCATGGTGCGGCGGCCTTGGCCGATGATCTGGCCGAAGAGCTTCAGAACAAGCCATTAAACAG
NM_001204213.1	*NOS1*	1201-1301	GGCGCTGGTGGAGATCAATATCGCGGTTCTCTATAGCTTCCAGAGTGACAAAGTGACCATTGTTGACCATCACTCCGCCACCGAGTCCTTCATTAAGCAC
Established arterial EC genes			
NM_018419.2	*SOX18*	1475-1575	CCTGCGAGGGTGCCTGGAGTTCCCACGTGTCCCGGGGCTTTTCCAGGAAGCCCGAGCCCAGGACCTGTTGGCAGAGTTGCCAGGGTTACATTTTTGAAGC
NM_004093.2	*EFNB2*	620-720	TTGTAAAACCAAATCCAGGTTCTAGCACAGACGGCAACAGCGCCGGACATTCGGGGAACAACATCCTCGGTTCCGAAGTGGCCTTATTTGCAGGGATTGC
NM_017617.3	*NOTCH1*	735-835	CTGCCAGGCTTCACCGGCCAGAACTGTGAGGAAAATATCGACGATTGTCCAGGAAACAACTGCAAGAACGGGGGTGCCTGTGTGGACGGCGTGAACACCT

Probe set ID	Gene	Targeted region	Target sequence
Established arterial EC genes, continued			
NM_000214.2	*JAG1*	915-1015	TTGCTTGTGGAGGCGTGGGATTCCAGTAATGACACCGTTCAACCTGACAGTATTATTGAAAAGGCTTCTCACTCGGGCATGATCAACCCCAGCCGGCAGT
NM_004429.4	*EFNB1*	137-237	CGAGGCTTCGGGGGCGCAAACTAATGGGACTGGCTCGCTCGGCAGCATCTCCCCGCTCTTCTAAGTACACTGAGCAGGGCCCGCGCTGAAGTAGAAGCTG
NM_019074.2	*DLL4*	893-993	AATGACCACTTCGGCCACTATGTGTGCCAGCCAGATGGCAACTTGTCCTGCCTGCCCGGTTGGACTGGGGAATATTGCCAACAGCCTATCTGTCTTTCGG
NM_145159.1	*JAG2*	4225-4325	ATTTTTGTAAAGTTTCCGTGCGTGGCACTCGCTGTATGAAAGGAGAGAGCAAAGGGTGTCTGCGTCGTCACCAAATCGTAGCGTTTGTTACCAGAGGTTG
NM_001001392.1	*CD44*	429-529	ACACCATGGACAAGTTTTGGTGGCACGCAGCCTGGGGACTCTGCCTCGTGCCGCTGAGCCTGGCGCAGATCGATTTGAATATAACCTGCCGCTTTGCAGG
NM_031439.2	*SOX7*	2635-2735	CTGTGAGAATTTGTCTTCCTCACCAGCCAGGTCCTCAGGCAAAGTCCTCAGCCAGTGCTTTAGAGCAACTTCCCGCAAATCAGAAACTCACTGTGATTCC
NM_012258.3	*HEY1*	398-498	AACAGTTTGTCTGAGCTGAGAAGGCTGGTACCCAGTGCTTTTGAGAAGCAGGGATCTGCTAAGCTAGAAAAAGCCGAGATCCTGCAGATGACCGTGGATC
Established venous EC genes			
NM_201264.1	*NRP2*	805-905	TCTCACCTGGGTTTTCTTAGCCCTCTACTTTTCAAGACACCAAGTGAGAGGCCAACCAGACCCACCGTGCGGAGGTCGTTTGAATTCCAAAGATGCTGGC
NM_021005.2	*NR2F2*	1530-1630	CCATAGTCCTGTTCACCTCAGATGCCTGTGGTCTCTCTGATGTAGCCCATGTGGAAAGCTTGCAGGAAAAGTCTCAGTGTGCTTTGGAAGAATACGTTAG
NM_004444.4	*EPHB4*	1680-1780	GTCCTGACTTCACCTATACCTTTGAGGTCACTGCATTGAACGGGGTATCCTCCTTAGCCACGGGGCCCGTCCCATTTGAGCCTGTCAATGTCACCACTGA
General EC genes			
NM_000442.3	*PECAM1*	1365-1465	ATCTGCACTGCAGGTATTGACAAAGTGGTCAAGAAAAGCAACACAGTCCAGATAGTCGTATGTGAAATGCTCTCCCAGCCCAGGATTTCTTATGATGCCC
NM_000552.3	*VWF*	8115-8215	CACCTGCAACCCCTGCCCCCTGGGTTACAAGGAAGAAAATAACACAGGTGAATGTTGTGGGAGATGTTTGCCTACGGCTTGCACCATTCAGCTAAGAGGA
NM_000459.2	*TEK*	615-715	CGAGTTCGAGGAGAGGCAATCAGGATACGAACCATGAAGATGCGTCAACAAGCTTCCTTCCTACCAGCTACTTTAACTATGACTGTGGACAAGGGAGATA
NM_001795.3	*CDH5*	3405-3505	TCTCCCCTTCTCTGCCTCACCTGGTCGCCAATCCATGCTCTCTTTCTTTTCTCTGTCTACTCCTTATCCCTTGGTTTAGAGGAACCCAAGATGTGGCCTT
NM_001114753.1	*ENG*	1480-1580	GTCCTTGATCCAGACAAAGTGTGCCGACGACGCCATGACCCTGGTACTAAAGAAAGAGCTTGTTGCGCATTTGAAGTGCACCATCACGGGCCTGACCTTC
Housekeeping genes			
NM_000194.1	*HPRT1*	240-340	TGTGATGAAGGAGATGGGAGGCCATCACATTGTAGCCCTCTGTGTGCTCAAGGGGGGCTATAAATTCTTTGCTGACCTGCTGGATTACATCAAAGCACTG
NM_001101.2	*ACTB*	1010-1110	TGCAGAAGGAGATCACTGCCCTGGCACCCAGCACAATGAAGATCAAGATCATTGCTCCTCCTGAGCGCAAGTACTCCGTGTGGATCGGCGGCTCCATCCT
NM_022551.2	*RPS18*	256-356	TGCAGAATCCACGCCAGTACAAGATCCCAGACTGGTTCTTGAACAGACAGAAGGATGTAAAGGATGGAAAATACAGCCAGGTCCTAGCCAATGGTCTGGA
NM_002046.3	*GAPDH*	35-135	TCCTCCTGTTCGACAGTCAGCCGCATCTTCTTTTGCGTCGCCAGCCGAGCCACATCGCTCAGACACCATGGGGAAGGTGAAGGTCGGAGTCAACGGATTT

SUPPLEMENTARY INFORMATION – ITEM 4: Table S3

Table S3. ***Probe set intensities in HUAECs/HUVECs of genes contained within the arteriovenous fresh profile.***

