Supplemental Figures

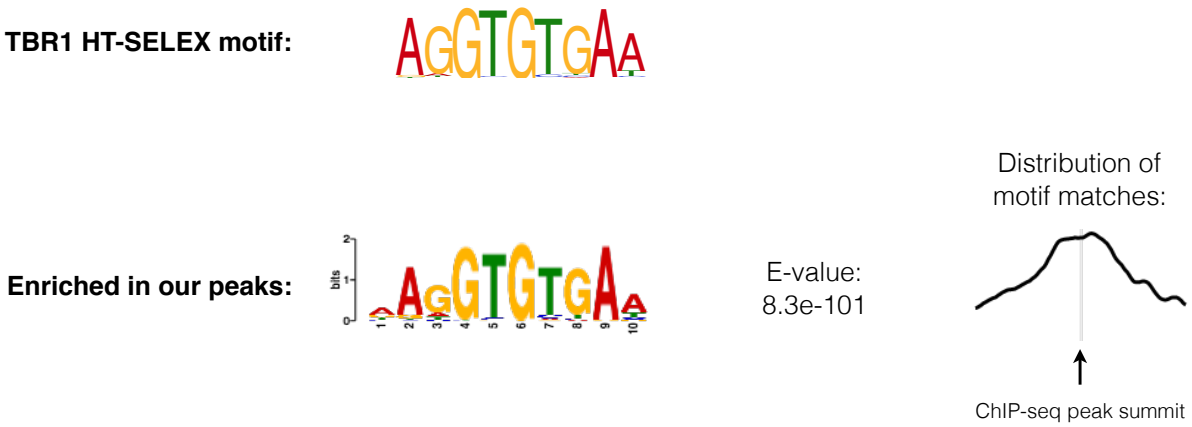

Figure S1 MEME-ChIP (Machanick and Bailey 2011) motif discovery recovers the known TBR1 motif (Jolma et al. 2013) enriched at the summits of ChIP-seq peaks.

