# Supplemental Data

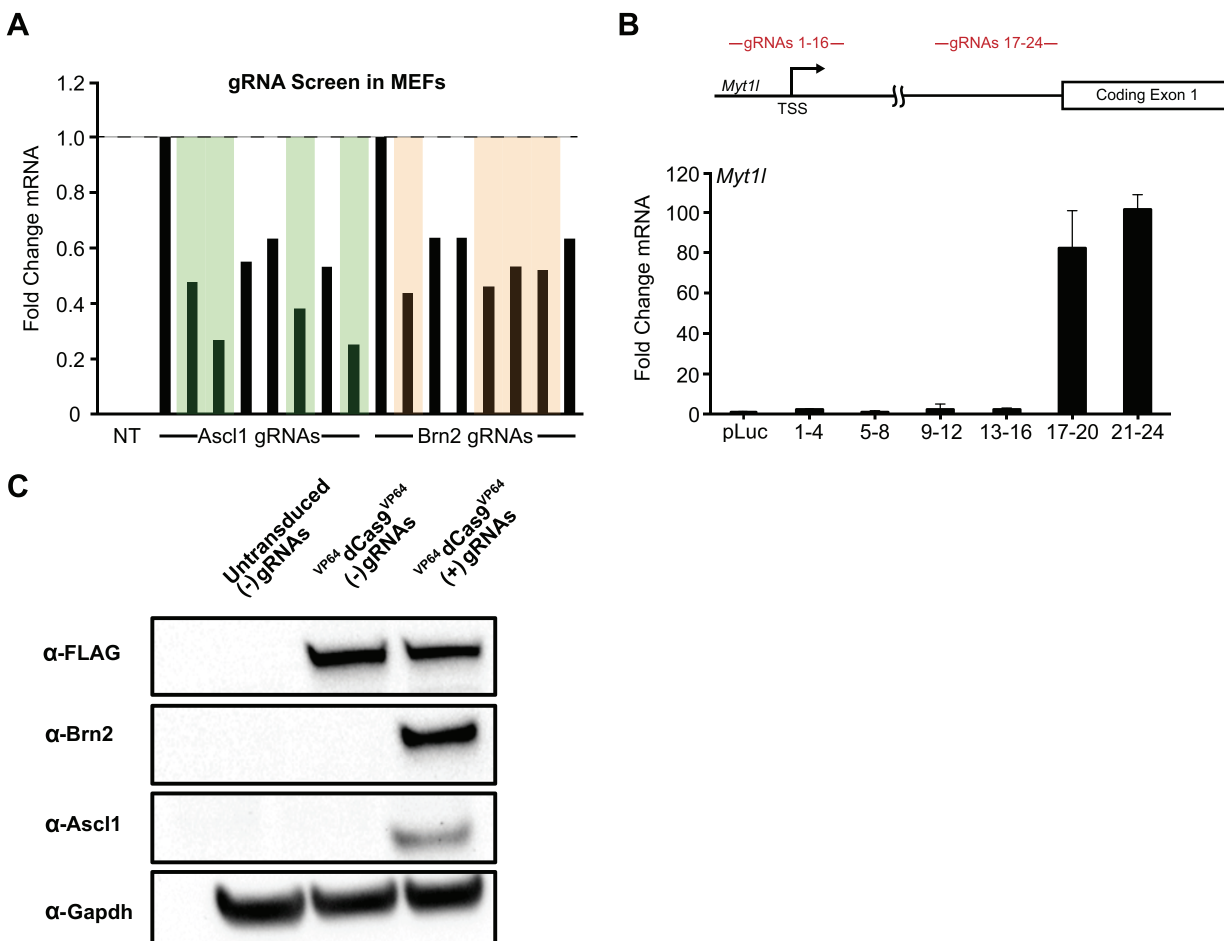
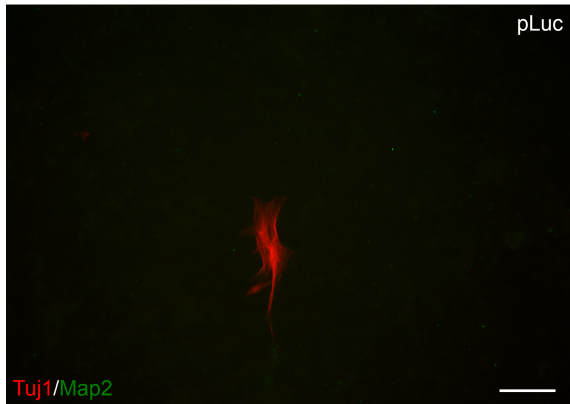



**Figure S1. gRNA optimization and target gene expression in PMEFs (relates to Figure 1).** (A) Selection of gRNAs with highest activity by elimination screening. Each gRNA was eliminated from a pool of eight gRNAs, and the four whose absence resulted in the largest drop in activity were selected for use in this study. (B) Activation of *Myt1l* in PMEFs determined by qRT-PCR with gRNAs targeting an intragenic region adjacent to the first coding exon. Target sites proximal to the TSS did not induce a detectable increase in expression (n = 2 biological replicates). (C) An N-terminal FLAG epitope tag was used to verify expression of $^{VP64}$dCas9$^{VP64}$ in transduced PMEFs. Brn2 and Ascl1 protein was only detected in cells transduced with $^{VP64}$dCas9$^{VP64}$ and transfected with gRNA expression plasmids.