		A	B	C	D	E	F	G	H
Probe set ID	Gene	HUAEC-F	HUVEC-F	HUAEC-C	HUVEC-C	B-A	C-A	D-B	D-C
				Arterial probe sets					
213764_s_at	*MFAP5*	12,0	3,9	6,0	4,1	-8,1	-6,0	0,2	-1,9
229459_at	*FAM19A5*	11,6	3,7	4,1	3,6	-7,9	-7,5	-0,1	-0,5
213765_at	*MFAP5*	11,4	3,9	5,7	4,2	-7,6	-5,7	0,4	-1,5
206084_at	*PTPRR*	11,4	4,0	6,7	4,6	-7,4	-4,6	0,6	-2,2
224646_x_at	*H19*	13,5	6,3	6,6	6,8	-7,1	-6,9	0,4	0,2
209758_s_at	*MFAP5*	11,3	4,6	6,9	5,6	-6,7	-4,4	0,9	-1,3
237094_at	*FAM19A5*	10,1	3,5	3,8	3,7	-6,6	-6,4	0,1	-0,1
224997_x_at	*H19*	11,6	5,1	5,3	5,1	-6,6	-6,4	0,0	-0,2
227265_at	*FGL2*	10,4	4,1	4,0	3,8	-6,3	-6,5	-0,4	-0,2
219789_at	*NPR3*	10,0	3,9	4,8	4,1	-6,1	-5,3	0,1	-0,7
229831_at	*CNTN3*	9,6	3,5	7,3	5,0	-6,0	-2,3	1,4	-2,3
210675_s_at	*PTPRR*	10,2	4,2	5,4	4,2	-6,0	-4,7	0,1	-1,2
228109_at	*RASGRF2*	11,0	5,0	7,9	7,4	-6,0	-3,1	2,4	-0,5
219689_at	*SEMA3G*	10,5	4,6	5,1	6,0	-6,0	-5,4	1,4	0,8
232424_at	*PRDM16*	9,3	3,4	4,8	3,6	-5,9	-4,6	0,2	-1,2
204834_at	*FGL2*	10,0	4,1	4,3	4,0	-5,9	-5,7	0,0	-0,2
205413_at	*MPPED2*	9,1	3,2	3,3	3,4	-5,8	-5,7	0,2	0,1
230943_at	*NA*	10,3	4,5	7,8	8,9	-5,8	-2,6	4,4	1,1
217757_at	*A2M*	10,7	4,9	4,6	4,7	-5,8	-6,1	-0,2	0,1
237727_at	*FREM1*	10,1	4,4	4,0	4,3	-5,7	-6,1	-0,2	0,3
211966_at	*COL4A2*	10,8	5,3	10,0	8,6	-5,5	-0,9	3,3	-1,4
219743_at	*HEY2*	9,8	4,3	6,2	5,8	-5,5	-3,6	1,5	-0,5
204105_s_at	*NRCAM*	9,3	3,9	10,3	8,6	-5,5	1,0	4,7	-1,7
211981_at	*COL4A1*	12,2	6,8	10,8	9,9	-5,4	-1,4	3,1	-0,9
203789_s_at	*SEMA3C*	9,6	4,3	10,1	7,3	-5,3	0,5	3,1	-2,8
209955_s_at	*FAP*	11,2	5,9	9,9	6,8	-5,3	-1,3	0,9	-3,1
222921_s_at	*HEY2*	10,9	5,6	8,5	8,0	-5,3	-2,3	2,5	-0,5
228737_at	*TOX2*	9,8	4,5	6,2	7,6	-5,2	-3,6	3,1	1,5
219054_at	*NPR3*	12,0	6,8	6,9	6,3	-5,2	-5,1	-0,5	-0,6
203934_at	*KDR*	10,6	5,5	7,4	8,7	-5,1	-3,2	3,2	1,3
1554062_at	*XG*	8,8	3,7	7,2	4,5	-5,1	-1,5	0,8	-2,7
229523_at	*TMEM200C*	10,3	5,4	7,4	7,1	-5,0	-2,9	1,7	-0,3
218723_s_at	*RGC_32*	9,1	4,2	6,0	7,9	-4,9	-3,1	3,6	1,9
216840_s_at	*LAMA2*	8,7	4,0	4,5	4,5	-4,8	-4,2	0,5	0,0
234994_at	*TMEM200A*	8,6	3,9	9,1	8,8	-4,7	0,5	4,9	-0,4
236031_x_at	*FREM1*	8,4	3,7	3,6	3,7	-4,7	-4,8	0,0	0,1
229309_at	*ADRB1*	8,3	3,6	4,3	4,1	-4,7	-4,0	0,5	-0,2
209278_s_at	*TFPI2*	12,0	7,4	8,8	11,3	-4,6	-3,2	3,9	2,5
219993_at	*SOX17*	8,8	4,3	6,3	7,8	-4,6	-2,5	3,6	1,5
202498_s_at	*SLC2A3*	10,6	6,2	5,4	5,8	-4,4	-5,1	-0,5	0,3
209277_at	*TFPI2*	10,8	6,4	7,7	10,0	-4,3	-3,0	3,5	2,2
232914_s_at	*SYTL2*	9,4	5,1	5,5	4,7	-4,3	-3,9	-0,5	-0,9
228233_at	*FREM1*	8,8	4,5	3,9	4,1	-4,2	-4,8	-0,4	0,2
228167_at	*KLHL6*	8,1	3,9	5,2	5,3	-4,2	-2,9	1,5	0,1
1553808_a_at	*NKX2-3*	8,1	4,0	4,4	4,1	-4,1	-3,7	0,1	-0,3
232080_at	*HECW2*	8,6	4,4	9,7	9,0	-4,1	1,1	4,5	-0,7
226809_at	*CYP4F30P*	8,5	4,5	4,4	4,4	-4,0	-4,1	-0,1	0,0
201579_at	*FAT1*	9,7	5,8	11,8	9,0	-3,9	2,0	3,1	-2,8
215363_x_at	*FOLH1*	9,9	6,0	4,6	4,6	-3,9	-5,4	-1,4	0,0
213519_s_at	*LAMA2*	8,8	4,9	5,2	5,3	-3,9	-3,6	0,3	0,1
1554079_at	*GALNTL4*	9,7	5,8	5,4	6,1	-3,9	-4,3	0,2	0,7
206306_at	*RYR3*	7,2	3,3	3,4	3,3	-3,9	-3,8	0,0	0,0
227198_at	*AFF3*	7,3	3,6	3,8	3,8	-3,7	-3,5	0,2	-0,1
222088_s_at	*SLC2A3*	11,6	8,0	6,2	6,8	-3,7	-5,5	-1,2	0,6
205247_at	*NOTCH4*	9,6	5,9	5,3	6,5	-3,7	-4,3	0,5	1,1
205116_at	*LAMA2*	8,6	4,9	5,3	5,2	-3,7	-3,3	0,3	-0,1
221950_at	*EMX2*	7,0	3,4	4,8	4,0	-3,6	-2,2	0,6	-0,8
224772_at	*NAV1*	10,4	6,8	10,4	9,6	-3,5	0,0	2,8	-0,8
219842_at	*ARL15*	9,2	5,6	6,6	5,6	-3,5	-2,5	0,0	-1,0
225496_s_at	*SYTL2*	8,8	5,4	5,3	5,3	-3,4	-3,5	-0,1	0,0
211303_x_at	*FOLH1*	7,6	4,3	3,8	3,7	-3,3	-3,8	-0,5	0,0
228423_at	*MAP9*	6,9	3,6	8,1	5,7	-3,3	1,2	2,1	-2,4
1556128_a_at	*RASGRF2*	9,3	6,1	7,2	7,9	-3,2	-2,0	1,8	0,6
225602_at	*GLIPR2*	8,1	4,9	9,7	9,4	-3,2	1,6	4,5	-0,2
237716_at	*APO*	8,0	4,8	4,4	4,2	-3,2	-3,6	-0,6	-0,2
226795_at	*LRCH1*	8,5	5,3	8,1	7,7	-3,2	-0,3	2,4	-0,4
205932_s_at	*MSX1*	8,4	5,3	7,3	7,9	-3,1	-1,1	2,6	0,6
201250_s_at	*SLC2A1*	11,3	8,2	9,5	8,6	-3,1	-1,9	0,4	-0,8
229655_at	*FAM19A5*	8,0	4,9	5,0	5,2	-3,0	-2,9	0,3	0,2
206204_at	*GRB14*	10,5	7,5	6,6	6,8	-3,0	-4,0	-0,7	0,2
220133_at	*ODAM*	6,8	3,8	5,0	4,2	-3,0	-1,9	0,3	-0,8
223316_at	*CCDC3*	13,8	10,9	8,0	7,9	-2,9	-5,7	-3,0	-0,1
201249_at	*SLC2A1*	7,1	4,3	5,5	4,7	-2,8	-1,7	0,4	-0,8
238169_at	*NA*	6,4	3,7	4,3	5,4	-2,7	-2,1	1,6	1,0
202497_x_at	*SLC2A3*	10,3	7,7	6,2	7,0	-2,7	-4,1	-0,6	0,8
206775_at	*CUBN*	8,6	5,9	6,3	6,0	-2,7	-2,3	0,1	-0,2
228151_at	*NA*	12,7	10,0	9,9	8,8	-2,6	-2,8	-1,3	-1,1
228962_at	*PDE4D*	7,6	5,0	6,1	5,6	-2,6	-1,5	0,6	-0,4
241703_at	*RUNDC3B*	6,2	3,6	6,7	4,5	-2,6	0,5	0,9	-2,2
225604_s_at	*GLIPR2*	6,3	3,9	8,3	8,3	-2,4	2,0	4,4	0,0
213484_at	*NA*	7,2	4,9	4,8	5,1	-2,3	-2,4	0,2	0,3
231747_at	*CYSLTR1*	6,5	4,2	3,9	4,1	-2,3	-2,6	-0,1	0,2
227828_s_at	*FAM176A*	7,0	4,7	8,8	8,8	-2,3	1,7	4,1	0,0
209227_at	*NA*	6,0	3,8	6,4	5,6	-2,2	0,4	1,8	-0,8
222257_s_at	*ACE2*	6,8	4,6	4,5	4,6	-2,2	-2,3	0,0	0,1
228601_at	*LOC401022*	8,8	6,7	7,4	7,8	-2,2	-1,4	1,1	0,4
227502_at	*LCHN*	9,0	6,9	6,3	6,3	-2,1	-2,7	-0,6	0,0
218574_s_at	*LMCD1*	11,7	10,1	8,3	7,7	-1,6	-3,4	-2,4	-0,7
207020_at	*HSF2BP*	6,5	4,9	6,5	6,0	-1,5	0,0	1,1	-0,4
				Venous probe sets					
205259_at	*NR3C2*	5,6	7,3	5,6	5,3	1,7	0,0	-2,0	-0,3
219416_at	*SCARA3*	7,6	9,4	6,9	8,1	1,8	-0,7	-1,3	1,2
219250_s_at	*FLRT3*	4,8	6,7	7,4	5,9	1,9	2,6	-0,8	-1,5
226435_at	*PAPLN*	4,2	6,2	4,5	4,4	1,9	0,3	-1,8	-0,1
229004_at	*NA*	5,3	7,3	6,7	5,5	2,0	1,4	-1,9	-1,3
227312_at	*SNTB2*	6,2	8,4	6,1	5,5	2,2	-0,2	-3,0	-0,6
228195_at	*MGC13057*	5,0	7,2	3,8	3,9	2,2	-1,2	-3,3	0,1
203571_s_at	*APM2*	5,8	8,1	5,8	6,2	2,3	-0,1	-1,9	0,4
227522_at	*CMBL*	6,6	9,0	8,8	9,6	2,4	2,2	0,6	0,8
218162_at	*OLFML3*	5,1	7,6	5,2	7,0	2,4	0,1	-0,6	1,8
224918_x_at	*MGST1*	4,0	6,6	8,5	9,6	2,6	4,5	2,9	1,0
238778_at	*MPP7*	5,0	8,4	5,1	3,3	3,3	0,0	-5,1	-1,8
239132_at	*NOS1*	6,6	10,0	5,0	4,9	3,4	-1,6	-5,0	-0,1

SUPPLEMENTARY INFORMATION – ITEM 5: Table S4

Table S4. ***Additional information regarding the genes contained in the arteriovenous fresh profile.***

Gene	Synonym(s)	Full name	Subcellular localization/function	Tg mice reported?	Phenotype	Other
Arterial genes						
A2M	CPAMD5, A2MD	α2-macroglobulin	Cell surface (proteinase inhibitor)	Yes (targeted KO) (5)	-viable and fertile -vascular phenotype not reported	anti-angiogenic by binding FGF-2 (6) and VEGF-A (7)
ACE2	ACEH, metalloprotease MPROT15	Angiotensin I converting enzyme (peptidyl-dipeptidase-A) 2	Cell surface (receptor)	Yes (targeted KO) (8)	-viable and fertile -cardiac contractility defect -upregulation of hypoxia-induced genes in heart -increased angiotensin II levels	predominantly expressed in vascular ECs of kidney, heart and testis
				Yes (targeted KO) (9)	-viable and fertile -normal cardiac function -altered blood pressure responses (depending on genetic background)	
ADRB1	ADRB1R, B1AR	Adrenergic, beta-1, receptor	Cell surface (receptor)	Yes (targeted KO) (10)	-prenatal death (strain dependent penetrance) -surviving adult mice lack chronotropic and inotropic responses	-
AFF3	LAF4, MLLT2-like	AF4/FMR2 family, member 3	Intracellular (nucleus; TF)	Not reported	-	-preferentially expressed in lymphoid tissue
APO	AOPEP, AP-O	Aminopeptidase O	Intracellular (nucleoli; metalloprotease)	Yes (gene trap) (11)	-viable, fertile -no sprouting defects in aortic ring assay -there could be functional redundancy among aminopeptidase family or the fusion protein resulting from the gene trap could be functional	-expressed predominantly in embryonic (dorsal aorta, intersomitic vessels) and adult blood vessels (aorta, ECs and SMCs), cultured HUVECs (11) -cleaves one of the peptides of the renin angiotensin pathway (angiotensin III) (12)
ARL15	ARFRP2	ADP-ribosylation factor-like 15	?	Not reported	-	-associated with CHD (13,14) -is a tumor suppressor gene (15)
CCDC3	Favine (fat/vessel-derived secretory protein)	Coiled-coil domain containing 3	Secreted ('adipocytokine')	Not reported	-	-highly (specifically) expressed in murine aorta and in adipose tissue (16) -higher in HUVECs than in aortic SMCs -mRNA not upregulated in atherosclerotic aorta or aorta from obese/diabetic mice
CNTN3	PANG, BIG-1	Contactin 3	Cell surface (lipid-anchored cell adhesion protein)	Not reported	-	-associated with AAA (17) -detected in aorta (normal + AAA) (18) **-its family member, F3/contactin acts as a functional ligand for Notch (19)**
COL4A1	arresten	Collagen type IV, alpha 1	Secreted ECM protein; localized to the BM	Yes (targeted; double KO with COL4A2) (20)	-lethal around E10.5-E11.5 -growth retardation -bleeding and dilated blood vessels -normal SMC coating	-mutations in humans are associated with recurrent stroke and cataract (21)

