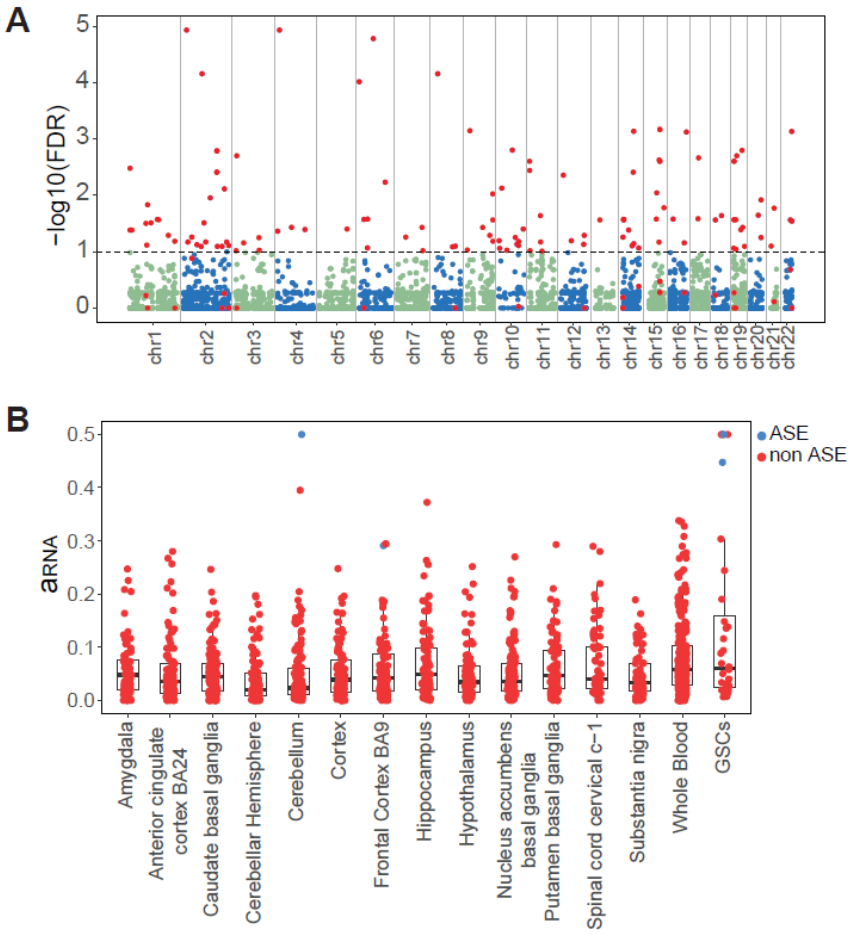
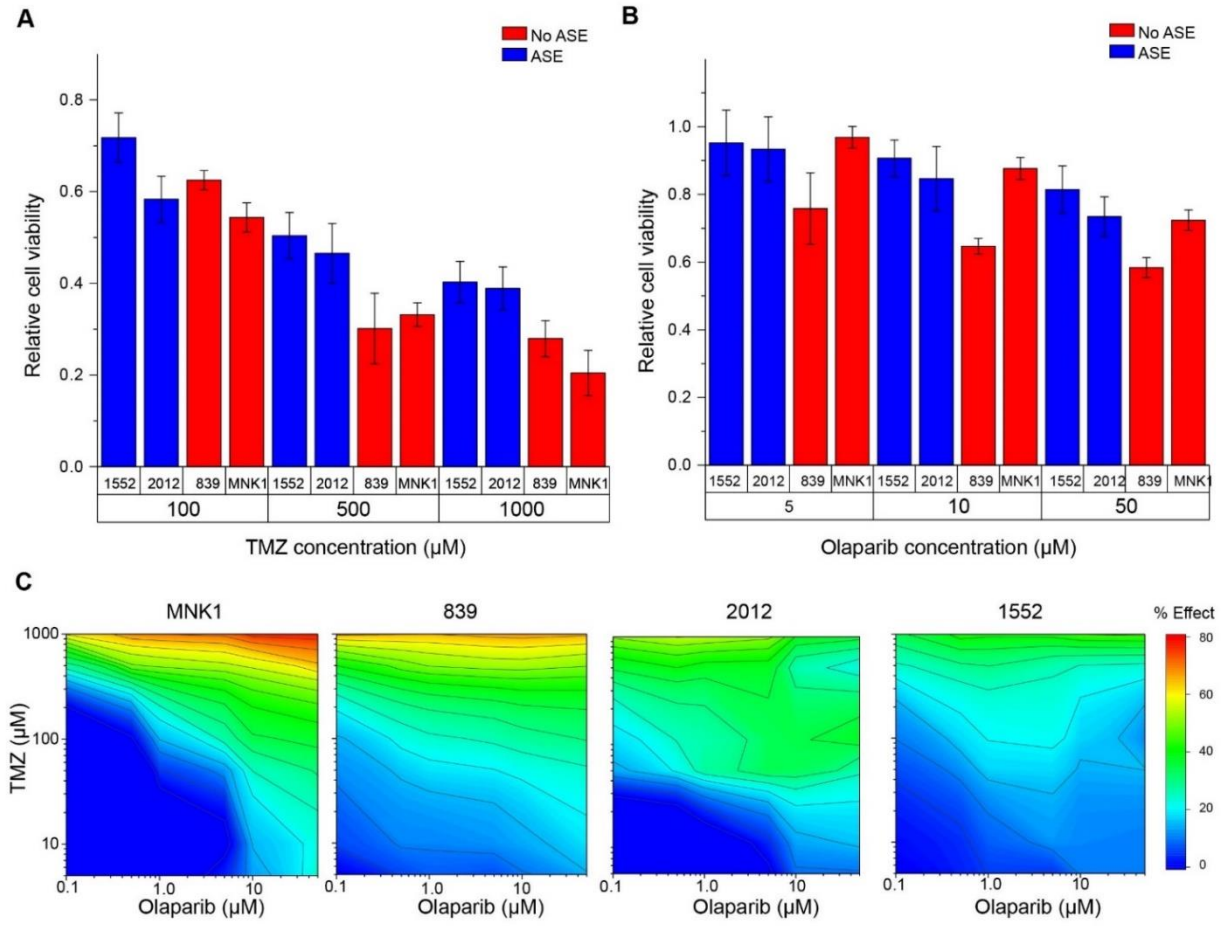
