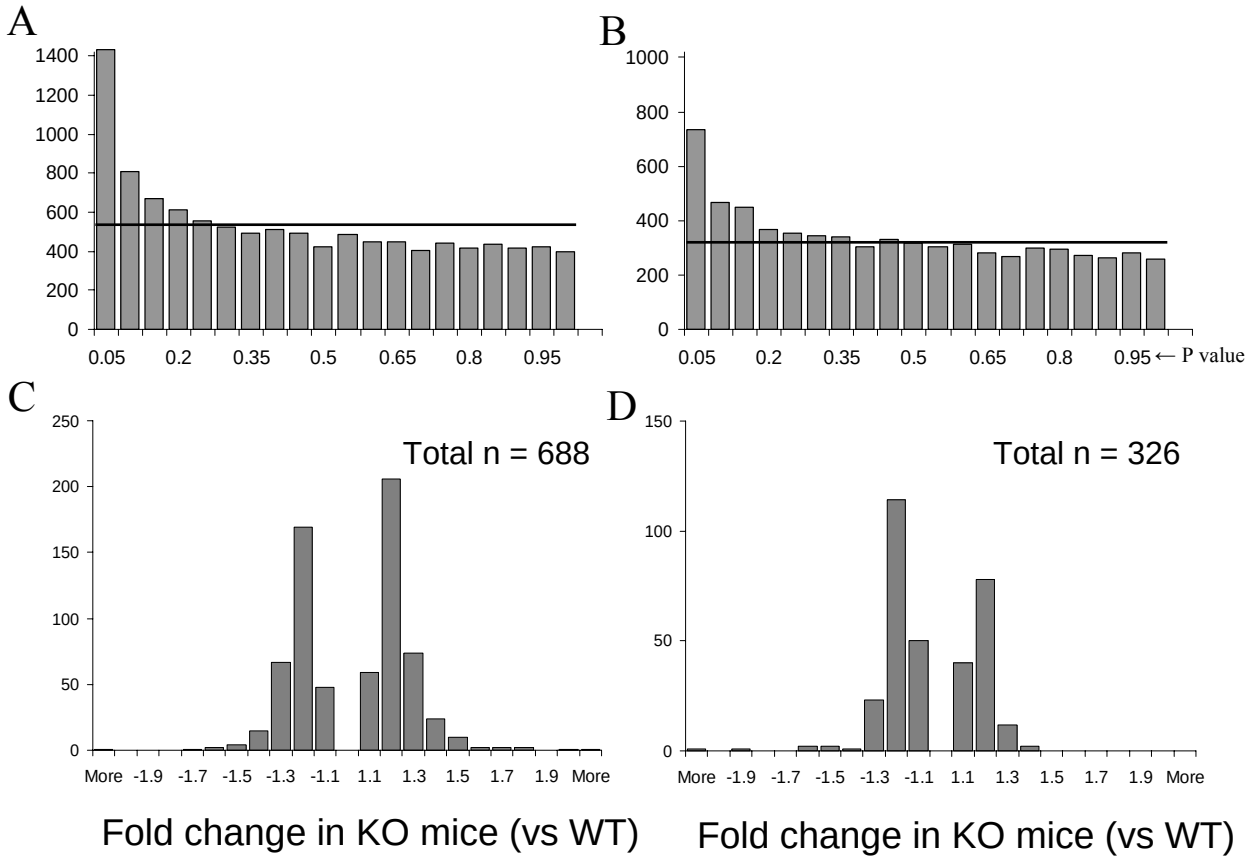
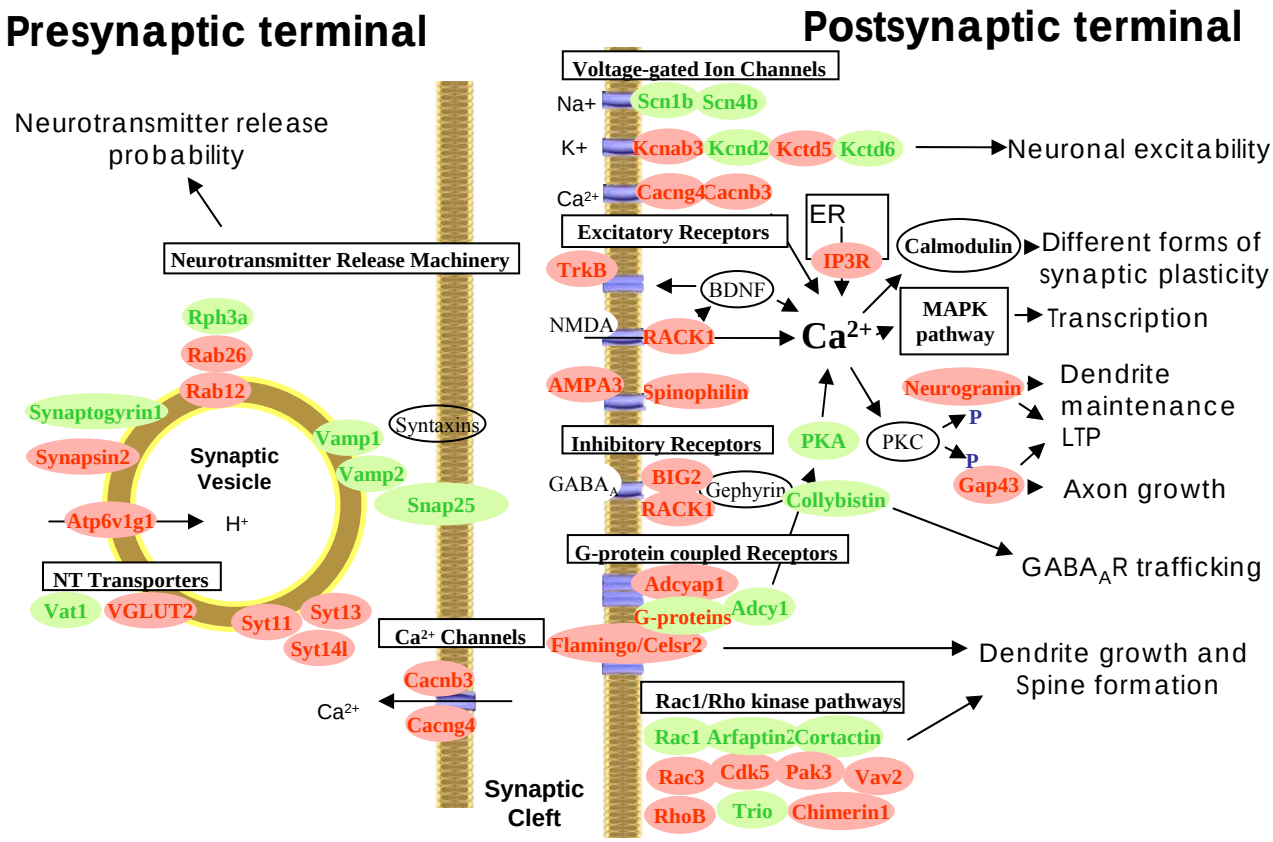