				Yes (mutant) (22)	-homozygous mice die at mid-gestation -heterozygous mice lethal around P1 -perinatal cerebral hemorrhage -disruption of BM in brain vessels	
				Yes (mutant) (23)	-intraorbital hemorrhages -homozygosity is lethal in 9/9 strains	
COL4A2	Canstatin	Collagen type IV, alpha 2	Secreted ECM protein; localized to the BM	Yes (targeted double KO with COL4A2) (20)	-lethal around E10.5-E11.5 -growth retardation -bleeding and dilated blood vessels -normal SMC coating	
				Yes (mutant) (23)	-intraorbital hemorrhages -homozygosity is lethal in 2/3 mutant strains	
CUBN	IFCR, MGA1	Cubilin (intrinsic factor-cobalamin receptor)	Intracellular (peripheral membrane protein receptor)	Yes (targeted KO) (24)	-lethal between E7.5 and E13.5 -allantois and heart are formed -enlarged yolk sac blood islands -yolk sac blood vessels fail to undergo remodeling -no somite formation -paired dorsal aortae are formed -aberrant vasculature in the allantois	-cubulin gene defect leads to megaloblastic anemia 1 (25) -cubilin ligands: vitamin B12, apoA-1, transferrin
CYP4F30P	-	Cytochrome P450, family 4, subfamily F, polypeptide 30, pseudogene	-	Not reported	-	-
CYSLTR1	CYSLT1, LTD4 receptor, HG55	Cysteinyl leukotriene receptor 1	Cell surface (receptor)	Yes (targeted KO) (26)	-viable and fertile -reduced vascular permeability during acute inflammation	-expressed in HUVECs (27)
EMX2	-	Empty spiracles homeobox 2	Intracellular (nucleus; TF)	Yes (targeted KO) (28)	-die perinatally (°severe urogenital problems) -reduced size of cerebral hemispheres -dentate gyrus is missing	-in *Xenopus*, Emx2 is expressed in branchial arches, specifically in skeletal neural crest cells -no expression in heart (29) -has been suggested to be involved in adipose tissue function (30) **-microarrays on hippocampus tissue from Emx2-/- E18.5 mice reveals genes related to angiogenesis, Notch and Wnt pathways** (31) -large blood vessels of the fissure region of the hippocampus are missing in Emx2-/- E18.5 pups (31)
				Yes (mutant) (29)	-homozygous mutants die perinatally -middle and inner ear defects in heterozygous mutants	
				Yes (targeted KO) (32)	-die perinatally because of severe urogenital alterations -dentate gyrus missing -hippocampus reduced	
				Yes (mutant) (33)	-defects in the thalamocortical system (misrouted axons)	
FAM176A	TMEM166	Family with sequence similarity 176, member A	Intracellular (ER membrane; involved in programmed cell death)	Not reported	-	-

FAM19A5	TAFA5	Family with sequence similarity 19 (chemokine (C-C motif)-like), member A5	Secreted (neuropeptide)	Not reported	-	-mainly expressed in the nervous system, including the hypothalamic paraventricular nucleus; not in heart; also in subcutaneous adipose tissue (34) -may be involved in fluid homeostasis (34)
FAP	seprase	Fibroblast activation protein, alpha	Cell surface (protease)	Yes (targeted KO, LacZ reporter) (35)	-viable and fertile -no vascular phenotype reported, no obvious phenotype in general	-first expressed in somites at E10.5 -on crossections, LacZ expression was restricted to somites (dermomyotome), myotubes and perichondral mesenchyme from cartilage primordial (35)
				Yes (targeted KO) (36)	-viable and fertile -no vascular phenotype reported, no obvious phenotype in general	
FAT1	FAT, CDHF7, CDHR8, ME5	FAT tumor suppressor homolog 1	Cell surface, protocadherin (non-classical cadherin)	Yes (targeted KO, LacZ reporter) (37)	-perinatal lethality due to renal defects -defects in forebrain development -defects in eye development	-has two potential β-catenin binding regions -has a putative PDZ domain -widely expressed in epithelial tissues
FGL2	T49, fibroleukin	Fibrinogen-like 2	Secreted (prothrombinase)	Yes (targeted KO, LacZ reporter) (38)	-lethal from E10.5 onwards -maternal and embryonic hemorrhages at implantation site at E8.5 -adult survivors have no obvious phenotype	-expressed in ECs and macrophages (38) -cleaves prothrombin to thrombin -is involved in MHV-3 infection in liver (38)
				Yes (targeted KO) (39)	-viable and fertile -no evidence for role as critical mediator of type 1 immunity-associated coagulation	
FOLH1	PSMA, GCP2	Folate hydrolase (prostate-specific membrane antigen) 1	Cell surface (membrane-bound glutamate carboxypeptidase)	Yes (targeted KO) (40)	-viable and fertile -normal neurological responses	-cleaves neurotransmitter NAAG -expressed in the neovasculature of many tumors in humans (41-43), but not in prostate tumors in mice (44)
				Yes (targeted KO) (45)	-homozygous lethal from E8.0 onwards	-most likely the truncated protein functions as a dominant negative in homozygous mice that can also block the function of additional glutamate carboxypeptidases
FREM1	TILRR, QBRICK, BNAR	FRAS1 related extracellular matrix 1	Secreted ECM protein; localized to the BM	Yes (mutant) (46)	-cryptophthalmos -blebs around eyes and sides of the head from E13.5 onwards -unilateral renal agenesis -limb syndactyly	-expressed in epidermal appendages and in mesenchyme surrounding the proximal renal tubules
				Yes (targeted KO) (47)	-Frem1 KO have less Frem2/Fras1 in BM -cryptophthalmos -blebs around eyes and sides of the head from E13.5 onwards -unilateral renal agenesis -limb syndactyly -subepidermal blistering	-while Frem2 and Fras1 are expressed at the epithelial side, Frem1 is expressed at the mesenchymal side
GALNTL4	GALNT15, GalNAc-T15, GALNT18	UDP-N-acetyl-alpha-D-galactosamine:polypetide N-acetylgalactosaminyl-transferase-like 4	Intracellular (Golgi; glycosylation catalyzer)	Not reported	-	-ubiquitously expressed in human tissues (48)

GLIPR2	GAPR-1	glioma pathogenesis-related 2	Intracellular (Golgi)	Not reported	-	-selectively expressed in epithelial cells (49) -promotes EMT -expression increased in fibrotic kidneys (49) -expressed in peripheral blood cells (50,51) -expressed in the trunk vessels of zebrafish (52)
GRB14	-	Growth factor receptor-bound protein 14	Intracellular (cytoplamsic; Golgi; adapter-type signaling protein)	Yes (targeted KO, reporter) (53)	-viable and fertile -improved glucose tolerance and lower circulating insulin levels in adult males -differential effects on insulin signaling in liver, skeletal muscle and white fat tissue	-associates with the insulin receptor -highly expressed in heart and liver, moderate expression in white adipose tissue and low expression in skeletal muscle -has been associated with hypertension in humans (54,55)
H19	ASM1	Imprinted maternally expressed transcript (non-protein coding)	-	Yes (targeted KO) (56)	-somatic overgrowth (due to a gain of function of IGF-2)	-imprinted maternally expressed gene -regulates IGF-2 imprinting and thereby controls growth -precursor for miR-675 (57)
HECW2	NEDL2, NEDD (neural-precursor-cell-expressed developmentally down-regulated)4-related E3 ubiquitin ligase NEDL2	HECT, C2 and WW domain containing E3 ubiquitin protein ligase 2	Intracellular (cytoplasm)	Not reported	-	**-in the Drosophila wing, Nedd4 ubiquitin ligases dNedd4 and Suppressor of Deltex Su(dx) are negative regulators of Notch signaling (58)** -Nedd4-like ubiquitin E3 ligases target angiomotin, a protein that controls EC migration, TJ formation, polarity and angiogenesis (59) -stabilizes p73, a p53 family member regulating cell growth and apoptosis (60) -predominantly expressed in brain, lung and heart (60)
HEY2	HERP1, HRT2	Hairy/enhancer-of-split related with YRPW	Intracellular (nucleus; TF)	Yes (targeted KO, reporter) (61)	-no defects in aortic development -post-natal cardiac hypertrophy (leading to death within 10 days after birth)	**-is a downstream Notch target**
				Yes (targeted KO, combined with Hey1 KO) (62)	-homozygous lethal after E9.5 -global lack of vascular remodeling, hemorrhage -defect in arterial EC cell fate decision	
				Yes (targeted KO, combined with Hey1 KO)	-homozygous lethal from E10.5 -defects in arteriovenous specification -cardiac septation/cushion formation defect	
HSF2BP	-	Heat shock transcription factor 2 binding protein	?	Not reported	-	-specifically expressed in testis (63)
KDR	FLK1, VEGFR2	Kinase insert domain receptor	Cell surface (a type III receptor tyrosine kinase)	Yes (targeted KO, reporter) (64)	-homozygous lethal between E8.5 and E9.5 -absence of yolk sac blood islands -aberrant vasculogenesis	-required cell autonomously for EC development and primitive and definitive hematopoiesis (65) **-Notch is activated downstream of a signaling cascade involving VEGFR2 (66)**
KLHL6	-	Kelch-like 6 (Drosophila)	Intracellular (perinuclear)	Yes (targeted KO) (67)	-viable -defect in B-cell development -normal blood vessel development	-lymphoid tissue-restricted expression in adult mice -expression in embryonic but not adult ECs