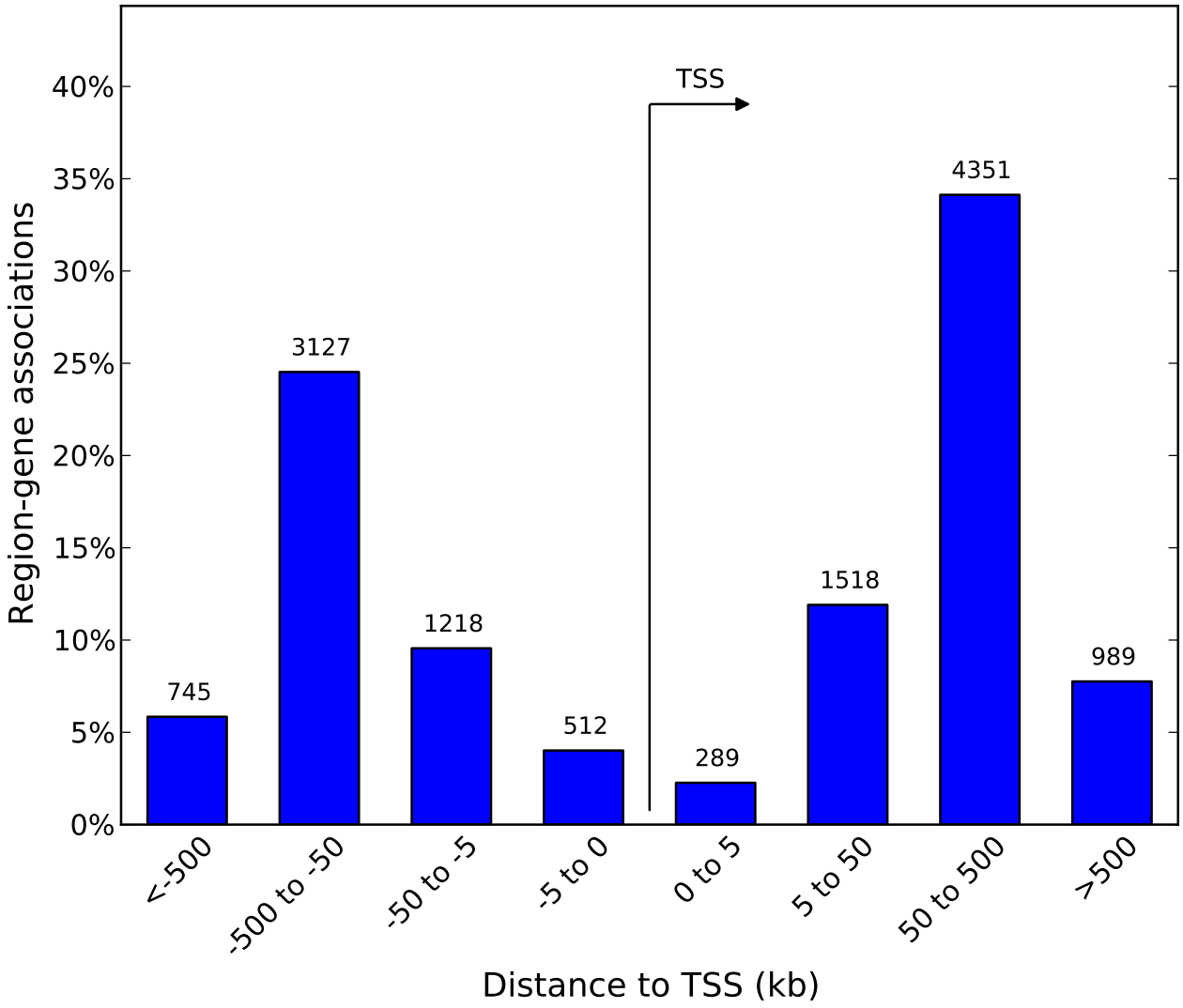

Figure S2 Distribution of TBR1 ChIP-seq peak distances to the associated transcription start sites.

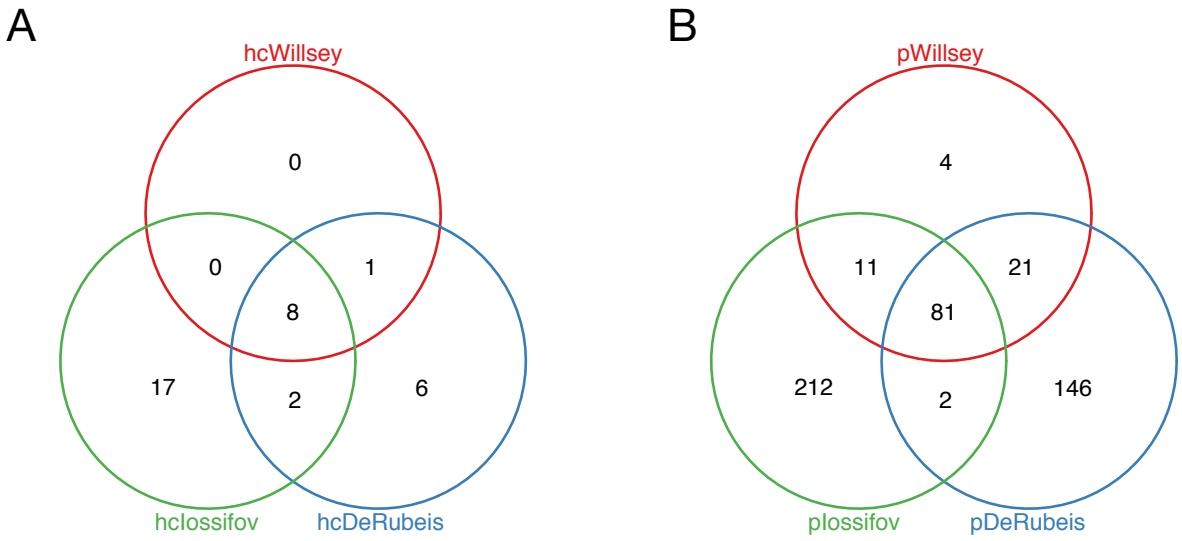

Figure S3 Overlaps among the mouse orthologs of high-confidence (A) and probable (B) ASD genes from different studies. *SYNGAP1* was not mapped from the high-confidence human ASD gene set to mouse.