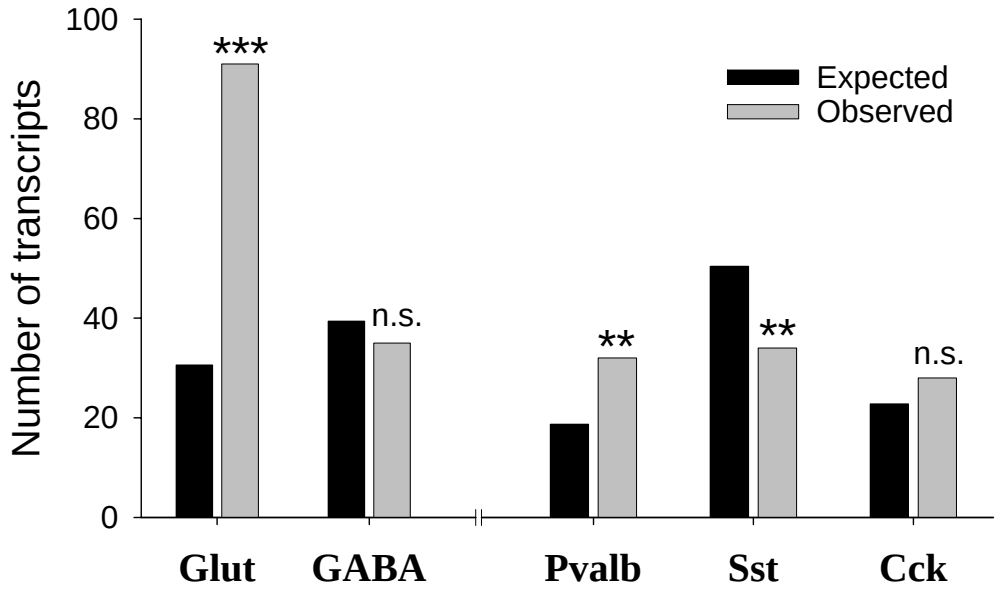


