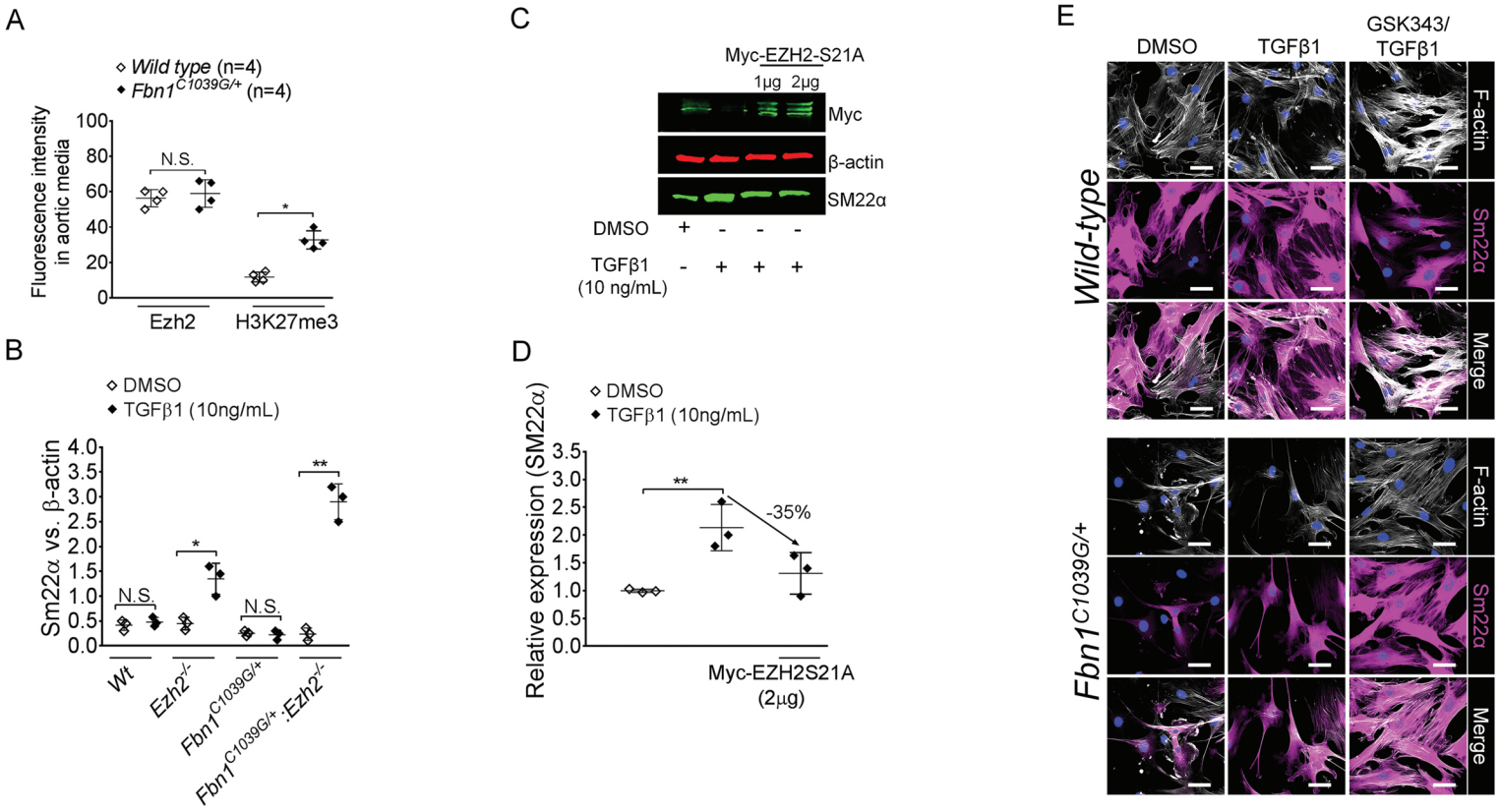
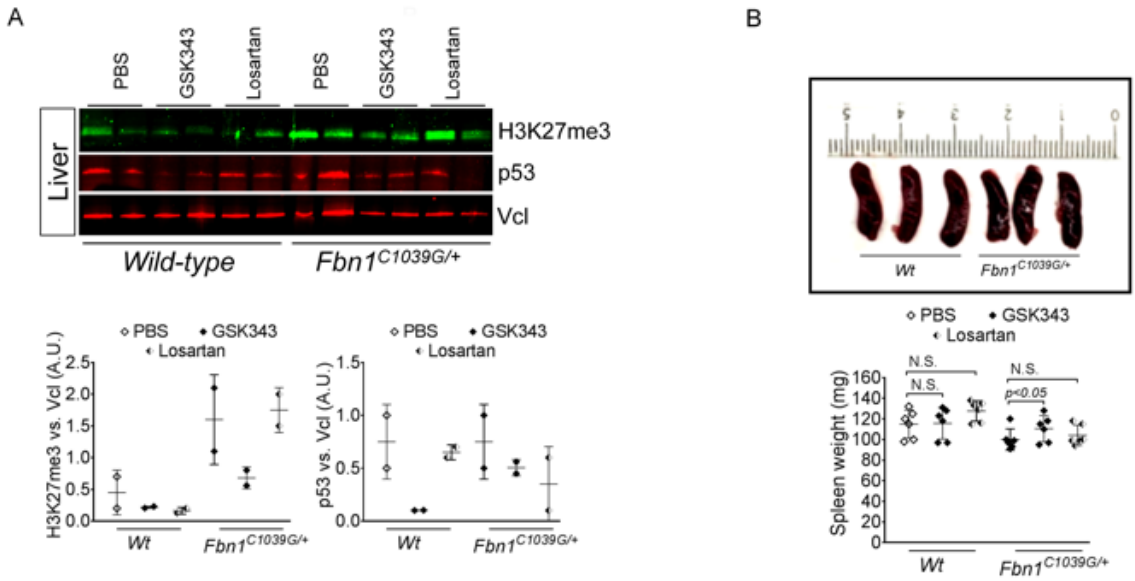


