SUPPLEMENTARY FIGURE LEGENDS

Figure S1.
(A) Unsupervised clustering of Pearson correlation analysis of RNA-seq samples
(B) Measurements of BAM factors expression in RNAseq samples
(C) Principal Component analysis for RNAseq samples

Figure S2.
(A) Chromatin fractionation of MEFs+BAM 48hrs cells. Left: Immunoblots probed using antibodies against BAM factors. Right: Immunoblots probed against chromatin marker histone 3 (H3) and cytoplasmic marker tubulin.
(B) Left: Heat maps of normalized tag densities across numerous Brn2 ChIP-seq attempts during protocol optimization in MEFs. Right: Summary of distinct conditions used for Brn2 ChIP protocol optimization
(C) Left: Heat maps of normalized tag densities across numerous Myt1l ChIP-seq attempts during protocol optimization in MEFs. Right: Summary of distinct conditions used for Myt1l ChIP protocol optimization

Figure S3.
(A) Heat maps of normalized tag densities for Ascl1 ChIP-seq in single infected MEFs, BAM infected MEFs, NPCs overexpressing Ascl1, and in endogenous NPCs. Top, middle, and bottom panel show tag density across data sets based on peaks identified in each of the data sets respectively.
(B) Gradient Pearson correlation for Ascl1 peaks across data sets.
(C) Gradient Pearson correlation for endogenous Ascl1 peaks in NPCs and Ascl1 overexpressing NPCs.

Figure S4
(A) Frequency of distribution of Ascl1 (E-box) and Brn2 (POU-like) motif across the mouse genome.
(B) Quantification of the total neurite length of Ascl1 only, and Ascl1 with the addition of Brn2 at different time points after dox administration (one day before (-1d), two and five days after). n=3 randomly selected fields of view, ** $p<0.01$

Figure S5.
(A) Nucleosomal DNA of uninfected MEFs measured as enrichment of H3 ChIP-seq signal over input at Ascl1 and Brn2 target sites based on NPC binding. This assay nicely complements the FAIRE signal which measures nucleosome-free DNA.

Figure S6.
(A) Characterization of MEFs ChromHMM analysis at control sites
(B) Distribution of Ascl1 peaks across MEFs chromatin states
(C) Chromatin co-Immunoprecipitation of chromatin marks present at trivalent chromatin state using mononucleosomes.
(D) The trivalent chromatin state of MEFs is significantly less enriched at those sites that are bound by Ascl1 in NPCs but not in MEFs after infection with BAM compared to the sites that are bound by Ascl1 in both cell types.
(E) Immunostaining for Map2 and Tuj1 in skeletal muscle myoblasts and osteoblasts after 20 days of Ascl1 expression. Image shown in 10x magnification.
(F) Measurement of H3K9me3 levels at the loci of 7 Ascl1 target genes in MEFs before and after Ascl1 expression.

(G) Representative tracks of the Dgkd loci with chromatin state 5, also known as the trivalent chromatin state, and bound by Ascl1 across data sets (MEFs+Ascl1, MEFs+BAM, and NPCs).
(H) Characterization of NPCs ChromHMM analysis. Left: identification of existing chromatin states in NPCs. Enrichment of NPCs chromatin states based on Ascl1 (middle) and Brn2 (right) binding in NPCs.
(I) Quantification of the distinct chromatin states' enrichment at Ascl1 and Brn2 binding in NPCs.

Figure S7
(A) Representative traces of Hes6, which is bound and transcriptionally regulated by Ascl1 as determined by ChIP-seq and RNA-seq.
(B) Example traces of sodium and potassium currents from cells in Ascl1+ Myt1l (middle panel) and Zfp238+Myt1l (bottom panel) conditions recorded in voltage-clamp mode (V_{hold} = -70 mV) in response to a voltage-pulse step protocol (top panel). Insets in middle and bottom panel show higher magnification of sodium currents as indicated with the box. Bar in the middle panel represents the timeframe of potassium currents used to calculate average values.
(C) Average values of sodium (I_{Na}) and potassium (I_K) currents (I) from cells capable of firing AP in Ascl1+ Myt1l (n = 18 cells / 3 batches) and Zfp+Myt1l (n = 14 cells / 3 batches) conditions plotted against step voltages (V) (i). No significant difference found at any step-size tested ($P > 0.05$).
(D) Model