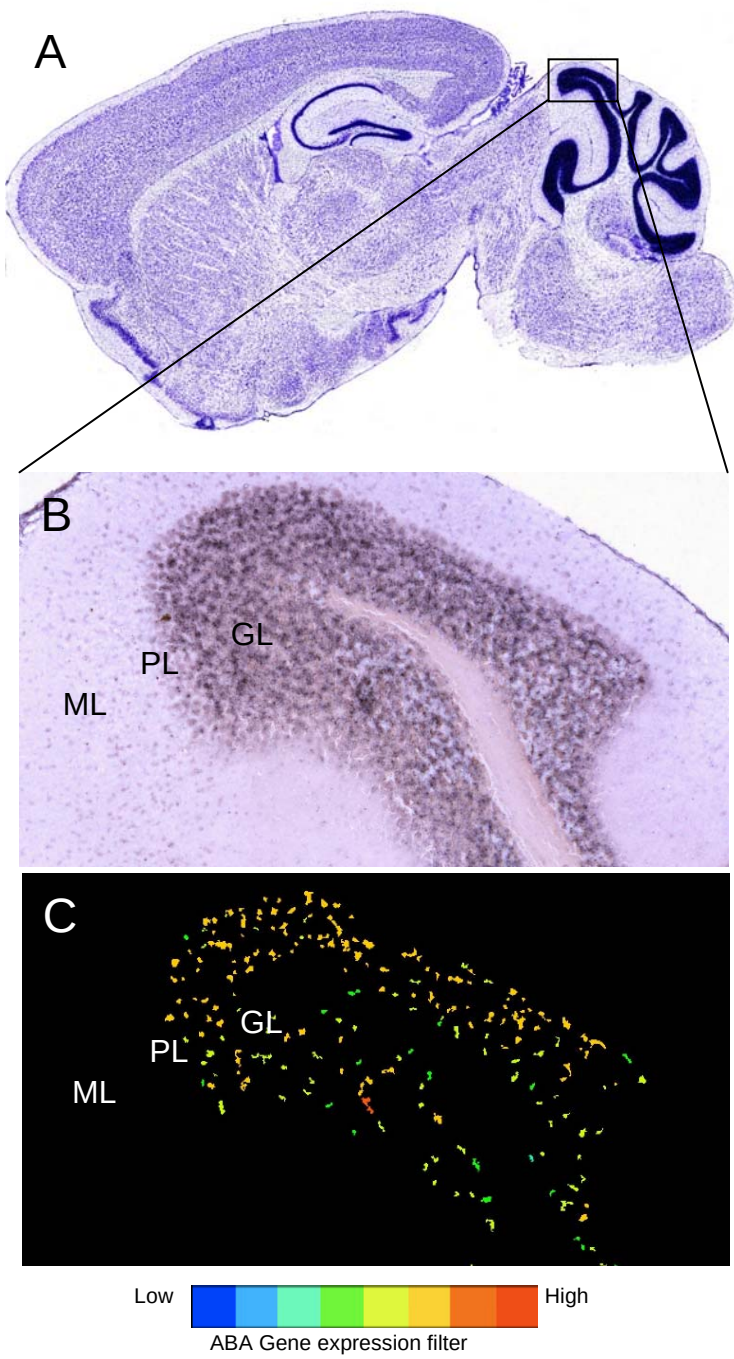

Supplemental figure 1. Overall results of t-tests for data sets collapsed across two knock-out lines. From nearly 17000 genetic elements present on the array, 10834 and 6852 transcripts passed "expression" filtering threshold for cerebellum and cortex, respectively. The p-value distributions shown for cerebellum (A) and cortex (B) were used to estimate FDR and calculate a q-value for each gene. A solid line in the middle of each graph represents chance distribution. The number of transcripts with $p<0.05$ was over 1400 and 700 and the false positive rate did not exceed 28% and 35% for cerebellum and cortex, respectively. A greater number of transcripts with lower p-values indicate a non-random regulation of genetic expression caused by the deletion of $GABA_A$ α1 gene. To reduce the number of nonspecific changes driven by a single knock-out line, we applied an additional filter requiring the same direction and at least a strong tendency ($p<0.2$) for transcriptional changes in both mutant lines. The number of transcripts that passed this statistical threshold was 688 and 326 for cerebellum (C) and cortex (D) respectively. Negative fold change values indicate down-regulation and positive up-regulation of mRNA in knock-out lines. More transcript up-regulation was detected in knock-out cerebellum, while analyses of cortex revealed the opposite tendency (both significant by chi-square test, $p<0.02$).



