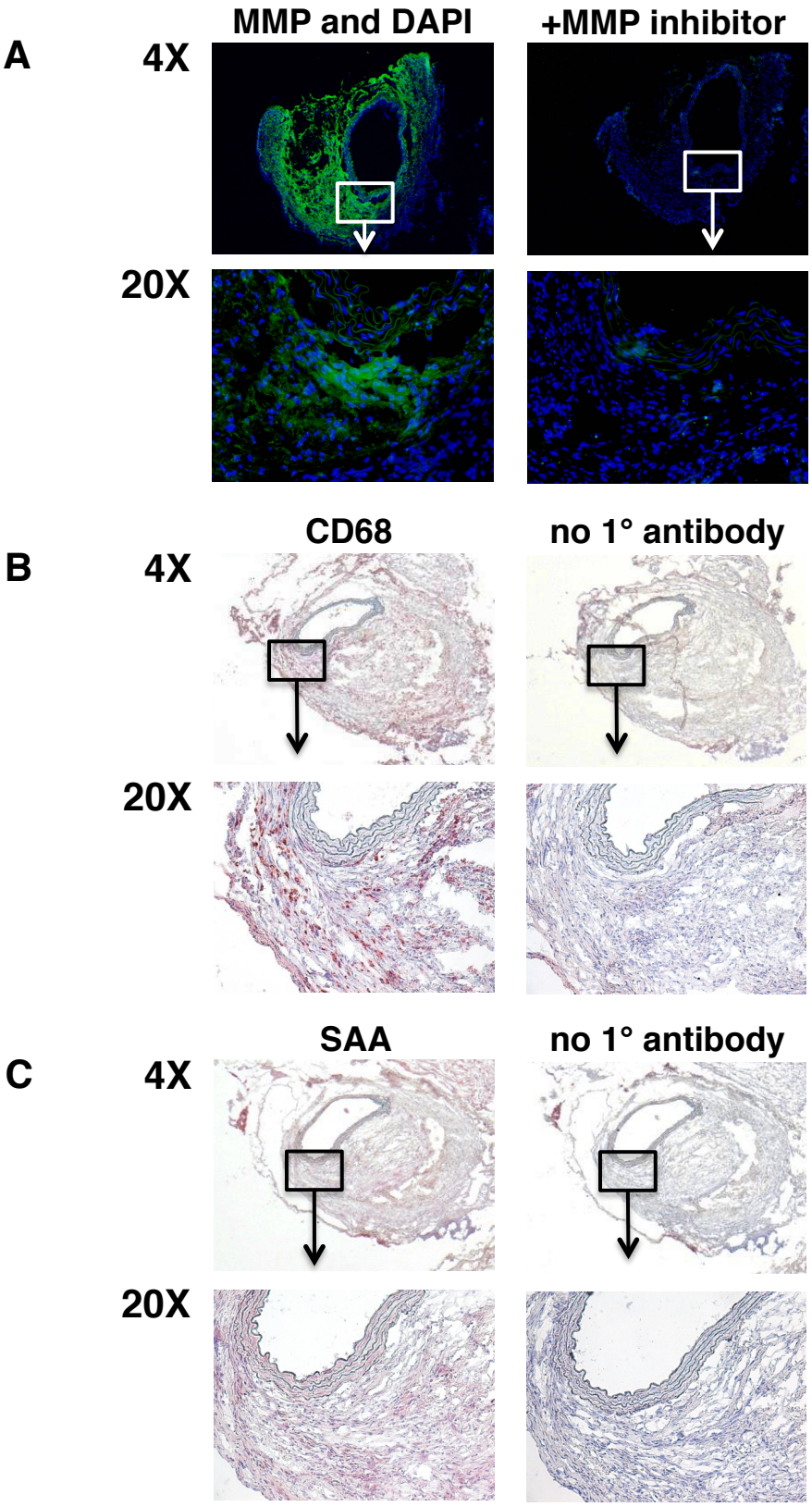