EXTENDED EXPERIMENTAL PROCEDURES

Cell culture and viral production

Embryonic fibroblasts were isolated as previously described from the distal extremities of E13.5 heterozygous TauEGFP mice embryos (Vierbuchen et al., 2010). iN cells were generated following the previously described protocol (Vierbuchen et al., 2010). ES cell-derived NPCs (line NS5) were obtained from A. Smith, Cambridge, UK and cultured as reported (Conti et al., 2005). Lentivirus was produced following standard procedures, following transfection of TetO-FUW lentiviral backbones containing the respective cDNAs (available from us via Addgene) together with the 3rd generation packaging plasmids from the Trono laboratory (Dull et al., 1998). Media was changed 16h after transfection and viral supernatant collected twice after another 24h and 48h. Supernatant was pooled and concentrated by ultra-centrifugation and cells were infected in a range of 3 different titers. All infected MEF populations that were passed on for genomic analysis were verified by immunofluorescence to contain a range of 80-90% Ascl1-, 70-80% Brn2-, and 40-50% Myt1l-positive nuclei, 2 days after dox treatment. The fraction was determined by visual counting immune-positive nuclei over all DAPI-positive nuclei. These numbers are not necessarily equivalent to the actual infected cells or actual stoichiometric ratios, because the immunofluorescence assay is not 100% sensitive and depends on antibody affinity. Empirically, though, these numbers are optimal for iN cell reprogramming. A manuscript describing this protocol in detail is under consideration at Nature Protocols. All tagged versions of the transcription factors have been verified to function in the iN cell reprogramming assay.

Establishing a dox-inducible Ascl1 ES cell line

A CAGGS promoter driven rtTA-t2a-puroR cassette was targeted into the ROSA26 locus of the V6.5 ES cell line using the protocol previously described (Beard et al., 2006). A successfully targeted clone was then infected with TetO-flagAscl1 lentivirus to establish a dox-inducible flagAscl1 ES cell line. Successfully infected clones were confirmed by immunofluorence using the anti-FLAG M2 antibody, as well as by an iN reprogramming assay to check that the cells can successfully reprogram into neurons under the presence of dox and neural media.

Generating dox-inducible Ascl1 MEFs

Following superovulation, morulae were flushed from uteri of 2.5dpc B6D2F1 females and cultured in KSOM droplets (EmbryoMax Chemicon MR-020P-D) under mineral oil (intestinal lubricant laxative, NDC 67618-108-16 Distributed Purdue Products LP) overnight. At 3.5dpc, 40 fully expanded blastocysts were isolated and about 10-15 Ascl1-inducible ESCs were injected into each blastocyst cavity. Injected blastocysts are cultured in KSOM at 37°C and 5% CO_2 until being transferred into two 2.5dpc pseudopregnant CD1 females. 20 blastocysts were injected per female, 10 blastocysts per uterine horn. Embryos were then harvested at E13.5 for MEFs and puro-selected for cells that contained the rtTA and Ascl1 transgenes (which we will call Ascl1-inducible MEFs).

Jmjd2d cloning and cell generation

We obtained JmjD2d WT and H189A in pcDNA plasmid (Zhu et al., 2012) and cloned them into pCDH-EF1-MCS-T2A-Puro vector (System BiosciencesCD527A-1). We then co-infected P3 MEFs with either JmjD2d WT or H189A lentivirus, together with Ascl1. Puromycin (4ug/mL, Fisher, ICN10055280) was added 16hrs after infection and kept in the media for 2 days to select for cells that were successfully infected with JmjD2d

WT/H189A, after which doxcycline was added to initiate iN reprogramming. N3 media is added the day after dox-induction of transgenes, and the cells were fixed 11 days after addition of dox and stained for presence of Tuj1 positive cells.

