Supplementary Information:

Supplementary Discussion

Included here are several important points of discussion that could not be included in the main text due to space constraints. The reference numbers refer to Supplementary References at the end of Supplementary Information.

ATP–dependent chromatin remodeling has long been shown to be important in orchestrating changes in nucleosome architecture and chromatin state to affect important cellular processes such as transcription, DNA replication, and DNA repair[1,2], in addition to being implicated in many diseases[3-6]. Here we provide the first mechanistic study to causally implicate ATP–dependent chromatin remodeling factors in a psychiatric disorder. In particular, we show that upregulation of the ACF complex in NAc neurons is necessary for pro–susceptibility to stress and depressive–like behavior. The associated repression of a subset of genes is correlated with altered TSS nucleosome architecture at these genes, which suggests active nucleosome remodeling as a potential mechanism for the observed gene repression and behavioral effects. Due to the lack of technology to directly remodel nucleosomes at precise genomic loci in vivo, we cannot establish causality between active nucleosome remodeling at TSSs per se with the observed gene repression and stress susceptibility. However, our study is in agreement with

previous work demonstrating that yeast homologs of the ISWI and other families of chromatin remodeling complexes repress transcription by altering nucleosome positioning, in particular around TSSs[7-9]. Several studies in other systems have further confirmed ACF's transcriptional repressive role[10-14].

Knowledge of upstream regulators of chromatin remodeling factors, particularly in the nervous system, remains limited. Here we identified *Baz1a* as an activity–regulated gene (**Fig. 2a, b**), which has broader implications for the neuroscience field. Furthermore, we demonstrate, both in cultured striatal neurons as well as in NAc *in vivo*, that BDNF increases *Baz1a* expression (**Fig. 2a,d**). These findings support the scheme that increased burst firing of VTA neurons and the release of BDNF in NAc, both previously causally implicated in mediating susceptibility to social defeat stress[15,16], may be responsible for the persistent upregulation of BAZ1A in NAc of susceptible mice. Detailed dissection of the upstream signaling pathways underlying CSDS-mediated induction of BAZ1A is an important question for future studies.

While upregulated BAZ1A is necessary for susceptibility to social defeat stress (**Fig. 3c,e**), it is, by itself, not sufficient to induce depressive–like behaviors without the co–occurrence of stress (**Fig. 3d**, **Supplementary Fig. 4a,b**). We show that BAZ1A induction after CSDS is associated with increased levels of ACF complex formation with SMARCA5 (**Supplementary Fig. 1f**), either through recruitment of SMARCA5 from the reserve pool or through competition with other ISWI accessory subunits for SMARCA5 binding. We disprove the latter hypothesis at least for BAZ1B–SMARCA5 complexes, although this could not be ruled out completely for other ISWI accessory subunits due to the lack of suitable antibodies. However, in our behavioral studies, the overexpression of both BAZ1A and SMARCA5 was required to increase susceptibility to subthreshold social defeat stress (**Fig. 3a,b**). One possible explanation for this

observation is that CSDS induces multiple molecular changes in NAc, in addition to induction of BAZ1A per se, which enables it to induce susceptibility. These changes could include post–translational modifications that would favor the formation of ACF and their binding to their gene targets. It is possible that some of these molecular changes do not occur in the subthreshold defeat paradigm, such that the co–overexpression of both BAZ1A and SMARCA5 is needed to overcome the lack of these other molecular changes. Ultimately, future experiments are needed to address the biochemical properties of the ACF and its recruitment to target genes. Another key point to note here is that, although ACF induction is a key regulator for susceptibility to stress–induced depressive–like behavior, it is not the only regulator. Additionally, those genes that are repressed by the ACF likely represent only a subset of genes repressed in susceptible animals after CSDS. Furthermore, other regulators mediate induced gene expression in susceptible animals that are unexamined here. For example, our analysis of nucleosome remodeling factors alone revealed several other regulators that may contribute to stress susceptibility or resilience (**Supplementary Fig. 1a**). This study is thus only the beginning of examining ATP–dependent nucleosome remodelers in the pathophysiology of depression.

In contrast to NAc, we showed no effect of CSDS on *Baz1a* levels in medial prefrontal cortex (mPFC), and no effect of ACF complex overexpression in mPFC on stress susceptibility. It will be interesting in future studies to examine a possible role for ACF complexes in other depression–related brain regions. As well, all of our CSDS studies were performed by necessity on male mice (CSDS does not work for C57BL/6J females), while <25% of our human postmortem samples were female. This small sample size did not give us statistical power to separately analyze *BAZ1A* regulation specifically in depressed females. Therefore, to address this important question, we utilized a second mouse model of depression—chronic unpredictable

stress—and found similar induction of *Baz1a* in NAc of stressed female and male mice (**Fig. 1d**), which suggests the involvement of BAZ1A in female depression as well. Finally, the majority of our human depression patients died from suicide, which may represent a specific subpopulation of depressed patients. These factors should be examined in future studies as more postmortem human depression samples become available.

Although ChIP–seq with nucleosome remodeling factors, especially for microdissected brain samples, are notoriously difficult and typically generate noisy data, we have succeeded here in generating quality data that successfully identified *bona fide* targets of the ACF. As reported in Results, we conducted several important quality control analyses to ensure good signal to noise ratio and good reproducibility between replicates of the same condition. Instead of using traditional peak calling methods typically used for histone marks, we used ChromHMM both to deal with the sometimes broad and diffuse signals of chromatin remodeling factors and to identify genomic loci with coinciding enrichment of both BAZ1A and SMARCA5 (ACF complex). Bioinformatics analysis as well as visual inspection of the ChIP–seq tracks (**Fig. 4a,b**, **Supplementary Fig. 5c–f**) revealed ACF enrichment in NAc of susceptible animals at many genic and intergenic loci. Additionally, we provided transcriptional validation of identified ACF targets: ~60% showed repressed expression in NAc of susceptible animals and ~40% of those showed reversal of repression upon BAZ1A knockdown (**Fig. 5a,b**). Furthermore, we provided functional validation—which is rarely incorporated into next generation sequencing studies: several of the validated targets control CSDS behavior (**Fig. 5c,d**). By contrast, the observation that none of randomly generated genes, which did not show ACF enrichment by ChIP–seq, showed transcriptional and functional validation is further proof of the specificity and utility of our ChIP–seq dataset (**Supplementary Fig. 7e–g**).

The nucleosome maps generated here will serve as an important resource for future studies. As discussed above, the increased ACF in susceptible animals is associated with nucleosome positioning/shift events at the NDR and the –1 nucleosome. However, additional nucleosome occupancy and shift changes were observed after CSDS (**Fig. 4c**, **Supplementary Fig. 6b,c**), for example, the general decrease in nucleosome occupancy at the +1 nucleosome, an effect observed for genes in susceptible and resilient animals independent of changes in ACF binding (**Supplementary Fig. 6d**). Such effects could be mediated by other chromatin remodeling factors that are regulated by CSDS (**Supplementary Fig. 1a**), an interesting subject for future investigations.

Supplementary Figure Legends

Supplementary Figure 1 Regulation of ATP–dependent chromatin remodelers after social defeat. **(a)** NAc mRNA levels of genes from families of ATP–dependent chromatin remodelers and their accessory units 10 days after CSDS. Numbers in boxes indicate fold change compared to control animals. Red and green represent increases and decreases, respectively, in mRNA levels compared to control; intensity of color is representative of fold change. **(b)** NAc mRNA levels of *Baz1a*, *Baz1b*, and *Smarca5* 10 days after CSDS. **(c)** NAc protein levels of BAZ1A, BAZ1B, and SMARCA5 10 days after CSDS. **(d)** NAc mRNA levels of *Baz1a*, *Baz1b*, and *Smarca5* 28 days after CSDS. **(e)** NAc protein levels of BAZ1A, BAZ1B, and SMARCA5 28 days after CSDS. (f) Immunoprecipitation of SMARCA5–containing complexes in NAc 48 hours after CSDS. (g) ISWI mRNA in NAc 48 hours after a single 5–min defeat session. (h) mPFC mRNA levels of *Baz1a*, *Baz1b*, and *Smarca5* 48 hours after CSDS. (i) mPFC mRNA levels of *Baz1a*, *Baz1b*, and *Smarca5* 10 days after CSDS. Post–hoc Student's t–test * $P < 0.05$ compared to respective controls.

Supplementary Figure 2 Regulation of *Baz1a* mRNA levels and social interaction by optogenetic stimulation. (a) *Baz1a* mRNA levels in NAc after chronic optogenetic stimulation of medial prefrontal cortex. (b) *Baz1a* mRNA levels in NAc after chronic optogenetic stimulation of hippocampus. (c) Social interaction after chronic VTA to NAc optogenetic stimulation in control animals. Paradigm for the experiment was as shown on the left panel.

Supplementary Figure 3 Regulation of social interaction behavior by ACF. (a) (b) Validation of HSV–BAZ1A and HSV–SMARCA5, respectively, for their protein overexpression in NAc *in*

vivo. (c) ACF overexpression in mPFC does not affect social interaction (SI, left panel) or sucrose preference (SP, right panel) after subthreshold defeat. Experimental paradigm is shown in the panel above. (d) Validation of AAV–BAZ1A miR in NAc *in vivo*. (e) (f) Correlation between *Baz1a* mRNA levels in NAc and (e) social interaction ratio and (f) sucrose preference ratio. The same animals were used as in Fig. 3c. Post–hoc Student's t–test * $P < 0.05$ vs respective controls.

Supplementary Figure 4 Lack of regulation of behavior by ACF manipulation in control animals. Experimental paradigms were shown above each panel. (a) Overexpression or ACF in NAc of control, non–stressed animals does not alter social interaction (SI, left panel), anxiety–related behavior in elevated plus maze (EPM, middle panel), and sucrose preference (SP, right panel) behaviors. (b) Knockdown of BAZ1A in NAc of control, non–stressed animals does not alter social interaction (SI, left panel), anxiety–related behavior in elevated plus maze (EPM, middle panel), or sucrose preference (SP, right panel) behaviors. (c) ACF overexpression in NAc does not affect locomotor behaviors in control animals. (d) ACF overexpression in NAc does not affect freezing behavior in contextual fear conditioning.

