Expanded View Figures

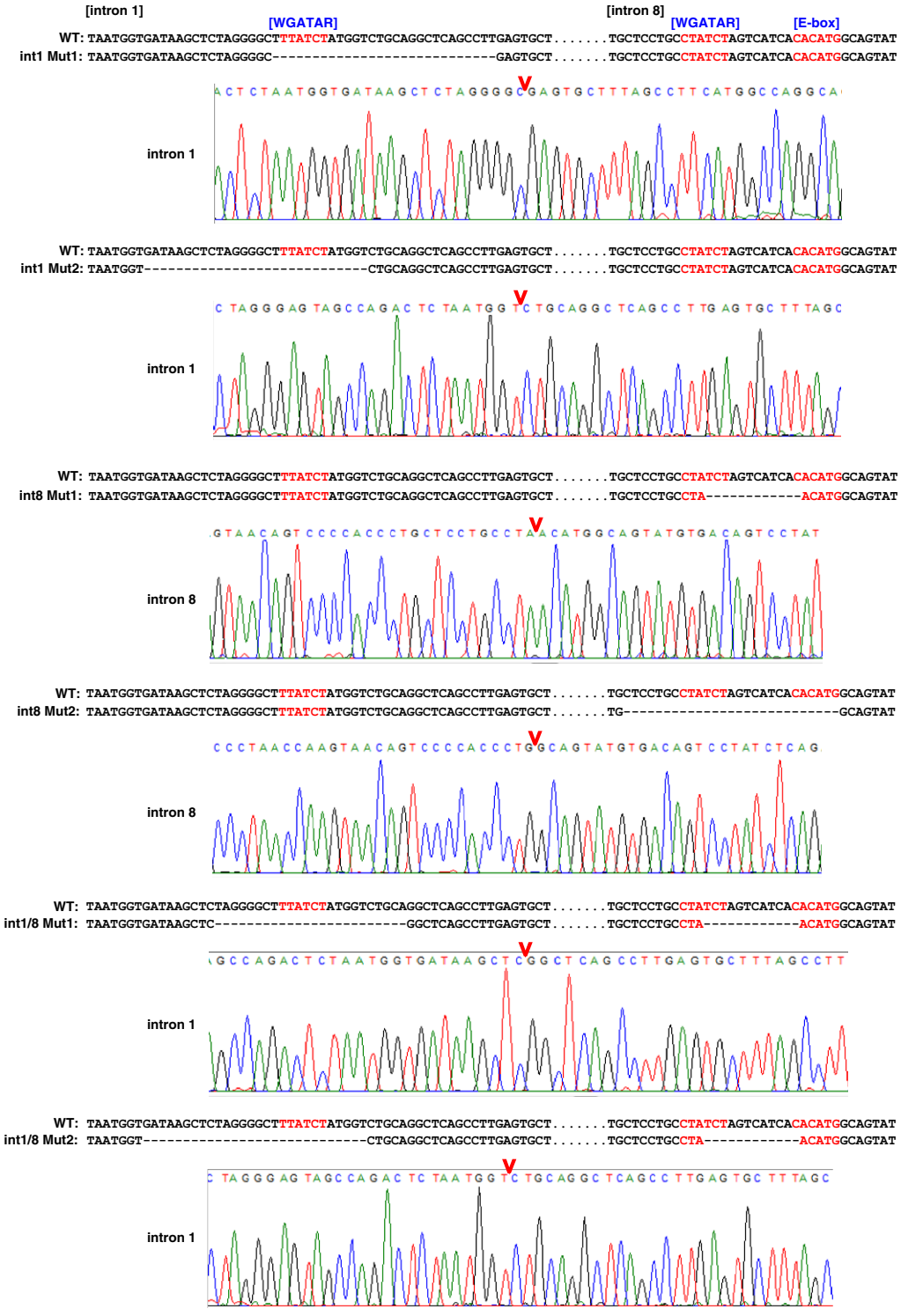
