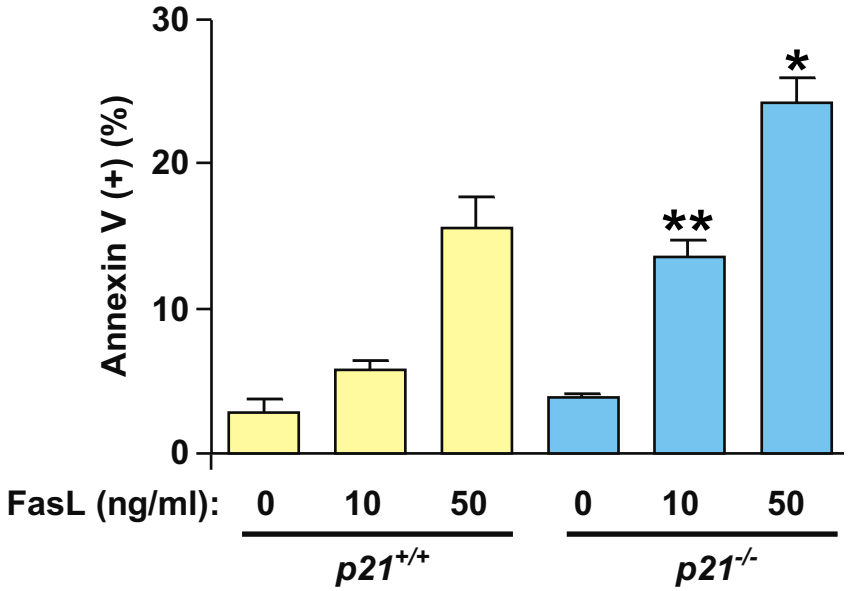
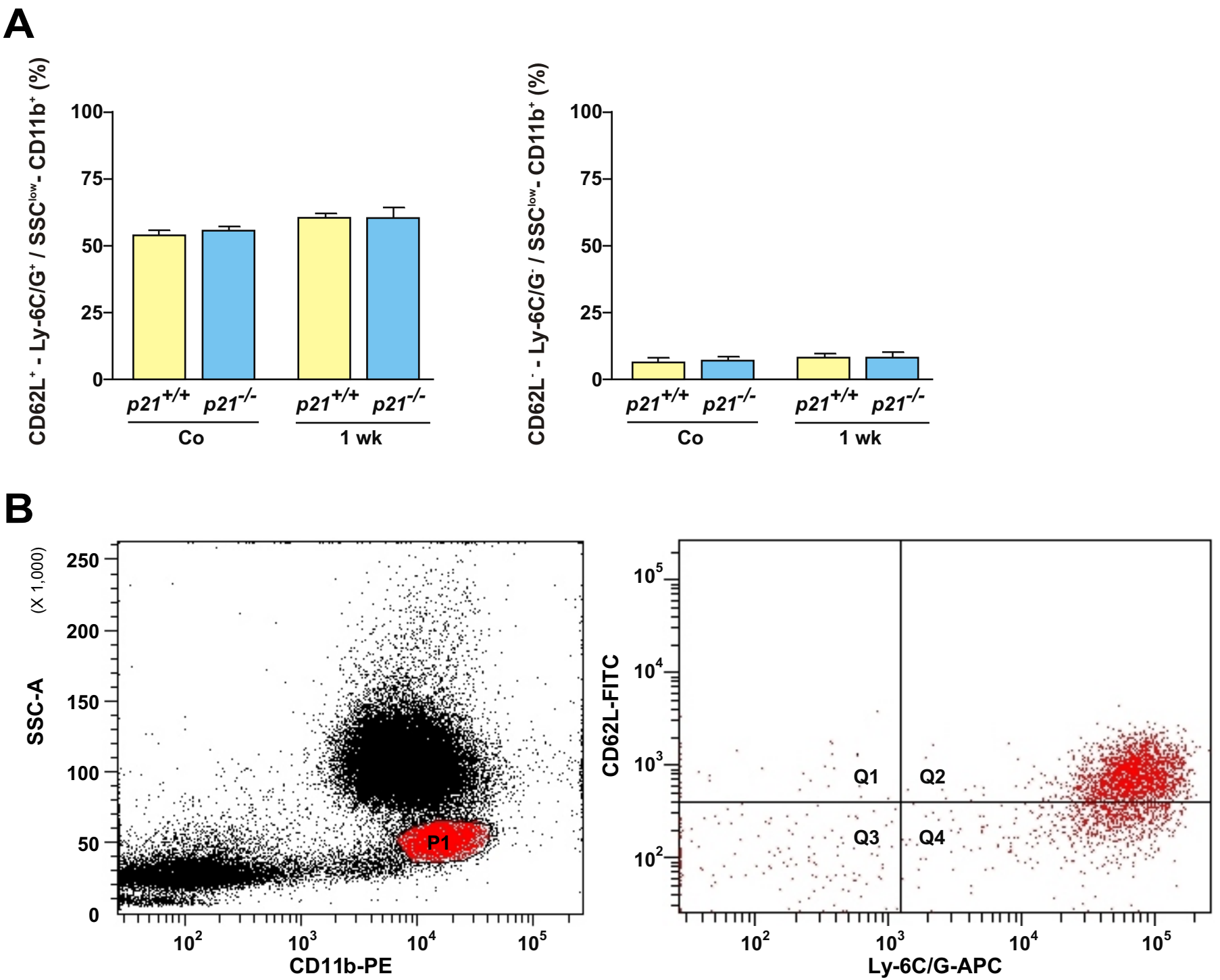