**A**




**B**

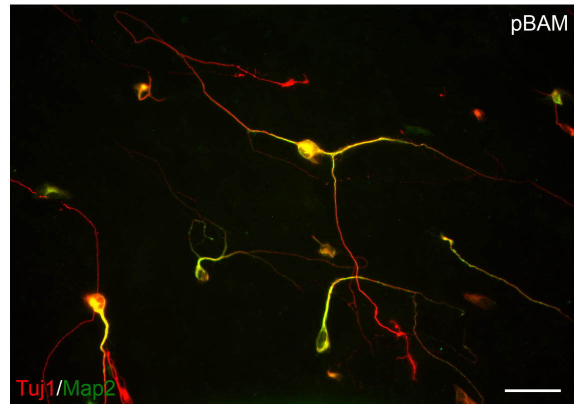



**Figure S2. Immunofluorescence staining of cells transfected with control plasmids (relates to Figure 2).** (A) PMEFs transfected with firefly luciferase (pLuc) did not generate any Tuj1$^+$Map2$^+$ cells with neuronal morphologies. There were sparse cells with fibroblastic morphologies that stained positive for Tuj1 (scale bar = 50 μm). (B) Ectopic expression of the reprogramming factors (pBAM) generated numerous Tuj1$^+$Map2$^+$ cells with elaborate neuronal morphologies (scale bar = 50 μm).