RNA-seq

Total RNA was purified with Trizol following supplier's recommended method. Poly-A RNA was isolated from 0.5-3µg total RNA using the MicroPoly(A)Purist Kit (Ambion). Samples were processed according to the manufacturer's protocol. Subsequently, libraries were prepared following the dUTP protocol (Levin et al., 2010). Sequencing reads (100bp) were generated on Hi-Seq 2000 Illumina platforms. Paired-end reads were aligned to the mouse reference sequence NCBI Build 37/mm9 with the TopHat (v1.1.3) algorithm (Trapnell et al., 2009). Expression levels of RefSeq annotated genes were calculated in unit of fragments per kilobase of exon model per million mapped fragments (FPKM). Genes with low FPKM (average log2 FPKM across all samples less than 1) were removed. Differential expression analysis was performed using Students' t-Test function "t.test" in R, and genes with p-value<0.05 and at least 2-fold expression change were defined as significant. Gene ontology analysis was performed using DAVID *(david.abcc.ncifcrf.gov) (Huang da et al., 2009a; Huang da et al., 2009b).*

Chromatin- Immunoprecipitation followed by quantitative PCR or sequencing (ChIP-seq and ChIP-qPCR)

ChIP-qPCR and ChIP-seq were carried out following the Farnham protocol (O'Geen et al., 2011) in Ascl1- and BAM-infected MEFs 48 hours after dox, and in Ascl1-infected NPCs 18 hours after induction. Full length Ascl1 cDNA was cloned in frame with the ERT2 domain (C-terminal fusion, with HA and Flag Tags at Ascl1 N-terminus) in the pBabe-IRES-Gfp Puro retroviral vector. NPCs were infected with retrovirus and after 24h, puromycin was added to select for transduced cells. Selected cells were expanded for 3 days and then induced with 4-OHT. Cells were infected with a pool of Ascl1, V5- or FLAG-tagged Brn2, and V5- or FLAG-tagged Myt1l, and the transgenes were induced with dox the day after. After 48 hours we performed ChIP-seq for each of the BAM factors. Approximately 50-100x10^6 cells were used for each ChIP-seq experiment. Crosslinking conditions were optimized for the respective transcription factor (see below). Chromatin was isolated using swelling buffer (0.1M Tris pH 7.6, 10mM KOAc, 15mM MgOAc, 1% NP40, and protease inhibitors) for 20 minutes on ice, centrifuged 2,500x g for 5 minutes at 4°C. Subsequently, we incubated pellet in Nuclei Lysis Buffer for 10 minutes on ice (50mM Tris pH 8.0, 10mM EDTA, 1% SDS, and protease inhibitors). Chromatin was sheared using bioruptor sonicator until DNA was fragmented to 200-1000 bp, and incubated with 5 µg of corresponding antibody overnight at 4ºC. Staph A cells were blocked overnight at 4ºC with 10 mg/mL BSA and were incubated with chromatin for 15 minutes at room temperature. Immunoprecipitate fraction was washed twice with dialysis buffer (2 mM EDTA, 50 mM Tris-Cl pH 8.0, 0.2% Sarkosyl, PMSF) and twice with washing buffer (100 mM Tris-Cl pH 9.0, 500 mM LiCl, 1% Igepal, 1% Deoxycholic Acid, PMSF). Subsequently, the immunoprecipitate fraction was eluted from Staph A cells by vortexing for 30 minutes at room temperature using elution buffer (50 mM Tris-Cl pH 8.0, 10 mM EDTA, 1% SDS, PMSF). We reversed crosslinking by overnight incubation at 67ºC. The isolated DNA was RNase treated and purified using Qiagen columns. For qPCR analysis we used Roche's Lightcycler. For sequencing libraries we followed Illumina's protocol.

Optimized crosslinking, tagging conditions and antibodies were for the Ascl1, Brn2, and Myt1l ChIP-seq experiments: For Ascl1 ChIP-seq in MEFs, cells were crosslinked in 1% formaldehyde for 10 minutes and Ascl1 was immunoprecipitated with