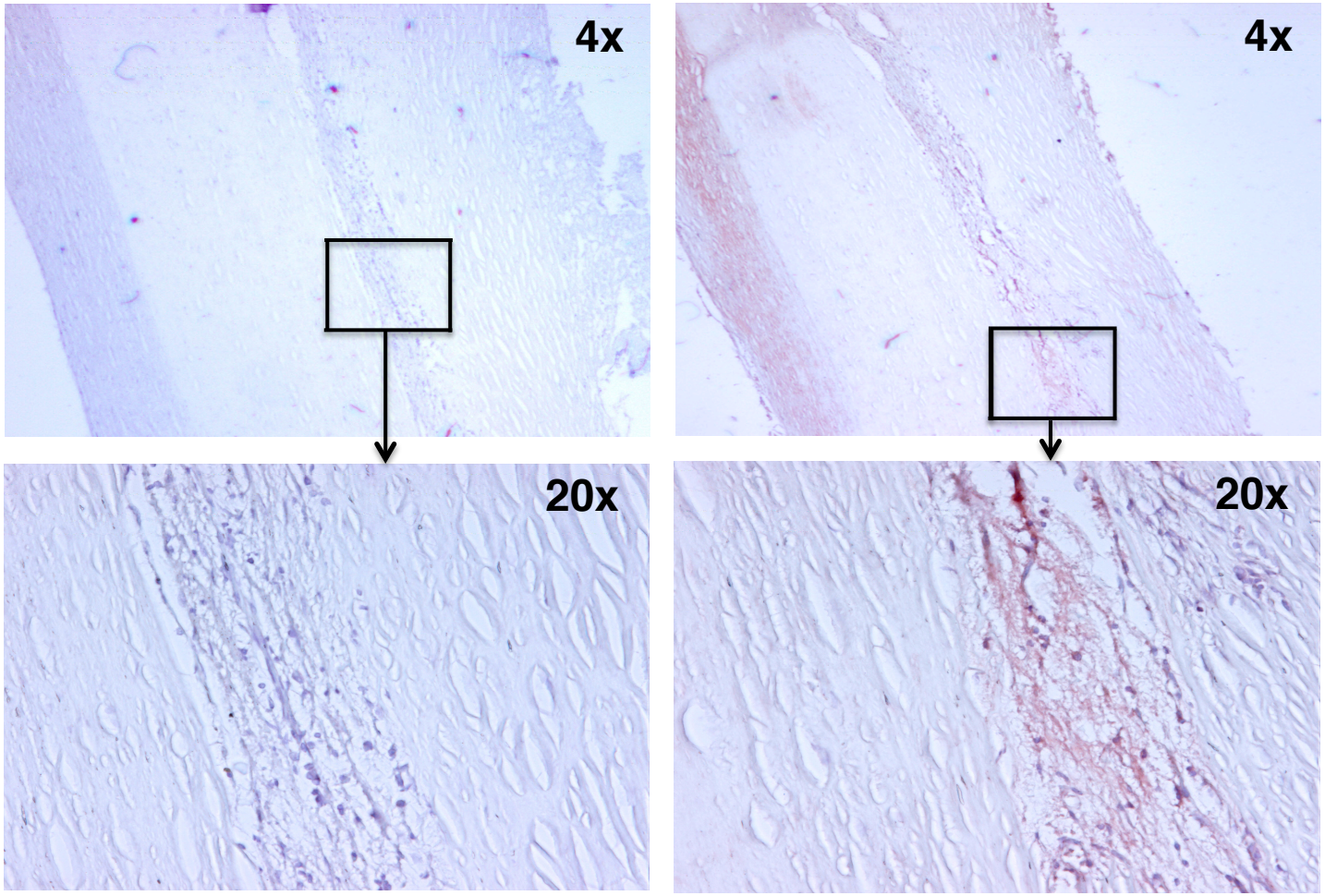
Supplemental Figure I



