

Supplemental Figure Legends

Supplemental Figure S1:

A: Multiple C-terminal GLI1 domains mediate reporter transcriptional expression. Chimeric Gal4-GLI1 plasmid constructs were transfected into PANC-1 cells and the transcriptional activity was measured as the luminescence signal for each sample normalized to the total protein as measured by the Bradford protein assay, $n=3$. Results are expressed as means \pm SEM. B: A representative Western Blot showing the expression of the transfected plasmids, Antibodies used are GLI1, GAL4 and Vinculin as the loading control. $n=3$. All fragments were detected with either the GAL4 or GLI1 antibodies. Some chimeric proteins could not be detected with the antibody to the GAL4 epitope. C: A CHIP-PCR showing the binding of the expressed GAL4-GLI1 chimeric proteins to the UAS sequence of the stably transfected reporter in the HEK293G cells. The minimal fold enrichment of the constructs at the reporter sequence was 7x greater than the IgG control. Notably, the fold enrichment for un-transfected samples was less than 1.0 for the GAL4 and GLI1 antibodies indicating the binding at the UAS was specific for the ectopically expressed proteins (data not shown). Results are expressed as means \pm SEM. D: A representative Western Blot showing the expression of the GLI1-TAD and the cofactors, $n=3$. Vinculin is the loading control. E. The relative transcriptional activity from co-expression of cofactors with the pM (grey bars) or GLI1-TAD (blue bars) in PANC-1 cells. A representative experiment is shown for 3 technical replicates where the luminescence signal for each sample was normalized to the total protein. The error bars represent the SEM.

Supplemental Figure S2:

A: A representative Western Blot showing the expression of GLI1 and SMARCA2 for the MNase digestion, $n=3$. B-actin is the loading control. B: Level of GLI1 and SMARCA2 knockdown of RNA and protein for the ATAC-seq samples and the RNA-seq samples, $n=3$. Results are expressed as means \pm SEM. The Western Blots are representative examples of knockdowns at the protein level. C: The location of all the ATAC-seq peaks for the GLI1 and SMARCA2 groups is determined relative to gene transcription start sites (TSS) using the GREAT package with basal parameters. All peaks included the combined peak lists generated by MACS2 for the knock down and corresponding non-targeting controls from 3 replicates. Overlapping peaks were then merged to create a non-redundant list of peaks.

Supplemental Figure S3:

Chromatin accessibility peaks at the HHIP locus overlaps with an H3K4me1 peak from Encode. ATAC-seq peaks upstream of the HHIP TSS regulated by GLI1 and SMARCA2 knockdown (from Figure 1A) were aligned with ENCODE layered tracks for H3K4me1 and H3K27ac that often mark regulatory elements. Highlighted with a red arrow is the chromatin peak that decreases in accessibility in response to GLI1 and SMARCA2 knockdown as determined statistically using the DiffBind package. This site overlaps with a H3K4me1 mark in data from HUVEC cells.