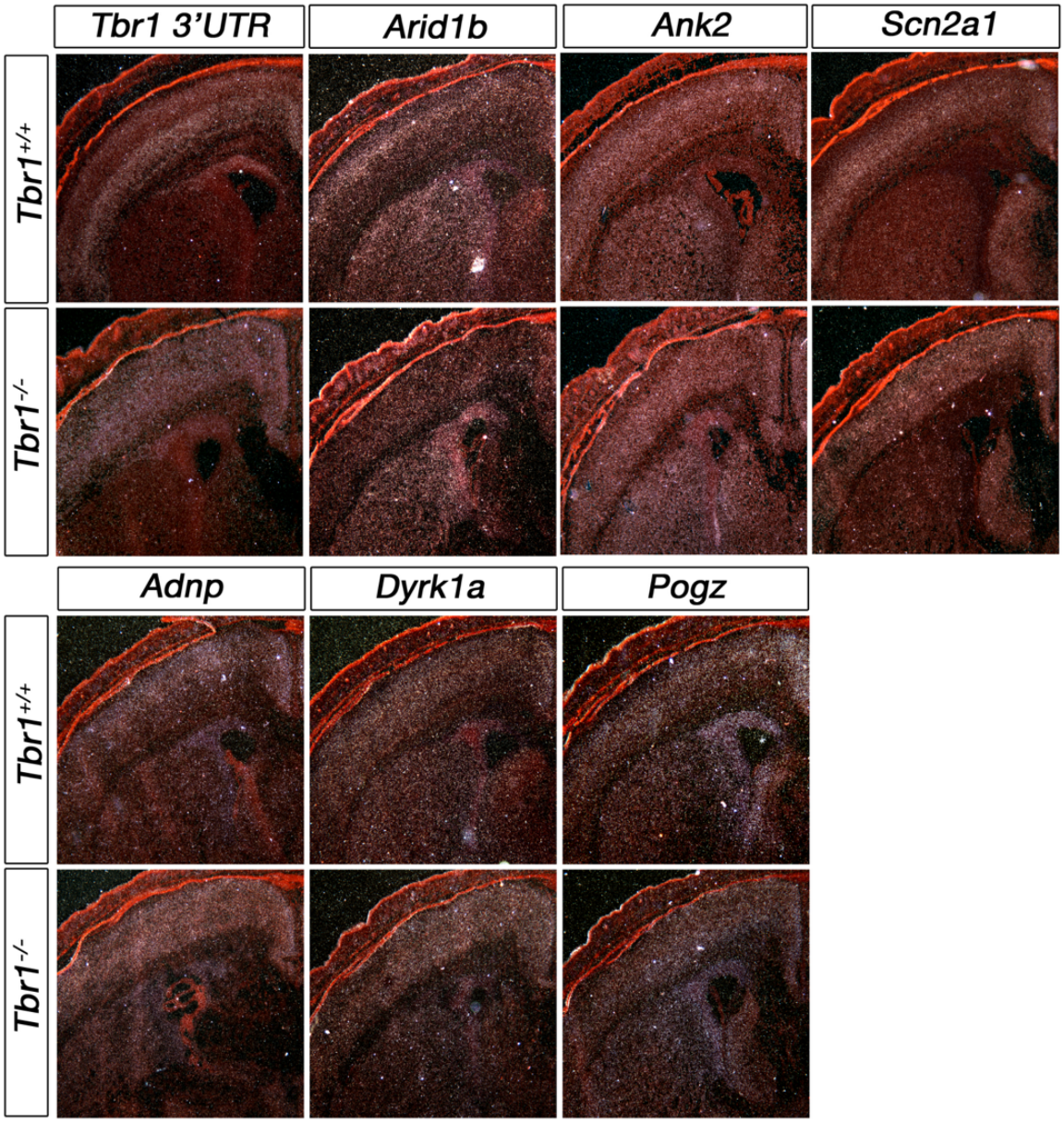

Figure S4 Radioactive *in situ* hybridization (RISH) of high-confidence ASD genes at P0 in *Tbr1*$^{+/+}$ and *Tbr1*$^{-/-}$ cortices reveals expression differences.