Supplementary Figure 5 BAZ1A and SMARCA5 ChIP–seq visualization. (a) IGV browser track view of BAZ1A and SMARCA5 ChIP–seq peaks in NAc with enrichment over input. (b) Corrgram package analysis was conducted in R for the correlation. The upper right panel is the reflection of correlation r value. The size of the pie reflects the value of r, blue reflects positive correlation. The lower left panel is a variation of a correlation scatterplot. The correlation ellipses are the reflection of the scatterplot of two variables, showing the confidence intervals, and the

red lines are the LOWESS smoothed curve. $P < 2.2 \times 10^{-16}$ for correlation. For (c)–(f), IGV visualization of BAZ1A and SMARCA5 ChIP–seq tracks at specific genomic loci (c–*Rab3* locus, d–*Agtr1b* locus, e/f–intergenic loci) in either separate tracks (left panel) or overlaid tracks (right panel) in NAc of control/susceptible/resilient animals. IGV 2.3 (Broad Institute) was used for visualization of all ChIP–seq track.

Supplementary Figure 6 BAZ1A and SMARCA5 ChIP–seq analysis. (a) Breakdown of ACF complex enriched loci in NAc identified by ChromHMM for control/susceptible/resilient animals into intergenic and genic regions. (b) Breakdown of nucleosome occupancy changes for both susceptible vs control (upper panel) and resilient vs control (lower panel) comparisons. In addition to separating changes into intergenic and genic regions, the exact numbers of increased/decreased nucleosome occupancy events are also shown beside each section. The sizes of the sections are roughly proportional to the absolute amount of occupancy change events. (c) Breakdown of nucleosome shift/positioning changes for both susceptible vs control (upper panel) and resilient vs control (lower panel) comparisons. In addition to separating changes into intergenic and genic regions, the exact numbers of upstream/downstream shifts in nucleosome positioning are also shown beside each section. The sizes of the sections are roughly proportional to the absolute amount of shift change events. (d) Promoter nucleosome profile in NAc around TSS and NDR "height". Average nucleosome profile around TSSs for genes enriched for ACF binding sites in NAc of susceptible animals (upper left panel; for gene list, see Supplementary Table 4) as well as a randomly generated group of ~500 genes that do not exhibit ACF enrichment in susceptible animals (upper right panel; for gene list, see Supplementary Table 6). The promoter nucleosome profile for control/susceptible/resilient animals are overlaid for each

respective set of genes. NDR "height" for each set of genes was calculated by subtracting the normalized read count from top of the +1 nucleosome by the normalized read count from the bottom of the TSS and plotted on the bottom two panels. Where we defined the top of the +1 nucleosome and the bottom of the TSS was indicated on the overlaid nucleosome profile by blue and red arrows respectively. (e) Average (from 3 replicates) binding profile of BAZ1A in NAc 1000 bp up– and downstream of TSSs for genes as a function of their expression levels under control conditions, categorizing genes as low, medium, or high expression

Supplementary Figure 7 Regulation of depressive–like behavior by downstream target and non–target of ACF. (a)–(c) Validation of HSV–Rab3b and HSV–Agtr1b in NAc *in vivo*. (d) Correlation between RAB3B protein levels and how well the animal recovered from CSDS (post–surgery SI ratio – pre–surgery SI ratio). These were the same animals as in Fig 5c. (e) Lack of gene regulation in NAc by CSDS for 10/10 of these randomly generated non–targets of ACF complex. (f) Lack of regulation of these non–targets by BAZ1A knockdown in NAc. (g) PRMT5 overexpression did not rescue susceptibility after CSDS. Post–hoc Student's t–test * $P < 0.05$, *** $P < 0.001$ vs respective controls.

Supplementary Tables Legends

Supplementary Table 1 Demographic information for human depression patient cohort 1 and 2. For cohort 1, NAc tissue, obtained from the Dallas Brain Collection at UT Southwestern, was analyzed for depressed subjects and matched controls. Ht–height; Wt–weight; BMI: body mass index. For sex, M–male; F–female. For race: B–African–American; C–Caucasian. Y–Yes; N–No. For cohort 2, NAc tissue, obtained from the Quebec Suicide Brain Bank (QSBB; Douglas Mental Health Institute, Verdun, Québec), was analyzed for depressed subjects and matched controls. M–male; F–female, AD–antidepressant.

Supplementary Table 2 Quality control for ChIP–seq libraries. ChIP–seq data were aligned to the mouse genome (mm9) by CASAVA 1.8, and only unique reads were retained for analysis by ELAND. FastQC was applied for quality control, and then SAMTools was used to remove potential PCR duplicates[17]. PhantomPeak was applied to estimate the quality and enrichment (NSC and RSC) of the ChIP–seq dataset.

Supplementary Table 3 Correlation analysis between individual BAZ1A and SMARCA5 ChIP–seq replicates from NAc. The genome is split into non–overlapping bins 1 kb long and the number of short reads overlapping each bin is counted. Each read is assigned to the bin that is closest to its mapping location and is not double counted. To avoid unmappable regions and background regions that represent noise, an arbitrary cutoff of 2 across all replicates is used as a filter for each bin. The read counts are normalized to a standard value representing a library size of one million. Pearson correlations between all replicates are then calculated.

Supplementary Table 4 ACF binding sites in the NAc of control, susceptible, and resilient animals after CSDS. Coincident BAZ1A and SMARCA5 binding sites were extracted from ChromHMM analysis of ChIP–seq data as described in Methods. Control–enriched sites were defined as sites that were only found in control conditions but not within 2 kb in either susceptible or resilient conditions. The chromosomal locations of these sites are shown in the Table as Start to Finish, and annotated to the genome accordingly.

Supplementary Table 5 Nucleosome occupancy and shift events after CSDS. Nucleosome occupancy and shift events after CSDS were computed from H3 ChIP–seq data using DANPOS as described in Methods. The chromosomal locations of these events were shown in the Table as Start to Finish, and were then annotated to the genome accordingly.

Supplementary Table 6 List of randomly generated genes that do not have ACF enrichment in susceptible animals.

Supplementary Table 7 Complete qPCR primer list.

Detailed Figure Statistics

Fig 1.

(b) *Baz1a*: one–way ANOVA: $F(2,26) = 3.030$, $P = 0.066$; susceptible vs control: $t(20) = 2.389$, $P < 0.05$. *Baz1b:* one–way ANOVA: $F(2,26) = 1.947$, $P = 0.163$. *Smarca5*: one–way ANOVA: $F(2,26) = 0.081$, $P = 0.923$. Control: n = 10; Susceptible: n = 11; Resilient: n = 7.

(c) BAZ1A: one–way ANOVA: $F(2,17) = 3.176$, $P = 0.067$; susceptible vs control: $t(11) = 2.384$, $P < 0.05$. BAZ1B: one–way ANOVA: $F(2,18) = 1.444$, $P = 0.262$. SMARCA5: one–way ANOVA: $F(2,18) = 0.455$, $P = 0.642$. Control: n = 6–7; Susceptible: n = 7–8; Resilient: n = 6–7.

(d) Two–way ANOVA, Interaction: $F(1,25) = 0.1754$, $P = 0.679$; Stress: $F(1,25) = 12.84$, $P = 0.0014$; Sex: $F(1,25) = 5.180$, $P = 0.0317$. Control male vs Stressed male: $t(12) = 2.46$, $P < 0.05$. Control female vs Stressed female: $t(13) = 2.64$, $P < 0.05$. Control male vs Control female: $t(13) = 1.29$, $P = 0.22$. Stressed male vs Stressed female: $t(12) = 1.954$, $P = 0.074$. Control male: n = 7; Stressed male: n = 7; Control female: n = 8; Stress female: n = 7.

(e) Cohort 1 *BAZ1A*: $t(19) = 2.239$, $P < 0.05$. Control: n = 10–11; Depressed: n = 9–10. Cohort 2 *BAZ1A*: $t(27) = 2.490$, $P < 0.05$. Control: n = 9; Depressed: n = 20.

Fig 2.

(a) BDNF: $t(4) = 3.651$, $P < 0.05$; KCl: $t(4) = 5.413$, $P < 0.01$. Control: n = 3; KCl: n = 3; BDNF: n = 3.

(b) *Baz1a*: $t(24) = 2.26$, $P < 0.05$. Control: n = 18; ChR2: n = 8.

(c) Control vs ChR2: $t(14) = 3.913$; $P < 0.01$. n = 8 for all groups

(d) *Baz1a*: $t(15) = 2.156$, $P < 0.05$. Control: n = 8; BDNF: n = 9.

Fig 3.

(a) Left panel –one–way ANOVA: $F(3,76) = 2.888$; $P = 0.041$; GFP vs BAZ1A + SMARCA5: $t(39) = 2.797$, $P = 0.008$; BAZ1A vs BAZ1A+SMARCA5: $t(32) = 2.714$, $P = 0.011$; SMARCA5 vs BAZ1A + SMARCA5: $t(31) = 2.035$, $P = 0.050$. GFP: n = 27; BAZ1A: n = 20; SMARCA5: n = 19; BAZ1A + SMARCA5: n = 15.

Right panel – one–way ANOVA: $F(3,30) = 4.269$; $P = 0.0127$; GFP vs BAZ1A + SMARCA5: $t(14) = 2.420$, $P = 0.030$; BAZ1A vs BAZ1A + SMARCA5: $t(14) = 1.972$, $P = 0.069$; SMARCA5 vs BAZ1A + SMARCA5: $t(14) = 2.849$, $P = 0.013$. GFP: n = 9; BAZ1A: n = 9; SMARCA5: n = 9; BAZ1A + SMARCA5: n = 7.

(b) Left panel – $t(32) = 2.177$, $P = 0.037$; GFP: n = 23; BAZ1A + SMARCA5: n = 11. Middle panel – $t(15) = 2.660$, $P = 0.018$; GFP: n = 8; BAZ1A + SMARCA5: n = 9. Right panel – $t(15) = 1.884$, $P = 0.079$; GFP: n = 7; BAZ1A + SMARCA5: n = 10.