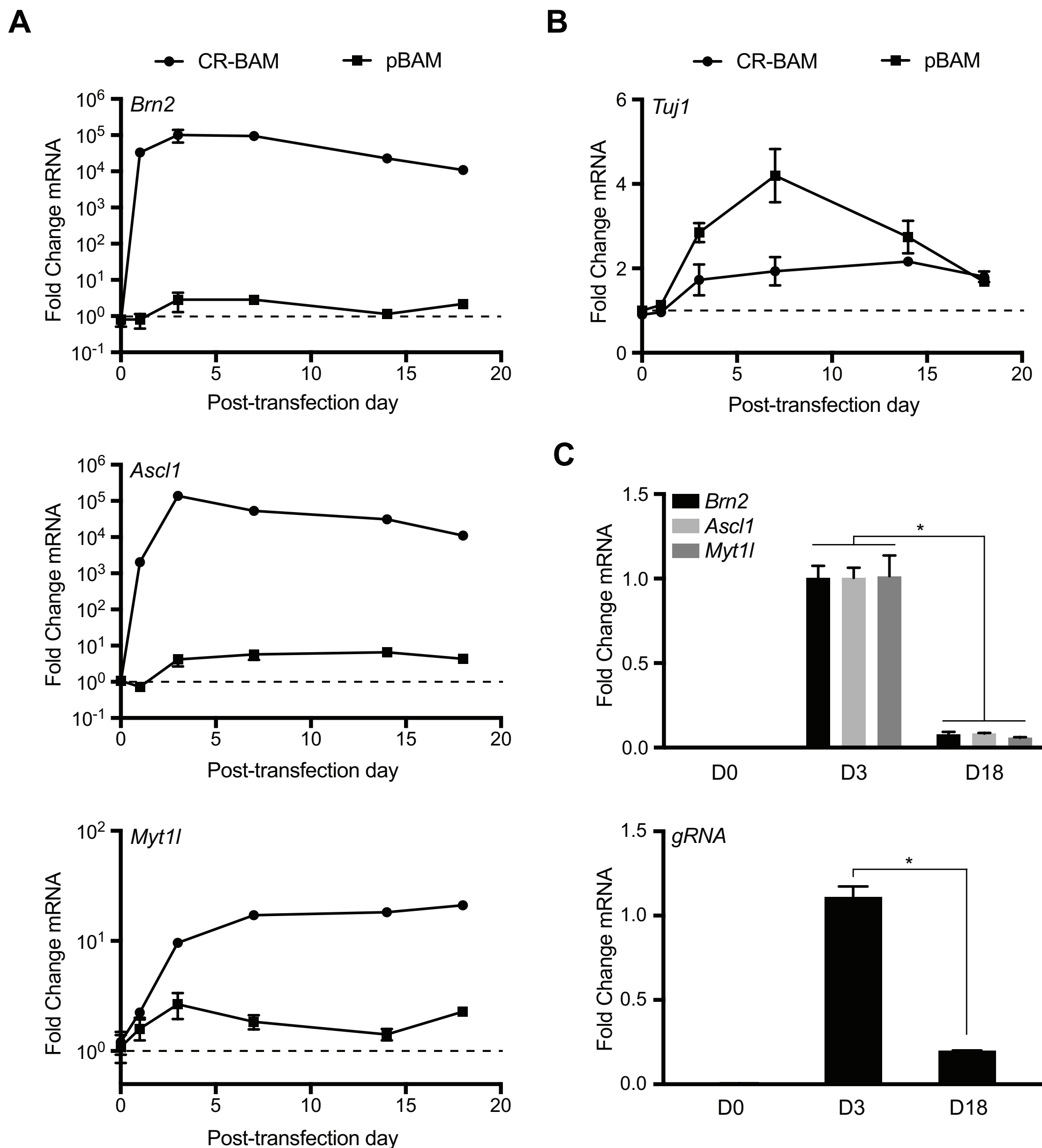



**Figure S3. Targeted Activation Leads to Rapid and Sustained Endogenous Gene Expression (relates to Figure 3).** (A) Activation of the endogenous BAM factors in PMEFs measured by qRT-PCR for 18 days after transfection of gRNAs or ectopic reprogramming factors. Targeted activation of the endogenous genes via $^{VP64}$dCas9$^{VP64}$ and gRNAs is rapid and sustained. Expression of all three endogenous genes is significantly higher with targeted activation than ectopic overexpression throughout the time course of the experiment ($p<0.0004$). Expression of the factors via ectopic plasmids generated a significant and sustained increase in endogenous *Ascl1* ($p < 0.0002$) and *Myt1l* ($p < 0.0005$, two-way ANOVA, $n = 3$ biological replicates). Fold change in mRNA expression is relative to transfection of a plasmid encoding firefly luciferase (pLuc). (B) *Tuj1* mRNA expression is significantly up-regulated throughout the time course of the experiment for both pBAM ($p < 0.0001$) and CR-BAM ($p < 0.0003$) transfection. pBAM transfection induced a higher level of *Tuj1* mRNA than CR-BAM transfection ($p < 0.02$, two-way ANOVA, $n \geq 3$ biological replicates). (C) The ectopic expression vectors for both pBAM (top) and CR-BAM (bottom) transfection are significantly depleted by day 18 post-transfection (pBAM: $p < 0.002$, CR-BAM: $p<0.0002$, one-way ANOVA with Holm-Bonferroni post hoc tests, $n = 3$ biological replicates). The ectopic BAM factors were detected with primers specific to the coding regions of the three genes. The gRNA expression vectors were detected with primers specific to the constant region of the chimeric gRNA sequence (See Table S3 for exact primer sequences).