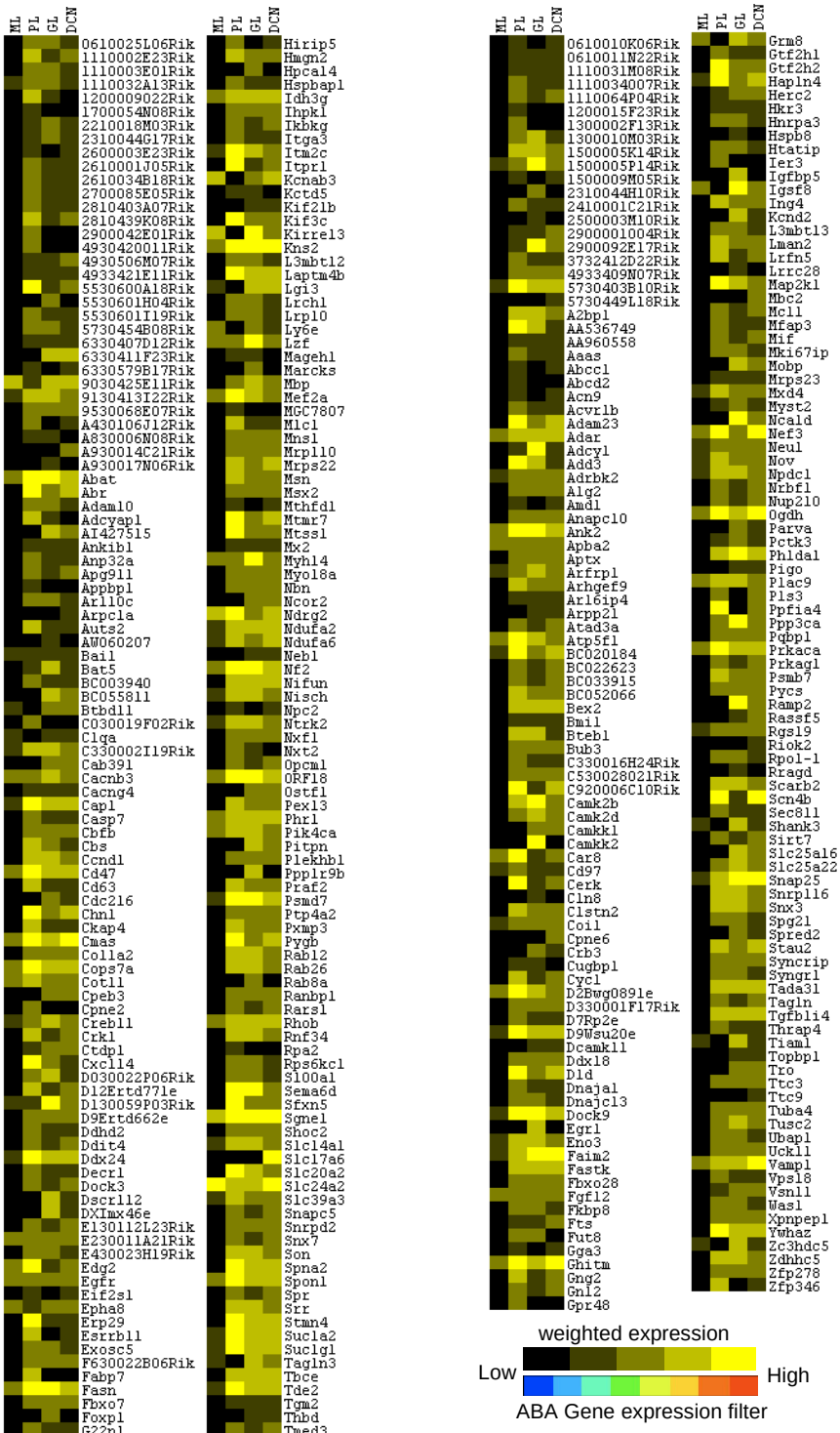

Supplemental figure 2. Schematic representation of a synapse with transcriptional changes that may underlie neuronal plasticity in α1 knock-out mice. Transcripts regulated in cortex and cerebellum are shown together for simplicity. Transcripts encircled in ovals represent single genes. Transcripts in green ovals are down-regulated and in red are up-regulated in knock-out lines. Ovals with white background are links in a pathway and represent transcripts not-regulated or not detected on array. Also shown are processes that have been implicated in synaptic plasticity.



