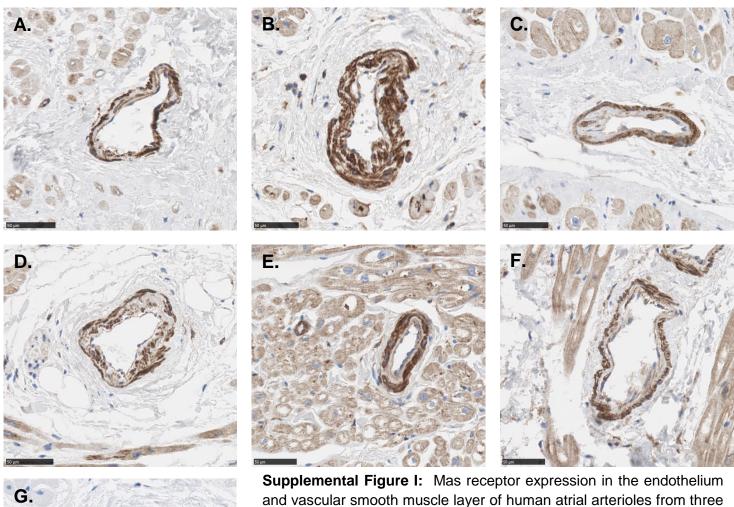
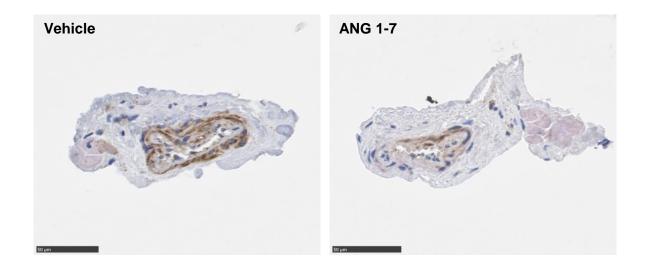
## Supplemental Figure I



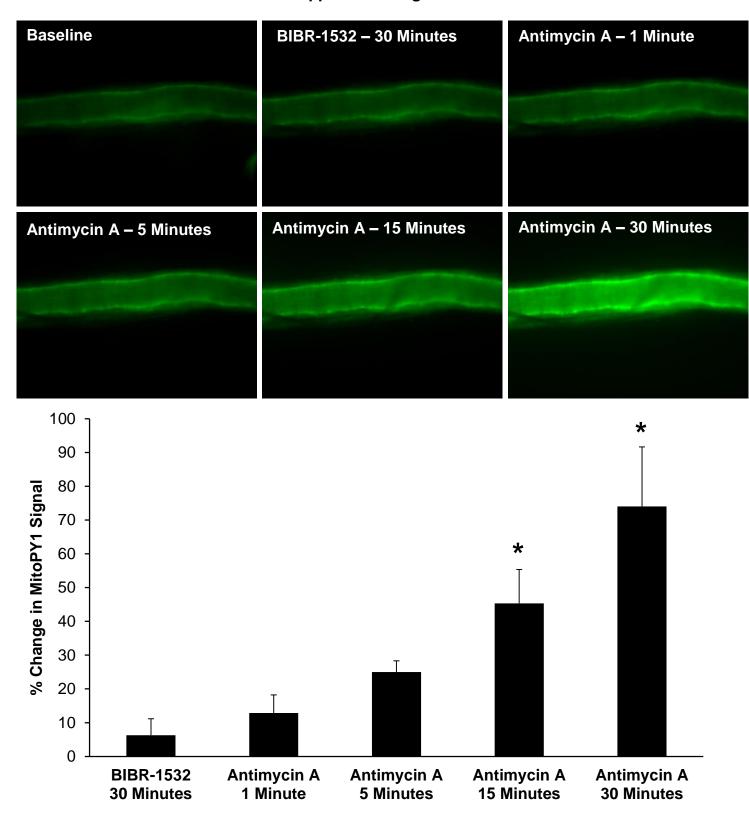
Supplemental Figure I: Mas receptor expression in the endothelium and vascular smooth muscle layer of human atrial arterioles from three different non-CAD (A-C) and CAD (D-F) subjects. Specificity of the antibody was examined by exclusion of the primary antibody (G). Scale bar =  $50 \mu m$ .