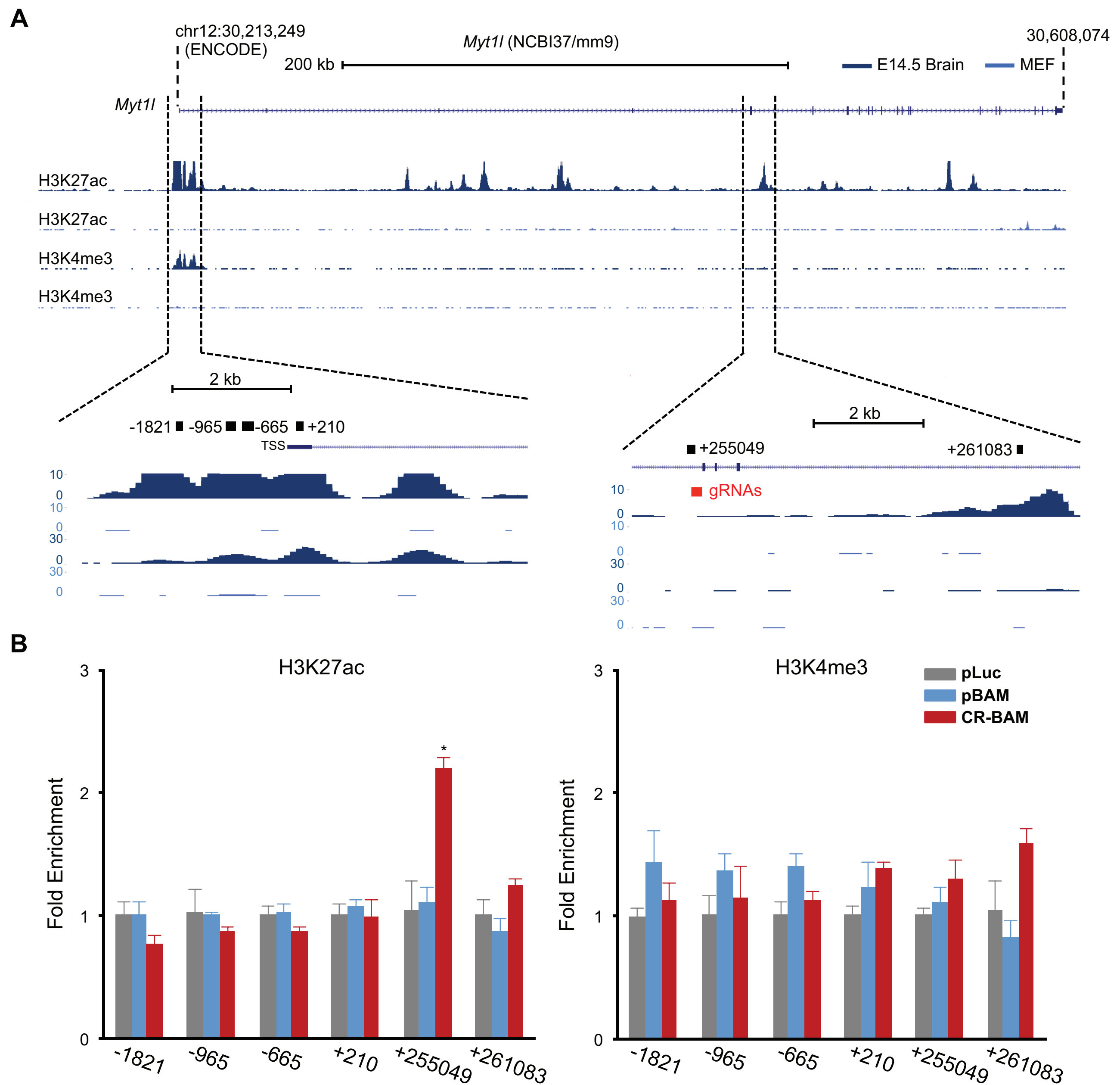



**Figure S4. Histone H3 modifications at the *Myt1l* genomic locus proximal and distal to the transcriptional start site (relates to Figure 3).** (A) Mouse genomic tracks depicting H3K27ac and H3K4me3 enrichment at the *Myt1l* locus in embryonic brain tissue and fibroblasts. Black bars indicate ChIP-qPCR amplicons, and the red bar indicates the gRNA target region. (B) Significant enrichment of H3K27ac is only detected at a region spanning the gRNA target sites in cells transfected with the gRNA cocktail ($p < 0.011$, one-way ANOVA with Holm-Bonferroni post hoc tests, n = 3 biological replicates).

**Table S1. gRNAs used in this study (relates to all Figures).**

| Target Gene | Protospacer Sequence (5' – 3') | Position Relative to TSS | Reference |
|---|---|---|---|
| *hASCL1* promoter | GCTGGGTGTCCCATTGAAA | -43 | Perez-Pinera et al., Nat. Methods, 2013 |
| | CAGCCGCTCGCTGCAGCAG | -103 | Perez-Pinera et al., Nat. Methods, 2013 |
| | TGGAGAGTTTGCAAGGAGC | -220 | Perez-Pinera et al., Nat. Methods, 2013 |
| | GTTTATTCAGCCGGGAGTC | -284 | Perez-Pinera et al., Nat. Methods, 2013 |
| *mAscl1* promoter | CAGCCGCTCGCTGCAGCAG | -103 | Perez-Pinera et al., Nat. Methods, 2013 |
| | TGGAGAGTTTGCAAGGAGC | -202 | Perez-Pinera et al., Nat. Methods, 2013 |
| | CCCTCCAGACTTTCCACCT | -392 | This study |
| | CTGCGGAGAGAAGAAAGGG | -523 | This study |
| *mBrn2* promoter | GAGAGAGCTTGAGAGCGCG | -51 | This study |
| | CCAATCACTGGCTCCGGTC | -185 | This study |
| | GGCGCCCGAGGGAAGAAGA | -222 | This study |
| | GGGTGGGGGTACCAGAGGA | -257 | This study |
| *mMyt1l* first coding exon | GTCTGGATTCACTGGACAA | +255188 | This study |
| | TAGAGCTACACAAGATTAA | +255120 | This study |
| | TACCTATGCTGCCCTATGG | +255087 | This study |
| | AGAGCAGGGAGAAGCCTAG | +255023 | This study |

**Table S2. Plasmids used in this study (relates to all Figures).**

| Plasmid Name | Addgene Plasmid # | Reference |
|---|---|---|
| pLV-hUbC-dCas9-VP64 | 53192 | Kabadi et al., Nucleic Acids Res., 2014 |
| pLV-hUbC-VP64-dCas9-VP64 | 59791 | Kabadi et al., Nucleic Acids Res., 2014 |
| pLV-4xSPgRNA | N/A | This study |
| pDM2.G | 12259 | Trono Lab |
| psPAX2 | 12260 | Trono Lab |
| pSPgRNA | 47108 | Perez-Pinera et al., Nat. Methods, 2013 |
| pUNO1-mBrn2 | N/A | This study |
| pUNO1-mAscl1 | N/A | InvivoGen, San Diego, CA |
| pUNO1-mMyt1lb | N/A | InvivoGen, San Diego, CA |
| VR1255C (pLuc) | N/A | Vical, San Diego, CA |
| pLV-hSyn-RFP | 22909 | Nathanson, et al., Neuroscience, 2009 |
| Tet-O-FUW-Ascl1 | 27150 | Vierbuchen et al., Nature, 2010 |
| Tet-O-FUW-Brn2 | 27151 | Vierbuchen et al., Nature, 2010 |
| Tet-O-FUW-Myt1l | 27152 | Vierbuchen et al., Nature, 2010 |
| FUW-M2rtTA | 20342 | Hochemeyer et al., Cell Stem Cell, 2008 |