(c) Left panel – $t(19) = 2.320$, $P = 0.032$; GFP: n = 11; BAZ1A miR: n = 10. Middle panel – $t(19) = 1.851$, $P = 0.080$; GFP: n = 11; BAZ1A miR: n = 10. Right panel – $t(17) = 2.127$, $P = 0.048$ GFP: n = 10; BAZ1A miR: n = 9.

(d) GFP: n = 7; BAZ1A + SMARCA5: n = 7.

(e) two–way ANOVA, pre/post surgery: $F_{1,28} = 9.292$, $P < 0.01$; virus: $F_{1,60} = 4.795$, $P < 0.05$; interaction: $F_{1,28} = 5.120$, $P < 0.05$; AAV GFP pre vs post surgery: $t(14) = 0.5093$, $P = 0.618$; AAV–BAZ1A miR pre– vs. post–surgery: $t(14) = 4.171$, $P < 0.001$; post–surgery AAV–GFP vs AAV–BAZ1A miR: $t(14) = 2.816$, $P = 0.014$. GFP: n = 8; BAZ1A miR: n = 8.

Fig. 4

(a) $\chi^2(df = 2) = 225.638$, $P < 0.0001$. Control vs susceptible: $\chi^2(df = 1) = 128.48$, $P < 2.2 \times 10^{-16}$; susceptible vs resilient: $\chi^2(df = 1) = 165.893$, $P < 2.2 \times 10^{-16}$; control vs resilient: $\chi^2(df = 1) = 2.724$, $P = 0.099$. **(c)** Occupancy: $\chi^2(df = 1) = 15171.88$, $P < 2.2 \times 10^{-16}$. Shift: $\chi^2(df = 1) = 2434.98$, $P < 2.2 \times 10^{-16}$.

Fig. 5

(a) *Sdk2*: one–way ANOVA F(2,20) = 3.141, $P = 0.065$, susceptible vs control: t(13) = 2.112, $P = 0.055$, resilient vs control, t(14) = 0.0660, $P = 0.066$. *Rab3b*: one–way ANOVA F(2,11) = 2.083, $P = 0.061$, susceptible vs control: t(10) = 2.856, $P < 0.05$. *Runx1*: one–way ANOVA F(2,21) = 5.010, $P = 0.017$, susceptible vs control: t(14) = 2.644, $P < 0.05$. *Prdm16*: one–way ANOVA F(2,17) = 2.666, $P = 0.098$, susceptible vs control: t(10) = 2.856, $P < 0.05$. *Nit1*: one–way ANOVA F(2,20) = 2.447, $P = 0.112$, susceptible vs control: t(13) = 1.770, $P = 0.10$, resilient vs control: t(14) = 2.273, $P < 0.05$. *Kcnj6*: one–way ANOVA F(2,20) = 3.783, $P = 0.040$, susceptible vs control: t(13) = 2.925, $P < 0.05$. *Grid2*: one–way ANOVA F(2,20) = 4.203, $P = 0.030$, susceptible vs control: t(13) = 2.719, $P < 0.05$. *Cadps2*: one–way ANOVA F(2,20) = 3.068, $P = 0.069$, susceptible vs control: t(13) = 2.816, $P < 0.05$. *Zbtb7c*: one–way ANOVA F(2,21) = 2.293, $P = 0.126$, susceptible vs control: t(14) = 2.159, $P < 0.05$. *Agtr1b*: one–way ANOVA F(2,19) = 3.991, $P = 0.036$, susceptible vs control: t(12) = 3.343, $P < 0.01$. *Dmrt1*: one–way ANOVA F(2,19) = 4.284, $P = 0.029$, susceptible vs control: t(12) = 4.084, $P < 0.01$; resilient vs control: t(12) = 2.341, $P < 0.05$. *Plcz1*: one–way ANOVA F(2,20) = 3.458, $P = 0.051$, susceptible vs control: t(13) = 2.381, $P < 0.05$. *Espnl*: one–way ANOVA F(2,21) = 4.007, $P = 0.034$, susceptible vs control: t(14) = 2.803, $P < 0.05$. *Mad1l1*: one–way ANOVA F(2,19) = 3.059, $P = 0.071$, susceptible vs control: t(12) = 2.602, $P < 0.05$. *Zfp438*: one–way ANOVA

F(2,22) = 3.433, $P = 0.052$, susceptible vs control: t(13) = 1.978, $P = 0.07$, resilient vs control: t(13) = 2.172, $P < 0.05$. n = 5–8 for control, susceptible and resilient.

(b) *Rab3b*: t(19) = 3.319, $P < 0.01$. *Prdm16*: t(18) = 2.014, $P = 0.059$. *Zbtb7c*: t(20) = 2.166, $P < 0.05$. *Agtr1b*: t(19) = 2.091, $P = 0.05$. *Plcz1*: t(14) = 2.934, $P < 0.05$. *Espnl*: t(14) = 2.209, $P < 0.05$. GFP: n = 11–12; BAZ1A miR: n = 9–10.

(c) two–way ANOVA, pre/post surgery: $F_{1,36} = 5.02$, $P < 0.05$; virus: $F_{2,36} = 3.50$, $P < 0.05$; interaction: $F_{2,36} = 3.04$, $P = 0.05$; HSV–RAB3B pre vs post surgery: t(12) = 1.80, $P = 0.097$; HSV–AGTR1B pre– vs. post–surgery: t(10) = 1.95, $P = 0.080$; post–surgery HSV–GFP vs HSV–RAB3B: t(13) = 2.43, $P < 0.05$; post–surgery HSV–GFP vs HSV–AGTR1B: t(12) = 3.39, $P < 0.01$. GFP: n = 8; RAB3B: n = 7, AGTR1B: n = 6. **(d)** one–way ANOVA: F(2,12) = 3.81, $P = 0.051$; HSV–RAB3B vs HSV–GFP: t(8) = 1.938, $P = 0.089$; HSV–AGTR1B vs HSV–GFP: t(8) = 2.233, $P = 0.056$; n = 5 for all groups. **(e)** *RAB3B*: t(27) = 1.899, $P < 0.05$. *AGTR1*: t(27) = 0.887, NS. Control: n = 9; Depressed: n = 20.

Supplementary Fig. 1

(a) n = 8–10 for control, susceptible, or resilient.

(b) *Baz1a*: one–way ANOVA: F(2,43) = 3.674, $P = 0.034$; susceptible vs control: t(33) = 2.502, $p < 0.05$. *Baz1b:* one–way ANOVA: F(2,43) = 2.238, $P = 0.119$. *Smarca5*: one–way ANOVA: F(2,43) = 0.024, $P = 0.976$. Control: n = 18; susceptible: n = 17; resilient: n = 11.

(c) BAZ1A: one–way ANOVA: F(2,31) = 6.048, $P = 0.006$; susceptible vs control: t(19) = 3.748, $P < 0.05$; resilient vs control: t(22) = 2.305, $P < 0.05$. BAZ1B: one–way ANOVA: F(2,31) = 0.107, $P = 0.899$. SMARCA5: one–way ANOVA: F(2,33) = 0.408, $P = 0.668$. Control: n = 10–11; susceptible: n = 11–12; resilient: n = 13.

(d) *Baz1a*: one–way ANOVA: $F(2,22) = 3.193$, $P = 0.061$; susceptible vs control: $t(16) = 2.366$, $P < 0.05$. *Baz1b*: one–way ANOVA: $F(2,22) = 0.198$, $P = 0.822$. *Smarca5*: one–way ANOVA: $F(2,22)=1.354$, $P = 0.279$. Control: n = 10; susceptible: n = 8; resilient: n = 7.

(e) BAZ1A: one–way ANOVA: $F(2,17) = 3.040$, $P = 0.074$; susceptible vs control: $t(13) = 2.636$, $P < 0.05$. BAZ1B: one–way ANOVA: $F(2,12) = 0.0.106$, $P = 0.900$. SMARCA5: one–way ANOVA: $F(2,10) = 0.045$, $P = 0.956$. n = 4–8 for control, susceptible, or resilient animals.

(f) BAZ1A: one–way ANOVA: $F(2,18) = 2.674$, $P = 0.096$; susceptible vs control: $t(12) = 2.240$, $P < 0.05$. BAZ1B: one–way ANOVA: $F(2,18) = 1.123$, $P = 0.347$. Control: n = 8; susceptible: n = 6; resilient: n = 7.

(g) Control: n = 15; single defeat: n = 13.

(h) *Baz1a* in the mPFC: one–way ANOVA $F(2,16) = 3.183$, $P = 0.069$; control vs resilient: $t(11) = 2.223$; $P = 0.048$. n = 7–8 for all groups.

(i) n = 7–8 for all groups.

Supplementary Fig. 2

(a) mPFC control: n = 14; mPFC ChR2: n = 10.

(b) HP control: n = 5; HP ChR2: n = 6.

(c) n = 9 for control; n = 10 for ChR2.

Supplementary Fig. 3

(a) HSV–BAZ1A: $t(14) = 2.71$, $P < 0.05$. n = 7–9 for all groups.

(b) HSV–SMARCA5: $t(13) = 2.41$, $P < 0.05$. n = 7–9 for all groups.

(c) n = 7 for all groups

(d) $t(10) = 2.451$, $P = 0.034$. n = 5–7 for all groups.

(e) r = –0.23, $P = 0.1632$, n = 20.

(f) r = –0.39, $P = 0.048$, n = 19.

Supplementary Fig. 4

(a) n = 6–7 for all overexpression groups.

(b) n = 9–10 for all knockdown groups.

(c) n = 7 for all groups.

(d) n = 10 for all groups.

Supplementary Fig. 7

(a) HSV–RAB3B: $t(8) = 2.57$, $P < 0.05$. n = 5 for all groups

(b) HSV–AGTR1B: $t(8) = 5.10$, $P < 0.001$. n = 5 for all groups.

(c) HSV–RAB3B: $t(13) = 2.565$, $P < 0.05$. n = 8 for HSV–GFP, n = 7 for HSV–RAB3B.

(d) r = 0.6461, $P < 0.01$, n = 15

(e) n = 5–8 for control, susceptible and resilient.

