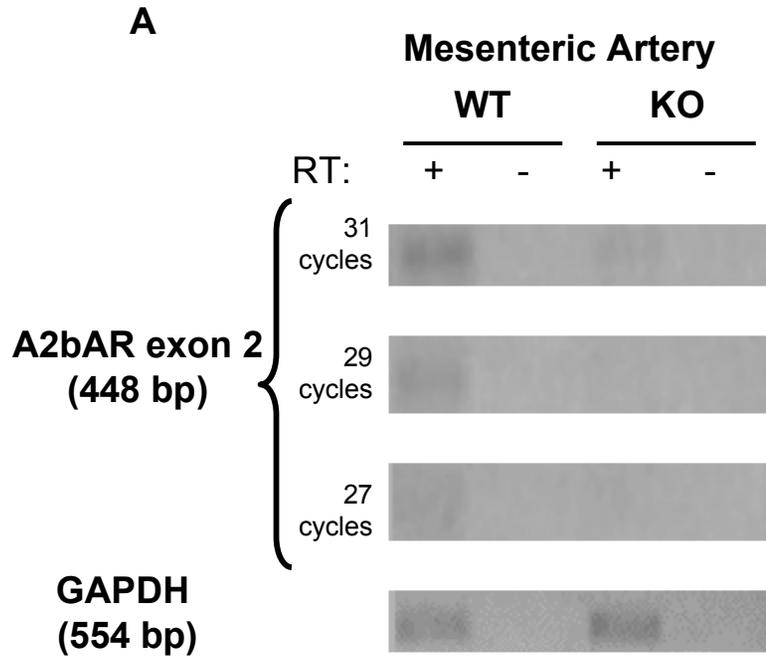
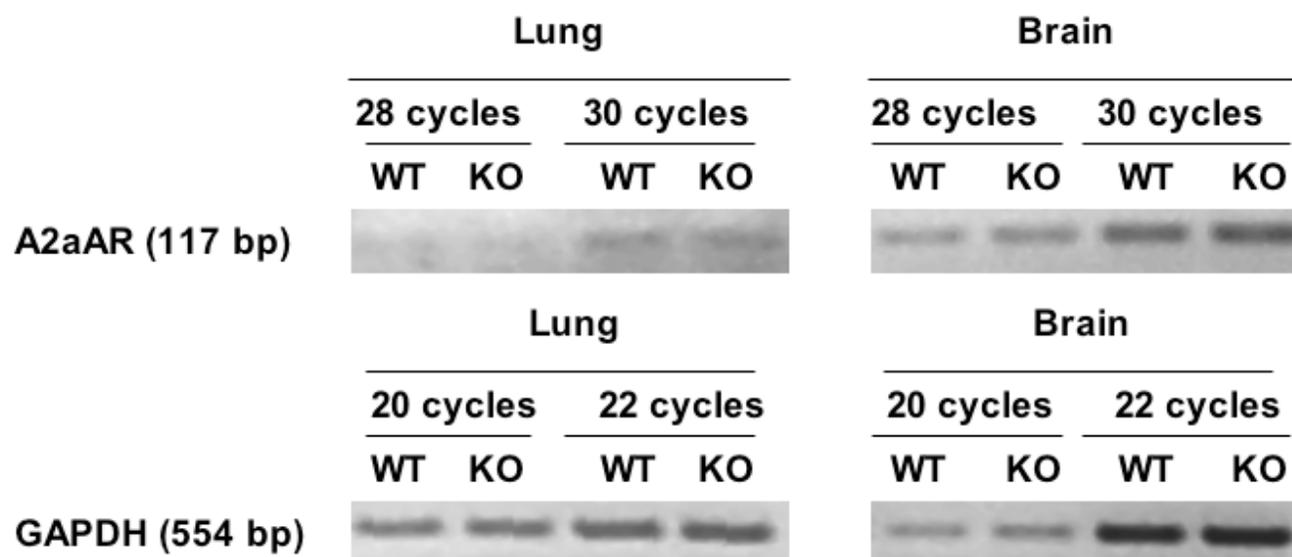