**Table S3. Quantitative RT-PCR and ChIP-qPCR primers and conditions (relates to all Figures).**

| Target | Forward Primer (5' – 3') | Reverse Primer (5' - 3') | Cycling Conditions |
|---|---|---|---|
| endo *hASCL1* | GGAGCTTCTCGACTTCACCA | AACGCCACTGACAAGAAAGC | 95°C 5s<br>58°C 20s x45 |
| endo *mAscl1* | GGAACAAGAGCTGCTGGACT | GTTTTTCTGCCTCCCCATTT | 95°C 5s<br>60°C 20s x45 |
| total *mAscl1* | AGGGATCCTACGACCCTCTTA | ACCAGTTGGTAAAGTCCAGCAG | 95°C 5s<br>60°C 20s x45 |
| endo *mBrn2* | TTATTTTCCCGGTCCCTTAAA | GTACTCTCGCCTGCAAAGGT | 95°C 5s<br>60°C 20s x45 |
| total *mBrn2* | GACACGCCGACCTCAGAC | GATCCGCCTCTGCTTGAAT | 95°C 5s<br>60°C 20s x45 |
| endo *mMyt1l* | CTCGAGAGAGTACCTGCAGAC | AGACCCCCAAATGTGCAGAC | 95°C 5s<br>60°C 20s x45 |
| total *mMyt1l* | CGTGGACTCTGAGGAGAAGC | GTGCATATTTGCCACTGACG | 95°C 5s<br>60°C 20s x45 |
| *Tuj1* | TCCGAGTACCAGCAGTACCA | GGCTTCCGATTCCTCGTCAT | 95°C 5s<br>64°C 20s x45 |
| *gRNA* | GCAAGTTAAAATAAGGCTAGTCCG | GACTCGGTGCCACTTTTTCA | 95°C 5s<br>64°C 20s x45 |
| *mGapdh* | CCTGCTCCCGTAGACAAAATG | TGAAGGGGTCGTTGATGGC | 95°C 5s<br>60°C 20sx45 |
| *hGAPDH* | GAAGGTGAAGGTCGGAGTC | GAAGATGGTGATGGGATTTC | 95°C 5s<br>58°C 20s x45 |
| *mBrn2* ChIP<br>+2436 | AACGCGCATTTAGAGACACG | CAAAGAAGTGCTGATGCCCG | 95°C 5s<br>60°C 20s x45 |
| *mBrn2* ChIP<br>+1720 | ACTCCTCCCCGGCTCAATTA | TGTTTTTATCCCGCCCCAGG | 95°C 5s<br>60°C 20s x45 |
| *mBrn2* ChIP<br>+1348 | GTTACTCAAAAGCCAGGGCG | GAATTACAGCGCACAGGTGTC | 95°C 5s<br>60°C 20s x45 |
| *mBrn2* ChIP<br>+644 | AAGGACTGAGAAGACTGGGCG | GCGCCCTTTGATTTACGTGGA | 95°C 5s<br>60°C 20s x45 |
| *mBrn2* ChIP<br>+130 | AACAGAAGGCGTCGGAGC | GGTTAAAGGAGCCGCGCA | 95°C 5s<br>60°C 20s x45 |
| *mBrn2* ChIP<br>-228 | GTTTGCTCTATTCGCAG | GTTGCTGGTGTGGGTGA | 95°C 5s<br>60°C 20s x45 |
| *mBrn2* ChIP<br>-863 | AGACAAGATCGCAGCGCAA | GCTGTAAGCTGTCCGCGA | 95°C 5s<br>60°C 20s x45 |
| *mBrn2* ChIP<br>-1250 | CCACAGTCTTTCCTGGGACC | GTACTCTCGCCTGCAAAGGT | 95°C 5s<br>60°C 20s x45 |