LAMA2	LAMM, merosin heavy chain	Laminin, α2	Secreted ECM protein; localized to the BM	Yes (targeted KO) (68)	-growth retardation -muscular dystrophy -die by 5 weeks	-expression mainly in striated muscle, Schwann cells and throphoblasts
LCHN	KIAA1147	?	?	Not reported	-	-expression is induced in hippocampus upon induction of forebrain ischemia (69) -expressed in cultured neurons but not astrocytes -very well conserved across species
LMCD1	dyxin	LIM and cysteine-rich domains 1	Intracellular (cytoplasm, nucleus; TF co-factor)	Yes (Cardiomyocyte-specific LMCD1 overexpressing mice) (70, 71)	More hypertrophy and fibrosis upon aortic banding by activation of calcineurin/nFAT signaling.	-co-regulator of GATA TF; its expression is induced in cardiomyocytes by mechanical load in the heart (71, 72) -highly expressed in vascular SMCs/myocardium (73) -co-regulates SMC differentiation of mesoangioblasts with GATA6 (74)
LOC401022	-	HOXD cluster antisense RNA1 (non-protein coding)	?	Not reported	-	-
LRCH1	CHDC1, Neuronal protein 81	Leucine-rich repeats and calponin homology (CH) domain containing 1	?	Not reported	-	-
MAP9	ASAP (aster-associated protein)	Microtubule-associated protein 9	Intracellular (cytoplasm, cytoskeleton)	Not reported	-	-has a crucial role in mitotic spindle assembly and cytokinesis (75)
MFAP5	MAGP2, MP25	Microfibrillar associated protein 5	Secreted ECM protein; localized to the BM	Not reported	-	-has been associated with AAA (76) **-can activate Notch 1 signaling by inducing receptor cleavage in SMCs (only in cis, not in trans) (77)** **-(exogenous) MAGP2 blocks Notch signaling in ECs (and hence inhibits sprouting) (78)** -is expressed in the IEL and adventitia but much less so in the medial layer of large blood vessels in fetal lung and thoracic aorta and arterioles in heart, spleen, kidney and skeletal muscle (79)
MPPED2	FAM1B, 239FB	Metallophosphoesterase domain-containing protein 2	?	Not reported	-	-is associated with chronic kidney disease (80) -has a role in brain development and is associated with WAGR mental retardation syndrome; is expressed in certain regions of the fetal (not adult) brain (81) -functions as a metallo-phophodiesterase without activity towards cAMP or cGMP (82)
MSX1	HOX7, OFC5	Msh homeobox 1	Intracellular (nucleus; TF)	Yes (targeted KO) (83)	-die in the immediate postnatal period -cleft secondary palate -craniofacial bone abnormalities -failure of tooth development -no heart phenotype	-expressed at diverse sites of epithelial-mesenchymal interaction -expressed in cephalic neural crest cells -expressed in SMCs and a subset of ECs in embryonic aorta (84) -*Msx* genes define a population of mural cell precursors (85)
				Yes (SM22-Cre-Msx1fl;MSX2fl)	-abnormal branching of the cephalic vessels at E11.5	

				(85)	-increased caliber of carotid and vertebral arteries related to reduced SMC coverage -secondary effects on EC maturation	-*Alk2* deficiency in neural crest cells results in absence of Msx1 expression and leads to persistent truncus arteriosus and abnormal SMC maturation of the aortic arch (86) - expressed in retinal arteries, from P10 on restricted to branchpoints (87) - expressed in choroïdal vasculature: endothelium till P14, later on switched to mural cells (87) **-Msx1 strongly upregulated Notch 3 and Hey1 in a neuroblastoma cell line (88)**
				Yes (Tie2-Cre-MSX1fl;MSX2fl) (85, 87)	- no abnormal branching or caliber of the cephalic vessels (85) - no phenotype in the mouse retinal vasculature (87)	
NAV1	POMFIL3, steerin-1	Neuron navigator 1	Intracellular (cytoplasm, cytoskeleton)	Not reported	-	-expression largely restricted to developing nervous system, heart and somites (89) -has a role in netrin-1-induced migration (89)
NKX2-3	NKX2C, CSX3	NK2 homeobox 3	Intracellular (nucleus; TF)	Yes (targeted KO, reporter) (90)	-30% dies within 2 weeks after birth -defects in lymphoid organ development -intestinal malabsorption	-is expressed in ECs of the viscera (e.g. capillaries in stomach, jejunum, arterioles in pancreas) and regulates expression of MadCAM-1 (90) -not expressed in developing dorsal aorta -is associated with inflammatory bowel disease and regulates endothelin-1 and VEGF-PI3K/AKT-eNOS in HIMECs (91) **-inhibits Hey1/2 in HIMECs (91)** -there may be considerable redundancy among NKX family members (92), e.g. Nkx2-3 and Nkx2-5 are required for heart formation in a functionally redundant matter in *Xenopus* (93)
				Yes (targeted KO) (94)	-postnatal lethality -abnormal development of small intestine and spleen	
				Yes (targeted KO) (95, 96)	-its absence converts spleen ECs into peripheral lymph node ECs; its absence induces the formation of LYVE-1+ Prox1- cysts in the spleen (which otherwise lacks a lymphatic vasculature)	
				Yes (targeted KO) (92)	-defects in maturation and cellular organization of sublingual glands	
NOTCH4	INT3	-	Cell surface (receptor)	Yes (targeted KO) (97)	-viable and fertile -in combination with Notch1: severe defects in angiogenic vascular remodeling (especially in arteries)	**-Notch receptor**
NPR3	ANPRC, GUCY2B	Natriuretic peptide receptor C/guanylate cyclase C (atrionatriuretic peptide receptor C)	Cell surface (receptor)	Yes (targeted KO) (98)	-reduced ability to concentrate urine -mild diuresis -blood volume depleted -hypotension -skeletal deformities due to increased bone turnover	-has been associated with hypertension in humans (54, 99) -one of the ligands, CNP, is mainly expressed in brain and in ECs and it can act in a paracrine/autocrine way -has no GC activity and is rather a clearance receptor for natriuretic peptides
				Yes (mutants) (100)	-skeletal deformities due to increased bone turnover	
NRCAM	Bravo	Neuronal cell adhesion molecule	Cell surface (cell adhesion molecule)	Yes (targeted KO) (101)	-viable and fertile -size reduction in certain cerebellar lobes	-contactin is a ligand -expressed on granule and Purkinje cells in developing cerebellum
				Yes (targeted KO) (102)	-develop cataracts	
ODAM	APIN	Odontogenic, ameloblast associated	Secreted (basal lamina protein (103))	Not reported	-	-

PDE4D	PDE43, STRK1	Phosphodiesterase 4D, cAMP-specific	Intracellular (cytoplamsm, cytoskeleton)	Yes (targeted KO) (104)	-delayed growth -reduced viability (die between P0 and P14) -reduced female fertility	-
PRDM16	MEL1, PFM13	PR domain containing 16	Intracellular (nucleus; TF)	Yes (gene trap, reporter, mixed background) (105)	-lethal (no live homozygotes recovered after birth) -critical for the establishment and maintenance of the HSC pool during development -enhanced apoptosis of HSC	-associated with MDS, T-cell leukemia, cleft palate -expressed in neural crest cells (*Xenopus*) (106) -expressed in the dorsal aspect of the somites and branchial arches (107) -expressed by stem cells throughout the nervous and hematopoietic systems (108) -is expressed in the left ventricle of the heart and in the circulatory system; later in development expression spreads to the whole ventricle (left + right) (109) -preliminary histological examination failed to identify structural defects to major blood vessels (109) **-Hamlet (the Drosophila orthologue of Prdm16/Prdm3) modifies the accessibility of Suppressor of hairless to Notch target gene promoters (110)**
				Yes (gene trap, reporter, backcrossed to C57Bl/6) (108)	-die soon after birth -increased ROS levels -stem cell depletion -increased cell death -reduced HGF expression in NSC -reduced brain mass	
				Yes (mutation and gene trap) (109)	-cleft palate with abnormal positioning of the tongue; die within one day after birth -the occurrence of respiratory failure suggests that there is also problems with the respiratory and circulatory system -mutant: choroid plexus hypoplasia; abnormal retinal folds -lung size reduced -heart ventricles reduced in size	
PSMAL	FOLH2, GCP3, PSM	Prostate-specific membrane antigen-like protein	Intracellular (cytoplasm)	Not reported	-	-
PTPRR	PTPBR7, PTP-SL	Protein tyrosine phosphate, receptor type, R	Cell surface (receptor)	Yes (targeted KO) (111)	-viable and fertile -defects in fine motor coordination and balance skills leading to ataxia -no histological abnormalities in Purkinje cells	-expression pattern is species-specific (112) -mostly expressed in the brain (112) -in mice mostly expressed in cerebellar Purkinje cells -controls MAPK activity
RASGRF2	GRF2	Ras protein-specific guanine nucleotide-releasing factor 2	Variable (cell surface and intracellular, cytoplasm)	Yes (targeted KO) (113)	-viable and fertile -no functional overlap with RASGRF1 since no aggravated phenotype in double KO mice	-expression in the nucleus of the solitary tract (region implicated in breath control and oxytocin synthesis during lactation)
				Yes (targeted KO) (114)	-mediator of NMDAR activation in adult neurons (not in newborns) -maintains CREB activity with RASGFR1 -hippocampal learning defect (in single and double KO mice) -protective role in stroke-induced excitotoxicity (in double KO mice)	
RGC-32	RGCC	Response gene to complement 32	Intracellular (cytoplasm and nucleus)	Not reported	-	-induces EMT in a pancreatic cancer cell line (115) and in renal tubular cells (116)

