## Supplemental Table 1: Primer Sequences for qPCR

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Abbreviations for Supplemental Tables 1-4: LAD = left anterior descending coronary artery; RCA = right coronary artery; LCX = left circumflex artery; Lmain = proximal left coronary artery; pr = proximal region of vessel; m = mid region of vessel; di = distal region of vessel; CM = cardiomyopathy; V-tach = ventricular tachycardia; ASD = atrial septal defect; HD = heart defect; ARVC = arrhythmogenic right ventricular cardiomyopathy; Movat’s = Movat’s pentachrome stain; PSR = picrosirius red stain; SHG = second harmonic generation.

**numbers following vessel name (e.g. RCA2) refer to non-contiguous segments of the same vessel from the same patient.**
## Supplemental Table 3: Atherosclerotic Hyperplasia Coronary Artery Samples

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## Supplemental Table 4: Complex Plaque Coronary Artery Samples

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Supplemental Figure 1. Proximity Ligation Assay Showing Interaction of PTEN and Serum Response Factor (SRF) in the Nucleus of Medial Smooth Muscle Cells (SMCs). Proximity ligation assay (PLA) and confocal microscopy were used to detect PTEN-SRF interactions in medial SMCs of human non-atherosclerotic coronary arteries. (a-g). PLA using mouse anti-PTEN and rabbit anti-SRF primary antibodies and anti-mouse PLUS and anti-rabbit MINUS PLA probe demonstrates PTEN-SRF nuclear interactions in medial SMCs of human NAH coronary. Shown are several representative images from an N=4 vessels. (h). Positive PTEN-SRF PLA signal in SMCs of an adventitial vasa vasorum microvessel. * = red blood cells in lumen of microvessel. For panels a-h: Red = Positive PLA; Blue = DAPI for cell nuclei; Scale bars = 50 μm. (i). Nuclei of medial SMCs were scored for percent positive PLA signal. 9 representative images from N=4 individual non-atherosclerotic arteries from N=4 patients.
Supplemental Figure 2. Reduced PTEN and alpha-Smooth Muscle Actin (αSMA) in Medial Smooth Muscle Cells (SMCs) adjacent to atherosclerotic plaques. Paired vessel segments with and without atherosclerotic plaque (P, No P) from the same coronary artery were stained for PTEN and αSMA. Confocal microscopy was performed with the same image acquisition parameters used for single cell analysis of Figures 3 and 6. Four to five 63X images were acquired for each pair of atherosclerotic and non-atherosclerotic segments from the same coronary artery and independent heart. A) The mean gray values of αSMA and PTEN within the cell boundary were determined by Image J in 216 individual media cells of arteries with no plaque (open box) and 237 individual cells of media with adjacent atherosclerotic plaque (gray box) from N=6 vessels and independent hearts. Box and whisker data plots indicate the median gray value (bar), interquartile range (box boundary) and minimum to maximum range (error bars) of data values. B) The mean gray values for αSMA (circles) and PTEN (squares) of individual SMCs were averaged for each vessel (No P segment, filled; P segment, open). Exact P values are shown for Mann Whitney, two-tailed, t test comparisons of αSMA and PTEN gray values between coronary artery segments with and without adjacent plaque.
### Supplemental Table 6: Mouse Plasma Cholesterol and Triglyceride Levels

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All variables are median values with (interquartile range)
Mann Whitney t-test of WT versus KO for each diet condition.
Supplemental Figure 3. Matched Intima and Media Thickness Between Coronary Arteries Exposed or not Exposed to Continuous Flow Left Ventricular Assist Devices (LVAD). Coronary arteries from explanted hearts of non-LVAD or CF-LVAD patients were matched for NAH. **A.** Representative H&E images showing similar intimal non-atherosclerotic hyperplasia in non-LVAD-exposed (left) compared CF-LVAD-exposed (right) vessels, and similar medial thickness when normalized to lumen diameter. Dashed lines delineate the arterial media; M = arterial media; I = arterial intima; scale bars = 100 µm. **B.** Intima areas (left graph) and media areas (right graph) of the coronary arteries were measured by Image J and normalized to the area of the vessel lumen to control for differences in vessel caliber. N=20 individual vessels per group. Compared to non-LVAD vessels, CF-LVAD vessels exhibited no change in relative intima or media areas (Two tailed Student's t tests, P=0.5305 for intima; P=0.5222 for media). Error bar represents mean ± standard deviation.
Supplemental Figure 4. Significantly Differentiated Genes Within Previously Published Datasets of Chemokine- and Cytokine-Associated Genes. 1173 differentially expressed genes were compared to previously published datasets of chemokine- and cytokine-associated genes. 79 genes were identified as commonly expressed in the PTEN-deficient SMC microarray and previously published chemokine / cytokine datasets.
Supplemental Figure 5. Significantly Differentiated Genes Within Previously Published Datasets of Matrix-Associated Genes. 1173 differentially expressed genes were compared to previously published datasets of extracellular matrix- and matrix remodeling-associated genes. 118 genes were identified as commonly expressed in the PTEN-deficient SMC microarray and previously published extracellular matrix datasets.
Supplemental Figure 6. Representative Western blot showing decreased PTEN expression in PTEN-deficient Smooth Muscle Cells (SMCs). Pools of SMCs stably expressing control (Ctrl) or PTEN-specific shRNA were serum-restricted for 72 h. Whole cell lysates were analyzed for total PTEN levels. Parental (non-infected) SMCs were used as a control for shRNA viral infection. β-Actin was used as a loading control.
# Supplemental Table 7: Cytokine-associated Upregulated Genes In PTEN-Deficient SMCs

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### Supplemental Table 9: ECM-associated Upregulated Genes in PTEN-Deficient SMCs

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### Supplemental Table 10: ECM-associated Downregulated Genes In PTEN-Deficient SMCs

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## Supplemental Table 11: Cytokine and ECM-associated Genes Upregulated 1.8-2.0-fold in PTEN-Deficient SMCs

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