| Target | Forward Primer (5' – 3') | Reverse Primer (5' – 3') | Cycling Conditions |
|---|---|---|---|
| *mAscl1* ChIP +2259 | TCTGGATCAGGTAGCCCCAG | AAGCAAGTTGCAGTGTGCAG | 95°C 5s<br>60°C 20s x45 |
| *mAscl1* ChIP +1877 | GGGCAGCGGTGATTTATGGA | TCGCTTAGCAACACAAAGCC | 95°C 5s<br>60°C 20s x45 |
| *mAscl1* ChIP +1482 | GACTTTCCACCTAGGCACCC | CGGACTCCCGGCTGAATAAA | 95°C 5s<br>60°C 20s x45 |
| *mAscl1* ChIP +1230 | CGGTTAGGGAGGGCGA | AAAGCAGCCGCAAAAG | 95°C 5s<br>60°C 20s x45 |
| *mAscl1* ChIP +507 | CCTCCTTCTGCGCGTTT | CGGCTCCACTCTCCAT | 95°C 5s<br>60°C 20s x45 |
| *mAscl1* ChIP +145 | TCGTCCTCTCCGGAACTGAT | GTGGCAAAACCCAGGTTGAC | 95°C 5s<br>60°C 20s x45 |
| *mAscl1* ChIP -314 | TCCCCCAACTACTCCAA | CCACATGAAGCGTACC | 95°C 5s<br>60°C 20s x45 |
| *mMyt1l* ChIP -1821 | GCTCCTTGTGGAGTGGAGTC | TCTCTCTGAGCTGTGGCTCT | 95°C 5s<br>60°C 20s x45 |
| *mMyt1l* ChIP -965 | AGAATCGAGCAATCCGTCCC | TGGCTTACTGCCTTTCGGTT | 95°C 5s<br>60°C 20s x45 |
| *mMyt1l* ChIP -665 | ATTTTGCAGGATGTCCCCCT | GGTTTCATGAAGGACCGGCT | 95°C 5s<br>60°C 20s x45 |
| *mMyt1l* ChIP +210 | CCCTGCAGTCTTCTTGGAGG | GGCAGGGAAGGTTGCTTTTG | 95°C 5s<br>60°C 20s x45 |
| *mMyt1l* ChIP +255049 | AGAGGAGAGCTGGATCCCTG | GCCTCCATAGGGCAGCATAG | 95°C 5s<br>60°C 20s x45 |
| *mMyt1l* ChIP +261083 | CAGCCGGTGGGTGAATAATTG | CAGAATCCAAATGAGGCGTGC | 95°C 5s<br>60°C 20s x45 |
| *mGapdh* ChIP +611 | GTATTAGGAACAACCCCACGC | TATGCACCTCACAACGCCAT | 95°C 5s<br>60°C 20s x45 |

# Supplemental Experimental Procedures

**Lentiviral Production.** HEK293T cells were acquired from the American Tissue Collection Center (ATCC) and purchased through the Duke University Cancer Center Facilities and were cultured in Dulbecco's Modified Eagle's Medium (Invitrogen) supplemented with 10% FBS (Sigma) and 1% penicillin/streptomycin (Invitrogen) at 37°C with 5% $CO_2$. Approximately 3.5 million cells were plated per 10 cm TCPS dish. Twenty-four hours later, the cells were transfected using the calcium phosphate precipitation method with pMD2.G (Addgene #12259) and psPAX2 (Addgene #12260) second generation envelope and packaging plasmids. The medium was exchanged 12 hours post-transfection, and the viral supernatant was harvested 24 and 48 hours after this medium change. The viral supernatant was pooled and centrifuged at 600 g for 10 minutes, passed through a 0.45 μm filter, and concentrated using Lenti-X Concentrator (Clontech) in accordance with the manufacturer's protocol.

**Cell Culture, Transductions and Transfections.** For lentiviral transduction of $^{VP64}$dCas9$^{VP64}$ (Addgene #59791), 40,000 PMEF-HL (Millipore) cells were plated into viral supernatant with 4 μg/ml polybrene and incubated for 20-24 hours before medium exchange. The cells were cultured for an additional three days in PMEF medium: Dulbecco's Modified Eagle's Medium (Invitrogen), 10% FBS (Sigma), 25 μg/ml gentamicin (Invitrogen), and 1x sodium pyruvate, GlutaMAX, nonessential amino acids, and β-mercaptoethanol (Invitrogen). The cells were then re-plated for transfection at 100,000 cells per well of a 24-well TCPS plate. Twenty-four hours after plating, the cells were transfected with either pmax-GFP to monitor efficiency, VR1255C (pLuc) to serve as a negative control, pUNO-BAM to serve as a positive control, or CR-BAM gRNA plasmid cocktails. Transfections were carried out using Lipofectamine 3000 (Invitrogen) in accordance with the manufacturer's protocol. Briefly, Lipofectamine 3000 and DNA (0.5 μg DNA per well of 24-well TCPS plate) were mixed at a 3:1 volume-to-mass ratio in serum-free Opti-MEM (Invitrogen). Transfections were carried out in complete PMEF medium with antibiotic for 12-15 hours before exchanged for fresh PMEF medium. Twenty-four hours later, PMEF medium was replaced with N3 neural induction medium: DMEM/F-12 Nutrient Mix (Invitrogen), B-27 serum-free supplement (Invitrogen), N-2 serum-free supplement (Invitrogen), 10 μM SB-431542 (Sigma), 0.5 μM LDN-193189 (Sigma), 2 μM CHIR99021 (Stemgent), and 25 μg/ml gentamicin (Invitrogen). The medium was half-exchanged every 48 hours for the first 10 days, and then every 24 hours for the remainder of the time in culture.