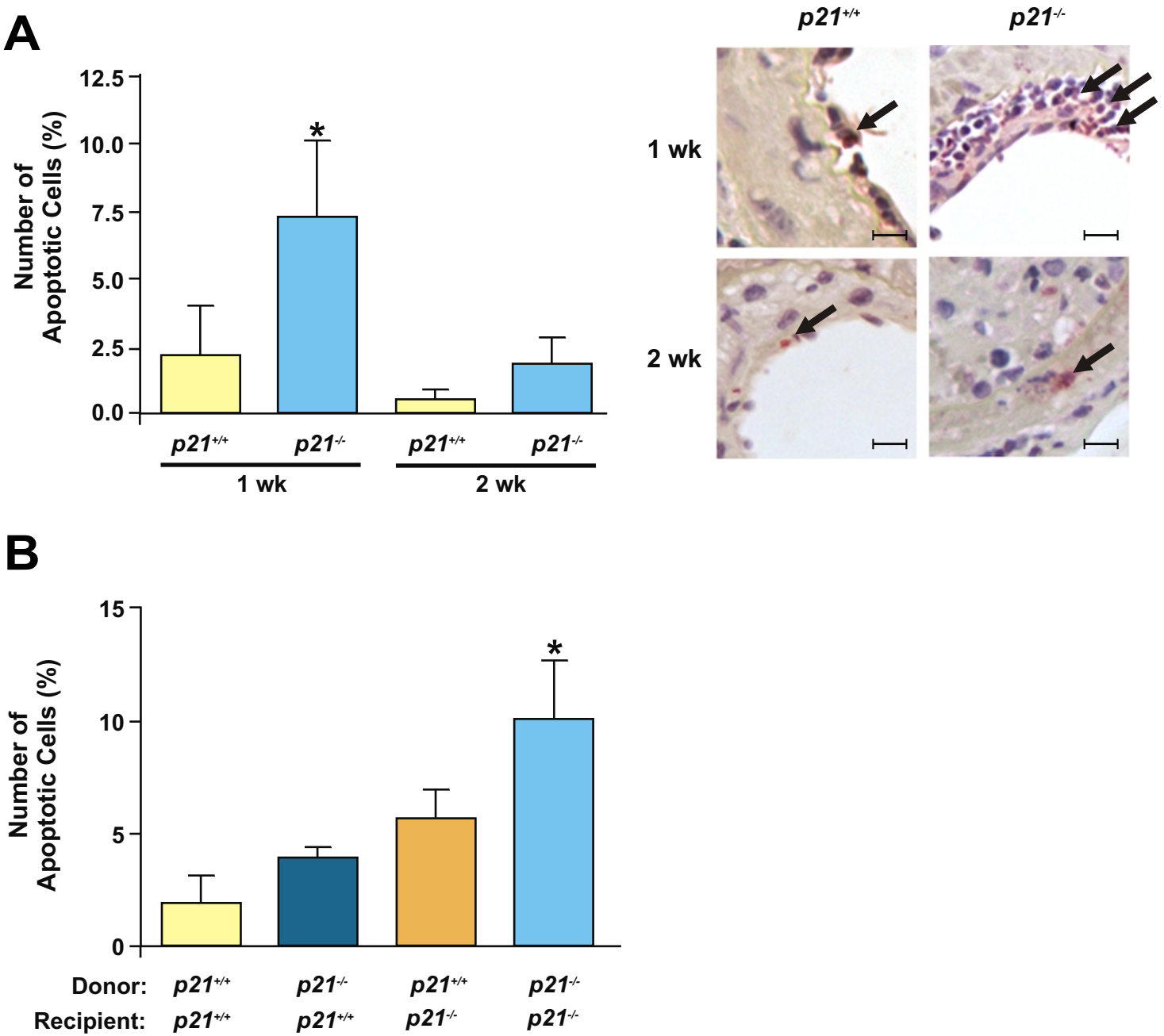
Supplemental Fig 1