# Supplementary Figure 1



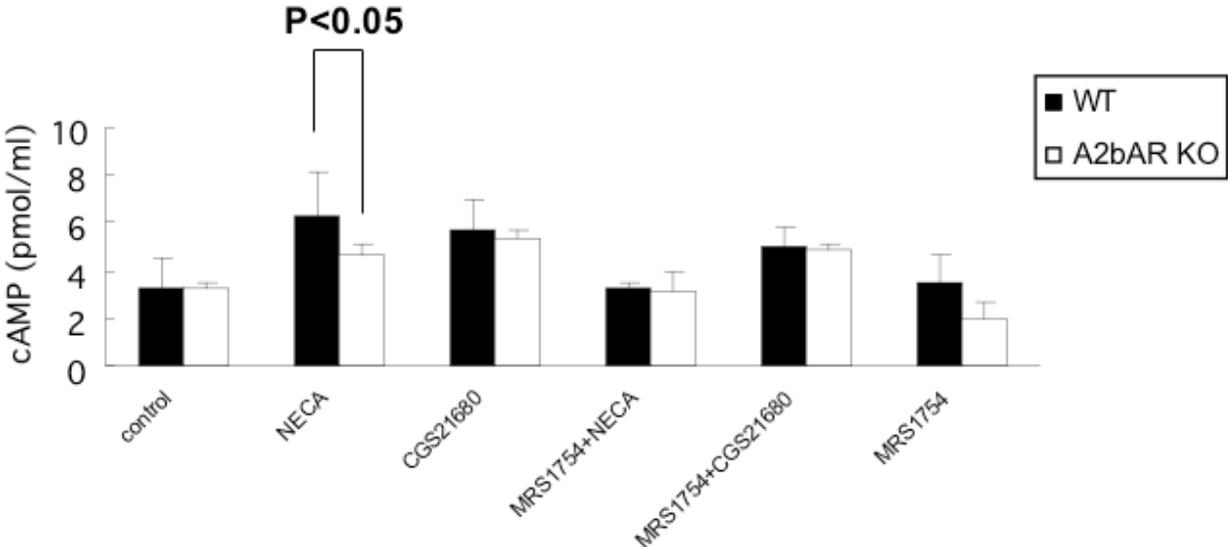
# Supplementary Figure 1

B



Supplementary Figure 1

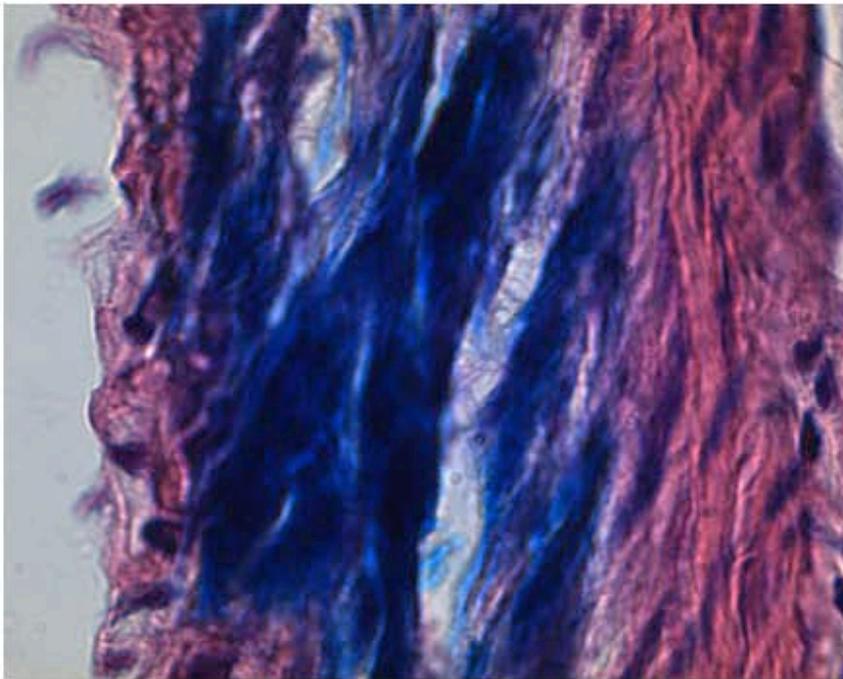