(f) GFP: n = 12; BAZ1A miR: n = 9.

(g) n = 5 for all groups.

Supplementary References

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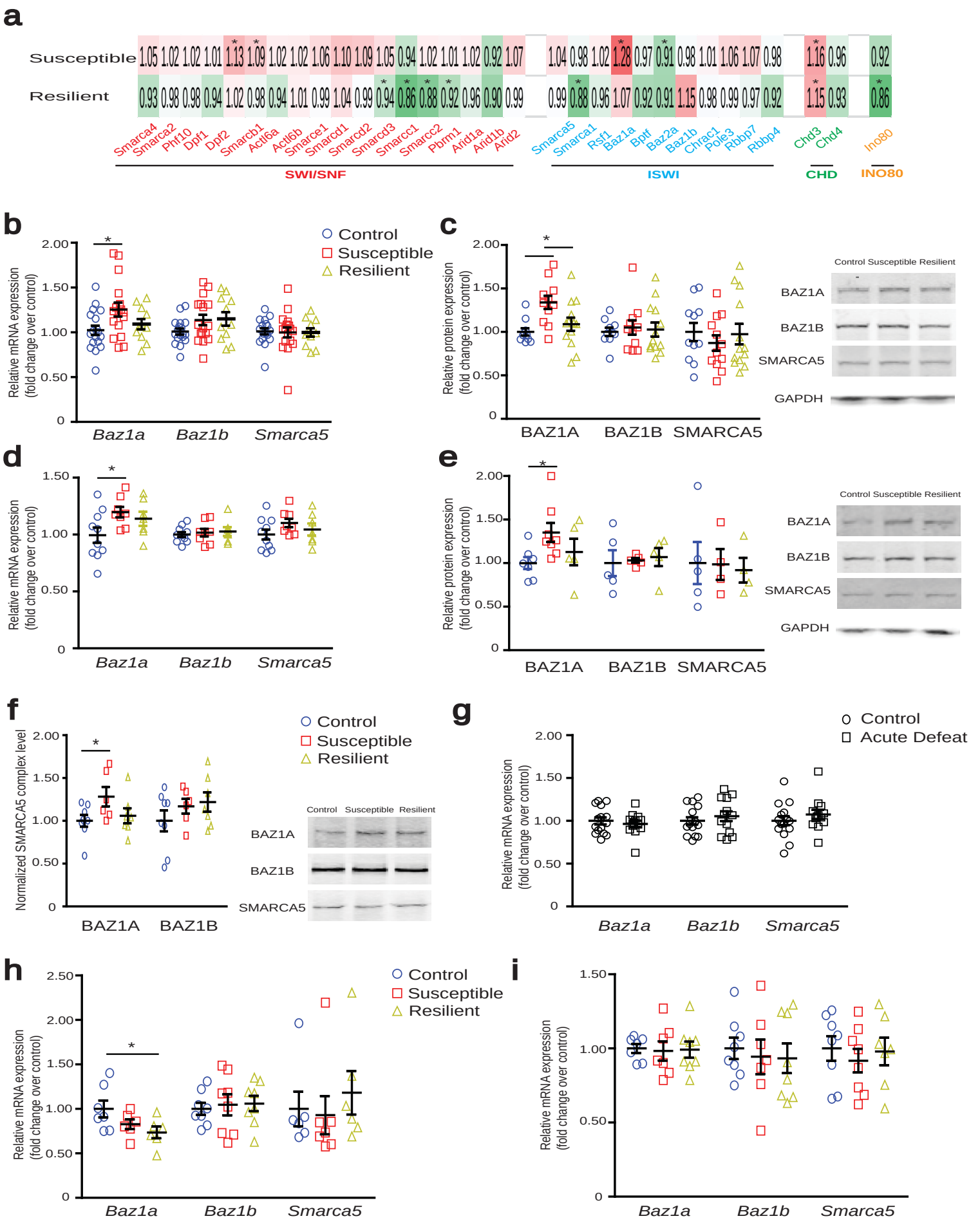
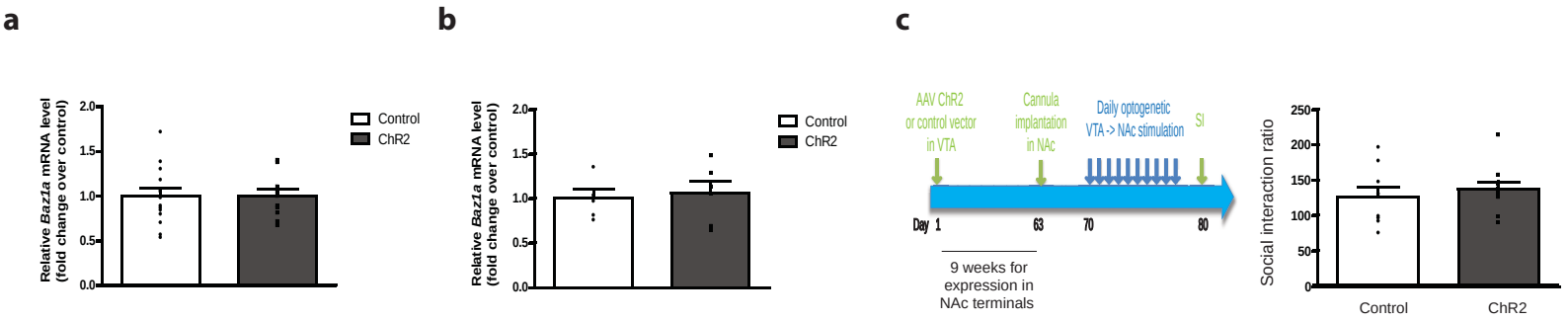
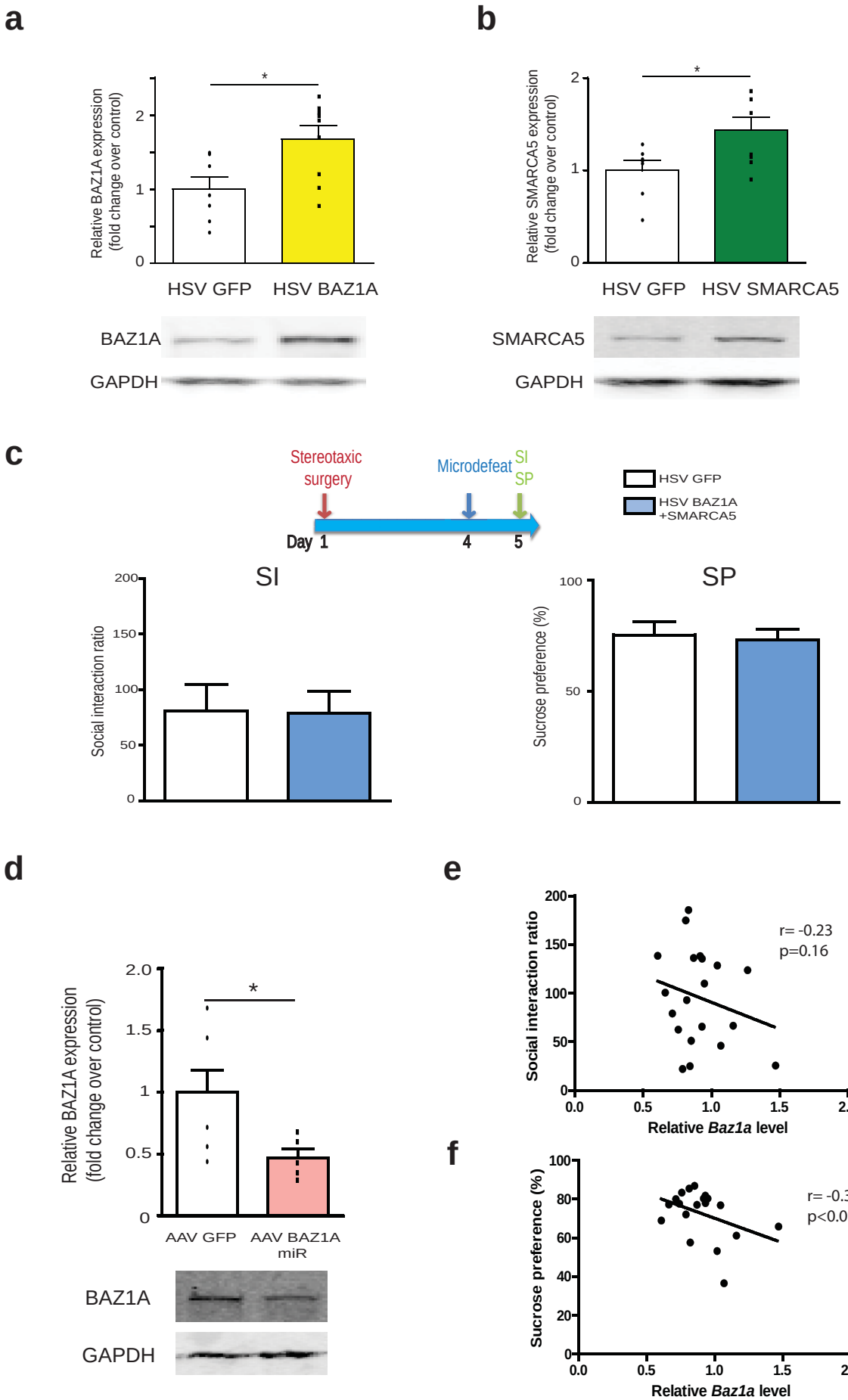