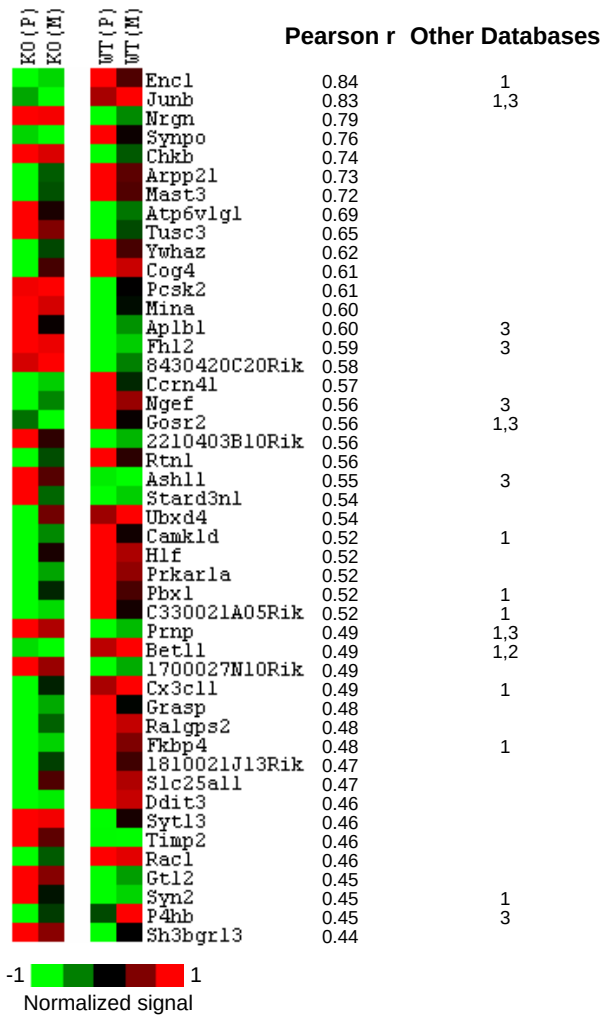

Supplemental figure 3. Cellular distribution of transcripts differentially regulated in cortex between KO and WT mice. Left panel: Number of transcripts enriched in either all glutamate (Glut) or all GABA neuronal populations. (MNED; t-test, $p<0.05$). Right panel: Number of transcripts enriched in Pvalb-positive, Sst-positive or Cck-positive GABA interneurons (enrichment of each GABA subclass was calculated based on comparisons with the other two GABA subclasses; MNED; t-test, $p<0.05$). The number of expected changes was calculated as a proportion based on pair-wise comparisons across all transcripts on the arrays used in MNED (n=22,690). Compared to chance, Glut-enriched and Pvalb-enriched genes were over-represented, Sst-enriched genes were under-represented. The abundance of all-GABA-enriched and Cck-enriched genes did not differ from chance. $\chi^2$ test: *** = $p<0.001$; ** = $p<0.01$; n.s. = not significant.