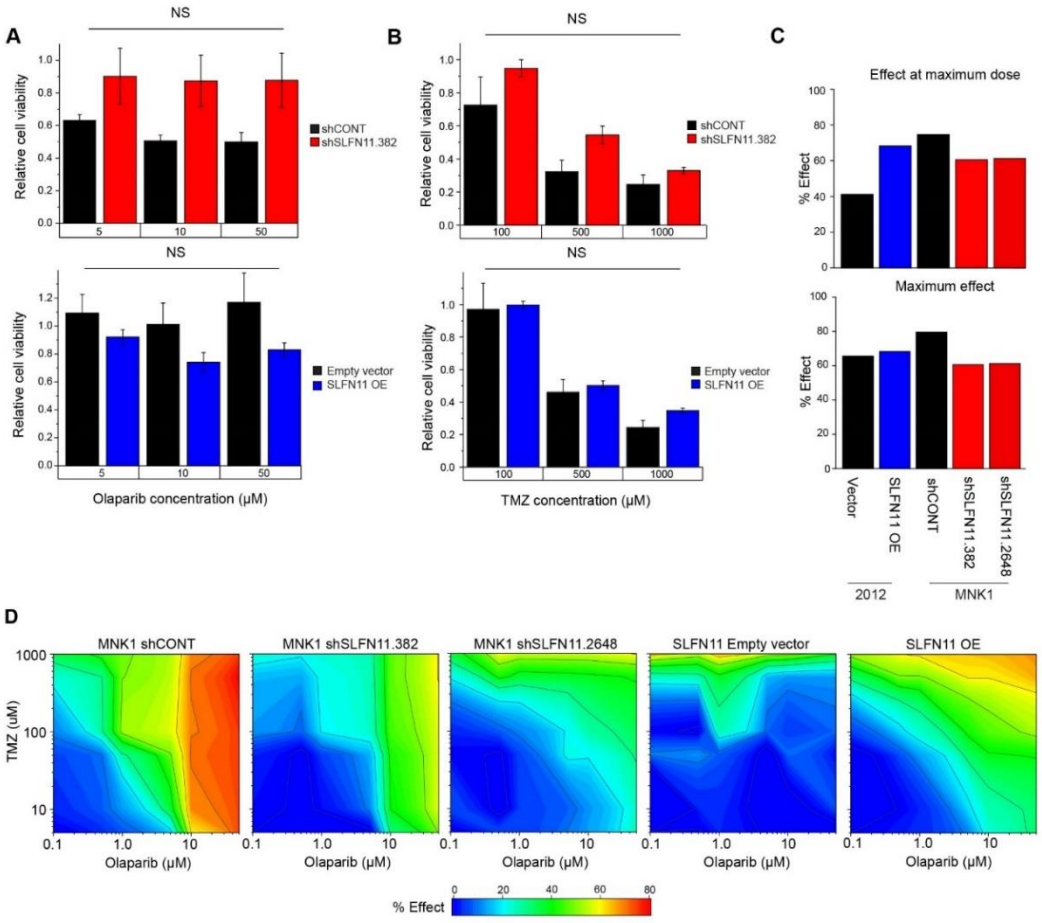
Supplemental Data