						-promotes vascular lesion formation (upon carotid artery balloon-injury in rats) by stimulation of SMC proliferation and migration (117) -plays a role in SMC differentiation of neural crest cells (118) -inhibits angiogenesis in matrigel implantation assay and inhibits EC proliferation in vitro (119) -is required for C5b-9-induced cell cycle activation in aortic ECs (120) and induces cell cycle activation in aortic SMCs (121) -regulated by PDGF-BB in SMCs (117)
RUNDC3B	RPIB9, RPIP9	RUN domain containing 3B	?	Not reported	-	-activated in breast carcinoma and correlates with poor prognosis (122)
RYR3	HBRR	Ryanodine receptor 3	Intracellular (calcium release channel)	Yes (targeted KO) (123)	-viable and fertile -increased locomotor activity -no obvious defects in the circulatory systems	-
				Yes (targeted KO) (124)	-viable and fertile -skeletal muscle contraction is impaired during the first weeks after birth but not in adult stage	
				Yes (targeted KO) (125)	-viable and fertile -improved spatial learning -increased hippocampal LTP	
SEMA3C	SEMAE	Sema domain, immunoglobulin domain (Ig), short basic domain, secreted (semaphorin) 3C	Secreted	Yes (targeted KO, reporter) (126)	-die within hours after birth from congenital cardiovascular defects (interruption of the aortic arch and improper septation of the cardiac outflow tract)	-promotes neural crest cell migration (126) -since NRP1-/- mice also have persistent truncus arteriosus, the co-receptor for SEMA3C may be NRP1 (126)
				Yes (mutant) (127)	-same as above -skin hypopigmentation	
SEMA3G	Sem2	Sema domain, immunoglobulin domain (Ig), short basic domain, secreted (semaphorin) 3G	Secreted	Yes (targeted KO, reporter) (128)	-viable and fertile -no overt vascular phenotype	-expressed in angiogenic and cultured ECs but not by SMCs (128) -highly expressed in arteries during development and thoughout adolescence (but no longer in adult stage) (128) -full-length SEMA3G binds to NRP2 -autocrine effects on EC/paracrine effects on SMCs (128)
SLC2A1	GLUT1	Solute carrier family 2 (facilitated glucose transporter), member 1	Cell surface (receptor)	Yes (targeted KO) (129)	-homozygotes are lethal around E14 -heterozygotes have epilepsy, impaired motor activity, incoordination, microencephaly, decreased brain glucose uptake, hypoglycoracchia	
				Yes ('antisense transgenic mice') (130)	-homozygotes are lethal around E18.5 -neural tube defects, caudal regression, headless state, microphthalmia	

SLC2A3	GLUT3	Solute carrier family 2 (facilitated glucose transporter), member 3	Cell surface (receptor)	Yes (targeted KO) (131)	-homozygous embryos die between E8.5 and E9.5; loss of GLUT1 expression in the basolateral surfaces of trophectoderm -early pregnancy loss at neurulation stage -heterozygotes show fetal growth restriction but survive until adulthood	-trophoblastic facilitative glucose transporter -GLUT1 and GLUT3 may need to be coordinately expressed to allow adequate glucose uptake by blastocysts -GLUT3 has an important role in trophectoderm/placenta whereas GLUT1 has an important role in the embryo proper -expressed in fetal ECs of term human placenta (132) -expressed in most brain areas
				Yes (gene trap, reporter) (133)	-heterozygotes have enhanced cerebrocortical activity but normal neuronal function, feeding behavior and energy balance	
SOX17	VUR3	SRY (sex determining region-Y)-box 17	Intracellular (nucleus; TF)	Yes (Sox17icre knock-in mouse) (134)	-viable (due to compensation through an alternative shorter mRNA)	-Sox17icre knock-in mouse reveals arterial EC-specific expression (with exception of the umbilical vein); two different promoters drive Sox17 expression in endoderm and vascular system (134) -is co-expressed with Sox7 and Sox18 in the vasculature (135-137) -loss of Sox17 is associated with the acquisition of an adult surface marker phenotype by HSCs -lineage tracing (using a Sox17-2A-icre knock-in mouse) (138) reveals expression in EC by E8.5 (dorsal aorta, heart endocardium, blood vessels in head region and allantois; E9.5 ISV; all vasculature by E10.5, including cardinal veins) -Sox17 plays a crucial role downstream of Norrin/Fz4/Lrp which induce an angiogenic program in retina and cerebellum (139)
				Yes (targeted KO, reporter) (140)	-embryonic lethal by E13.5 due to severe fetal hematopoietic defects (is specifically expressed in fetal/neonatal HSCs but not in adult HSCs)	
				Yes (targeted KO, reporter) (141)	-die around E10.5 -lack definitive gut endoderm	
				Yes (targeted KO; double with Sox18) (136)	-single Sox17-/-: aberrant heart looping, enlarged cardinal vein (due to blood stagnation), mild defects in anterior dorsal aorta -double KO: more severe defects in anterior dorsal aorta formation, head/cervical vasculature, aberrant differentiation of endocardial cells	
				Yes (targeted KO; double with Sox18) (135)	-Sox17+/-:Sox18-/-: 50% dies before P21; reduced vascularization in liver and kidney -vascular abnormalities in reproductive organs in females leading to infertility	
SYTL2	SLP2, EXO4	Synaptotagmin-like protein 2	Cell surface	Yes (targeted KO) (142)	-reduced number of mucus granules and reduction of mucus secretion by gastric primary cells	-most abundantly expressed in stomach
TFPI2	PP5, REF1	Tissue factor pathway inhibitor 2	Secreted	Not reported	-	-
TMEM200A	TTMC	Transmembrane protein 200A	membrane	Not reported	-	-
TMEM200C	TTMA	Transmembrane protein 200C	membrane	Not reported	-	-
TOX2	GCX-1	TOX high mobility group box family member 2	Intracellular (nucleus; TF)	Not reported	-	-expression is restricted to organs related to reproduction: hypothalamus, pituitary, testis, uterus, and ovary (143) -TOX is associated with cardiovascular disease (144)
XG	PBDX	Xg blood group	Cell surface	Not reported	-	-

Gene	Synonym(s)	Full name	Subcellular localization/function	Tg mice reported?	Phenotype	Other
Venous genes						
APM2	-	Adipose most abundant gene transcript 2 protein	?	Not reported	-	-
CMBL	-	Carboxymethylenebutenolidase homolog (Pseudomonas)	Intracellular (cytoplasm)	Not reported	-	-Ubiquitous expression in various tissues (145) -Involved in bioactivation of drugs (145)
FLRT3	-	Fibronectin leucine rich transmembrane protein 3	Cell surface	Yes (targeted KO) (146)	-highly disorganized BM in the anterior visceral endoderm -EMT of anterior epiblast cells and upregulation of mesodermal genes	-*Xenopus* Flrt3 complexes with FGF receptors (147)
MGC13057	-	?	?	Not reported	-	-
MGST1	GST12, MGST	Microsomal glutathione S-transferase 1	Intracellular (ER, mitochondrial membrane)	Not reported	-	-Confers resistance to oxidative stress (148) -not expressed in cultured HUVECs (27)
MPP7	-	Membrane protein, palmitoylated 7 (MAGUK p55 subfamily member 7)	Cell surface (cell junctions)	Not reported	-	-
NOS1	nNOS	Nitric oxide synthase 1 (neuronal)	Cell surface (sarcolemma)	Yes (targeted KO) (149)	-viable and fertile -enlarged stomach -no overt central nervous system abnormalities	
				Yes (targeted KO) (150)	-hypogonadism and infertility (both male and female) -pyloric stenosis	-this KO has no residual nNOS activity (unlike the first one)
NR3C2	MLR, MR, mineralocorticoid receptor 1	Nuclear receptor subfamily 3, group C, member 2	Variable; has transcription factor properties, functions both as a nuclear and a membrane receptor (the latter at least in neurons) (151)	Yes (EC-specific overexpression) (152)	-EC MR has a direct effect on blood pressure independent of the kidney -develop moderate hypertension at baseline -have increased acute BP response to angiotensin II or endothelin I	-MR activation may cause vascular aging and atherosclerosis (153) -physiologic ligand for MR is still controversial -MR promotes vascular oxidative stress (153) -is expressed in (arterial) ECs and SMCs (152) -adosterone may be beneficial in healthy vessels but may be detrimental (e.g. causing oxidative stress) in diseased vessels when there is another pathological challenge in addition to vascular MR activation
OLFML3	OLF44, mONT3	Olfactomedin-like 3	Secreted (signaling protein)	Yes (targeted KO, reporter) (154)	-viable and fertile	-expressed in allantois (E7.25), lateral plate mesoderm (E8.0), CNS and heart (E8.5), neural tube, branchial arches, mesenchymal tissues dorsal to the telencephalon, ventral protion of diencephalon and midbrain (E10.5) (154)
PAPLN	-	Papilin,	Secreted (ECM protein;			-may modulate metalloproteinases during organogenesis

		proteoglycan-like sulfated glycoprotein	primarily part of BM (155))			(156)
SCARA3	CSR, MSLR1, APC7	Scavenger receptor class A, member 3	Intracellular (ER, Golgi)	Not reported	-	-
SNTB2	Syntrophin-3, β2-syntrophin	Syntrophin, β2 (dystophin-associated protein A1, 59kDa, basic component 2)	Intracellular (adaptor protein)	Yes (targeted KO) (157)	-no overt phenotype -combined KO with α-syntrophin results in more severe abnormalities in neuromuscular junctions	-nNOS is a syntrophin PDZ domain ligand *in vitro* and *in vivo*, but only *in vitro* for β2-syntrophin

SUPPLEMENTARY INFORMATION – ITEM 6: Table S5

Table S5. ***Expression of genes from the arteriovenous fresh profile in DLL4-Fc treated HUAECs.***

Arterial genes	BSA			DLL4-Fc		
MFAP5	100	±	8	169	±	8
FAM19A5	100	±	34	107	±	4
PTPRR	100	±	14	185	±	22
H19	100	±	18	1298	±	258*
FGL2	100	±	27	3	±	8
NPR3	100	±	1	233	±	64
CNTN3	100	±	2	241	±	46
RASGRF2	100	±	18	115	±	14
SEMA3G	100	±	33	757	±	141*
PRDM16	100	±	22	76	±	13
MPPED2	100	±	55	152	±	109
A2M	100	±	28	194	±	21
FREM1	100	±	33	145	±	81
COL4A2	100	±	15	315	±	33*
HEY2	100	±	17	992	±	43*
NRCAM	100	±	3	64	±	3
COL4A1	100	±	15	428	±	47*
SEMA3C	100	±	26	159	±	48
FAP	100	±	6	133	±	4
TOX2	100	±	9	116	±	6
KDR	100	±	15	46	±	2*
TMEM200C	100	±	25	111	±	35
XG	100	±	18	209	±	13*
RGC-32	100	±	4	43	±	0.2*
LAMA2	100	±	2	117	±	8
TMEM200A	100	±	9	52	±	6*
ADRB1	100	±	24	88	±	26
TFPI2	100	±	4	180	±	21*
SOX17	100	±	3	91	±	8
SLC2A3	100	±	4	109	±	12
SYTL2	100	±	7	89	±	6
KLHL6	100	±	7	102	±	7
NKX2-3	100	±	15	412	±	290
HECW2	100	±	9	178	±	10*
CYP4F30P	100	±	11	222	±	22
FAT1	100	±	7	311	±	36*
FOLH1	100	±	10	242	±	35*
GALNTL4	100	±	24	133	±	5
RYR3	100	±	10	164	±	25*

