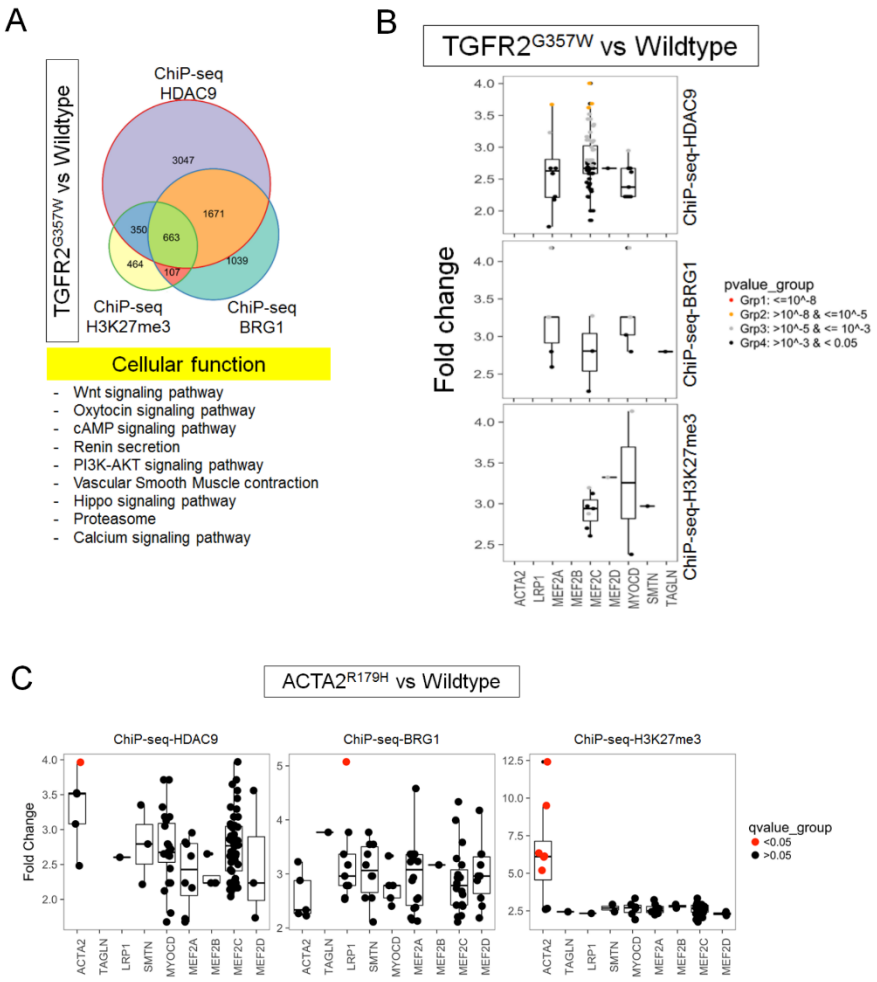
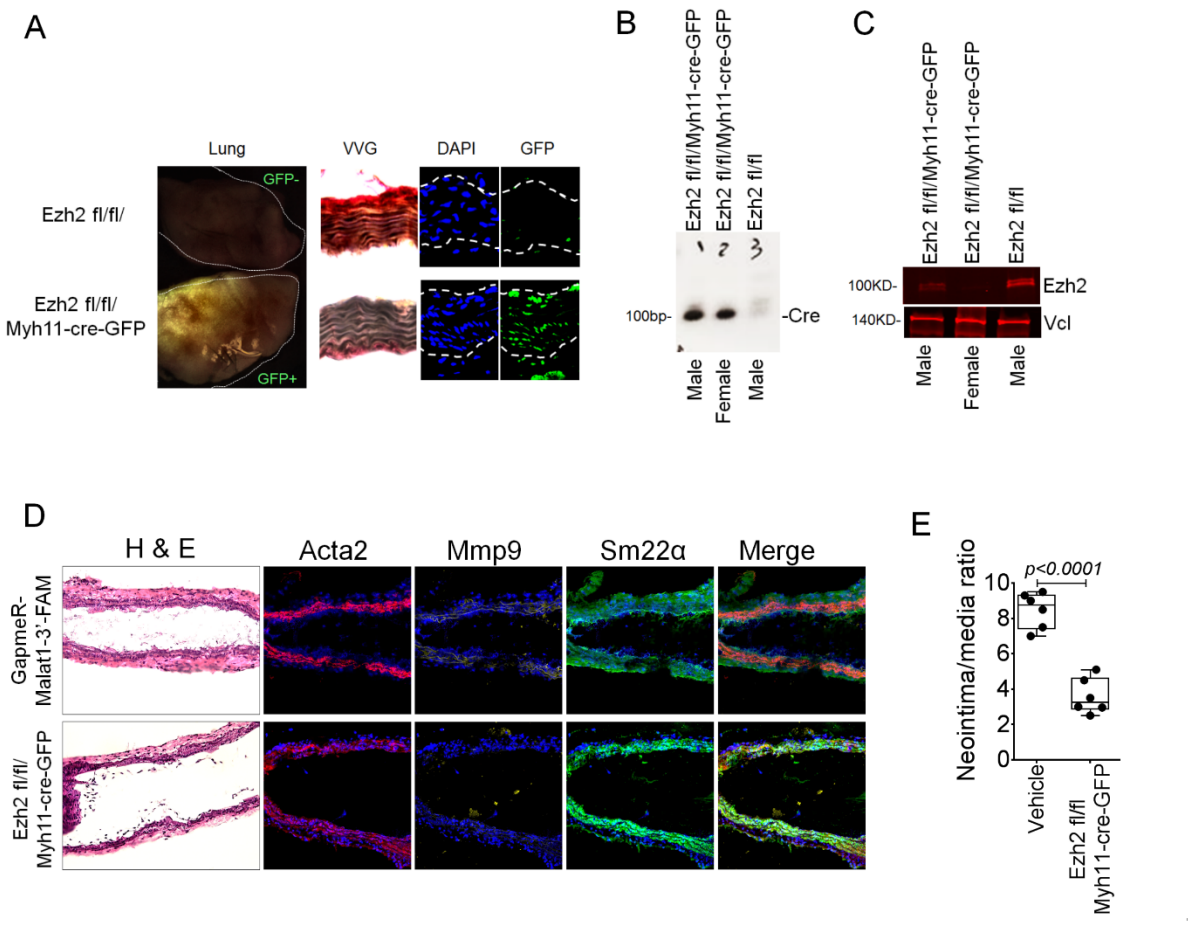


Supplementary Figure 1. TGFR2^{G357W} mutation induces HDAC9-BRG1-MALAT1 complex interaction with genetic loci associated with VSMC contractility. (A) Venn diagram describing ChIP-Seq experiment in human VSMCs under conditions of with TGFR2^{G357W} expression shows overlapping enrichment of HDAC9, BRG1 and H3K27me3 modification at 663 gene loci versus wild-type cells. Pathway analysis demonstrates multiple cellular functions involving smooth muscle cell contraction. (B) Box plots show fold change enrichment of HDAC9, BRG1 and H3K27me3 at locus of SMCs contractile elements in TGFR2^{G357W} mutant versus wild-type cells. Color coded nominal p-values of individual genomic site within locus. P value annotation of the peaks from macs2 was performed using the ChipSeeker package in R. (C) Color coded p-values of selected genomic sites in the promoters of listed genes after Benjamini-Hochberg correction for multiple testing of with ACTA2^{R179H} expressing cells.



Supplementary Figure 2. Deletion of Ezh2 in vascular smooth muscle compartment in carotid ligation. (A) Detection of Myh11-cre-GFP positive in lung and aorta tissues. (B) Gel image of PCR genotyping. (C) Immunoblot assays shows protein levels of Ezh2 in male and female Myh11-cre mice compared with not cre mouse. (D) Histological and immunofluorescence analysis of ligated carotids of GapmeR-Malat1-3'FAM (n=3) and Ezh2fl/fl:Myh11-cre-GFP (n=6). Acta2 (red), Mmp9 (yellow), Sm22α (green) and nucleus (blue). (E) Ratio of neointima vs media of ligated carotids of wild type (n=6) and Ezh2fl/fl/Myh11-cre-GFP (n=6) mice. Significance was calculated using 1-way ANOVA with a Tukey's multi comparisons test.