C



**Supplementary Figure 2**

**A**

**Aortic arch ( 1000X )**



**B**

**Mesenteric artery ( 1000X )**

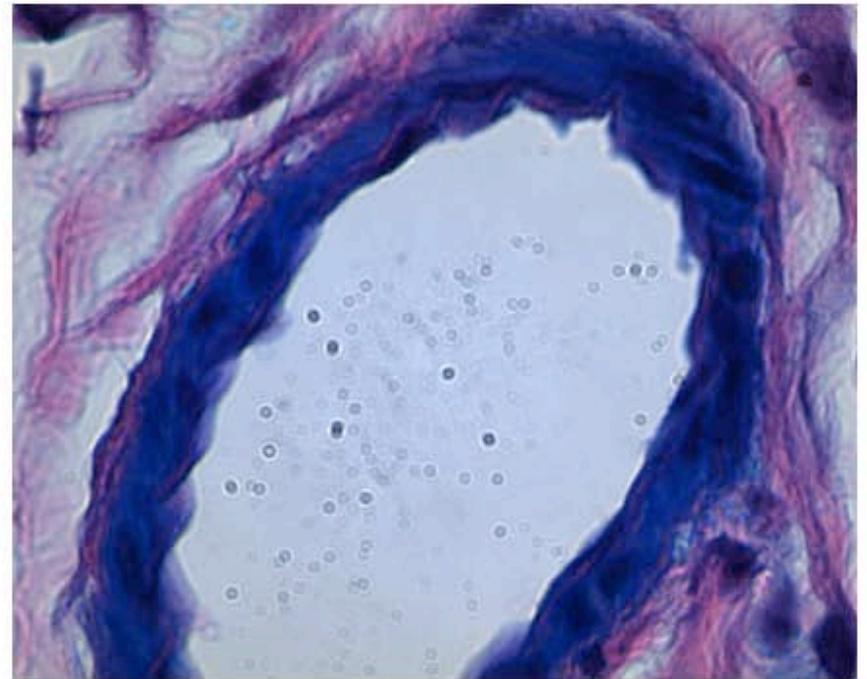
