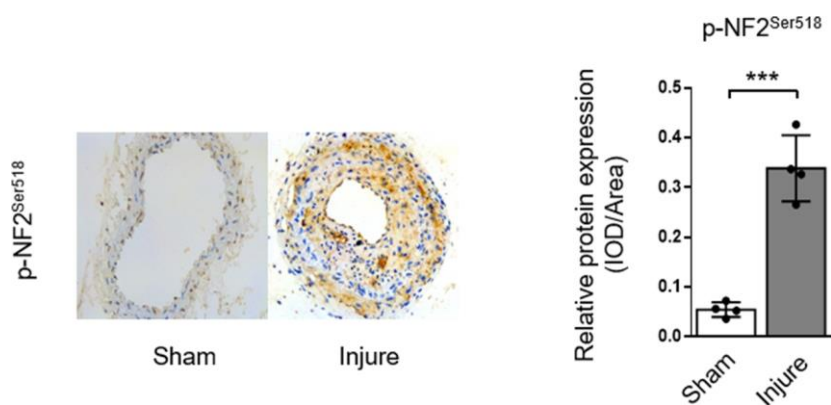
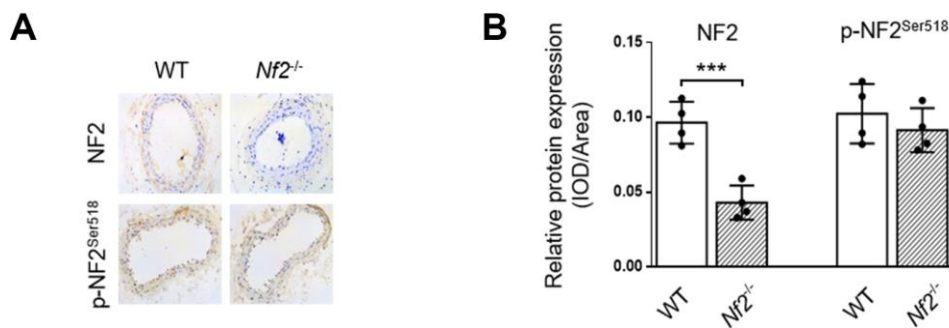


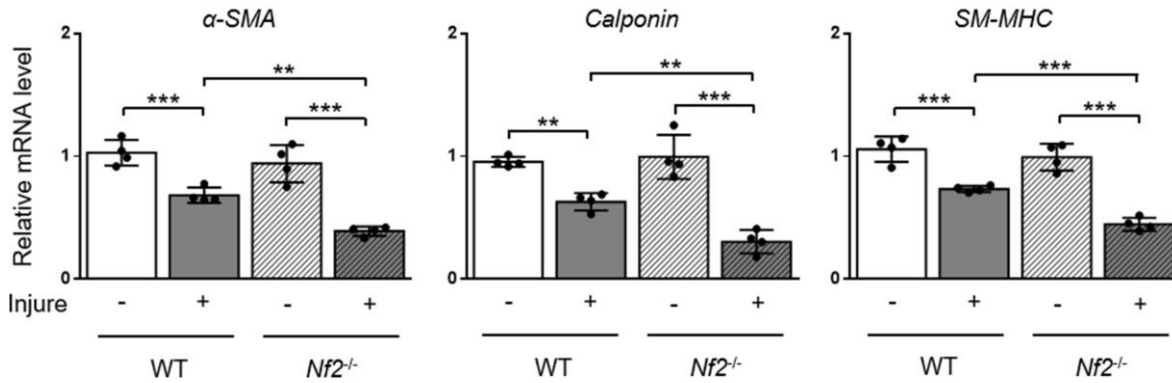
## SUPPLEMENTARY FIGURES



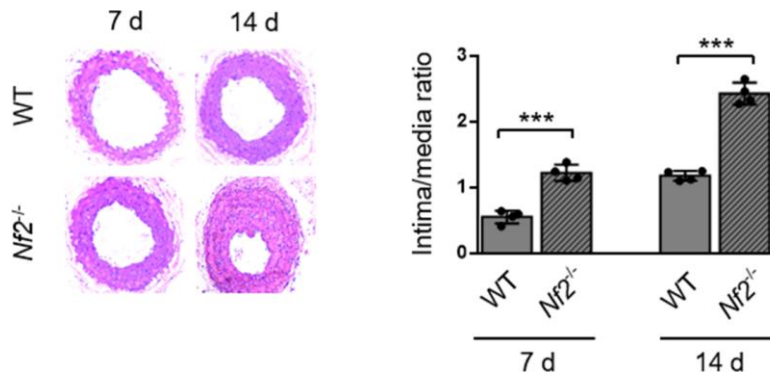
**Supplementary Figure 1. Vascular injury induces enhanced phosphorylation of NF2 in neointima.** The relative protein expression of p-NF2<sup>Ser518</sup> by immunohistochemistry in carotid artery of mice at day 28 after sham operation or wire injury (left) and corresponding quantification (right) were shown (n=4). Magnification 200×. Data are shown as mean ± S.D. \*\*\**P*<0.001 denote statistical comparison between the two marked groups, respectively.