Supplemental Figure I: Negative controls for MMP activity and antibody staining.
Male apoE$^{-/-}$ mice were infused with 1,000 ng kg^{-1} min^{-1} AngII for 10 days. Adjacent sections from AAA were analyzed in parallel to allow direct comparisons. A) *In situ* zymography demonstrates the lack of fluorescent staining when tissues are processed in the presence of an MMP inhibitor. Autofluorescence of elastin fibers (green fluorescence) is evident in some regions. B,C) Immunostatining demonstrates the lack of red chromogen in the absence of primary antibodies for macrophages and SAA.

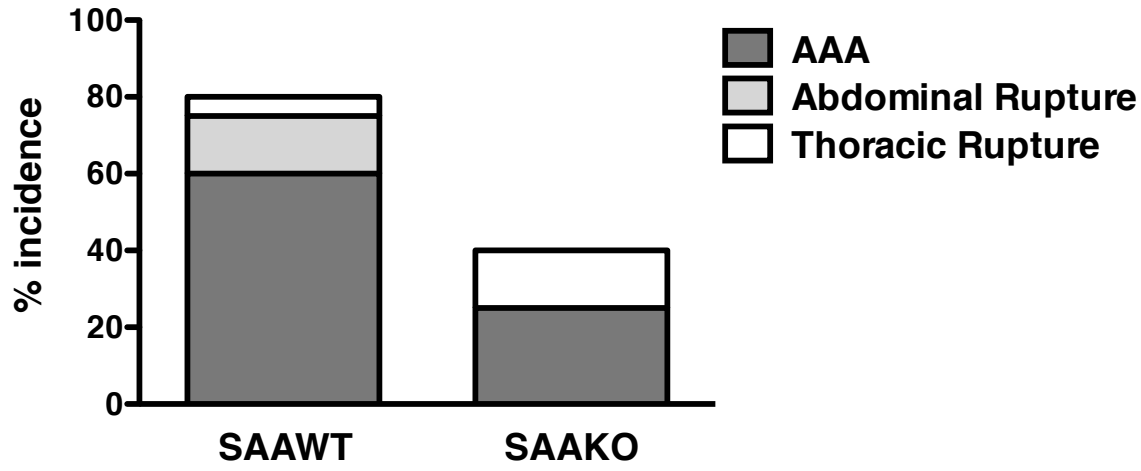
Supplemental Figure II



Supplemental Fig II: SAA is present in human AAA

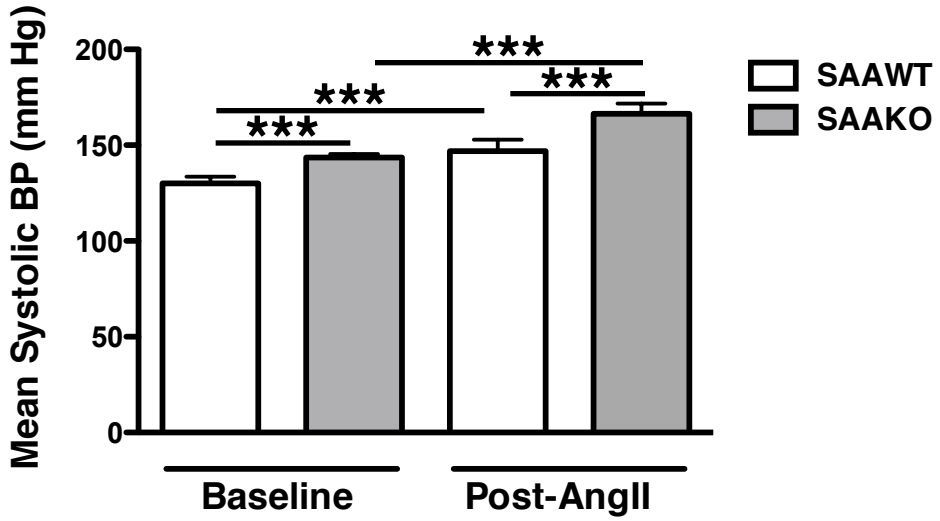
Sections of aortic tissue removed from a patient during surgical repair of AAA were immunostained with anti-human SAA or control antisera. Magnification = 4X and 20X. The presence of SAA is indicated by red staining.