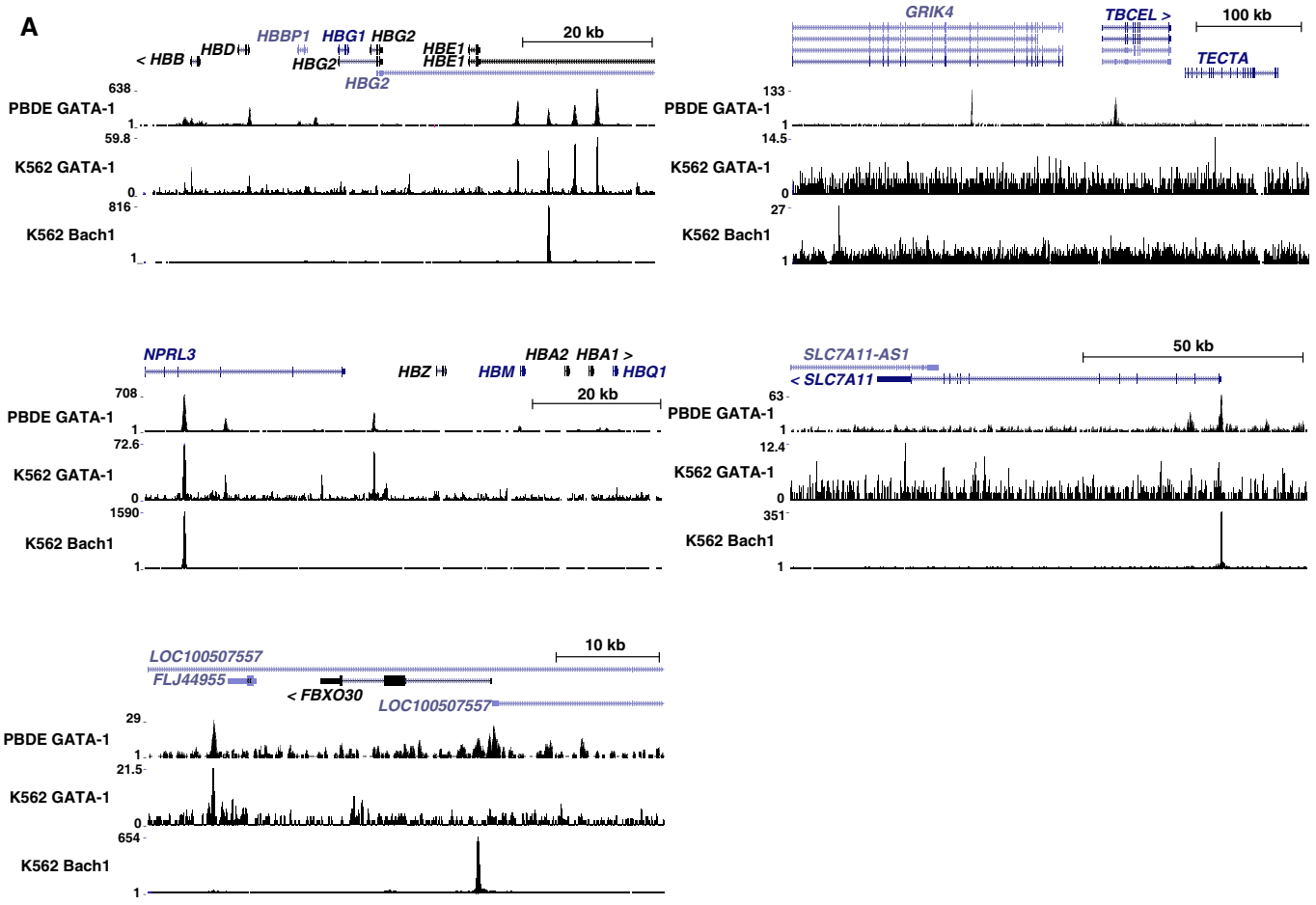