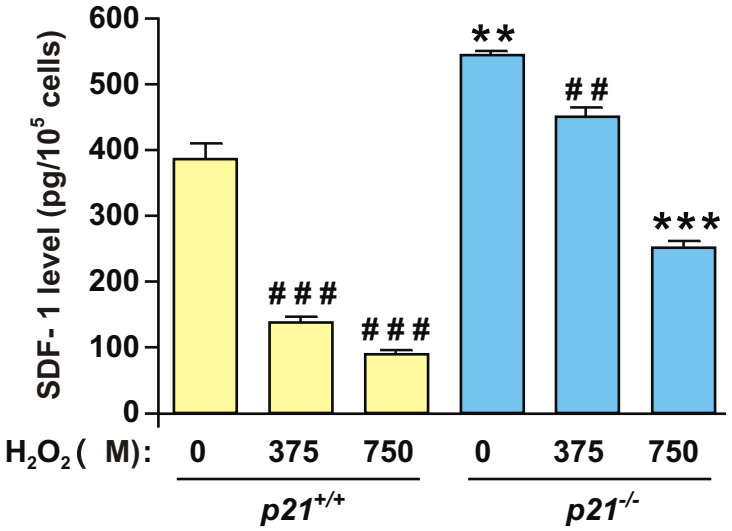
Supplemental Fig 2



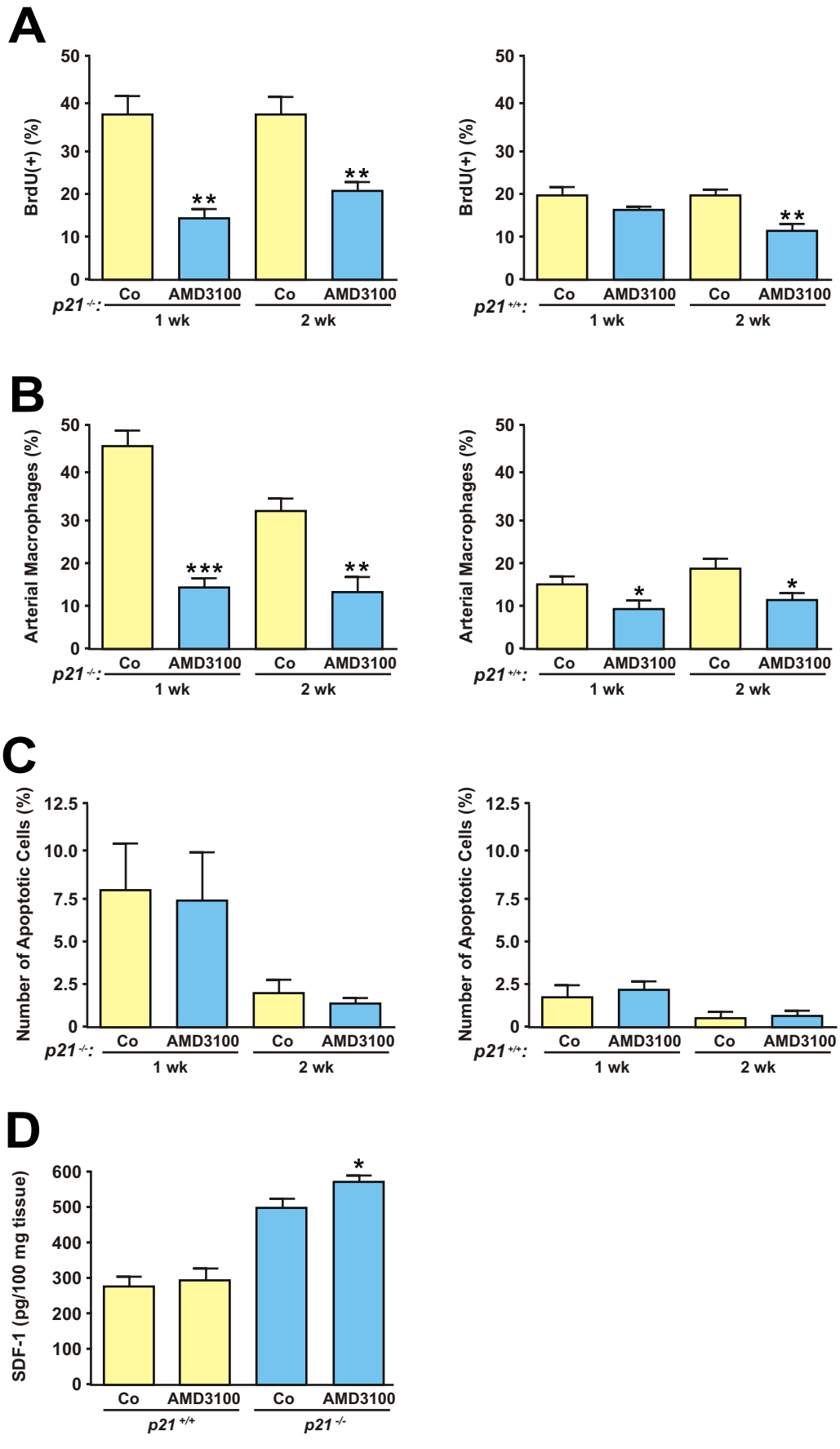
Supplemental Fig 3



Supplemental Fig 4



Supplemental Fig 5



Supplemental Figure legends:

Supplemental Figure 1: Accelerated apoptosis in *p21*$^{-/-}$ VSMCs in vitro. *p21*$^{-/-}$ VSMCs in culture (blue bars) displayed increased apoptosis compared to *p21*$^{+/+}$ VSMCs (yellow bars) with in vitro administration of 0, 10 or 50 ng / ml of FasL (n=3;*P < 0.05, **P < 0.01 for comparison of *p21*$^{-/-}$ to *p21*$^{+/+}$ at identical FasL doses).

Supplemental Figure 2: Immunophenotyping of peripheral blood before and after wire vascular injury in *p21*$^{+/+}$ and *p21*$^{-/-}$ mice. **(A)** Peripheral blood leukocytes were isolated and stained with CD11b-PE, Ly-6C/G-APC and CD62L-FITC and the percentages of Ly-6C/G^{+}/CD62L^{+} and Ly-6C/G^{-}/CD62L^{-} monocytes were determined (n=3). **(B)** Representative dot blots. Left: acquisition of the side scatter-A (SSC-A) vs. CD11b-PE. P1 represents the gated CD11b-PE positive SSC-A low cells. Right: acquisition of Ly-6C/G-APC and CD62L-FITC cells present in the CD11b-PE population. No differences were observed between *p21*$^{+/+}$ and *p21*$^{-/-}$ mice.