## **Supplemental Figure II**



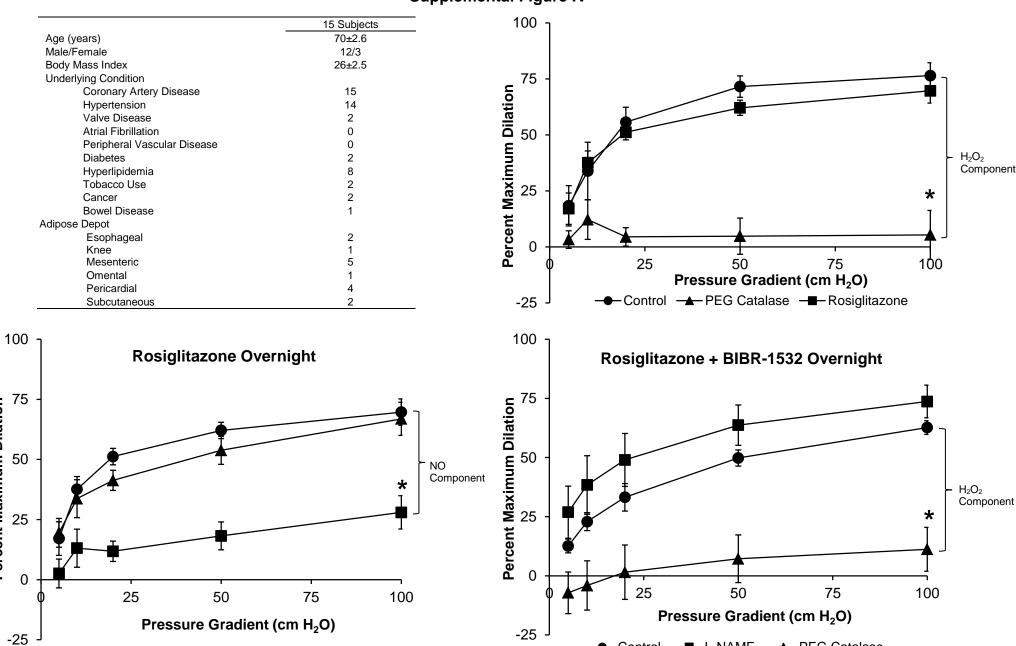
**Supplemental Figure II:** Mas receptor expression in the endothelium and vascular smooth muscle layer of human atrial arterioles from a single CAD subject treated either with ANG 1-7 ( $10^{-9}$  M) or vehicle overnight. Scale bar = 50 µm.

## **Supplemental Figure III**



**Supplemental Figure III.** BIBR-1532 (10  $\mu$ M; n=5) had no effect on mitochondrial H<sub>2</sub>O<sub>2</sub> levels in isolated human adipose arterioles when it was added to the organ bath for 30 minutes. The mitochondrial electron transport chain uncoupler antimycin A (1  $\mu$ M; n=4) increased mitochondrial H<sub>2</sub>O<sub>2</sub> in a time-dependent manner. n=number of vessels, each from a different individual. \*P<0.05 vs. BIBR-1532, one-way ANOVA.

## **Supplemental Figure IV**



Supplemental Figure IV. Tissue from 15 subjects with CAD was used to examine the effects of rosiglitazone (a PPARy activator) on FMD in adipose arterioles. Overnight rosiglitazone (10 µM) treatment had no effect on the magnitude of dilation (top, right), but restored the NO-component of dilation in arterioles from subjects with CAD (bottom, left). When the telomerase inhibitor BIBR-1532 (10 µM) was co-incubated with rosiglitazone, NO-mediated dilation was no longer restored (bottom, right). n=4-7, all groups. \*P<0.05 vs. Control.

-Control

---L-NAME

→ PEG Catalase

**Percent Maximum Dilation** 

Control

■ L-NAME

PEG Catalase