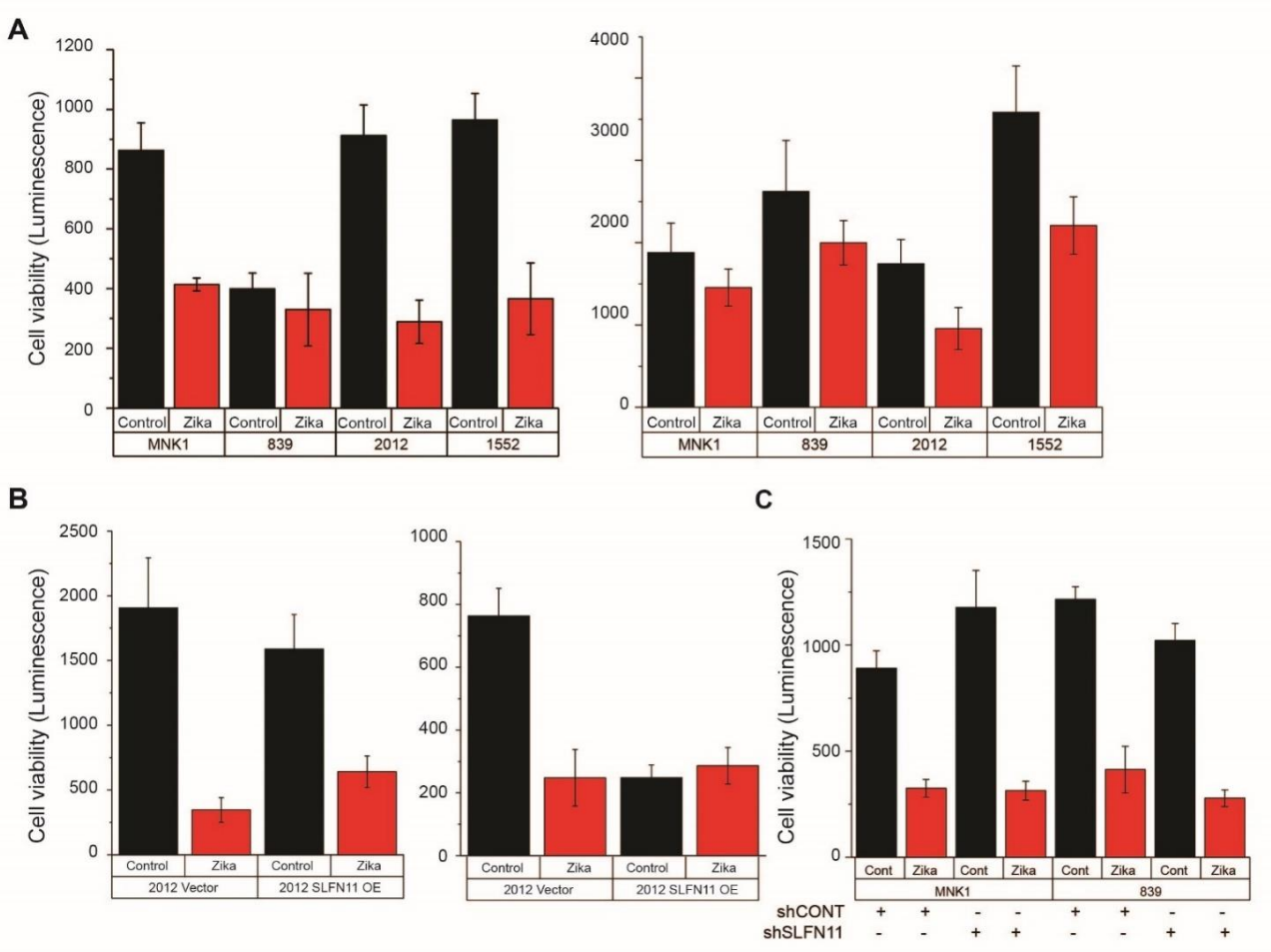
Supplemental Figure S1: Recurrent allele-specific expression within glioblastoma stem cells. **A)** Manhattan plot demonstrating that genes with recurrent ASE in glioblastoma stem cells (GSCs) are not localized to any single genomic locus. The x-axis is the gene start position for all tested genes and y-axis is the -log10 transformed FDR corrected Fisher Exact Test p-value. Genes significant under an FDR of 10% are highlighted in red. **B)** Comparison of estimated ASE for *IP6K2* for GSCs, normal brain and whole-blood samples from GTEx.