Supplemental Figure 3: **Cell apoptosis in injured arteries from *p21*$^{+/+}$ and *p21*$^{-/-}$ mice.** (**A**) Left panel: percentage of apoptotic cells in arteries from *p21*$^{+/+}$ and *p21*$^{-/-}$ mice 1 and 2 weeks after vascular injury (n=5; *P < 0.05 *p21*$^{+/+}$ vs. *p21*$^{-/-}$ 1 wk). Right panel: TUNEL staining of the arteries following vascular injury in *p21*$^{-/-}$ and *p21*$^{+/+}$ mice 1 and 2 weeks after injury. Arrows indicate apoptotic cells. Scale bar = 7 μm. (**B**) One week after femoral artery wire injury, the number of TUNEL positive cells was decreased in arterial tissue from *p21*$^{+/+}$ mice receiving *p21*$^{+/+}$ BM compared to *p21*$^{-/-}$ mice receiving

$p21^{-/-}$ BM (n=5; *P < 0.05). No other between group comparisons reached statistical significance.

Supplement Figure 4: SDF-1 levels are decreased in $p21^{-/-}$ and $p21^{+/+}$ VSMCs upon oxidative stress. Increased SDF-1 levels were observed in $p21^{-/-}$ compared to $p21^{+/+}$ VSMCs prior to oxidative stress. The induction of oxidative stress, with 375 or 750 μM of H_2O_2, caused a reduction in SDF-1 levels in both $p21^{-/-}$ (blue bars) and $p21^{+/+}$ (yellow bars) VSMCs (n=3; **P < 0.01 vs. $p21^{+/+}$ 0 μM, $^{\#\#}P$ < 0.01 vs. $p21^{-/-}$ 0 μM,***P < 0.001 vs. $p21^{-/-}$ 0 μM, $^{\#\#\#}P$ < 0.001 vs. $p21^{+/+}$ 0 μM).