Supplemental Figure 1. Inhibition of the methyltransferase, EZH2 restores SM22α expression. (A) Quantification (left) and localization correlation (right) of immunofluorescent staining of Ezh2 and H3K27me3 (shown in Figure 3H) in *Fbn1*$^{C1039G/+}$ and wild type mouse aortas. (B) Quantification of immunoblotting (shown in Figure 3J) of Sm22α (versus β-actin) in mouse VSMCs isolated from wild type, *Ezh2*$^{-/-}$, *Fbn1*$^{C1039G/+}$, and *Fbn1*$^{C1039G/+}$*:Ezh2*$^{-/-}$ cells treated with or without TGF-β1 (10ng/mL). (C&D) Immunoblotting (C) and transcriptional (D) analysis of Sm22α in cells overexpressing mutant Serine to Alanine 21 EZH2 (EZH2S21A). Cell overexpressing EZH2 show suppression of SM22 expression which cannot be overcome with TGF-β stimulation (10 ng/ml). (E) Immunofluorescent staining of Sm22α (magenta) and F-actin (grey) in *Fbn1*$^{C1039G/+}$ and wild type mouse VSMCs treated with EZH2 inhibitor (GSK343 10μM) and TGF-β1 (10ng/mL), Bar=10 μm.



Supplemental Figure 2. *In vivo* inhibition of EZH2 activity palliates experimental aortic aneurysm. (A) Immunoblotting of murine liver for H3K27me3, p53, and vinculin (VCL) proteins in wild type and *Fbn1*$^{C1039G/+}$ mice treated with either PBS, GSK343, or losartan. Quantification of immunoblotting demonstrated below. (B) Lack of splenic enlargement in wild type and *Fbn1*$^{C1039G/+}$ mice treated with either PBS, GSK343, or losartan.

Figure 1b.

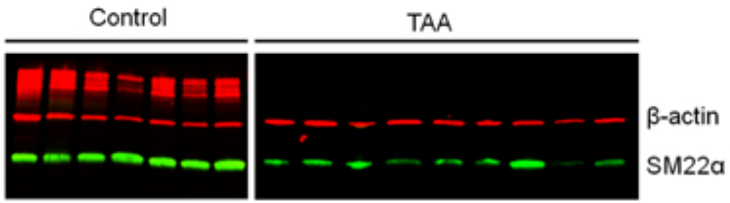


Figure 1h.

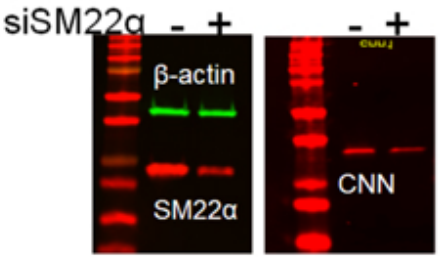


Figure 1i.

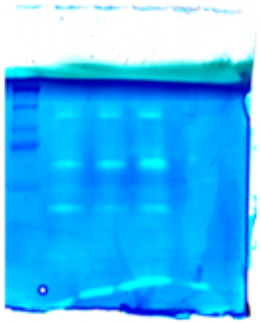


Figure 3c.