a validated Rabbit anti-Ascl1 antibody (Abcam ab74065). For Brn2 ChIP-Seq in MEFs, Brn2 was tagged with V5 in both N- and C- terminus. Brn2 infected cells were crosslinked with 1% DSG for 45 minutes followed by 1% formaldehyde for 10 minutes. We used a rabbit anti- V5 tag antibody (Abcam ab15828). The optimal condition for Myt1l ChIP-Seq in MEFs was the use of a FLAG-tagged version of Myt1l at both N- and C- terminus, crosslinking with 1% formaldehyde for 10 minutes, and use of a mouse anti-FLAG M2 antibody (sigma F3165). For Ascl1 ChIP-Seq in ES cell-derived NPCs (line NS5) cells were crosslinked in 1% formaldehyde for 15 minutes. ChIP was carried out with an anti-HA tag antibody. For Brn2 ChIP-Seq in NPCs, cells were crosslinked in 1% DSG for 45 minutes followed by 1% formaldehyde for 15 minutes. ChIP was carried out with a goat anti-Brn2 antibody (C-20) (Santa Cruz Biotechnology SC-6029).

Sequencing reads (36-50bp) were generated on Illumina GAIIX Genome Analyzer or on Hi-Seq 2000 platforms. Raw reads were uniquely mapped to mouse reference genome (NCBI37/mm9) using bowtie (version 0.12.6) allowing maximum one mismatch (Langmead et al., 2009). Peaks for each sample were called using MACs algorithm (version 1.4.2) against its corresponding input with p-value cutoff 1^{e-5} (Zhang et al., 2008).

For ChIP-seq peak classification the following criteria was used. Promoter: +/- 5kb around TSS, enhancer: 5-20kb upstream from transcriptional start site (TSS), genebody: encompassing exons and introns, gene tail: 2kb downstream of transcriptional end site (TES), intergenic: none of the above. Heatmaps of ChIP-seq signals were generated for occupancy profiles around peak summits (+/- 2kb region) were calculated using a 50bp sliding windows through the following formula:

$$signal = \log_2\left(\sum_{i=1}^{50} \frac{\mathrm{cov}erage\_of\_base_i}{number\_of\_unique\_mappable\_reads} \times 10,000,000\right)$$

Average diagrams were generated by averaging the normalized tag density signals in the ChIP-seq heatmaps. Sequences for top 1000 Ascl1 peaks in each dataset, top 500 Brn2 peaks in NPCs, all peaks for Brn2 in MEFs, all peaks for Myt1l in MEFs (ranked by fold enrichment), within ± 200bp around peak summits were extracted and de novo motif analysis against these sequences was performed using MEME (Bailey et al., 2009).

Co-localization between transcription factors was determined if two peaks from distinct ChIP-seq datasets overlapped by at least 1 bp. Transcription factor target genes were defined in this study if the corresponding ChIP-seq peak summit resided in a 10kb upstream to 2kb downstream window of that gene's TSS.

Chromatin Co-Immunoprecipitation (IP)

Chromatin Co-IP was carried out following a modified version of the Farnham protocol (O'Geen et al., 2011). After elution from Staph A cells as described above, SDS-containing dye was added to the immunoprecipitate fraction. The samples were boiled for 10 min and subjected to SDS-PAGE separation and subsequent immunoblotting. For chromatin marks co-IP, we followed the same protocol with the exception that chromatin was fragment to mononucleosomes.

Formaldehyde-Assisted Isolation of Regulatory Elements (FAIRE-seq)

Faire-seq protocol was followed as previously described (Giresi et al., 2007) in order to identify regions in MEFs genome that are nucleosomal-free and could be associated with regulatory activity. Approximately $5\text{-}10x10^6$ MEFs were crosslinked with 1% formaldehyde for 7 minutes.

Chromatin state discovery and characterization (ChromHMM)

To annotate the genome of distinct cells based on chromatin state we used the ChromHMM software (version 1.06) following the guidelines previously described (Ernst and Kellis, 2012). ChromHMM is a software that integrates chromatin datasets from different histone modification ChIP-seq experiments to uncover novel combinations using a multivariate Hidden Markov Model. Using aligned reads from bowtie (version 0.12.6) allowing maximum one mismatch (Langmead et al., 2009) SAM format files were generated and subsequently converted to bed files using “parseSam2Bed.pl” script. Next bed files were converted into binarized data files for model learning and optionally prints the intermediate signal files using “BinarizeBed” function in ChromHMM. 8-state and 9-state models for MEF and NPC chromatin data were then generated using “LearnModel” function in ChromHMM, and enrichment of Ascl1 and Brn2 binding sites (or any sites) were obtained by “NeighborhoodEnrichment” function in ChromHMM. The enrichment of each state and a segmentation file, which indicates for every genomic locus what state was occupied, were generated automatically in ChromHMM. A self-developed script was used to calculate the percentage of genomic bases within a +/-200bp window around the top 1000 transcription factor peak summits that were occupied by the given state. The frequency of Ascl1 biding sites across states was calculated by overlapping Ascl1’s peak summits with ChromHMM states segments. To obtain the normalized frequency of Ascl1 peaks in each state, the total number of Ascl1 peaks in each state was divided by the length of the corresponding state's segmentation size, and multiplied by 1 Megabases (Mb) to get the frequency of observed Ascl1 peaks in each state within every 1 Mb.