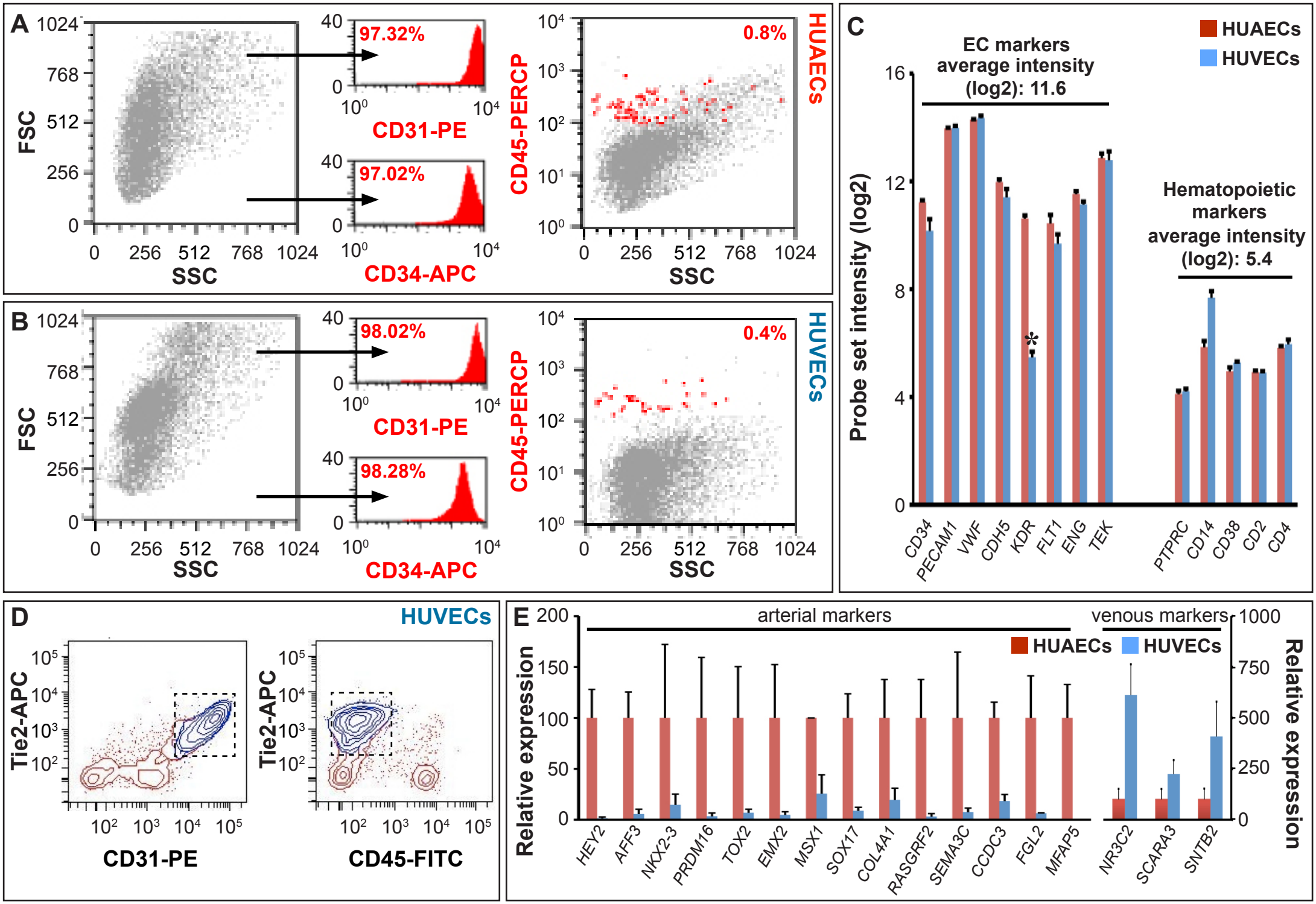
Arterial genes	BSA			DLL4-Fc		
AFF3	100	±	85	71	±	15
NOTCH4	100	±	12	129	±	7
EMX2	100	±	17	137	±	31
NAV1	100	±	3	95	±	7
ARL15	100	±	8	125	±	5
PSMAL	100	±	11	246	±	14*
MAP9	100	±	19	179	±	134
GLIPR2	100	±	8	132	±	8
APO	100	±	4	103	±	11
LRCH1	100	±	4	105	±	2
MSX1	100	±	1	256	±	19*
SLC2A1	100	±	6	162	±	2*
GRB14	100	±	4	46	±	1*
ODAM	100	±	97	776	±	1807
CCDC3	100	±	18	2735	±	407*
CUBN	100	±	16	330	±	7*
PDE4D	100	±	2	113	±	5
RUNDC3B	100	±	11	94	±	9
CYSLTR1	100	±	8	584	±	40*
FAM176A	100	±	4	210	±	4*
ACE2	100	±	9	100	±	86
LOC401022	100	±	12	110	±	4
LCHN	100	±	16	351	±	30*
LMCD1	100	±	9	193	±	33*
HSF2BP	100	±	5	121	±	21
Venous genes	**BSA**			**DLL4-Fc**		
NR3C2	100	±	6	91	±	2
SCARA3	100	±	14	83	±	2
FLRT3	100	±	14	169	±	11*
PAPLN	100	±	17	161	±	15*
SNTB2	100	±	7	96	±	6
MGC13057	100	±	2	107	±	19
APM2	100	±	21	87	±	15
CMBL	100	±	2	115	±	3
OLFML3	100	±	30	21	±	95
MGST1	100	±	2	70	±	2
MPP7	100	±	1	112	±	13
NOS1	100	±	26	132	±	21

SUPPLEMENTARY INFORMATION – ITEM 7: Table S6

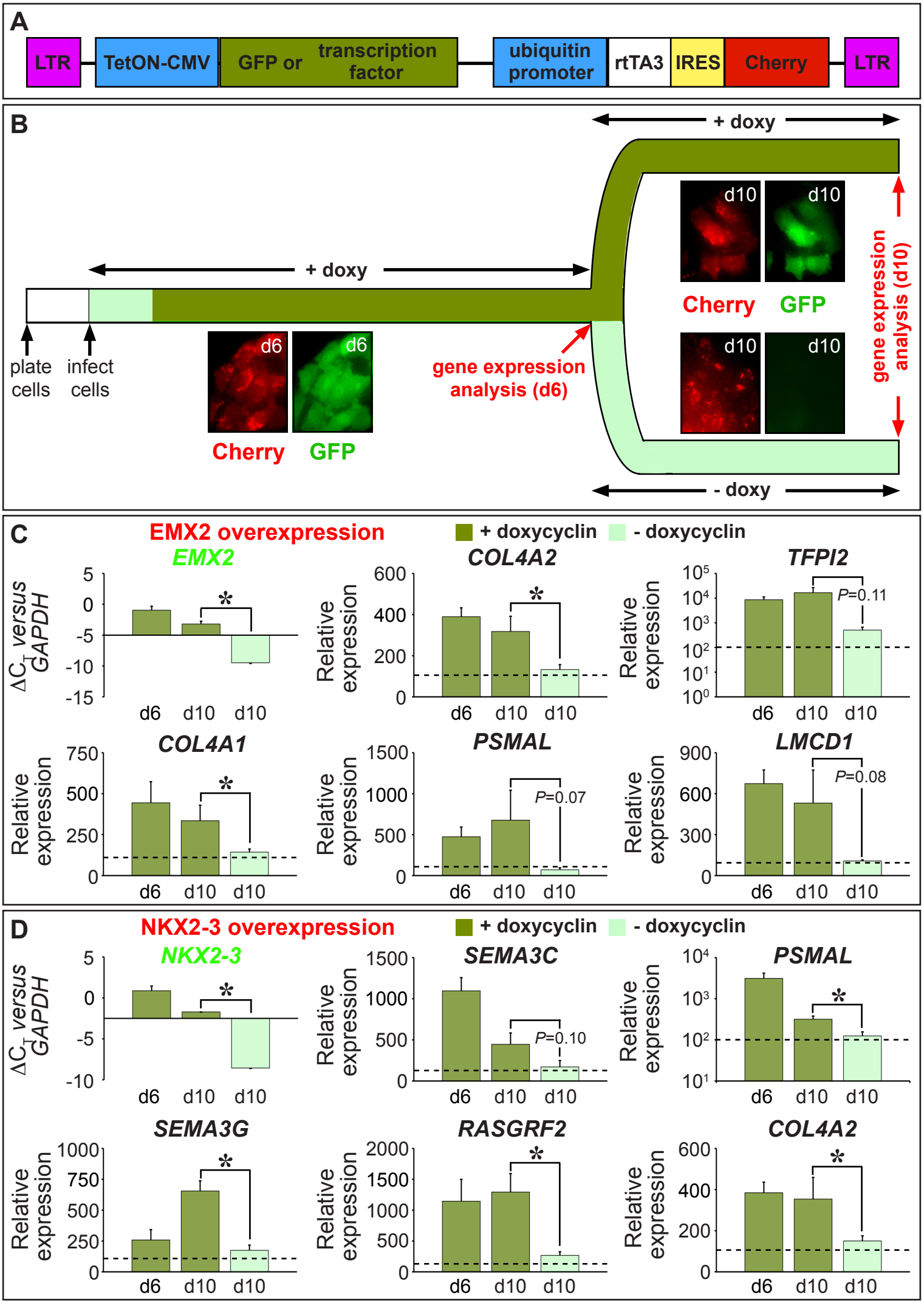
Table S6. ***Expression of genes from the arteriovenous fresh profile in cultured HUVECs overexpressing TFs.***