Supplement Figure 5: SDF-1 inhibition induced by AMD3100 administration prevents excessive proliferation during vascular wound repair. (A) AMD3100 treatment decreased cellular proliferation as assessed by BrdU incorporation at 1 and 2 weeks after vascular injury in $p21^{-/-}$ arteries and at 2 weeks after vascular injury in $p21^{+/+}$ arteries (left: n=4; **P < 0.01 vs. $p21^{-/-}$ 1 wk; **P < 0.01 vs. $p21^{-/-}$ 2 wk), (right: n=4; **P < 0.01 vs. $p21^{+/+}$ 2 wk). **(B)** AMD3100 treatment reduced the number of local arterial macrophages in $p21^{-/-}$ and $p21^{+/+}$ arteries at 1 and 2 weeks after vascular injury (left: n=5; ***P < 0.001 vs. $p21^{-/-}$ 1 wk; **P < 0.01 vs. $p21^{-/-}$ 2 wk), (right: n=10; *P < 0.05 vs. $p21^{+/+}$ 1 wk; *P < 0.05 vs. $p21^{+/+}$ 2 wk). **(C)** The number of local apoptotic TUNEL positive cells after vascular injury was unchanged after treatment with AMD3100. **(D)** One week after vascular injury, $p21^{-/-}$ mice treated with AMD3100 displayed marginally increased local vascular SDF-1 levels compared to control $p21^{-/-}$

mice (n=5; *P < 0.05 vs. Co $p21^{-/-}$). AMD3100 administration did not influence SDF-1 tissue levels in $p21^{+/+}$ mice.

Supplemental Table 1: Peripheral blood cell analysis of *p21*$^{+/+}$ and *p21*$^{-/-}$ mice before and after wire injury. Blood was collected from non-injured mice (Co) and 1 week following wire injury and analyzed using a Cell-dyne 3500 hematology analyzer (*n*=3).

	p21$^{+/+}$		*p21*$^{-/-}$	
	Co	1 wk	Co	1 wk
Monocytes/mm^3 Monocytes (%)	191.6 ± 47.7 4.8 ± 1.3	169.2 ± 45.7 4.2 ± 1.2	233.8 ± 55.7 5.9 ± 1.6	294 ± 94.7 5.2 ± 1.5
Neutrophils/mm^3 Neutrophils (%)	422.4 ± 108.9 10.4 ± 2.8	1362.0 ± 250.0 35.6 ± 6.7	664.6 ± 73.4 16.3 ± 1.8	1656.0 ± 202.3 42.1 ± 5.9
Lymphocytes/mm^3 Lymphocytes (%)	2538.0 ± 348.8 61.8 ± 9.1	3164.0 ± 130.9 59.3 ± 6.9	2462.0 ± 277.2 76.8 ± 3.3	2173.0 ± 243.3 52.4 ± 5.6

Supplemental Methods

Primer Sequences:

mp21^{Cip1}: forward 5'-GAC CTG GGA GGG GAC AAG AG-3'
reverse 5'- TTC TCT TGC AGA AGA CCA ATC-3'

mp53: forward 5'-TGA GGT TCG TGT TTG TGC CTG-3'
reverse 5'-GGT AGC TGG AGT GAG CCC TGC-3'

mSDF-1: forward 5'-ACA CTC CAA ACT GTG CCC TTC AGA-3'
reverse 5'-ATG CTG GCA AAC CTT AGC ATG ACC-3'

18S: forward 5'-TTT CGG AAC TGA GGC CAT GA-3'
reverse 5'-GCA AAT GCT TTC GCT CTG GTC-3'

ChIP mSDF-1 promoter STAT3 binding site:
forward 5'-ACC TGT TTG GTC TCT TTG CTC GGT-3'
reverse 5'-CTG TCA AAG GCA CAA GCC GTG AAA-3'

β-actin genomic control (ChIP):
forward 5'-TTT CCC TGA GCA GCT TGT CA-3'
reverse 5'-CTG GGC CGT TAG CTA GTG TC-3'