For experiments requiring stable integration of all expression vectors, PMEF-CD-1 (Stemcell) cells were thawed once and transduced following the protocol described previously. PMEFs were either transduced with vectors directly encoding the BAM factors under the control of doxycycline-inducible promoters (Addgene #27150, #27151 and #27152) along with the reverse tetracycline-controlled transactivator (Addgene 20342) or with $^{VP64}$dCas9$^{VP64}$ (Addgene #59791) and multiplex gRNA vectors (this study) and cultured in neurogenic medium. For electrophysiological analysis, cells were transduced with Syn-RFP (Addgene #22909) and were dissociated after 7-15 days in culture using Accutase (Stemcell) and replated onto a previously established monolayer of primary rat astrocytes on poly-D-lysine/laminin-coated glass coverslips (BD) and cultured for an additional 7 – 12 days. The medium was half-exchanged every 3 days.

**Glia cell isolation.** The cortices from postnatal day one Sprague-Dawley rat pup brains were dissected and sectioned into 1 $mm^3$ pieces. The tissue was incubated with a papain solution (1 vial papain (Worthington) in 20 ml D-PBS (Gibco) with 200 µl 0.4% DNase) for 45 minutes at 37°C. The solution was mixed by swirling every 15 minutes. The dissociated tissue was collected with repeated incubations with a low ovomucoid solution (2 ml 10X Low Ovo stock (Worthington) in 18 ml D-PBS with 200 µl 0.4% DNase) and a high ovomucoid solution (2 ml 6X High Ovo stock (Worthington) in 10 ml D-PBS). The cell mixture was centrifuged twice at 1100 rpm for 11 minutes, filtered through a 30 µm Nitex membrane, and centrifuged a final time and resuspended in Astrocyte Growth Media (500 ml DMEM (Gibco), 50 ml heat inactivated FCS (Gibco), 5 ml pen-strep (Gibco), 2 mM glutamine (Gibco), 1 mM sodium pyruvate (Gibco), 5 µg/ml NAC (Sigma), 5 µg/ml insulin (Sigma), and 10 µM hydrocortisone (Sigma)). Contaminating cells were removed by vigorous shaking and the addition of 10 µM Ara-C. The astrocytes were passaged two times before culturing with PMEF-derived iNs.

**Quantitative Reverse Transcription PCR.** Cells were lysed directly from a 24-well TCPS plate, and total RNA was isolated using RNeasy Plus and QIAshredder kits (Qiagen). Reverse transcription was carried out on 0.5 µg total RNA per sample in a 10 µl reaction using the SuperScript VILO Reverse Transcription Kit (Invitrogen). 1.0 µl of cDNA was used per PCR reaction with Perfecta SYBR Green Fastmix (Quanta BioSciences) using the CFX96 Real-Time PCR Detection System (Bio-Rad). The amplification efficiencies over the appropriate dynamic range of all primers were optimized using dilutions of purified amplicon in Herring Sperm DNA (Invitrogen). All amplicon products were verified by gel electrophoresis and melting curve analysis. All qRT-PCR results are presented as fold change in mRNA normalized to *Gapdh* expression.

**Western Blot.** Cells were lysed with RIPA buffer (Sigma) containing a protease inhibitor cocktail (Sigma). Protein concentration was assessed using a bicinchoninic acid (BCA) protein standard curve (Thermo Scientific) on the BioTek Synergy 2 Multi-Mode Microplate Reader. 25 μg of protein per sample was loaded into a NuPage 4-12% Bis-Tris Gel polyacrylamide gel (Invitrogen) and transferred to a nitrocellulose membrane. Following transfer, the membrane was washed briefly with TBST (50 mM Tris, 150 mM NaCl and 0.1% Tween-20) and blocked for 1 hour at room temperature in TBST with 5% nonfat milk. The membrane was washed three times for 15 minutes and then incubated with primary antibody in blocking solution overnight at 4° C. The following primary antibodies were used: rabbit anti-FLAG (1:1000 dilution, Sigma, F7425); rabbit anti-Brn2 (1:1000 dilution, abcam, ab94977); goat anti-Ascl1 (1:200 dilution, Santa Cruz, sc-48449); rabbit anti-GAPDH (1:5000 dilution, Cell Signaling, clone 14C10). The membrane was washed three times for 15 minutes and then incubated with secondary antibody in blocking solution for 1 hour at room temperature. The following secondary antibodies were used: anti-rabbit HRP-conjugated (1:5000 dilution, Sigma, A6154); anti-goat HRP-conjugated (1:5000 dilution, Santa Cruz, sc-2354). The membrane was washed with TBST for 1 hour at room temperature and then imaged using the ImmunStar WesternC Chemiluminescence Kit (Bio-Rad) and ChemiDoc XRS+ System (Bio-Rad).