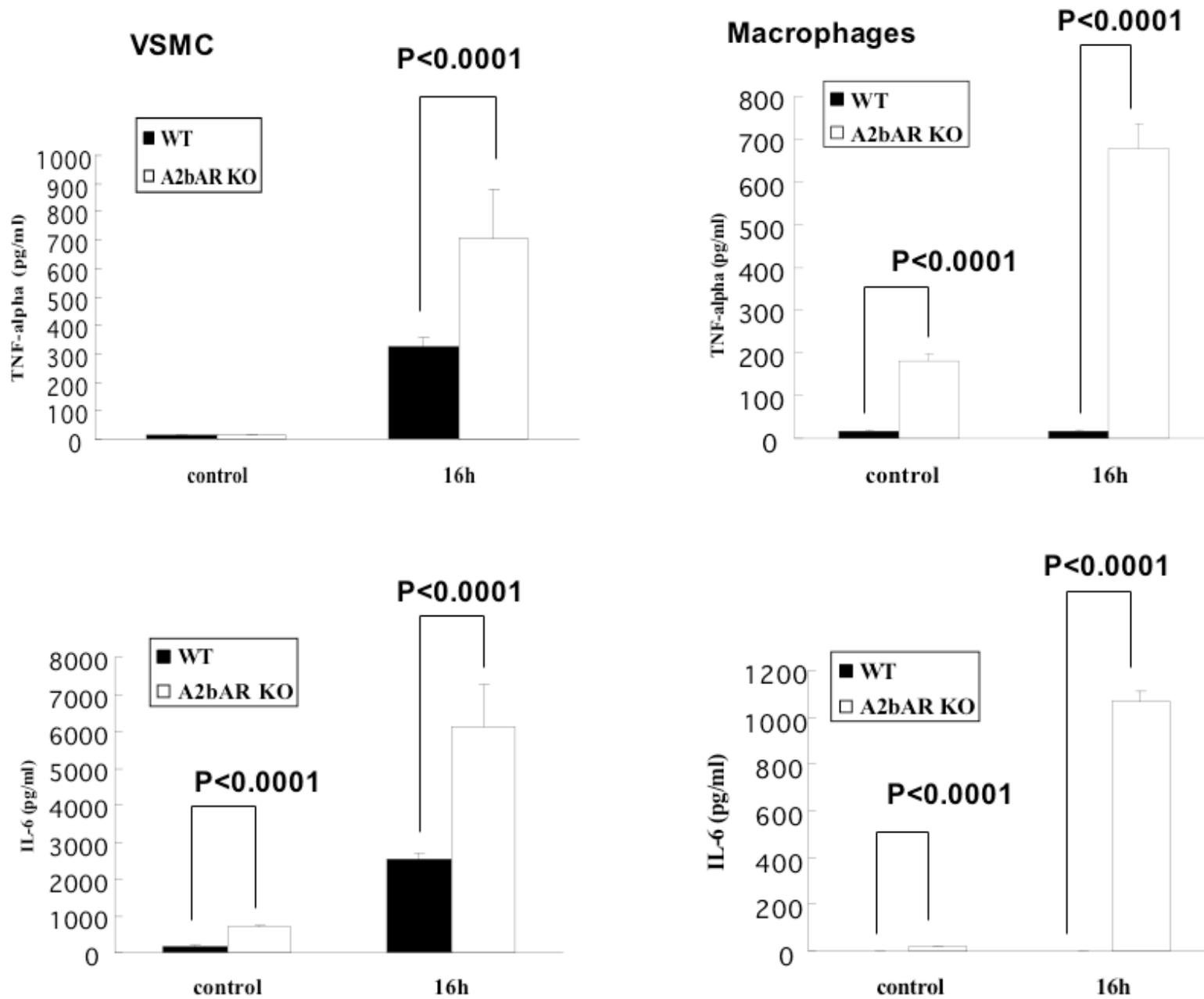
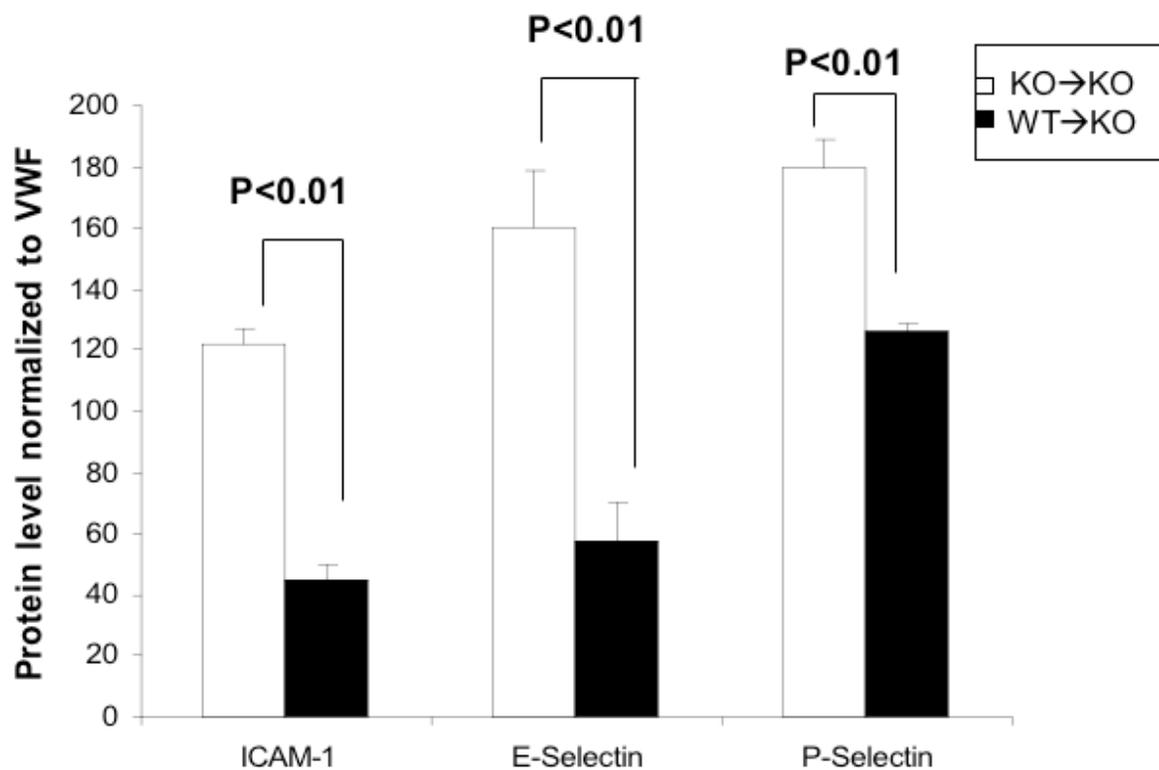
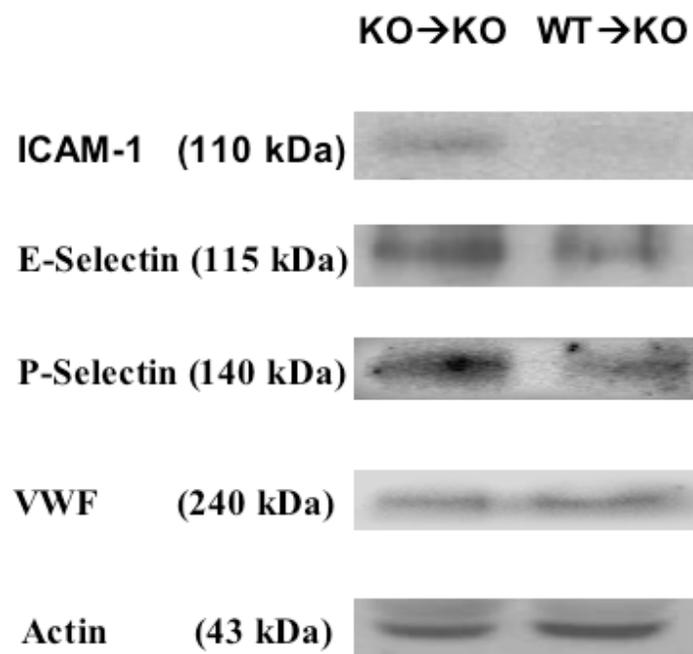