Supplementary Figure 1



Supplementary Figure 2



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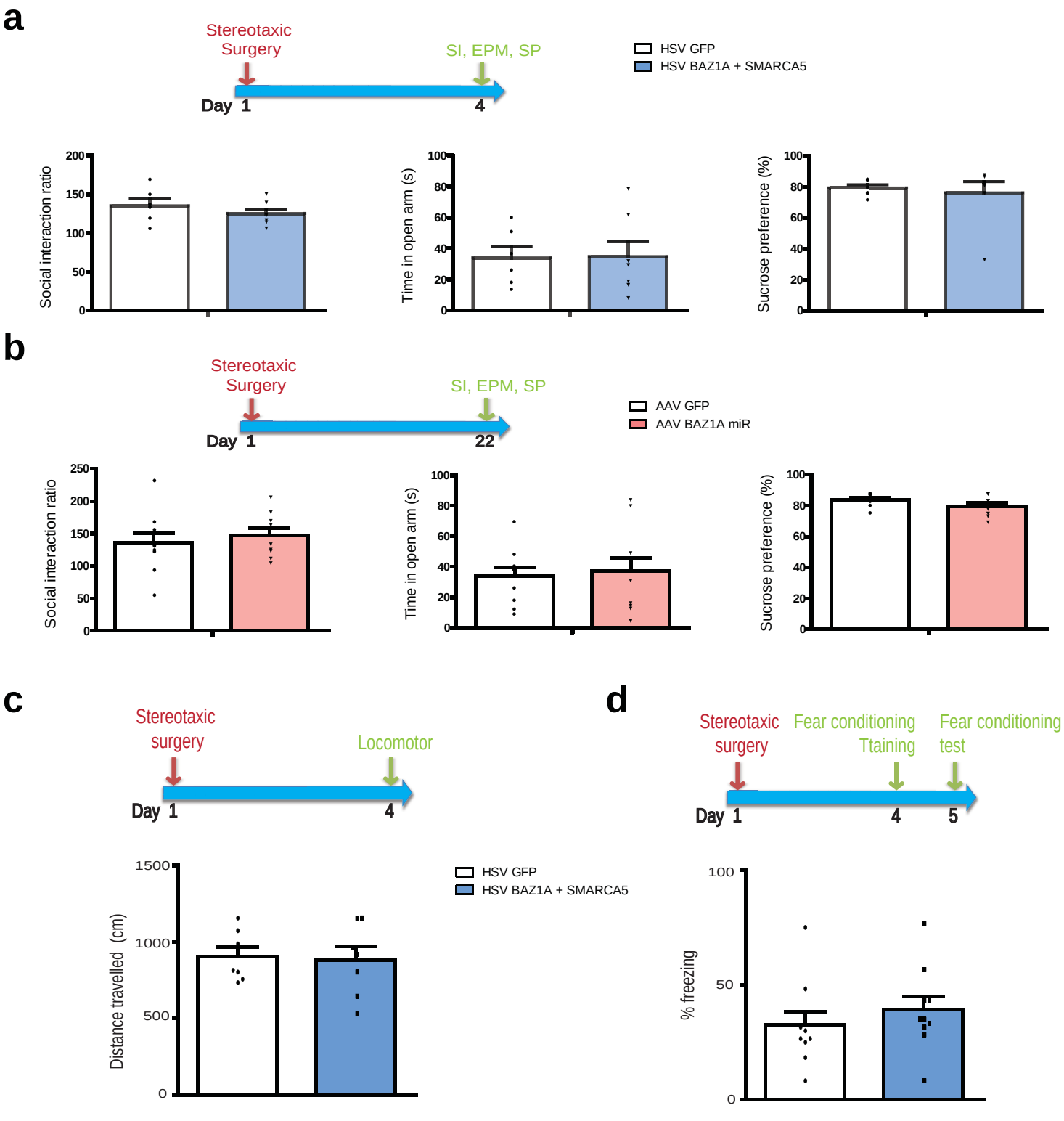
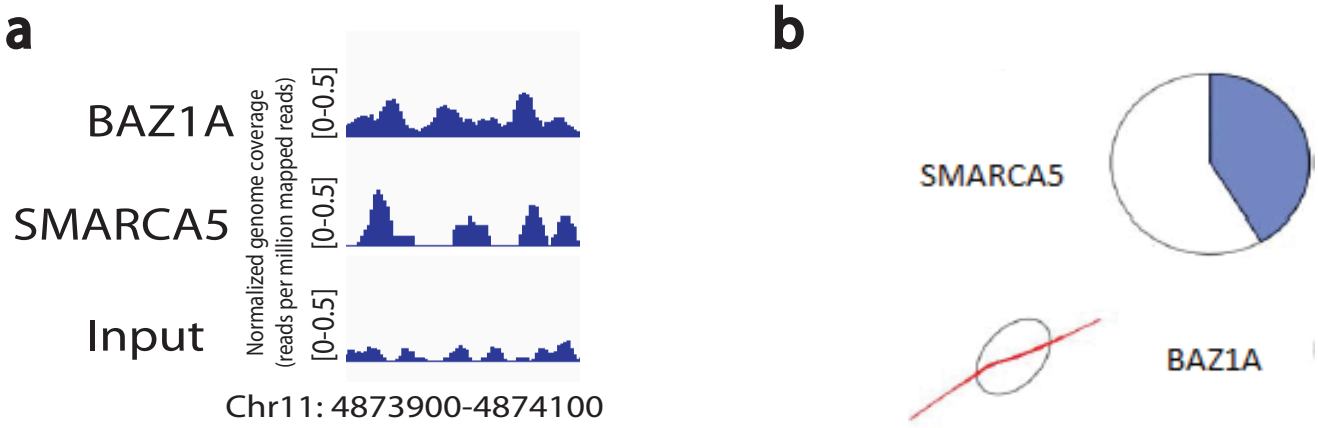
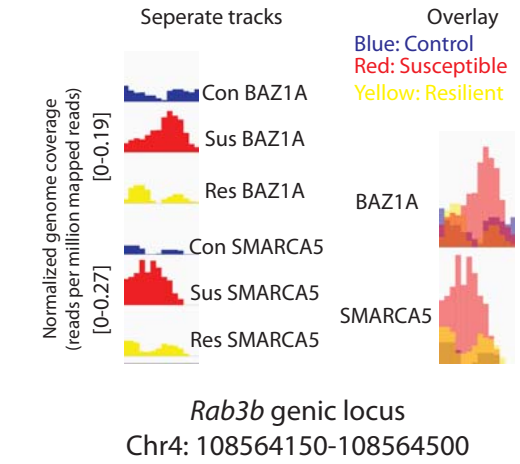
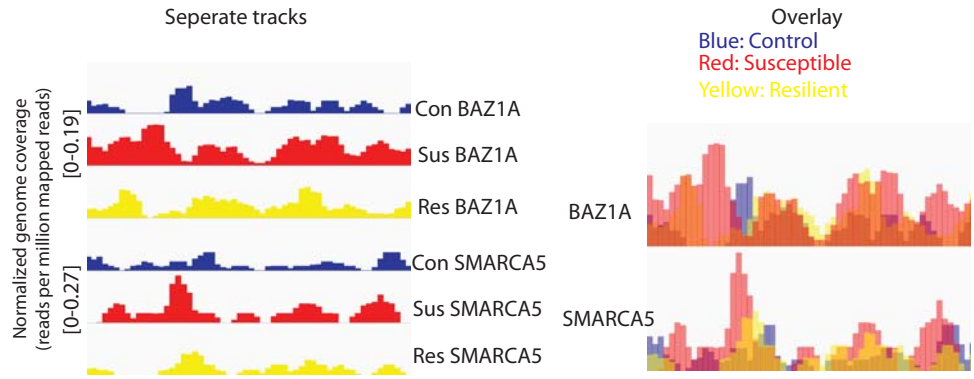


Supplementary Figure 4





Rab3b genic locus
Chr4: 108564150-108564500



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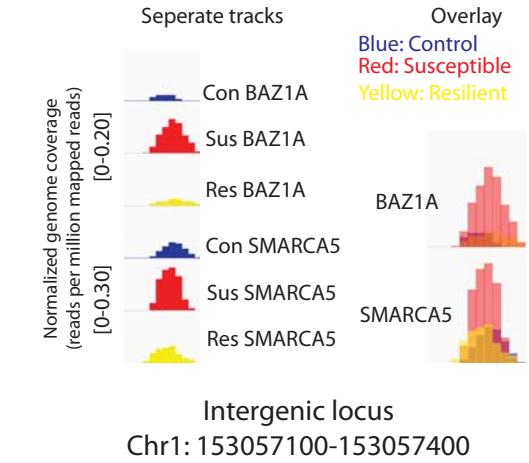