aEC genes	cherry			Hey2			Msx1			Emx2			Prdm16			Nkx2-3			Aff3			Sox17			Tox2			All		
MFAP5	100	±	69	919	±	229*	91	±	71	696	±	209	919	±	0*	178	±	125	382	±	236	5#	±	4*#	620	±	403	219#	±	25*#
FAM19A5	100	±	15	158	±	11	91	±	22	151	±	29	148	±	15	105	±	40	119	±	11	126	±	30	172	±	10	133	±	18
PTPRR	100	±	57	207	±	133	165	±	21	643	±	92	441	±	107	95	±	34	110	±	52	228	±	60*	94	±	41	595	±	127*
H19	100	±	4	129	±	44	196	±	68	310	±	85*	39540	±	10556*	97	±	7	122	±	22	247	±	118	99	±	16	11953	±	3906*
FGL2	100	±	71	706	±	539*	212	±	130	121	±	60	339	±	160	510	±	196*	146	±	58	238	±	146	459	±	215	2851	±	1436*
NPR3	100	±	77	798	±	403*	182	±	168	490	±	193*	419	±	64*	315	±	207	128	±	105	154	±	61	355	±	86	723	±	310*
CNTN3	100	±	96	180	±	111	51	±	28	27	±	22	546	±	63*	134	±	60	43	±	29	197	±	122	98	±	65	176	±	39
RASGRF2	100	±	37	919	±	378*	31	±	16	319	±	101	393	±	78*	1074	±	261*	139	±	50	331	±	26	105	±	22	323	±	189
SEMA3G	100	±	52	81	±	31	288	±	212	174	±	45	179	±	25*	373	±	160*	117	±	60	1037	±	333*	69	±	12	531	±	312*
PRDM16	100	±	20	208	±	38	184	±	44	177	±	42	125	±	27	119	±	24	135	±	21	139	±	51	114	±	5	186	±	28
MPPED2	100	±	79	371	±	605	197	±	237	222	±	275	217	±	370	153	±	119	213	±	186	141	±	303	1619	±	1481*	553	±	784
A2M	100	±	41	70	±	22	50	±	7	141	±	31	2061	±	394*	470	±	71*	55	±	13	156	±	6	259	±	52*	338	±	103*
FREM1	100	±	12	125	±	35	106	±	6	167	±	22	96	±	56	141	±	15	91	±	29	117	±	64	66	±	10	144	±	26
COL4A2	100	±	21	179	±	49	146	±	42	384	±	54*	77	±	8	190	±	61*	89	±	20	167	±	39	58	±	8	124	±	15
HEY2	100	±	116	152	±	144	746	±	272*	255	±	208	767	±	263*	307	±	54	56	±	32	179	±	97	73	±	24	275	±	99
NRCAM	100	±	39	221	±	62	55	±	42	43	±	7*	110	±	10	147	±	29	154	±	21	180	±	14	185	±	11	140	±	11
COL4A1	100	±	22	193	±	56	141	±	55	340	±	49*	86	±	10	165	±	67	95	±	25	198	±	40	59	±	8	148	±	11
SEMA3C	100	±	41	985	±	820*	356	±	75	43	±	19	15578	±	2529*	1255	±	392*	101	±	21	439	±	160	86	±	48	1238	±	1125*
FAP	100	±	9	125	±	4	117	±	12	115	±	10	129	±	8	120	±	8	96	±	4	100	±	4	81	±	3	108	±	7
TOX2	100	±	16	91	±	11	116	±	12	104	±	9	77	±	5	88	±	10	89	±	16	94	±	10	6848	±	1314*	2542	±	111*
KDR	100	±	28	61	±	16	120	±	33	65	±	10	29	±	3*	253	±	33*	89	±	14	95	±	20	182	±	4	86	±	13
TMEM200C	100	±	91	56	±	22	83	±	108	85	±	19	14	±	9*	29	±	43	24	±	13	17	±	5	18	±	5	22	±	9
XG	100	±	25	201	±	54	297	±	37*	222	±	39	373	±	71*	296	±	71*	107	±	24	81	±	11	157	±	33	553	±	120*
RGC-32	100	±	21	41	±	14*	82	±	22	113	±	20	169	±	8	163	±	20	73	±	16	69	±	20	54	±	11	161	±	8
LAMA2	100	±	12	107	±	10	122	±	12	159	±	8*	120	±	7	105	±	25	94	±	5	113	±	6	80	±	1	135	±	9
TMEM200A	100	±	31	130	±	15	64	±	20	52	±	4	53	±	2	93	±	15	124	±	6	88	±	8	102	±	7	40	±	5*
ADRB1	100	±	10	102	±	24	113	±	14	120	±	7	60	±	12	86	±	9	67	±	10	86	±	20	41	±	2*	64	±	9
TFPI2	100	±	27	342	±	268*	154	±	45	1706	±	197*	94	±	9	201	±	36	105	±	28	51	±	20	28	±	4	368	±	71*
SOX17	100	±	12	105	±	2	112	±	7	110	±	11	86	±	3	81	±	9	103	±	11	166	±	24*	78	±	2	160	±	34*
SLC2A3	100	±	5	102	±	14	103	±	10	139	±	14	77	±	6	145	±	11	89	±	7	118	±	8	95	±	7	103	±	7
SYTL2	100	±	43	479	±	143*	85	±	55	138	±	31	738	±	254*	499	±	94*	97	±	22	64	±	40	257	±	135	307	±	196
KLHL6	100	±	13	96	±	8	137	±	16	148	±	2	68	±	2	122	±	6	102	±	9	95	±	4	93	±	13	93	±	9
NKX2-3	100	±	6	124	±	38	117	±	21	119	±	27	48	±	18	93	±	13	83	±	14	107	±	25	72	±	5	69	±	16
HECW2	100	±	19	118	±	21	141	±	13	95	±	9	38	±	2*	48	±	4*	90	±	8	110	±	7	86	±	7	96	±	10
CYP4F30P	100	±	15	165	±	42	118	±	29	194	±	13*	123	±	37	93	±	20	66	±	8	107	±	36	68	±	6	108	±	13
FAT1	100	±	35	220	±	66*	349	±	49*	314	±	31*	177	±	14*	186	±	48*	115	±	18	138	±	25	285	±	31*	358	±	26*
FOLH1	100	±	61	252	±	92	256	±	102	172	±	83	43	±	42	278	±	100	106	±	53	125	±	72	148	±	97	153	±	95
GALNTL4	100	±	10	108	±	16	106	±	15	157	±	7*	112	±	11	165	±	6*	90	±	11	159	±	12*	145	±	23	136	±	15
RYR3	100	±	24	106	±	7	99	±	9	115	±	13	103	±	27	105	±	30	93	±	7	117	±	16	92	±	7	87	±	22
AFF3	100	±	22	89	±	38	246	±	64	162	±	145	47	±	41	85	±	37	132	±	23	68	±	9	45	±	15	76	±	17
NOTCH4	100	±	24	133	±	12	112	±	13	144	±	15	118	±	10	254	±	47	92	±	18	438	±	79	79	±	15	164	±	15