**Immunofluorescence Staining and Image Analysis.** Cells were washed briefly with PBS and then fixed with 4% paraformaldehyde (Sigma) for 20 minutes at room temperature. Cells were washed twice with PBS and then incubated with blocking buffer (10% FBS (Sigma), 3% wt/vol BSA, and 0.2% Triton X-100) for 30 minutes at room temperature. The following primary antibodies were used with incubations overnight at 4 °C: rabbit anti-Brn2 (1:500 dilution, Abcam, ab94977); goat anti-Ascl1 (1:100 dilution, Santa Cruz, sc-48449); rabbit anti-Tuj1 (1:500 dilution, Covance); mouse anti-Map2 (1:500 dilution, BD Biosciences). Cells were washed three times with PBS and then incubated with secondary antibody in blocking solution for 1 hour at room temperature. The following secondary antibodies were used: Alexa Fluor 488 goat anti-rabbit (1:500 dilution, Invitrogen); Alexa Fluor 594 goat anti-mouse (1:500 dilution, Invitrogen); Alexa Fluor 594 donkey anti-goat (1:500 dilution, Invitrogen). Cells were washed three times with PBS and imaged with a Leica DMI 3000 B fluorescence microscope.

Quantification of single-cell Brn2 and Ascl1 protein levels in transfected cells was assessed using a MATLAB script. Briefly, DAPI images were used to identify nuclei, and the mean fluorescence within the nucleus was recorded for the channels corresponding to Brn2 and Ascl1. Distributions of mean intensities were constructed by collecting all cells with intensities greater than two standard deviations above the mean intensity of cells transfected with firefly luciferase. $Tuj1^{+}Map2^{+}$ and $Tuj1^{+}Syn\text{-}RFP^{+}$ cells were counted manually from images at 10x magnification. Counting was performed on 10 randomly selected windows from 3 biological replicates for each treatment condition.

**Electrophysiology.** RFP expression from the Syn-RFP lentiviral reporter was used to identify the most mature iNs for patch clamping using a Nikon2000-U fluorescence microscope. Evoked action potentials and inward and outward currents were recorded in whole-cell configuration using an Axopatch 200B amplifier (Molecular Devices). The intracellular solution contained 120 mM potassium gluconate, 10 mM KCl, 5 mM $MgCl_2$, 0.6 mM EGTA, 5 mM HEPEs, 0.006 mM $CaCl_2$, 10 mM phosphocreatine disodium, 2 mM Mg-ATP, 0.2 mM GTP, and 50 units/ml creatine phosphokinase, pH 7.3 adjusted with KOH. The extracellular solution contained 119 mM NaCl, 5 mM KCl, 20 mM HEPES, 2 mM $CaCl_2$, 2 mM $MgCl_2$, 30 mM glucose, pH 7.3 adjusted with NaOH. Data were analyzed and prepared for publication using MATLAB.

**Chromatin Immunoprecipitation qPCR.** For transfection experiments, cells were transfected in 15 cm TCPS dishes and cultured in neurogenic medium for three days before fixation. For transduction experiments, cells were transduced in 15 cm TCPS dishes and fixed after 3 and 6 days post-transduction. Cells were fixed in 1% formaldehyde for 10 minutes at room temperature. The reaction was quenched with 0.125 M glycine, and the cells were lysed using Farnham lysis buffer with a protease inhibitor cocktail (Roche). Nuclei were collected by centrifugation at 2,000 rpm for 5 minutes at 4°C and lysed in RIPA buffer with a protease inhibitor cocktail (Roche). The chromatin was sonicated using a Bioruptor Sonicator (Diagenode, model XL) and immunoprecipitated using the following antibodies: anti-H3K27ac (abcam, ab4729) and anti-H3K4me3 (abcam, ab8580). The formaldehyde crosslinks were reversed by heating overnight at 65°C, and genomic DNA fragments were purified using a spin column. qPCR was performed using SYBR Green Fastmix (Quanta BioSciences) with the CFX96 Real-Time PCR Detection System (Bio-Rad). 500 pg of genomic DNA was used in each qPCR reaction. The data are presented as fold change gDNA relative to negative control and normalized to a region of the Gapdh locus. Primers were designed to target regions with H3K27ac and/or H3K4me3 enrichment in mouse embryonic day 14.5 brain tissue (Figures 3 and S4).

# Supplemental References

Nathanson, J.L., Yanagawa, Y., Obata, K., and Callaway, E.M. (2009). Preferential labeling of inhibitory and excitatory cortical neurons by endogenous tropism of adeno-assocated virus and lentivirus vectors. Neuroscience *161*, 441-450.

Hockemeyer, D., Soldner, F., Cook, E.G., Gao, Q., Mitalipova, M., Jaenisch, R. (2008). A drug-inducible system for direct reprogramming of human somatic cells to pluripotency. Cell Stem Cell 3(3):346-353.