Supplemental Figure S2: Cellular viability assays for GSCs. A) Relative cell viability plotted on the y-axis for each model treated with TMZ. Non-ASE models are in red and ASE models are in blue. **B)** Relative cell viability plotted on the y-axis for each model treated with olaparib. Non-ASE models are in red and ASE models are in blue. **C)** Cell viability relative to DMSO control is annotated on a rainbow scale with blue indicating high viability, or minimal drug effect, and red indicating low viability, or strong drug effect. Doses are scaled logarithmically on the x-axis (olaparib) and y axis (TMZ). Data are represented as mean +/- standard deviation. Panels A-B are averages of at least 6 technical replicates per model, with > two biological replicates for 2012, 1552 and MNK1, resulting in at least biological triplicates for each condition ASE vs non-ASE. Panel C is the average of two biological replicates per model. ASE: allele-specific expression; GSC: glioblastoma stem cell; KD: knockdown; OE: overexpression; TMZ: temozolomide.



Supplemental Figure S3: Cellular viability assays for GSCs with knockdown and overexpression of *SLFN11*. A) Relative cell viability plotted on the y-axis for MNK1 and 2012 GSC models treated with olaparib. Upper panel: MNK1 shCONT cells are in black and *SLFN11* KD in red; Lower panel: 2012 empty vector control cells are in black and *SLFN11* OE are in blue. **B)** Relative cell viability plotted on the y-axis for each model treated with TMZ. Upper panel: MNK1 shCONT cells are in black and *SLFN11* KD in red; Lower panel: 2012 empty vector control cells are in black and *SLFN11* OE are in blue. **C)** Top: effect of the maximum combinatorial drug dose (reduction of cell viability) for each comparison. Bottom: maximal effect achieved at any dose for each model. **D)** Cell viability relative to DMSO control is annotated on a rainbow scale with blue indicating high viability, or minimal drug effect, and red indicating low viability, or strong drug effect. Doses are scaled logarithmically on the x-axis (olaparib) and y axis (TMZ). Data are represented as mean +/- standard deviation. Panels A-B are average of two biological replicates per model. Panels C-D are the average of three biological replicates per model. GSC: glioblastoma stem cell; KD: knockdown; NS: non-significant; OE: overexpression; TMZ: temozolomide.



Supplemental Figure S4: Cellular viability assays for GSCs following treatment with Zika virus. Non-normalized cell viability data corresponding to Figure 6. **A)** Viability of ASE vs non-ASE cells following treatment with Zika vs. control. Data are presented as two biological replicates per model. **B-C)** Viability following overexpression **(B)** or **(C)** knockdown of *SLFN11* with Zika vs control treatment, with two biological replicates per condition (control vs. KD).

Supplemental Table Legends

Supplemental Table S1: Results for ASE analysis for GSCs.

Supplemental Table S2: Results for Fisher Exact Test (FET) which was used for discovering genes which show enrichment of ASE in GSCs compared to whole-blood from GTEx.

Supplemental Table S3: Results from Spearman's rank correlation between normalized gene expression and normalized coverage for H3K27ac ChIP-seq bins which are located within 100Kb of promoters of ASE genes.

Supplemental Table S4: Results from Spearman's correlation analysis between normalized gene expression and mean promoter methylation ($\beta_{promoter}$) for ASE genes.