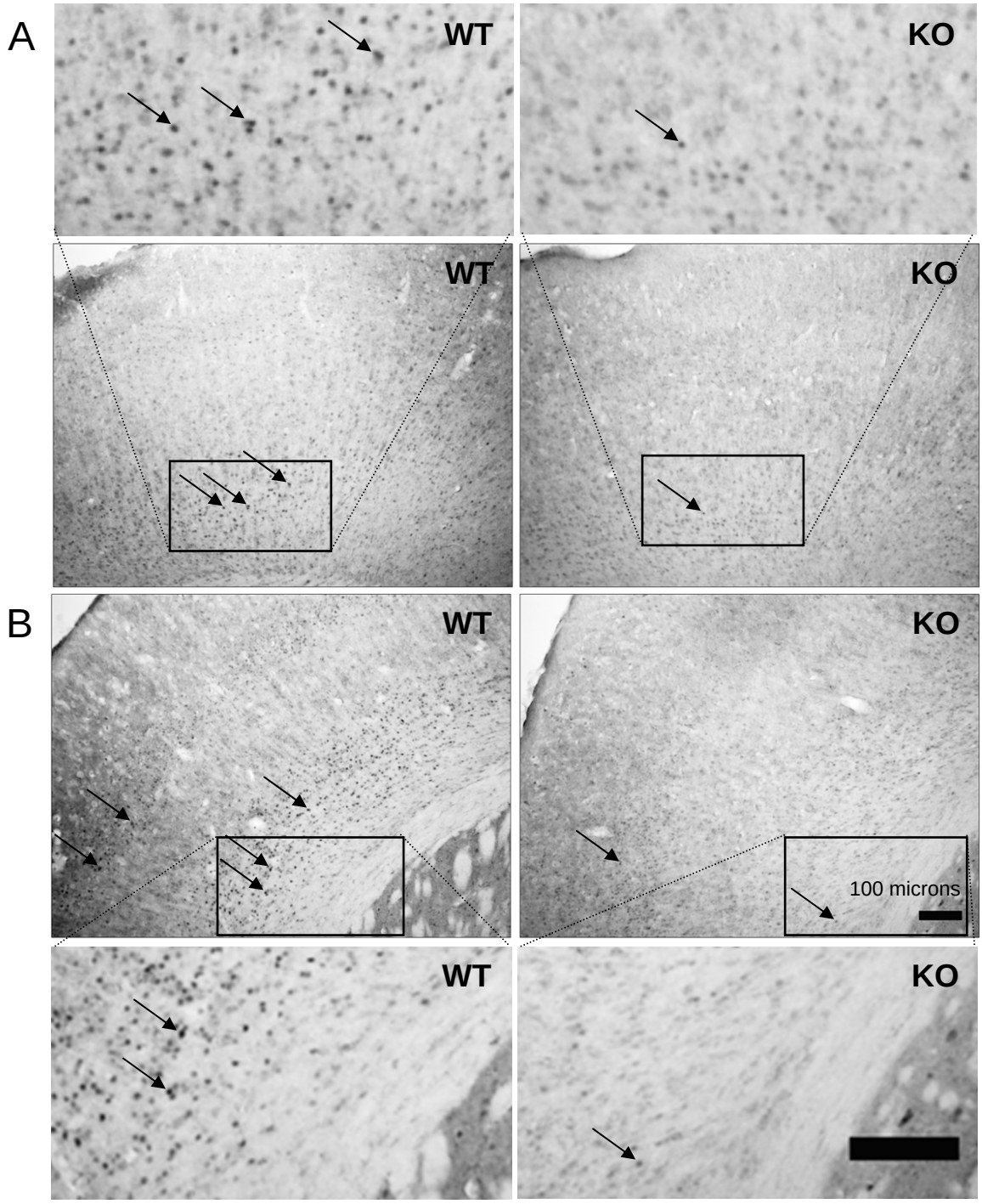

Supplemental figure 4. Distribution of $GABA_A$ α6 subunit in cerebellum shows a granule neuron pattern of expression. ABA images: A. whole brain, Nissl stain, B,C, cellular resolution of expression. ML: molecular layer, PL: Purkinje layer, GL: granule layer.




Supplemental figure 5. ABA expression of 425 cerebellum genes. Two left images: genes up-regulated in mutant mice. Two right images: genes down-regulated in mutant mice. ML: molecular layer; PL: Purkinje layer; GL: granule layer; DCN: deep cerebellar nuclei.




Supplemental figure 6. Potential targets of the transcription factor Egr1. Differential expression between knock-out (KO) and wild type (WT) lines is shown in pseudo-color. Average values for Pittsburgh (P) and Merck (M) lines are shown. Correlation coefficients (absolute values) with Egr1 abundance across 12 neuronal populations from MNED are shown in the middle (n = 12 times 3 replicates = 36; $r \geq 0.44$, $p < 0.01$). Evidence of correlated expression from other databases is listed on the right. Other databases: 1. Genes regulated in parallel with *Egr1* transcript in cortex following a subchronic exposure to ethanol in inhalation chambers; 2. Genes regulated in parallel with *Egr1* transcript in cortex following an acute injection of ethanol; 3. Genes correlated with *Egr1* transcript abundance in forebrain based on expression profiles of BXD RI strains. Data from sources 1 and 2 were collected in the lab of Dr. Susan Bergeson at the University of Texas at Austin and are submitted for publication (https://trip.icmb.utexas.edu/cgi-bin/genedb.pl) Source 3 data are from the GeneNetwork databases (http://www.genenetwork.org).




Supplemental figure 7. Representative immunohistochemistry slices for Egr1 show down-regulation in motor (A) and somatosensory (B) cortex of null mutants. Arrows indicate signal.