Intergenic locus
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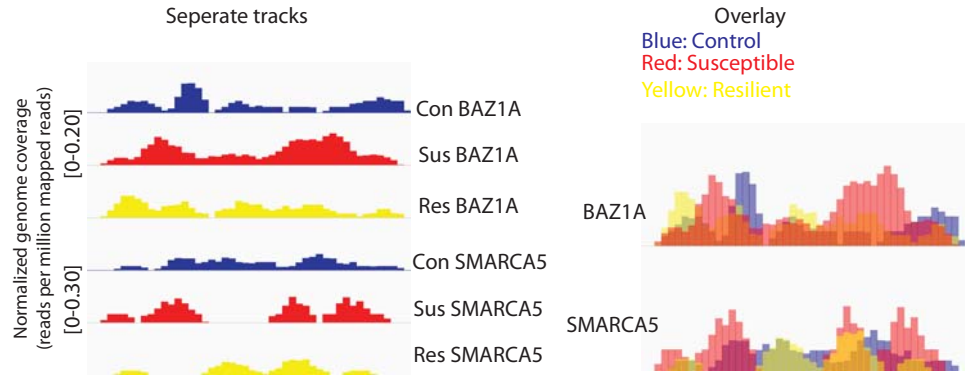
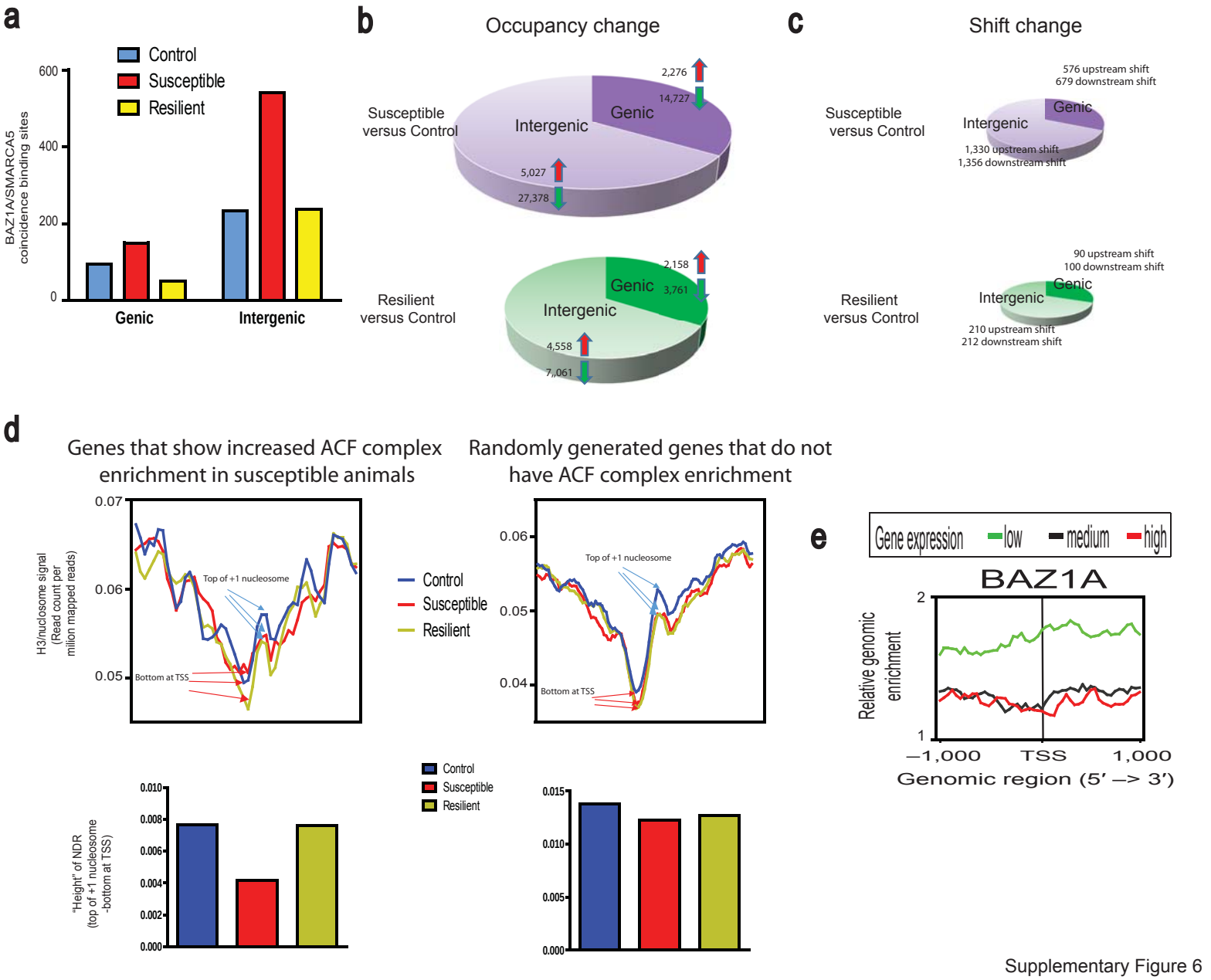
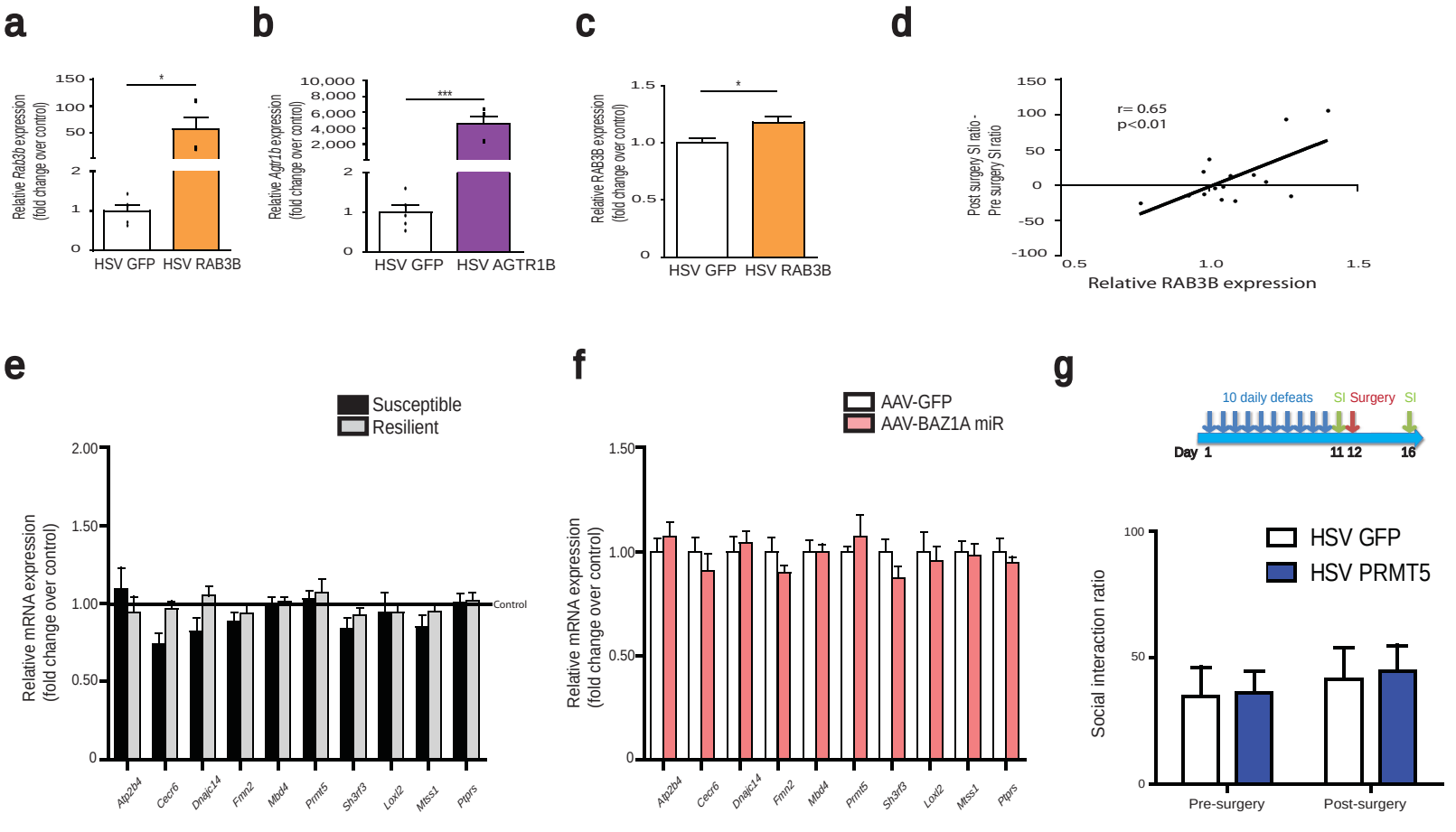


Intergenic locus
Chr12: 114699600-114700800

Supplementary Figure 5



Supplementary Figure 6



Supplementary Figure 7

Supplementary Table 1: Demographic information for depression human patients cohort 1 and 2