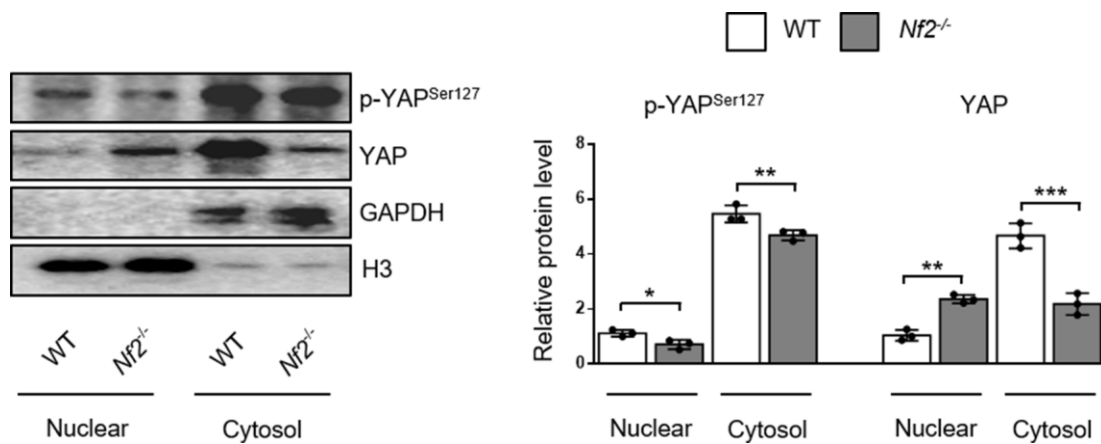
**Supplementary Figure 2. The relative protein expression levels of NF2 and p-NF2<sup>Ser518</sup> by immunohistochemistry in WT or *Nf2*<sup>-/-</sup> mice at the age of 10 weeks.** The representative picture (A) and corresponding quantification (B) for NF2 and p-NF2<sup>Ser518</sup> were shown (n=4). Magnification 200×. Data are shown as mean ± S.D. \*\**P*<0.01 and \*\*\**P*<0.001 denote statistical comparison between the two marked groups, respectively.



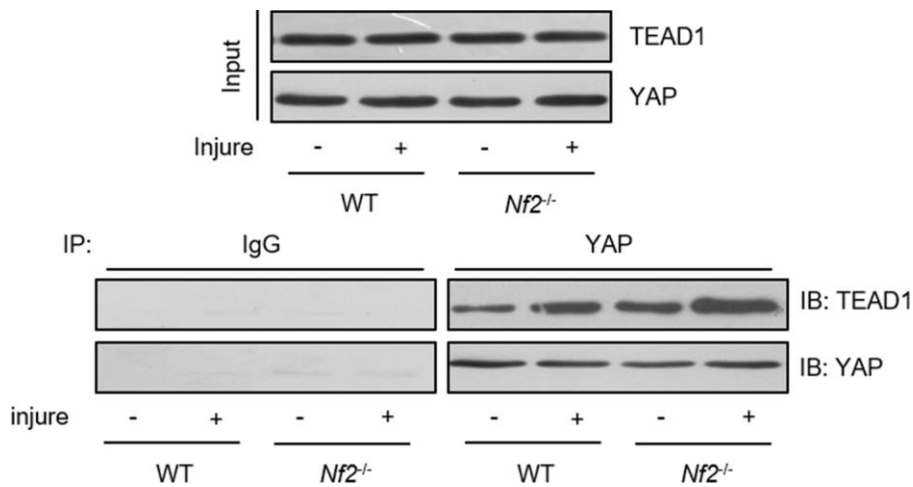
**Supplementary Figure 3. The mRNA levels of differentiation marker genes decline after injury in mice deficient for *Nf2*.** The mRNA levels of  $\alpha$ -SMA, Calponin and SM-MHC in carotid arteries from WT or  $Nf2^{-/-}$  mice at day 28 after sham operation or injury (n=4). Data are shown as mean  $\pm$  S.D. \*\* $P < 0.01$  and \*\*\* $P < 0.001$  denote statistical comparison between the two marked groups, respectively.



**Supplementary Figure 4. *NF2* knockdown also enhances injury-induced neointima hyperplasia at early and midway stage.** Representative H&E staining of carotid arteries from WT or  $Nf2^{-/-}$  mice at day 7 or 14 after wire injury (left) and corresponding quantification for ratio of intima/media (right) were shown (n=4). Magnification 200 $\times$ . Data are shown as mean  $\pm$  S.D. \*\*\* $P < 0.001$  denote statistical comparison between the two marked groups, respectively.



**Supplementary Figure 5. NF2 knockdown causes declined YAP phosphorylation in both nucleus and cytoplasm of PDGF-BB-treated VSMC.** VSMC from WT or *Nf2*<sup>-/-</sup> mice was treated by PDGF-BB (30 ng/mL) for 48 h. The nuclear and cytosolic-enriched fractions were then prepared. The relative protein expression levels of p-YAP<sup>Ser127</sup> and YAP were determined by immunoblotting (n=3). Data are shown as mean ± S.D. \**P*<0.05, \*\**P*<0.01 and \*\*\**P*<0.001 denote statistical comparison between the two marked groups, respectively.



**Supplementary Figure 6. NF2 knockdown enhances YAP-TEAD1 interaction in injured carotid artery.** Common carotid arteries from WT and *Nf2*<sup>-/-</sup> mice at day 28 after injury were subjected to immunoprecipitation using anti-YAP antibody or control IgG. Inputs and immunocomplexes were analyzed by immunoblotting.