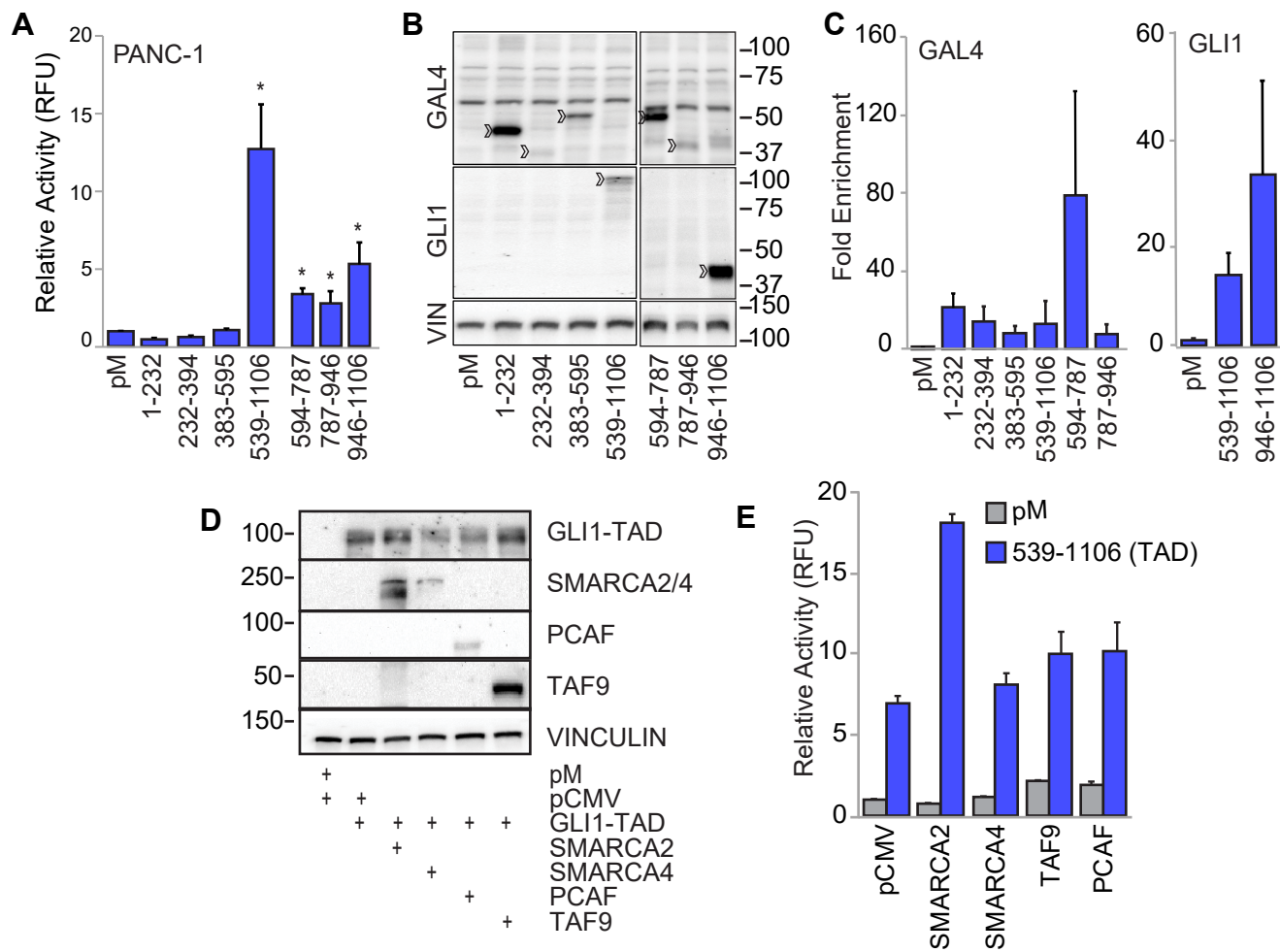


Figure S1

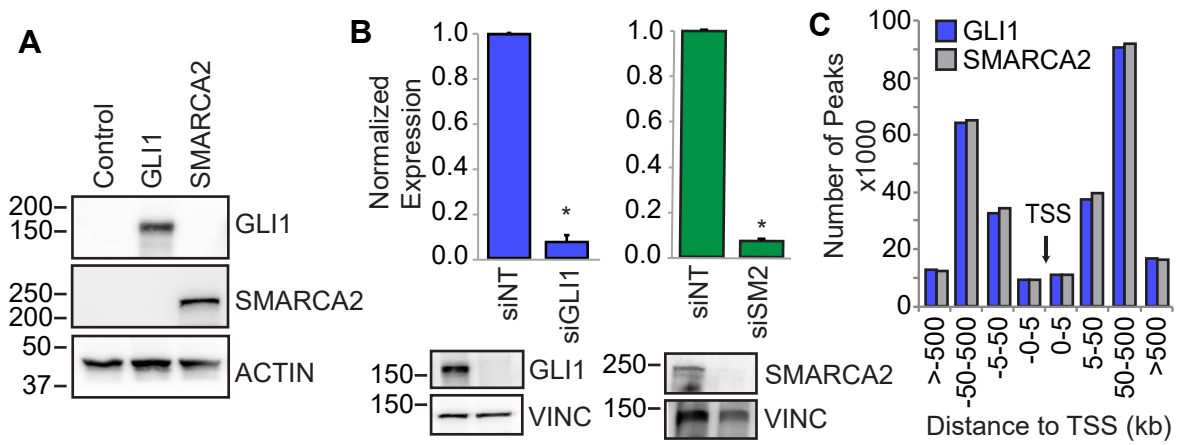


Figure S2

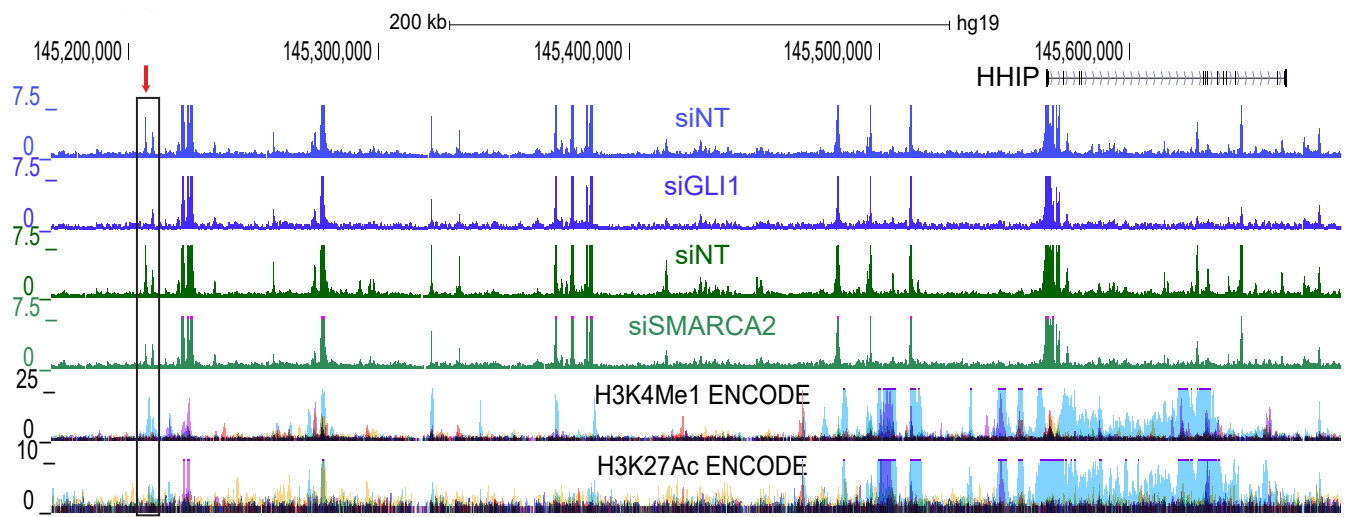


Figure S3