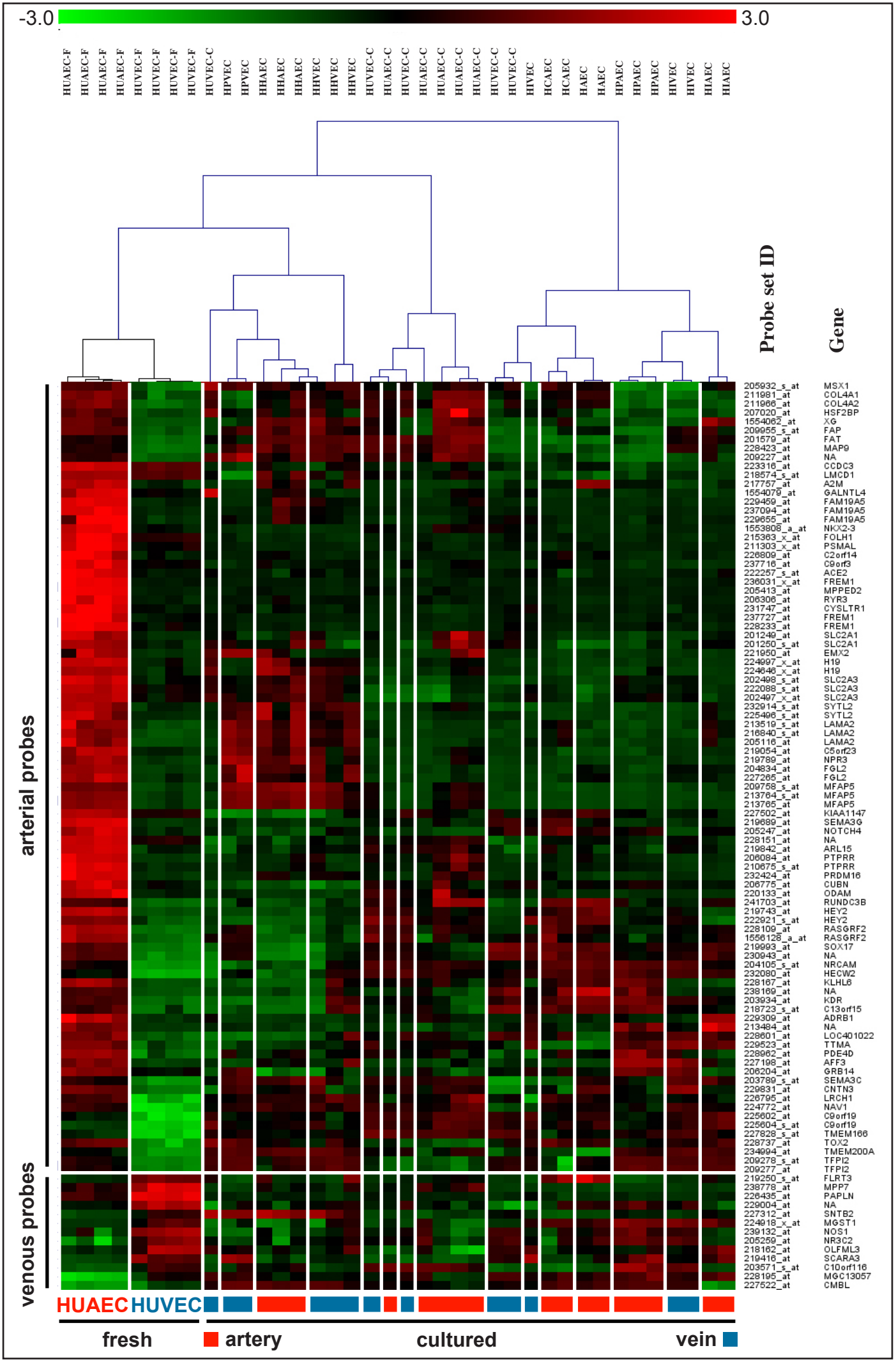
aEC genes	cherry			Hey2			Msx1			Emx2			Prdm16			Nkx2-3			Aff3			Sox17			Tox2			All		
EMX2	100	±	17	113	±	11	85	±	8	99	±	4	116	±	16	66	±	46	89	±	14	118	±	37	140	±	5	120	±	23
NAV1	100	±	14	113	±	12	100	±	14	109	±	4	111	±	8	94	±	7	100	±	13	112	±	10	90	±	7	103	±	6
ARL15	100	±	6	115	±	6	107	±	9	118	±	4	194	±	5*	148	±	8	99	±	7	136	±	10	107	±	2	143	±	13*
PSMAL	100	±	25	363	±	27*	523	±	65*	313	±	54	44	±	8	550	±	61*	209	±	75	133	±	13	151	±	31	259	±	78
MAP9	100	±	44	329	±	110*	104	±	27	125	±	40	857	±	345*	226	±	87	191	±	77	172	±	78	252	±	79	775	±	259*
GLIPR2-	100	±	25	94	±	14	189	±	45*	178	±	17*	98	±	5	53	±	4	92	±	7	99	±	12	53	±	6	116	±	11
APO	100	±	15	129	±	10	108	±	14	141	±	3	132	±	10	261	±	25*	87	±	5	114	±	10	108	±	2	123	±	6
LRCH1	100	±	24	100	±	11	123	±	13	162	±	14*	111	±	5	413	±	62*	69	±	13	129	±	14	151	±	6	147	±	16
MSX1	100	±	10	185	±	31*	142	±	21	227	±	29*	128	±	6	217	±	38*	116	±	11	165	±	32	183	±	42*	244	±	12*
SLC2A1	100	±	13	149	±	21	162	±	26	275	±	41*	162	±	11*	228	±	72*	97	±	11	102	±	17	77	±	4	221	±	19*
GRB14	100	±	13	63	±	11	112	±	32	156	±	9*	56	±	2	108	±	8	81	±	8	85	±	9	102	±	15	110	±	4
ODAM	100	±	54	1510	±	717*	133	±	77	162	±	117	2891	±	1008*	99	±	52	178	±	186	3762	±	664*	71	±	34	2569	±	3349*
CCDC3	100	±	31	115	±	56	101	±	28	32	±	26	78	±	26	189	±	29	116	±	42	174	±	44	242	±	83	200	±	101*
CUBN	100	±	16	224	±	40*	136	±	50	88	±	13	205	±	33*	246	±	33*	113	±	12	171	±	17	156	±	6	114	±	15
PDE4D	100	±	13	133	±	17	117	±	17	133	±	10	73	±	6	119	±	16	110	±	21	101	±	11	102	±	5	74	±	12
RUNDC3B	100	±	20	149	±	35	60	±	28	74	±	8	209	±	12*	196	±	46*	107	±	13	109	±	9	105	±	12	117	±	12
CYSLTR1	100	±	20	100	±	21	90	±	19	148	±	14	98	±	10	154	±	22	84	±	11	125	±	9	122	±	15	97	±	17
FAM176A	100	±	22	163	±	21	71	±	27	236	±	12*	116	±	4	261	±	12*	108	±	10	121	±	6	91	±	3	129	±	7
ACE2	100	±	68	241	±	175	184	±	98	75	±	165	556	±	155	962	±	128*	148	±	106	241	±	195	602	±	366	888	±	466*
LOC401022	100	±	33	90	±	21	42	±	11	102	±	21	231	±	27*	346	±	48*	67	±	15	115	±	19	79	±	20	122	±	53
LCHN	100	±	22	121	±	15	102	±	9	114	±	5	179	±	7*	167	±	21*	84	±	16	162	±	22*	102	±	7	126	±	16
LMCD1	100	±	24	163	±	47	181	±	34	767	±	204*	211	±	28*	155	±	30	84	±	7	170	±	16	73	±	3	325	±	33*
HSF2BP	100	±	17	112	±	13	105	±	28	171	±	12*	111	±	7	279	±	30*	104	±	13	117	±	30	98	±	4	125	±	15
vEC genes	**cherry**			**Hey2**			**Msx1**			**Emx2**			**Prdm16**			**Nkx2-3**			**Aff3**			**Sox17**			**Tox2**			**All**		
NR3C2	100	±	35	151	±	8	64	±	23	117	±	7	143	±	12	158	±	12	130	±	19,3	161	±	8	100	±	8	133	±	13
SCARA3	100	±	15	101	±	24	92	±	2	108	±	9	69	±	3	121	±	16	62	±	8,6	98	±	9	79	±	12	70	±	8
FLRT3	100	±	6	138	±	7	111	±	8	104	±	9	75	±	10	85	±	8	102	±	5,6	131	±	16	102	±	15	99	±	6
PAPLN	100	±	34	126	±	17	86	±	18	113	±	9	111	±	19	178	±	36	67	±	12,6	108	±	24	413	±	130*	162	±	36*
SNTB2	100	±	15	120	±	10	83	±	17	107	±	5	101	±	7	131	±	13	109	±	12	126	±	17	86	±	5	84	±	8
MGC13057	100	±	13	135	±	12	117	±	22	119	±	9	548	±	40*	152	±	23	84	±	17	88	±	14	139	±	9	369	±	22*
APM2	100	±	34	114	±	11	165	±	18	134	±	20	119	±	6	151	±	37	84	±	26	100	±	15	66	±	4	177	±	6
CMBL	100	±	14	91	±	7	120	±	18	129	±	8	90	±	5	96	±	16	100	±	4	89	±	7	103	±	4	100	±	10
OLFML3	100	±	66	444	±	63*	201	±	254	14703	±	5481*	222	±	65	190	±	63	58	±	17	60	±	14	119	±	43	2075	±	1394*
MGST1	100	±	6	105	±	6	86	±	10	63	±	6	99	±	3	134	±	15	96	±	6	94	±	6	67	±	5	77	±	5
MPP7	100	±	36	111	±	45	47	±	18	90	±	6	546	±	18*	139	±	43	92	±	27	95	±	31	230	±	94	301	±	152*
NOS1	ND			ND			ND			ND			ND			ND			ND			ND			ND			ND		



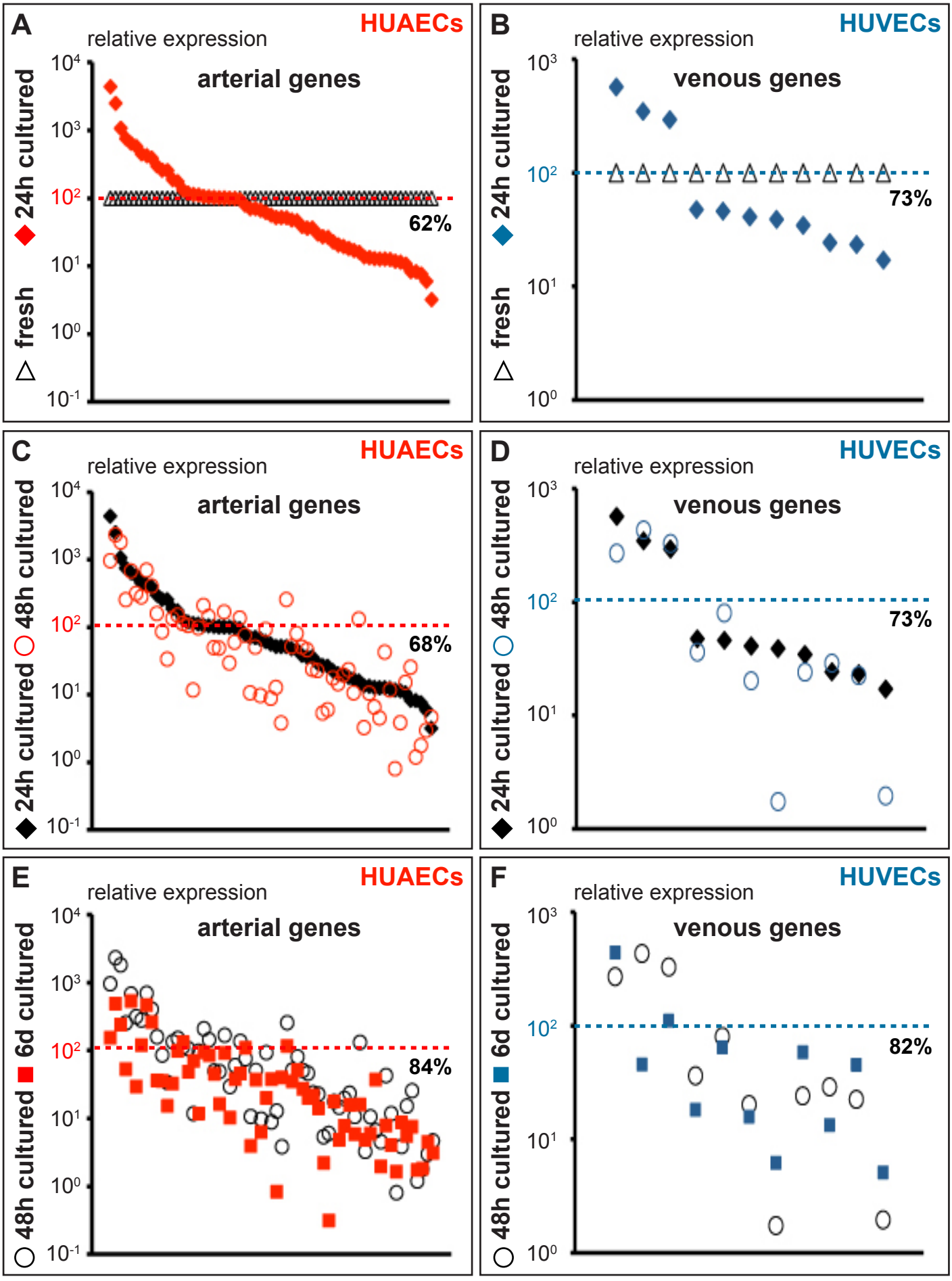
Aranguren *et al.* Figure S1



Aranguren *et al.* Figure S2



Aranguren *et al.* Figure S3



Aranguren *et al.* Figure S4