Supplemental Figure III



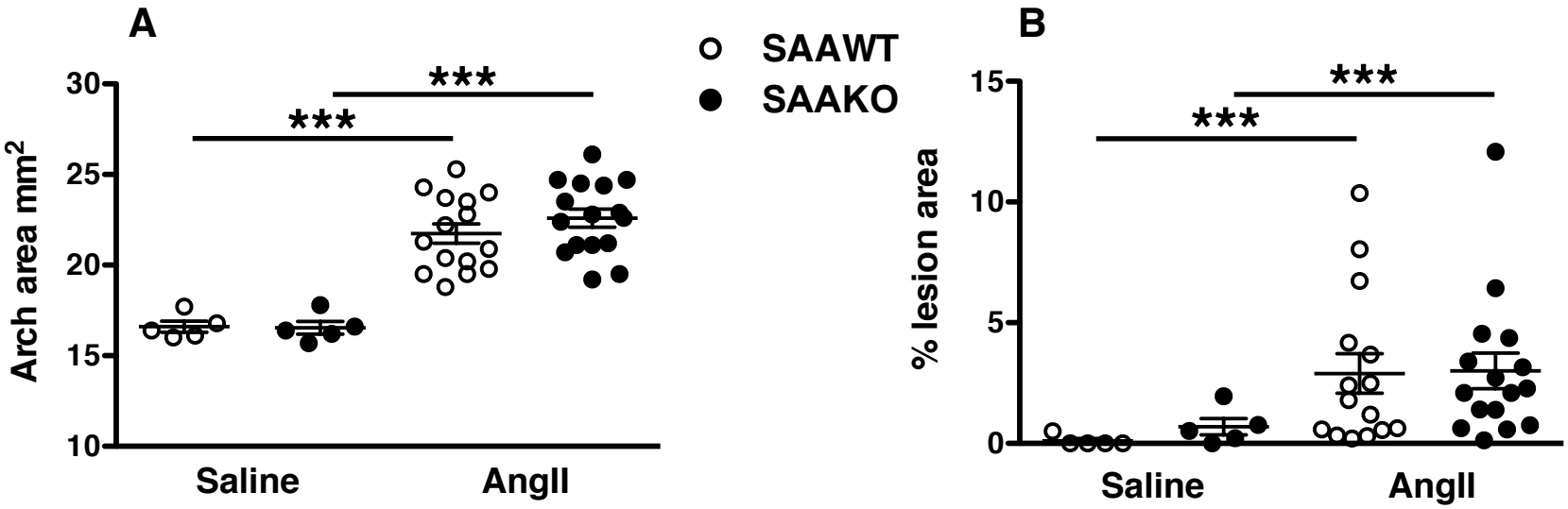
Supplemental Fig III: SAA deficiency reduces the incidence and severity of AngII-induced AAA. Male SAAWT and SAAKO mice were infused with 1,000 ng kg^{-1} min^{-1} AngII for 28 days (n = 20). Mice that died during the study due to a rupture of the abdominal or thoracic aorta are represented by lightly shaded or white sections of the bars, respectively. Mice that developed AAA, defined in surviving mice as a greater than 50% increase in luminal diameter of the abdominal aorta as assessed by *in vivo* US are indicated by darkly shaded region of the bars. *P<0.05

Supplemental Figure IV



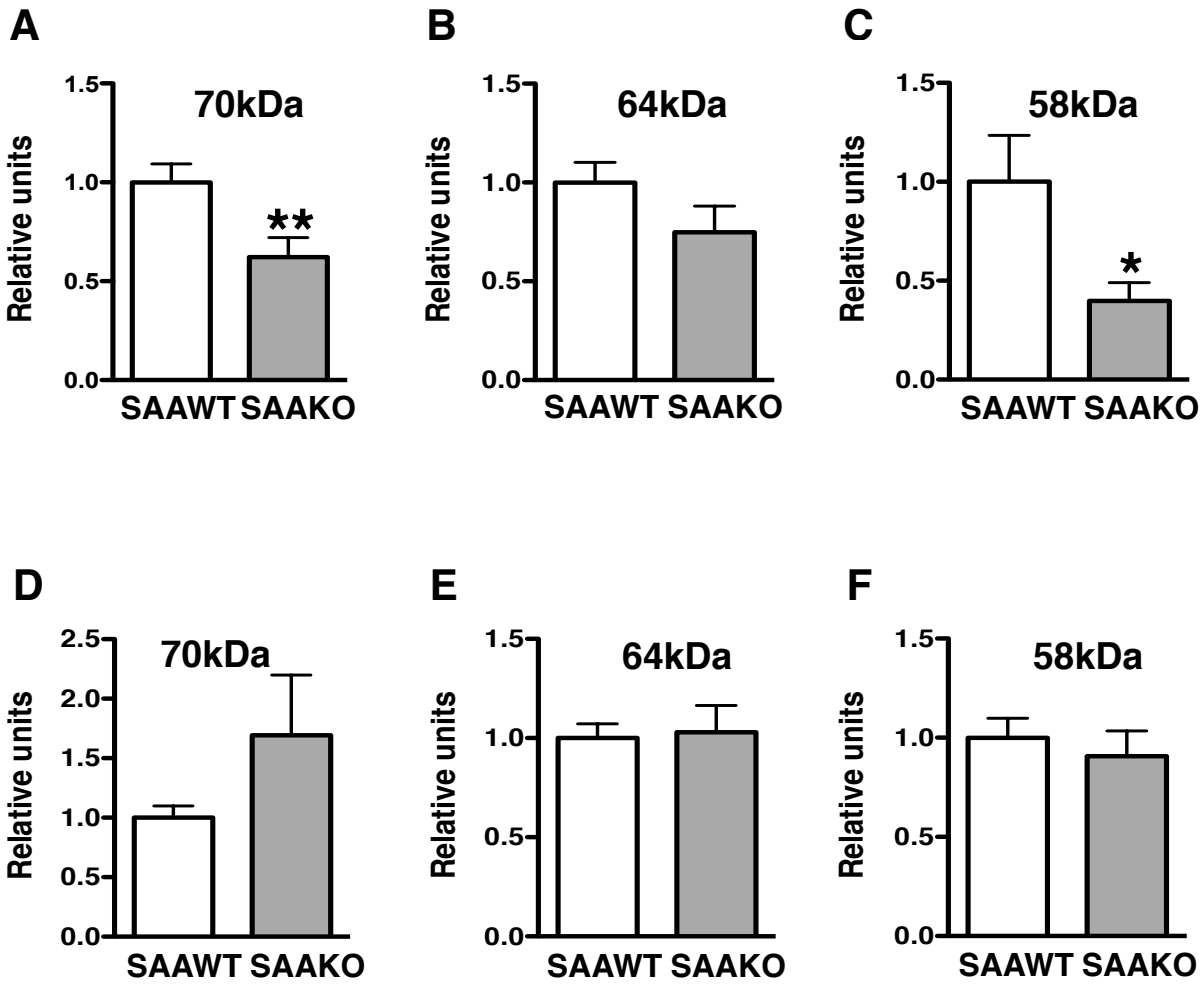
Supplemental Fig IV: SAA deficiency does not attenuate the hypertensive response to AngII. Systolic blood pressure in male SAAWT and SAAKO mice prior to (n = 24 – 25) and after 1,000 ng kg^{-1} min^{-1} AngII infusion (n = 16-17) for 28 days. Systolic blood pressure was measured consecutively for 5 days prior to pump implantation and again consecutively for 5 days prior to termination of experiment. Bars represent the mean ± SEM. ***P<0.001