Cohort 1

Sex	Age	Race	Psychiatric Diagnosis	PMI Hours	RIN	pH	Ht	Wt	BMI	Cause of Death	Verbal/Emotional Abuse	Physical Abuse	Sexual Abuse	Prescription and Non Prescription Drugs	Toxicology Results
F	81	C	MDD w/ Psychotic features, Alcohol Abuse	7	8.9	6.076	66	130	21.0	Natural - COPD & lung cancer	Y	Y: molested by uncle when growing up.	Y	None Reported	None
F	83	C	Normal Control	30	8.5	6.31	59	146	29.5	Suicide - sharp force injuries	N	N	N	Celebrex 200mg; lipitor 10mg; Lotrel 5/20mg; Atacand 32mg; Remicade infusion; Methotrexate eight tablets; Timolol eye drops; Xalatan eye drops; Pepto-bismol; Calcium; Natural Tears; Tylenol Arthritis	All Negative
F	46	C	MDD	11	8.3	6.439	61	198	37.4	Natural - Hypertensive cardiovascular disease with part II obesity contributing	N	N	N	Chra Cal; Super B Complex; Premarin 1.25 mg; Furosemide 40mg; triheptal 150mg; Klorcon M10; Risperdal 4mg; Nexium 40mg; Toprol XL 50mg; Lexapro 10mg; Wellbutrin SR 150mg; Effexor 75mg; Albuterol Inhaler: Lorazepam 1mg	0.07 mg/L bupropion; 0.09 mg/L erythrodihydrobupropion; 0.79 mg/L threodihydrobupropion; hydozybupropion detected; 0.10 mg/L promethazine; 0.09 mg/L citalopram; 8.5 mg/L meprobamate; 6.7 mg/L carisoprodol; 0.077 mg/L hydrocodone; 0.02 mg/L alprazolam; 147 mEq/L Na+; 85 mEq/L K+; 120 mEq/L Cl-; 38 mg/dL Glu; 20 mg/dL VUN
M	25	C	Depression NOS, Opiate Dependence	21	8.9	6.792	68	212	32.2	Suicide - Hanging	Y-picked on at school age 16 for announcing that he was gay.	N	Y-raped at age 14 by another male	Adderol 20mg; Klonopin 0.5 mg; Lexapro 10mg	1.4 mg/L Diphenhydramine; 0.02 mg/L Citalopram
M	42	C	Depression NOS	15	9.1	6.799	68	202	30.7	Suicide - Hanging	U	U	U		0.22% Ethanol
M	24	C	MDD, severe, recurrent, no psychosis, opiate abuse, alcohol dep.	23	9.1	6.446	61	186	35.1	Suicide - oxycodone overdose (maybe accidental)	U	U	U	Gabapentin 400mg; Sucralfate 1gm; Nexium 40mg; Cymbalta 60mg; Seroquel 100mg; Campral 333mg; Hydoxyzine 50mg; Seroquel 200mg; Desipramine 50mg; Propoxy-N/APAP 100-650; Hydromorphone 4mg; Oxycontin 20mg	0.24 mg/L Desipramine; 0.41 mg/L Oxycodone BLOOD: 3.87 mcg/mL Gabapentin; 120 ng/mL Duloxetine
M	40	C	MDD, Severe, Reccurent wo psychosis	18.25	6.9	6.741	68	168	25.5	Suicide - toxic effects of doxepin and citalopram	U	U	U	Doxepin 100mg; Citalopram 20mg	1.4 mg/L Doxepin; 0.14 mg/L Demethyldoxepin; 0.47 mg/L Citalopram
M	31	C	Normal Control	16.16	8.1	6.823	66	130	21.0	Natural - Hypertensive cardiovascular disease w/ diabetes mellitus & chronic kindey failure contributing	U	U	U	HTN medicine	0.03 mg/L Atropine
M	63	C	Normal Control	14	7.7	6.555	72	180	24.4	Natural - acute MI with prior MI's and hypertension	N	N	Y-raped age 9 by friend of his father	None Reported	None
F	35	C	Opiod dependence, depression NOS	9	7.4	6.344	68	134	20.4	Suicide - toxic effects of acetaminophen and hydrocodone	N	N	N	Depot Lupron; Xanax; Prozac; Estrace; Bentyl; Vicodin (21); Ativan	0.02 mg/L Dihyrocodeine; 11 mg/L Meprobamate; 1.9 mg/L Carisoprodol; 67 mg/L Acetaminophen; .20 mg/L Hydrocodone.
M	19	C	Normal Control	20	9	6.641	64	125	21.5	Homicide - gunshot	N	N	N	None Reported	None
M	48	C	Normal Control	14.65	10	6.822	68	200	30.4	Natural - mitral valve regurgitation	N	N	N	Benicar; Lipitor; folbee; Tessalon Perles; Concerta; Profen Forte; Soma	None
M	61	C	MDD	20	8.6	6.846	68	180	27.4	Suicide - suffocation w plastic bag and helium tank	N	N	N	Etodolac 500mgs	All Negative
F	57	C	Depression NOS/Opioid Abuse	16	6.6	6.437	67	185	29.0	Natural - cardio hypertrophy of unknown etiology	N	N	N	Hydocodone APAP5 500mg; Alprazolam 2mg; Fluoxetne 40mg; Trazodone 50mg; Imetrex; Gen-Sumatriptan 100mg; Unisome; Lexapro 20mg;	0.13 mg/L Norfluoxetine; 1.4 mg/L Diphenhydramnine; 0.35 mg/L Methadone; 0.25 mg/L Citalopram; 103 mg/L Salicylate; 0.06 mg/L Alprazolam; 0.046 mg/L Hydrocodone
F	65	C	Normal Control	11	6.4	6.838	64	140	24.0	Natural - cardiac tamponade due to hemopericardium due to aortic dissection due to arteriosclerotic cardiovascular disease	N	N	N	None Reported	0.06 mg/L atropine
M	43	C	Normal Control	15	9.5	6.886	75	325	40.6	Natural - Hypertensive cardiovascular disease associated with morbid obesity	N	N	N	Cardizem, Lipotrol, Lasix	None
F	59	C	Depression NOS	10.45	9	7.033	69	296	43.7	Natural - hypertensive - atherosclerotic cardiovascular disease w obesity contributing	U	U	U	Zoloft; Plavix; Cymbalta; Furosemide; Vitorin; Apapro; Metoprolol.	All Negative
M	61	C	Depression NOS	19	7.5	6.01	69	164	24.2	Suicide - gunshot wound to trunk	N	N	N	None Reported	1.0 mg/L Lidocaine; Etomidate detected
M	34	C	Normal Control	23.35	9.9	6.17	70	290	41.6	Natural - Hypertensive and atherosclerotic cardiovascular disease w/ contributing factors of uncontrolled diabetis mellitus	N	N	N	None Reported	0.03 mg/L Diphenhydramine; 0.05 mg/L Diltiazem; 0.02 mg/L Atropine
F	41	B	Depression NOS	13	2.9	5.77	63	198	35.1	Natural - diabetic ketoacidosis	U	U	U	Mirtazapine 30mg; Citalopram 40mg; furosemide 40mg;	0.003% Acetone; 0.02 mg/L Mirtazapine; 0.04 mg/L Citalopram
F	50	C	Normal Control	23.3	4.8	6.02	65	130	21.6	Natural - Adenocortical insufficiency	U	U	U	Hydocodone; Dexamethasone; Triameterine; Demerol, Phenergan	0.08 mg/L meperidine; 0.19 mg/L demethylmeperidine; 0.09 mg/L Promethazine; 0.03 mg/L Diazepam; 0.09 mg/L Demethyldiazepam
M	60	C	Normal Control	20	8.5	6.63	69	155	22.9	Accident - possible adverse reaction to hyprine during angioplasty surgery	N	N	N	Tetracycline 500mg; Altace 5mg; Ecotrin 81mg; Finacea; Protonix; Methocarbonal 750mg; Ecodolac 500mg; Gentak	None
M	60	C	Normal Control	30	8.7	6.7	72	210	28.5	Natural - Cardiac arrest	N	N	N	Benazepril 40mg; Digoxin 0.25mg; Lipitor 10mg; Furosemide 40mg; Micro-K 10mEq; Tekturna 150mg; Flonase; ASA 325mg	None

Cohort 2

Group	Cause_Death	Age	Gender	Weight (g)	PH	Refrig_Delay (hrs)
Control	Natural	47	M	1412	6.49	12
Control	Natural	41	M	1376	6	24
Control	Accident	32	M	1516	6.67	29.5
Control	Natural	46	M	1210	6.42	19.5
Control	Suicide	25	M	1600	6.73	36
Control	Suicide	46	M	1600	6.83	59
Control	Accident	42	M	1470	6.75	63
Control	Suicide	41	F	1355.2	6.5	3.5
Control	Accident	76	F	1146.9	-9	7
Depressed + Positive AD	Suicide	42	M	1446	6.4	21
Depressed + Positive AD	Suicide	48	M	1452	6.79	21.5
Depressed + Positive AD	Suicide	22	M	1490	6.68	24
Depressed + Positive AD	Suicide	53	M	1500	6.89	41
Depressed + Positive AD	Suicide	39	M	800	6.37	18.5
Depressed + Positive AD	Suicide	35	M	1380	6.87	31
Depressed + Positive AD	Suicide	40	F	1290	6.81	49.5
Depressed + Positive AD	Suicide	47	M	1690	6.79	2.5
Depressed + Positive AD	Suicide	63	M	1900	6.6	24
Depressed + Positive AD	Suicide	49	F	1350	7.5	14.5
Depressed + Positive AD	Suicide	64	M	1395.5	6.25	6.5
Depressed + Negative AD	Suicide	52	M	1370.5	6.3	29
Depressed + Negative AD	Suicide	53	M	1507	6.64	14
Depressed + Negative AD	Suicide	39	M	1264	6.6	25.5
Depressed + Negative AD	Suicide	49	M	1280	6.57	32
Depressed + Negative AD	Suicide	53	M	no info	6.91	33.5
Depressed + Negative AD	Suicide	68	M	1100	6.93	32
Depressed + Negative AD	Suicide	63	M	1420	6.95	50
Depressed + Negative AD	Suicide	55	F	1350	6.5	2.5
Depressed + Negative AD	Suicide	49	M	1432.8	6.7	2.5

Supplementary Table 2: Quality control for ChIP-seq libraries

Sample_id	Total Raw Reads	Uniquely mapped reads	Reads after deduplication	NSC	RSC
Input_C	55742015	40420806	30603518		
Input_R	89700862	64739824	57989097		
Input_S	71408627	50925269	44841921		
Con1_BAZ1A	57958460	41389290	11271883	1.23	1.35
Con2_BAZ1A	36320759	25696086	22620464	1.09	2.36
Con3_BAZ1A	73363435	53517331	32204009	1.07	2.17
Res1_BAZ1A	89762379	60353823	46965680	1.06	3.3
Res2_BAZ1A	32754553	23389019	19198326	1.11	4.15
Res3_BAZ1A	58452461	41878843	21335551	1.16	2.5
Sus1_BAZ1A	76287210	53921446	39166385	1.07	1.71
Sus2_BAZ1A	68188409	49048607	46829508	1.02	2.34
Sus3_BAZ1A	92411989	66387754	49700386	1.06	2
Con1_SMARCA5	59081146	36494423	5499690	1.86	1.51
Con2_SMARCA5	59372588	39726198	25824438	1.13	1.85
Con3_SMARCA5	92747357	63564841	37174774	1.11	1.72
Res1_SMARCA5	61312955	38704149	8386450	1.52	1.63
Res2_SMARCA5	103967904	69871399	44862383	1.09	2.24
Res3_SMARCA5	65156934	43466346	8421768	1.46	1.91
Sus1_SMARCA5	63906983	43373669	12990215	1.37	2.32
Sus2_SMARCA5	101966063	64887925	12691015	1.39	1.56
Sus3_SMARCA5	54414110	35390937	10943603	1.39	1.47
Con_input_H3	174568278	129375594	123003711		
Res_Input_H3	170821139	123553028	114844859		
Sus_Input_H3	158022981	118121722	105618955		
Con1_H3	165654843	112316768	107304879	1.02	1.57
Con2_H3	187081214	126971168	121307223	1.02	1.53
Con3_H3	171500107	119831682	114624211	1.03	1.55
Res1_H3	146335213	102707823	98206293	1.03	1.48
Res2_H3	182238067	124566704	118018203	1.02	1.57
Res3_H3	182762004	127435339	121242713	1.03	1.66
Sus1_H3	153894689	105881281	100366004	1.03	1.29
Sus2_H3	170035193	115562497	109212915	1.03	1.87
Sus3_H3	190117087	130767930	124422474	1.03	1.41

Supplementary Table 3: Correlation analysis between individual BAZ1A and SMARCA5 replicates