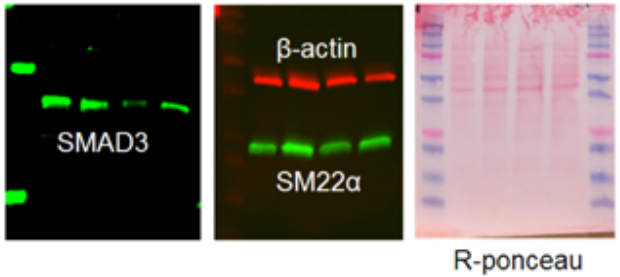


Figure 3g.

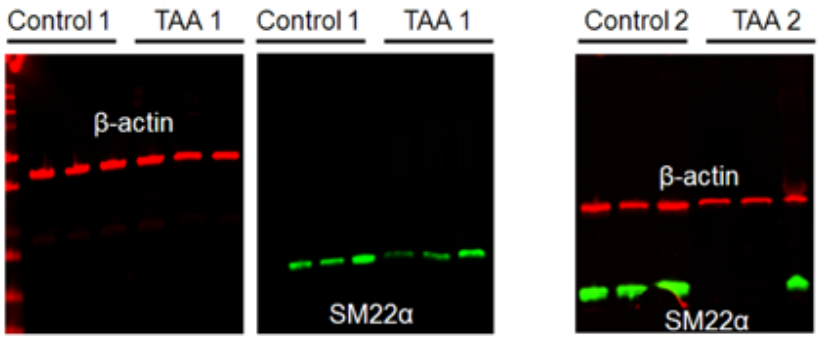
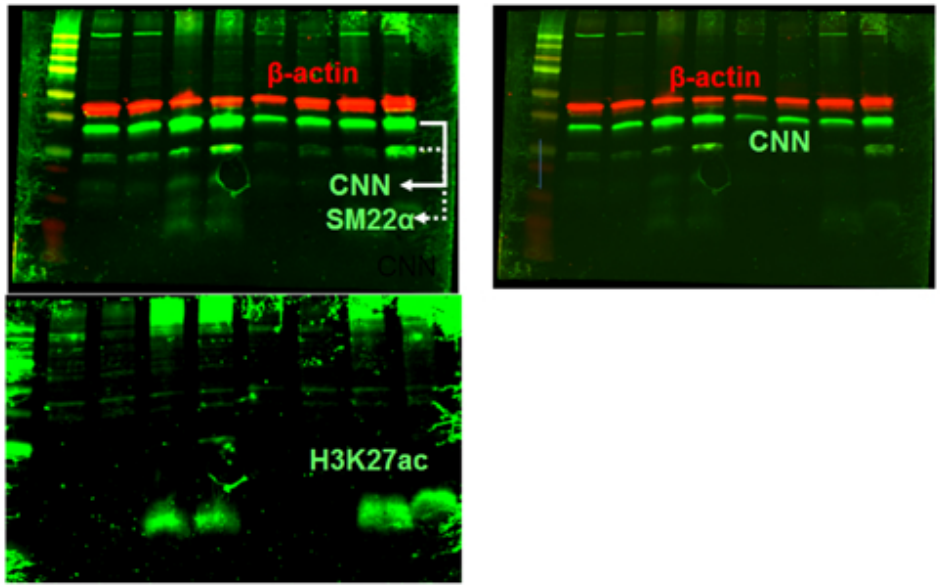
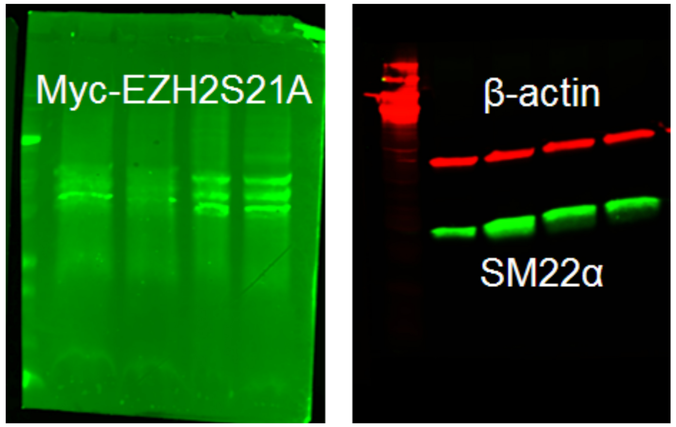


Figure 3j.



Supplementary Figure 1C.



Supplementary Figure 2A