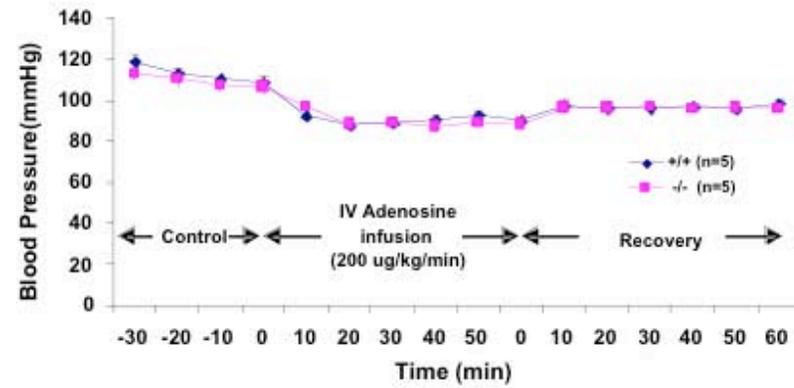
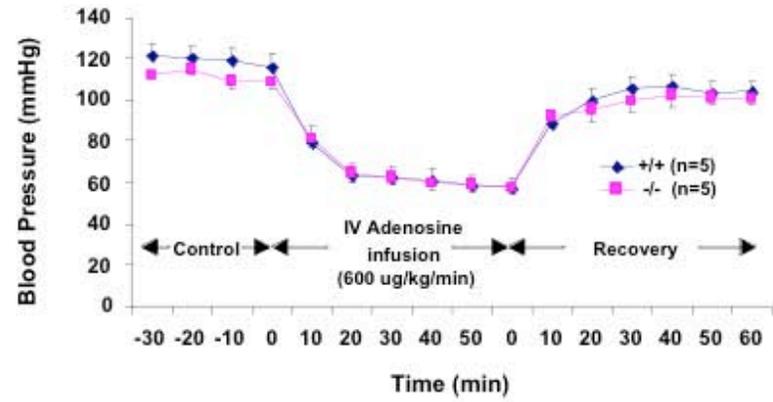
Figure 3 (supplementary)



Supplementary Figure 4



Supplementary Figure 5



**Table 1 (supplement)**

	<b>WBC</b> <b>(10<sup>3</sup>/μl)</b>	<b>RBC</b> <b>(10<sup>6</sup>/μl)</b>	<b>platelets</b> <b>(10<sup>3</sup>/μl)</b>
<b>WT ( n=15 )</b>	<b>0.38±0.17</b>	<b>8.96±1</b>	<b>1183±289</b>
<b>A2bAR KO ( n=15 )</b>	<b>0.34±0.19</b>	<b>8.82±1.3</b>	<b>1040±292</b>
<b>P</b>	<b>&gt; 0.05</b>	<b>&gt; 0.05</b>	<b>&gt; 0.05</b>