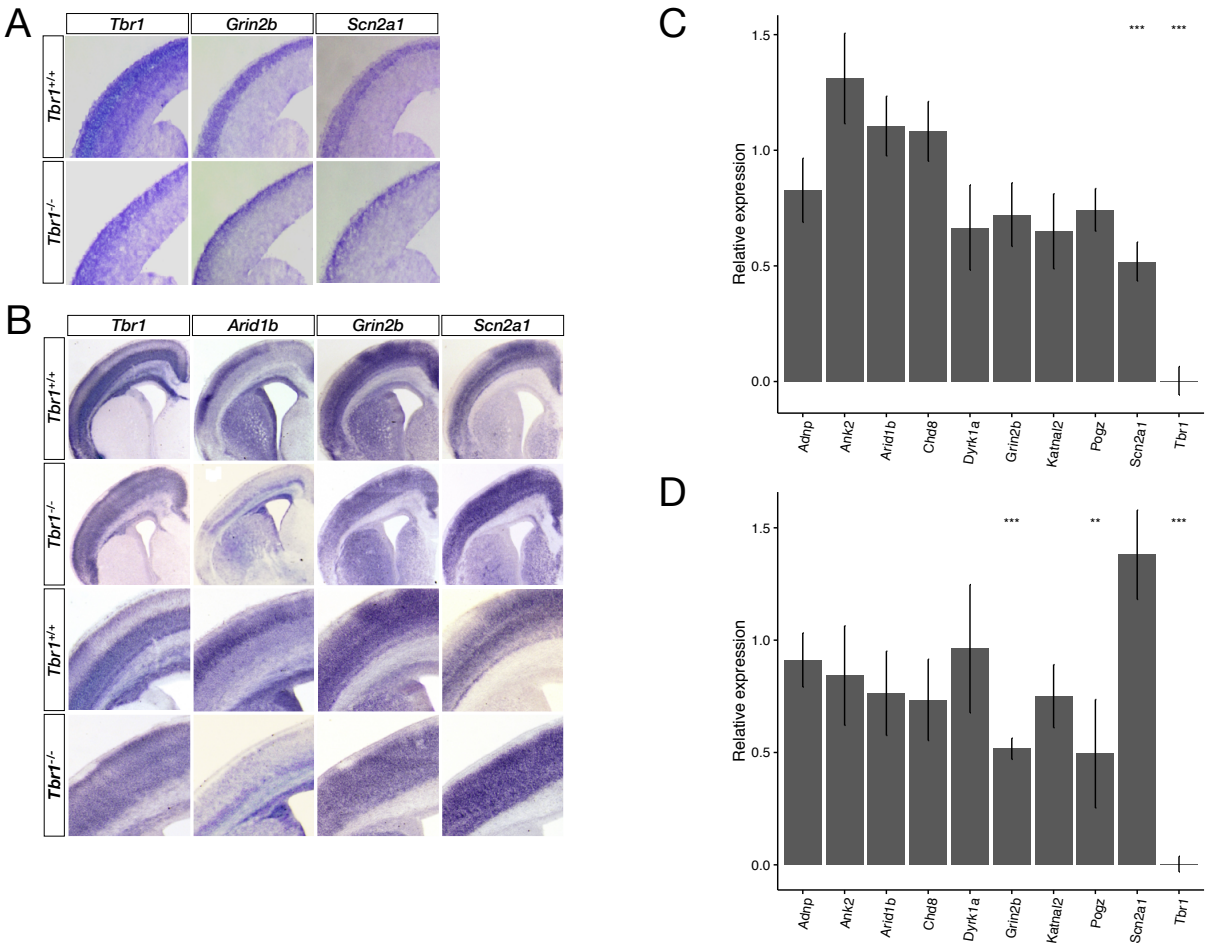

Figure S5 Digoxigenin *in situ* hybridization of high-confidence genes at E14.5 (A) and P0 (B) in *Tbr1*$^{+/+}$ and *Tbr1*$^{-/-}$ cortices reveals expression differences. Relative expression corresponds to quantitative real-time PCR (qRT-PCR) results comparing transcript expression levels in the cortex of the *Tbr1* mutant mice and wild-type littermates at E14.5 (C) and P0 (D). The error bars represent the standard error of the mean. 2-sided *t*-test. **p-value < 0.01; ***p-value < 0.001.