Principal component analysis (PCA)

PCA was performed using the PCA package in Microsoft Excel statistical add-in "XLSTAT". RPKM of all the mm9 RefSeq genes under all conditions was calculated, serving as an input data table for XLSTAT (initial variables). For all samples, XLSTAT calculates the factor loadings in the new space and the correlations between the initial variables and the components in the new space. The first two principle components consist of 77.0% and 9.9%, respectively, of the entire normalized variant between all samples. A 2-D PCA consists of the varimax factor loadings of each sample projected onto the first two principle component space. The values of varimax factor loadings were applied and displayed in Prism.

Electrophysiology

Cells were visualized using an X-cite 120Q fluorescence lamp (Lumen Dynamics) and an Olympus BX51WI microscope equipped with a Rolera-XR camera (Qimaging). Whole-cell patches were established at room temperature using MPC-200 manipulators (Sutter Instrument) and Multiclamp 700B amplifier (Molecular Devices) controlled by Clampex 10 Data Acquisition Software (Molecular Devices). Pipettes were pulled using PC-10 puller (Narishige) from borosilicate glass (OD: 1.5 mm, ID: 0.86 mm; Sutter Instrument) to a resistance of 2–3 MOhm and were filled with internal solution containing (for voltage-clamp, in mM) 135 CsCl2, 10 HEPES, 1 EGTA, 1 Na-GTP, and 1 QX-314 (pH adjusted to 7.4, 310 mOsm) or (for current-clamp, in mM) 130 KMeSO3, 10 NaCl, 10 HEPES, 2 MgCl2, 0.5 EGTA, 0.16 CaCl2, 4 Na2ATP, 0.4 NaGTP, and 14 Tris-creatine phosphate (pH adjusted to 7.3, 310 mOsm). The bath solution contained (in mM) 140 NaCl, 5 KCl, 2 CaCl2, 1 MgCl2, 10 HEPES, and 10 glucose (pH adjusted to 7.4). Resting membrane potential, input resistance, and capacitance was recorded for every cell. The current clamp mode was used to record action potentials. We set the

initial resting membrane potential (Vrest) to -60 mV using a small, constant holding current, and applied current-pulses of 10-50 pA with a step-size of 10 pA, to test the ability to generate action potentials. Statistical comparisons between cumulative plots were made using K-S (Kolmogorov-Smirnov) test. Average data are presented as means ± SEM. Cumulative plots are generated from parameters collected from individual cells under similar experimental condition.

Antibodies

The following antibodies were used for ChIP analysis: Rabbit anti-Ascl1 (Abcam ab74065), rabbit anti- V5 tag (Abcam ab15828), mouse anti- FLAG M2 (sigma F3165), rabbit anti-H3K27acetyl (Abcam ab4729), Goat anti-Brn2 (C-20) (Santa Cruz Biotechnology SC-6029), rabbit anti-IgG (Abcam ab37415).

The following antibodies were used for immunostaining: mouse anti-MAP2 (Sigma), rabbit anti-Tuj1 (Covance), goat anti-Brn2 (Santa Cruz Biotechnology), rabbit anti-Ascl1 (Abcam), mouse anti-FLAG (Sigma). Fluorophore-labeled secondary antibodies were purchased from Molecular Probes/Invitrogen.