	Con1_SMARCA5	Con2_SMARCA5	Con3_SMARCA5	Res1_SMARCA5	Res2_SMARCA5	Res3_SMARCA5	Sus1_SMARCA5	Sus2_SMARCA5	Sus3_SMARCA5
Con1_SMARCA5	1.00	0.81	0.75	0.94	0.68	0.95	0.84	0.92	0.91
Con2_SMARCA5	0.81	1.00	0.93	0.89	0.88	0.88	0.90	0.93	0.91
Con3_SMARCA5	0.75	0.93	1.00	0.87	0.97	0.85	0.94	0.87	0.92
Res1_SMARCA5	0.94	0.89	0.87	1.00	0.82	0.97	0.92	0.94	0.96
Res2_SMARCA5	0.68	0.88	0.97	0.82	1.00	0.80	0.93	0.79	0.88
Res3_SMARCA5	0.95	0.88	0.85	0.97	0.80	1.00	0.91	0.94	0.96
Sus1_SMARCA5	0.84	0.90	0.94	0.92	0.93	0.91	1.00	0.88	0.95
Sus2_SMARCA5	0.92	0.93	0.87	0.94	0.79	0.94	0.88	1.00	0.93
Sus3_SMARCA5	0.91	0.91	0.92	0.96	0.88	0.96	0.95	0.93	1.00

	Con1_BAZ1A	Con2_BAZ1A	Con3_BAZ1A	Res1_BAZ1A	Res2_BAZ1A	Res3_BAZ1A	Sus1_BAZ1A	Sus2_BAZ1A	Sus3_BAZ1A
Con1_BAZ1A	1.00	0.75	0.74	0.72	0.76	0.77	0.73	0.72	0.73
Con2_BAZ1A	0.75	1.00	0.76	0.80	0.79	0.77	0.80	0.80	0.78
Con3_BAZ1A	0.74	0.76	1.00	0.78	0.79	0.77	0.80	0.82	0.79
Res1_BAZ1A	0.72	0.80	0.78	1.00	0.79	0.76	0.83	0.83	0.80
Res2_BAZ1A	0.76	0.79	0.79	0.79	1.00	0.77	0.79	0.80	0.77
Res3_BAZ1A	0.77	0.77	0.77	0.76	0.77	1.00	0.77	0.77	0.77
Sus1_BAZ1A	0.73	0.80	0.80	0.83	0.79	0.77	1.00	0.83	0.81
Sus2_BAZ1A	0.72	0.80	0.82	0.83	0.80	0.77	0.83	1.00	0.82
Sus3_BAZ1A	0.73	0.78	0.79	0.80	0.77	0.77	0.81	0.82	1.00

Supplementary Table 7 qPCR Primer List

Mouse Primer	
Baz1a_F	taggcagagaccatccttgg
Baz1a_R	gaaagaagggccaaatctcc
Baz1b_F	agggctgggtccataatagc
Baz1b_R	gcagctcatccaattccttc
Smarca5_F	aggttggatggacagacacc
Smarca5_R	aaagcgaaacactcggacag
Smarca1_F	tcattcagccttcagcacag
Smarca1_R	gcggtcctcctttcacatag
Smarcd1_F	atgtctgcccagttgaatcc
Smarcd1_R	acatgctaactccgggtgac
Smarcd2_F	gaaggcaggaactggaacag
Smarcd2_R	agggtgacaggaaacacacc
Smarcd3_F	ggaaacagatgggttccaag
Smarcd3_R	tgtcgccattgatgtactcc
Smarca2_F	accctgaaccagatgattgc
Smarca2_R	caggtgagcctttccacttc
Smarca4_F	atccgaaaccacaagtaccg
Smarca4_R	ctttcctcgccttcactgtc
Actl6b_F	atgtcaagtccgagccaaac
Actl6b_R	acgatgccttgctgtaggac
Actl6a_F	tgtggtgctggagagagatg
Actl6a_R	atcccagtcttcaaccatgc
Phf10_F	gagcgcatggaagaaagaag
Phf10_R	ggcaagtaccgaagctcatc
Dpf1_F	ataagaaccggccaggactc
Dpf1_R	ttggctgtgtgtttcctctg
Dpf2_F	ggatggcagcagtttagagg
Dpf2_R	cgctttggcgtatcttcttc
Smarcc1_F	ttattcccagctacgcatcc
Smarcc1_R	aaggcatgaaccctcatcac
Smarcb1_F	ccaccattgcatacagcatc
Smarcb1_R	tctcggcatcagtcagtgtc
Pbrm1_F	atgacgaagatgggcaagac
Pbrm1_R	gcactttggaaggcagtttc
Smarcc2_F	tctacctggcgtatcggaac
Smarcc2_R	gtcggctctcagcatctacc
Smarce1_F	aatggttcatcagcggaaac
Smarce1_R	ccttctccctctcctcttgc
Arid1a_F	tcggaaacacctcacaacag
Arid1a_R	aatgccaaatggaaacttgc
Arid1b_F	agtgcatccgggtctacaag
Arid1b_R	ctggattctgactggcttcc
Arid2_F	caagccacttccttctcagc

Arid2_R	gacaggtgatggtgatgacg
Bptf_F	tctaagcacgcacaaacctg
Bptf_R	aacatcgtctgcactggttg
Baz2a_F	gcaagcccagactcaagttc
Baz2a_R	gccaagacaagaaggcagac
Rsf1_F	aatcaggcgagttcacaagc
Rsf1_R	tgccttcctcttcatcatcc
Rbbp7_F	gctgcatgagtccttgtttg
Rbbp7_R	agccagttgccagaatgaac
Rbbp4_F	aaggtggtggatgcaaagac
Rbbp4_R	ggcttggcttggaagtattg
Chrac1_F	acaatgaggacgatggaagc
Chrac1_R	cagtgggagagaggagatgg
Pole3_F	gaaaggaaagcgcaagactc
Pole3_R	tcttcgttctggtcgtcctc
Chd3_F	aacatctcccaccactcctg
Chd3_R	ttctagccgttctcccagtg
Chd4_F	gagacctgccctatgaccag
Chd4_R	ccagtttccgtagcttcacc
Ino80_F	tattgatgtctcgccagcag
Ino80_R	gggaaggaaagtgggaaatc
Gapdh_F	aggtcggtgtgaacggatttg
Gapdh_R	tgtagaccatgtagttgaggtca
Agtr1b_F	acattctgggcttcgtgttc
Agtr1b_R	ctacgtccgcaatttcacag
Sdk2_F	acattctgggcttcgtgttc
Sdk2_R	ctacgtccgcaatttcacag
Zbtb7c_F	acattctgggcttcgtgttc
Zbtb7c_R	ctacgtccgcaatttcacag
Nit1_F	acattctgggcttcgtgttc
Nit1_R	ctacgtccgcaatttcacag
Runx1_F	acattctgggcttcgtgttc
Runx1_R	ctacgtccgcaatttcacag
Rab3b_F	acattctgggcttcgtgttc
Rab3b_R	ctacgtccgcaatttcacag
Cadps2_F	acattctgggcttcgtgttc
Cadps2_R	ctacgtccgcaatttcacag
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Grid2_R	ctacgtccgcaatttcacag
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Prdm16_R	ctacgtccgcaatttcacag
Tusc3_F	ctacgtccgcaatttcacag
Tusc3_R	ctacgtccgcaatttcacag
Dmrt1_F	accagtggcagatgaagacc
Dmrt1_R	acacactggctttggcttct

Irx6_F	gcgcctcaagaaggagaac
Irx6_R	ggtcttccaggtcactcagc
Plcz_F	ttggagacttcctgctttcg
Plcz_R	cctccttgatccgtgatagg
Espnl_F	aatggacacatggagtgctg
Espnl_R	cagtggaggaggtggaaatg
Myo1g_F	tcaagtccacctgtgtgctc
Myo1g_R	agtccttgcagctcttggtc
Pth2r_F	ttccagggtttcttcgtgtc
Pth2r_R	gtgggtttgccagagatgag
Agbl4_F	atccttcaacagggatgctg
Agbl4_R	aaatgttcttgccacgttcc
Dffb_F	gagtacctgcgagtccttgg
Dffb_R	ggagtgcttggaaagacagc
Mad1l1_F	tcgtggttgagctacagcag
Mad1l1_R	tgccatctctctccttggtc
Dcc_F	tcaagcacggaatgtgaaag
Dcc_R	aatgggtggttcgttgagag
Fsip1_F	gatgcacagactcgacaagc
Fsip1_R	gtgtttcccaactcctcgtc
Meox_F	atcttcaacgagcagcatcc
Meox_R	gatcgtcccaagtaccatgc
Spef2_F	tgcacgattggaaagaactg
Spef2_R	attgttggagggtggtgaac
Zfp438_F	caaacctagcctgtggaagc
Zfp438_R	tgccttctgcatacttgctg
Mtss1l_F	acatcaccagccagaaatcc
Mtss1l_R	acagtgcctccaccagtagg
Dnajc14_F	ctgttctcttggctgggttc
Dnajc14_R	tcgggtcacttcctcttcag
Fmn2_F	gacctgacttgcctgagacc
Fmn2_R	tcgcactcctcatctgtgtc
Sh3rf3_F	ctggaggtggaagtctgagc
Sh3rf3_R	ctgttgggatggtggtatcc
Mbd4_F	acgtgtcggagcttctcaag
Mbd4_R	tgattctcccaaagccagtc
Atp2b4_F	tccgaaatgagaagggtgag
Atp2b4_R	atagcaaacccgacatctgc
Prmt5_F	ctgagtgtctggatggagca
Prmt5_R	gcatctcaaactgtgcctca
Ptprs_F	gagacggcctacgagttacg
Ptprs_R	agggtgagcccattgtactg
Loxl2_F	ggaagctgcgttactggaag
Loxl2_R	agggttgctctggcttgtag
Cecr6_F	ggtcgtgagctaccaacagc
Cecr6_R	cagagggaggagcaccatag

Human primer	
SMARCA5_F	gagcaacagcagcaacaaag
SMARCA5_R	gcagcaggttgaatgaaatg
BAZ1B_F	aaatgcaccttgggtcgtag
BAZ1B_R	ggatttctttgagggcttcc
BAZ1A_F	gagcttcgtttgagcaatcc
BAZ1A_R	gaactcctgctttgcctgtc
RAB3B_F	tcaatgctgtccaagactgg
RAB3B_R	tgtccagcgaatcagacatc
AGTR1_F	ctttgccactatgggctgtc
AGTR1_R	gatgatgcaggtgactttgg