Figure EV1. DNA sequences at *Alas2* intron 1 or 8 of mutant clonal cell lines.
CRISPR/Cas9-mediated mutations were detected by direct sequencing of the PCR amplicons of genomic DNA.



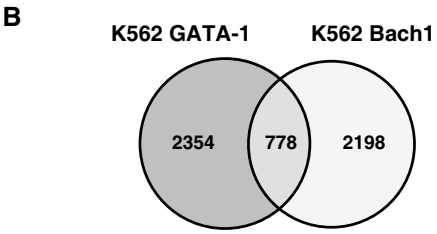


Figure EV2. GATA-1 and Bach1 ChIP-seq profiles.

A ChIP-seq data revealed GATA-1 and Bach1 occupancy at Bach1-sensitive genes in primary human erythroid cells (peripheral blood-derived erythroblasts) and K562 erythroleukemia cells (accession numbers: GSM935540, GSM935465, and GSM935576).

B Venn diagram depicting relationships between GATA-1 and Bach1 target genes in K562 cells. The definition of a "target" gene was as follows: the gene had a peak in one of the promoter regions (2 kb upstream of transcription start site (TSS) to 200 bp downstream of TSS), a peak in one of the introns, or a peak in the region 10 kb upstream of TSS, which was not in an intron or promoter of another gene. All target genes were listed in Table EV3.

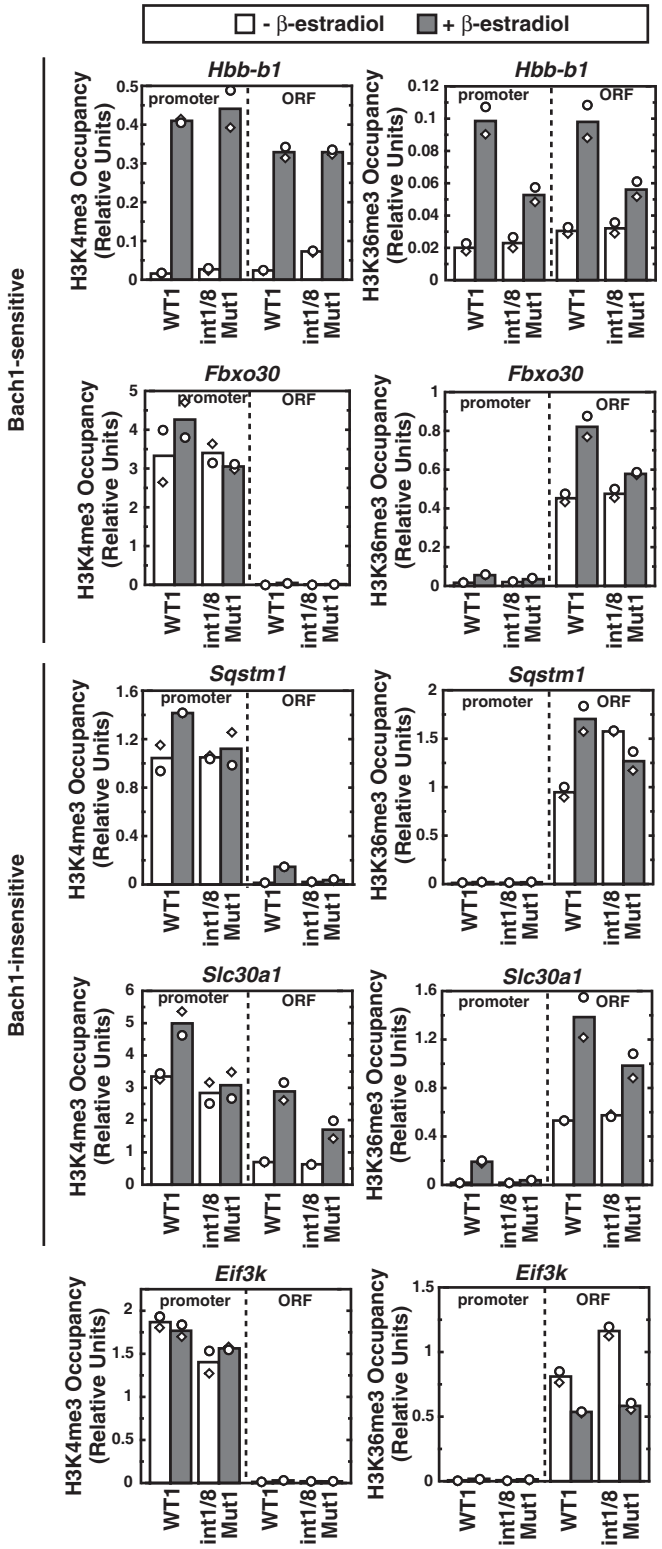


Figure EV3. Quantitative ChIP analysis of H3K4me3 and H3K36me3 levels at promoters and open reading frames of Bach1-sensitive and Bach1-insensitive genes.

Quantitative ChIP analysis revealed H3K4me3 and H3K36me3 levels in untreated or β-estradiol-treated WT1 and double-mutant clone 1 ($n = 2$; bars show the mean and individual data points are shown; each data point was based on two measurements).