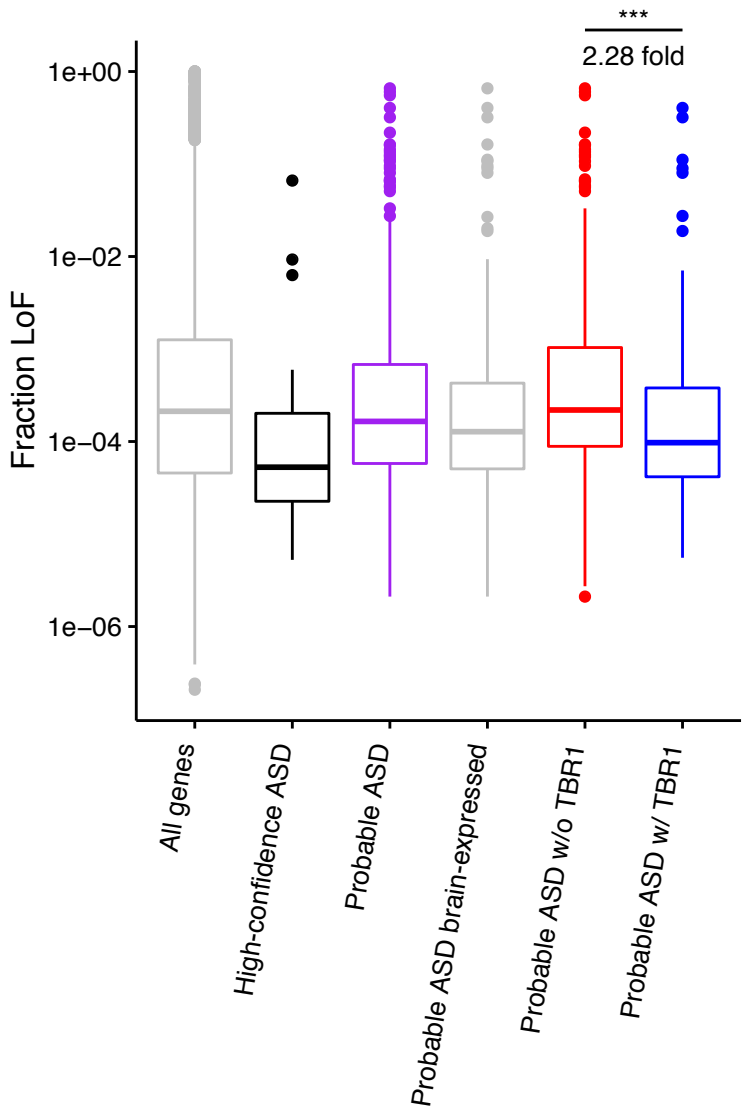

Figure S6 Probable ASD genes that are TBR1 targets are depleted for RVIS percentiles. Box-plots depicting the distributions of RVIS percentiles for different ASD gene lists (x-axis). Probable ASD genes with adjacent TBR1 ChIP-seq peaks in the developing cortex (blue) have lower RVIS percentiles than those without an adjacent TBR1 peak (red). Significance was determined using the 1-sided 2-sample Wilcoxon test. ***p-value < 0.001.

Supplemental Tables

Table S1 GREAT enrichments corresponding to the E15.5 TBR1 ChIP-seq, E14.5 neocortex EP300 ChIP-seq, E15.5 neocortex SATB2 ChIP-seq, and 28 ENCODE ChIP-seq sets including tissues at different developmental time-points and primary cell lines featured in Figure 1.

Table S2 Fraction LoF scores and RVIS percentiles for each gene. “NA” describes missing values.

Table S3 The number of TBR1 ChIP-seq peaks adjacent to the mouse ortholog, GREAT bionmial *p*-value, fraction LoF score, fraction LoF percentile among all genes, Icelandic knockout status, and limma *p*-value and fold for each high-confidence and probable ASD gene. “NA” describes missing values.

Table S4 Gene ontology enrichment analysis of the mouse orthologs of probable ASD genes with at least one TBR1 ChIP-seq peak against the background of all probable ASD genes.

Table S5 ChIP-seq replicate quality control information. NSC: normalized strand cross-correlation coefficient. RSC: relative strand cross-correlation coefficient.

Table S6 PCR Primers used to generate probes for radioactive *in situ* hybridization. The T7 promoter sequence (5’- gcgcgtaatacgactcactatagggc-3’) was added to the 5’ end of each reverse primer.

Table S7 Primers used for qRT-PCR experiments.

Table S8 cDNA clones used to synthesize DIG-labeled RNA probes for *in situ* hybridization on frozen sections.

Supplemental References

Jolma A, Yan J, Whitington T, Toivonen J, Nitta KR, Rastas P, Morgunova E, Enge M, Taipale M, Wei G, et al. 2013. DNA-binding specificities of human transcription factors. *Cell* **152**: 327–339.

Machanick P, Bailey TL. 2011. MEME-ChIP: motif analysis of large DNA datasets. *Bioinformatics* **27**: 1696–1697.