**Table 2 (supplement)****Hemodynamic characteristics of venules**

<b>Genotype</b>	<b>Venule Diameter (<math>\mu\text{m}</math>)</b>	<b>Centerline velocity (mm/s)</b>	<b>Shear rate (<math>\text{s}^{-1}</math>)</b>
<b>Large venule</b>			
<b>WT (n=9)</b>	<b>229.50<math>\pm</math>20.83</b>	<b>5.26 <math>\pm</math>0.20</b>	<b>122.55 <math>\pm</math>11.37</b>
<b>KO (n=8)</b>	<b>234.72 <math>\pm</math>34.14</b>	<b>5.67 <math>\pm</math>0.93</b>	<b>122.60 <math>\pm</math>13.15</b>
<b>Micro venule</b>			
<b>WT (n=4)</b>	<b>28.32 <math>\pm</math>5.13</b>	<b>1.53 <math>\pm</math>0.30</b>	<b>266.68 <math>\pm</math>23.68</b>
<b>KO (n=5)</b>	<b>33.20 <math>\pm</math>2.52</b>	<b>1.5 <math>\pm</math>0.16</b>	<b>234.16 <math>\pm</math>38.39</b>

**Table 3 (supplement)**

**Tail-cuff systolic blood pressure and heart rate measurements in WT, A2bAR KO male and female mice**

<b>Group</b>	<b>WT (n=17)</b>		<b>A2bAR KO (n=17)</b>	
	<b>Male (n=7)</b>	<b>Female (n=10)</b>	<b>Male (n=7)</b>	<b>Female (n=10)</b>
<b>BP(mmHg)</b>	<b>108 ± 1.56</b>	<b>111 ± 1.10</b>	<b>108 ± 2.00</b>	<b>108 ± 1.50</b>
<b>HR (beats/min)</b>	<b>654 ± 14.7</b>	<b>642 ± 8.9</b>	<b>640 ± 16.6</b>	<b>670 ± 18.2</b>

**all data are expressed as mean ± S.E.M. BP, blood pressure; HR, heart rate.**

**Supplementary Figure 1.** Expression of and activities of A2bAR and A2aAR in A2bAR KO mice. **A.** RT-PCR of A2bAR exon 2. RNA was prepared from the mesenteric artery or kidney, reverse transcribed and amplified by PCR as detailed under Methods. GAPDH amplification was used as a control. Reverse transcriptase (RT) was omitted in some samples to measure amplification of residual genomic DNA in the preparation. Low level amplification was noted in the KO mice under high PCR cycles compared to WT. Similar results were obtained with kidney RNA. This minute level of RNA in the KO mice is not expected to be translated to an active protein for reasons detailed under Methods (first paragraph). Direct measurement of A2bAR activity is also described in Figure 1 in the paper and here in panel C. **B.** RT-PCR of A2aAR in different cells. Details of amplification are as above and as described under Methods. **C.** Activity of the A2bAR and A2aAR in peritoneal macrophages derived from WT and A2bAR KO mice. Since macrophages are known to be activated by A2 adenosine receptor signaling, we also measured A2a and A2b AR activities in these cells (in addition to VSMC shown in Figure 1 in the paper). cAMP was determined in cells derived from WT or KO mice. Measurements were pursued after a 10-minute treatment with vehicle or with 5 $\mu$ M NECA (which stimulates both the A2aAR and A2bAR), or with 1 $\mu$ M CGS21680 (selective for A2aAR) in presence or absence of the A2bAR antagonist MRS1754 (at 5  $\mu$ M, preincubated for 10 minutes). Data shown are averages  $\pm$  SD for 3 experiments with duplicates. It is clear that A2aAR activation is similar in the A2bAR KO mice and WT mice.

**Supplementary Figure 2.** Histological examination of arteries from A2bAR KO and WT mice. **A.** Viewing at high magnification of  $\beta$ -Gal expression in aortic arch tissue

sections. **B.** Viewing at high magnification of  $\beta$ -Gal expression in mesenteric tissue sections. Tissues were prepared and stained from  $\beta$ -Gal as detailed under Methods. The magnification used is 1000x. The expression in smooth muscles is clear and distinct, although quite patchy.

**Supplementary Figure 3.** Primary cultures of vascular smooth muscle cells (VSMC) or peritoneal macrophages release cytokines. Cells derived from WT or KO mice were examined at base line (control) or 16 hours after treatment with LPS (30  $\mu$ g/ml). Results are averages +/- standard deviations of 4 measurements.

**Supplementary Figure 4.** Western blot analysis of samples derived from transplanted mice. Samples from KO bone marrow to KO mice were compared to WT bone marrow to KO mice. Methods are as the ones described for Figure 4 in the paper.

**Supplementary Figure 5. Blood pressure and heart rates are not significantly altered in the A2bAR compared to wild type mice. A,B.** Blood pressure changes in A2bAR WT or KO mice during intravenous (IV) adenosine infusion.