The following antibodies were used for immunoblotting: rabbit anti-H3K9me3 (Abcam ab6001), rabbit anti-H3K27acetyl (Abcam ab4729), rabbit anti-H3K4me1 (Abcam ab8895), rabbit anti-beta tubulin (Abcam ab6046),

Primers

For Ascl1 ChIP qPCR validation in mouse fibroblasts (5’ to 3’):
ActB (forward, CTCATCAAATGCCCACACC; reverse, GACTCTTTGCAGCCACATTC), *HoxD13* (forward, CAGAACGCTCTCAAGTCGTC; reverse, ATGTACTTCTCCACCGGGAA), *ColA* (forward, CGGAGTCAGGGTGCTAAGAT; reverse, TCCATGATCCCAGTCTTTGA), *Mfng* (forward, AGCCACCCACTGGAAGAG; reverse, ACTGTGAGGCATAGGAAGGC), *Ngfr* (forward, CTGCCCTCCTCATTGCTC; reverse, GAGCGGACTGAGCTAGAAGC), *Grip1* (forward, AGCAGATGTGTCTGGGTCTG; reverse, GACCAGATAATCAGCATGCAA), *Grin1* (forward, CGAGGTGCTGAAGCGTATT; reverse, GTAGGTCCCGCTCACGAC), *Syt3* (forward, AGATGGCTGGTTCTTGCTG; reverse, AAACACTGTGAGTCTGAGGCA), *Zfp238* (forward, GCTGGAGGTAGCATTTCCAT; reverse, CGCACACACTAACTCCCTGT).

For Ascl1 ChIP-qPCR validation in human NHDF and NHEK (5’ to 3’):
Site 1 (forward, CTTAGGAGTCCGGTCTCTGG; reverse, CCAACAACCAAGATGATCCA), site 2 (forward, CCAGCCGAACCTTAATCCTA; reverse, AACACATATGCGCTCTCTGC), site 3 (forward, TGCAGGAACACAATCTCCTC; reverse, CACTTGGCCTGCACACTAAT), site 4 (forward, GAGCACAGAGCCCACAGTTA; reverse, TCCCAGAGCTGGAGTTCTTT), site 5 (forward, ACACCCAACCATACAGCCTT; reverse, GAAGGTGTGATAGGGCCAAC), site 6 (forward, AGGCTTCCTGGGAGAGGT; reverse, GCCTCCACATCATTCTCCA), site 7 (forward, GAGCAGAAGGAAGAGCCAAA; reverse, CTTGGCAGGTGGTTAGAAGC), site 8 (forward, TGCCAGCCACTGTAGAGTTT; reverse, ACATGTCCCAGGGAAGAGAC), site 9 (forward, CTTAGTGGTGGCTTAGTAAATGGA; reverse, GGCAGAAAGGCAAGTTCAA), site 10 (forward, GCTGCTCTCGTGTATTCTGG; reverse, AAGTGGTTGGGAATGGTTGT), site 11 (forward, AGCACAAATAAATGAAACAGGAAC; reverse, TCCTCTTGCTGTTGGGAATAG), negative control is *EphA1* (forward, ATATGACAAACACGGCCCAT; reverse, GGTGGTTAACTTGGGGAACA), *Gapdh* (forward, GTCGGAGTCAACGGATTTG; reverse, TGGGTGGAATCATATTGGAA)

For measuring H3K9me3 levels in Ascl1 regulated loci in MEFs (5' to 3'):
Dgkd (forward, AACAACAAAGCCCAAACAAA; reverse, TGCTTGCCATCAAGTTCTTC), *Id3* (forward, GAAGCTTTCCCTAGCAGGTG; reverse, AAGGCACGGCACTATTTAGG), *Nt5dc3* (forward, TGATGGTGTTAGGTGTCGCT; reverse, CGAACCTGGGCTAGAAAGAC), *Angptl2* (forward, ACACCCAGCCAGCTAGAGTT; reverse, CAGGGCACTTTCTGAACTGA), *Pianp* (forward, AGAGCGAAGAGATGAGGGAA; reverse, CATCCTCATTGCTTGGTGAC), *Grasp* (forward, CCTTCTGGGATGTTCGAGAT; reverse, ACCGGAGCACTTGAGAGACT), *Kcnf1* (forward, GCAAACTTCAGCTTCCACTG; reverse, AGAGGCTCCACTTTGCAGAT).