Supplemental Figure V



Supplemental Fig V: SAA deficiency does not alter AngII-induced dilation or atherosclerosis of the arch region of the ascending AA. Male SAAWT and SAAKO mice were infused with saline or 1,000 ng kg^{-1} min^{-1} AngII for 28 days. A) Luminal surface area of the aortic arch region of saline-infused mice (n = 5) and surviving AngII-infused mice (n = 15 – 16); B) atherosclerosis quantified by *en face* analysis of the luminal surface of the aortic arch region of saline -and AngII-infused mice. Circles represent the values for individual mice. Bars represent the mean +/- SEM. ****P*<0.001

Supplemental Figure VI



Supplemental fig VI: Abdominal aortas, but not thoracic aortas, of AngII-infused SAAKO mice exhibit less MMP-2 activity compared to SAAWT mice. SAAWT and SAAKO mice were infused with 1,000 ng kg^{-1} min^{-1} AngII for 10 days. MMP activity by gel zymography, was evaluated in the abdominal (A-C) and thoracic (D-F) aortas of SAAWT (n = 24) and SAAKO (n = 18) mice to determine the relative abundance of 70kDa; 64kDa and 58 kDa MMP-2 isoforms. Values are the mean ± SEM *P<0.05, **P<0.01.

Supplemental Table I: Primer sequences

Gene	Accession number	Primers
GAPDH	NM_008084	5’-CTCATGACCACAGTCCATGCCA-3’
		5’-GGATGACCTTGCCCACAGCCTT-3’
TNF-α	NM 013693	5’-GGCAGGTCTACTTTGGAGTCATTG-3’
		5’-GTTAGAAGGACACAGACTGG-3’
MCP-1	NM 011333.3	5’-TTCCTCCACCACCATGCAG-3’
		5’-CCAGCCGGCAACTGTGA-3’
MMP-9	NM013599	5’-CAATCCTTGCAATGTGGATG-3’
		5’-AGTAAGGAAGGGGCCCTGTA-3’
MMP-13	BC 125320	5’-GCCATTTCATGCTTCCTGAT-3’
		5’-TTTTGGGATGCTTAGGGTTG-3’
MMP-2	NM 008610	5’-ACACTGGGACCTGTCACTCC-3’
		5’-TGTCACTGTCCGCCAAATAA-3’
SAA	NM_009117	5’-GCCATGGAGGGTTTTTTTCATTTGTT-3’
		5’-GAGCATGGAAGTATTTGTCTGAG-3’
MMP-12	NM_008605	5’-CATGAAGCGTGAGGATGTAGAC-3’
		5’-TGGGCTAGTGTACCACCTTTG-3’
TGF-β	NM_009367	5’-CTTCGACGTGACAGACGCT-3’
		5’-GCAGGGGCAGTGTAAACTTATT-3’