Supplementary figures
Figure 1. Representative photomicrographs of the thoracic aorta.
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Figure 1. Representative photomicrographs of the thoracic aorta stained for A) COX-1 (400X); B) COX-1 (600X) (arrow); C) Endothelial cells (von Willebrand factor; 600X) (arrow); D) COX-2 (100X); E) Smooth muscle cells (α-actin; 100X); F) COX-2 (400X); G) Macrophages (F4/80; 400X); H) Negative control for A (400X); I) Negative control for F and G (400X); L: lumen.

Figure 2. Representative photomicrographs of aortic root stained for A) Movat staining (100X); B) Macrophages (F4/80; 600X) (arrow head); C) COX-1 (600X) (arrow); D) COX-2 (600X) (arrow); F) Negative control (400X).

Figure 3. Positive correlation between aortic COX-2 expression and smooth muscle cell content in aortic root lesion. $R^2=0.4081$, P<0.008.

Figure 4. No changes of vasoconstriction in the presence of selective TXA2 inhibitor (SQ29548) after DE exposure. PE-concentration response curve shows that vasoconstriction of the thoracic aorta in DE exposure group was not different in the presence of TXA2 inhibitor. Values are mean±SEM.

Figure 5. The mRNA expression of COX-1, COX-2 and PGIS in the heart. A) The mRNA expression of COX-1 was not altered after exposure to DE, n=7 (filtered air), n=8 (DE), P>0.05; B) DE exposure significantly increased COX-2 mRNA expression, n=7 (filtered air), n=8 (DE), *P<0.006; C) The mRNA expression of PGIS significantly increased in DE exposure group (n=8), compared with filtered air exposure (n=7), *P<0.02. Values are mean±SEM.