Data sets used (publicly available)

MEFs:
H3K4me1: ENCODE (LICR histone)
http://genome.ucsc.edu/cgi-bin/hgFileUi?db=mm9&g=wgEncodeLicrHistone
H3K4me3: ENCODE (LICR histone)
http://genome.ucsc.edu/cgi-bin/hgFileUi?db=mm9&g=wgEncodeLicrHistone
H3K9me3: Bilodeau, S. SRX038922, GSM656317, GSE2657, PMID:
H3K27me3: Koche, R.P. SRX035873, GSM640756, GSE26099, PMID: 21211784.
Gro-seq Rep1: SRX040539, GSM665997, GSE27037, PMID: 21460038
Gro-seq Rep2: SRX040540, GSM665998, GSE27037, PMID: 21460038

NPCs:
Ascl1: GSE48336
H3K4me1: Stadler, M.B. et al. SRX095620, PMID: 22170606.
H3K4me3: Mikkelsen, T. SRX001938, GSM307613, GSE12241, PMID: 17603471.
H3K9me3: Mikkelsen, T. SRX001939, GSM307616, GSE12241, PMID: 17603471.
H3K27me3: Mikkelsen, T. SRX001936, GSM307614, GSE12241, PMID: 17603471.
H3K27acetyl: Creyghton, M.P. GSE24164, GSM94585, PMID: 21106759.

NHEK:
ENCODE (Broad Histone)
http://genome.ucsc.edu/cgi-bin/hgFileUi?db=hg19&g=wgEncodeBroadHistone

NHDF:
ENCODE (Broad Histone)
http://genome.ucsc.edu/cgi-bin/hgFileUi?db=hg19&g=wgEncodeBroadHistone

HepG2:
ENCODE (Broad Histone)
http://genome.ucsc.edu/cgi-bin/hgFileUi?db=hg19&g=wgEncodeBroadHistone

Hepatocytes: ENCODE (LICR histone)
http://genome.ucsc.edu/cgi-bin/hgFileUi?db=mm9&g=wgEncodeLicrHistone

Skeletal muscle myoblasts- HSMM
ENCODE (Broad Histone)
H3K4me1: GSM733761
H3K4me3: GSM733637
H3K9me3: GSM733730
H3K27me3: GSM733667

H3K27acetyl: GSM733755

Alveolar adenocarcinoma- A549
ENCODE (Broad Histone)
H3K4me1: GSM1003453
H3K4me3: GSM1003561
H3K9me3: GSM1003454
H3K27me3: GSM1003455
H3K27acetyl: GSM1003578

Lung fibroblasts- NHLF
ENCODE (Broad Histone)
H3K4me1: GSM733649
H3K4me3: GSM733723
H3K9me3: GSM1003531
H3K27me3: GSM733764
H3K27acetyl: GSM733646

Umbilical vein endothelial cells- HUVEC
ENCODE (Broad Histone)
H3K4me1: GSM733690
H3K4me3: GSM733673
H3K9me3: GSM1003517
H3K27me3: GSM733688
H3K27acetyl: GSM733691

T cell leukemia- Dnd41
ENCODE (Broad Histone)
H3K4me1: GSM1003460
H3K4me3: GSM1003468
H3K9me3: GSM1003551
H3K27me3: GSM1003521
H3K27acetyl: GSM1003462

CD14+ cells
ENCODE (Broad Histone)
H3K4me1: GSM1003460
H3K4me3: GSM1003468
H3K9me3: GSM1003551
H3K27me3: GSM1003521
H3K27acetyl: GSM1003462

Lymphoblastoid- GM12878
ENCODE (Broad Histone)
H3K4me1: GSM733772
H3K4me3: GSM733708
H3K9me3: GSM733664
H3K27me3: GSM733758
H3K27acetyl: GSM733771

Osteoblasts
ENCODE (Broad Histone)
H3K4me1: GSM733704
H3K4me3: GSM1003506
H3K9me3: GSM733681
H3K27me3: GSM1003466
H3K27acetyl: GSM733739

Mammary epithelial cells- HMEC
ENCODE (Broad Histone)
H3K4me1: GSM733705
H3K4me3: GSM733712
H3K9me3: GSM1003485
H3K27me3: GSM733722
H3K27acetyl: GSM733660

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