Contents

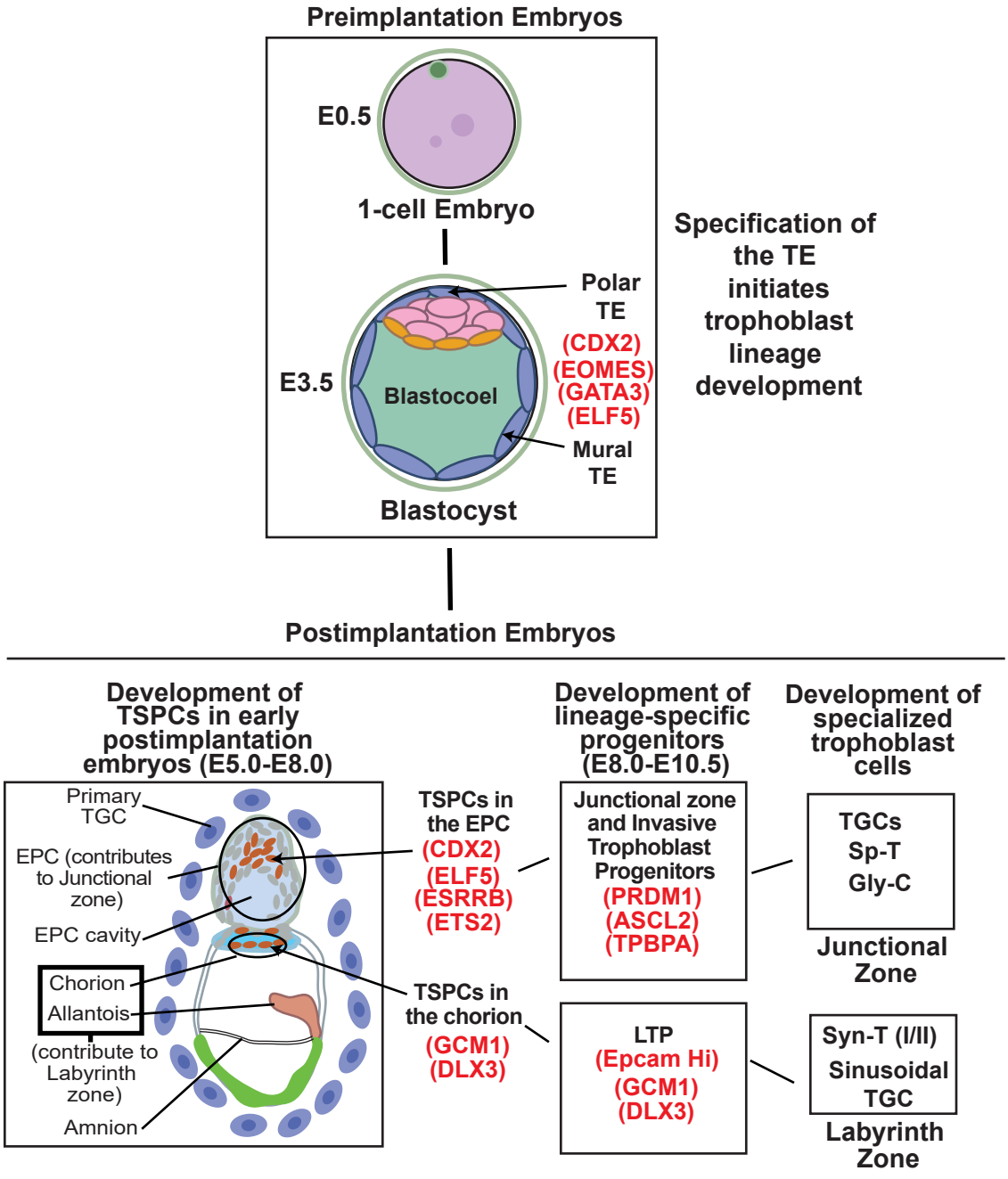

Fig. S1. Schematic presentation of different stages of trophoblast lineage development in mouse. Developmental stage-specific markers are indicated in red. In the preimplantation embryo, trophoblast development starts with the specification of TE-lineage, which mediates embryo implantation. In an early postimplantation embryo, trophoblast stem and progenitor cells (TSPCs) arise. BothTE cells and TSPCs express stem-state markers, like CDX2, EOMES and ELF5. GATA3 is also implicated in gene regulation within the TE. Later, TSPCs within the EPC and chorion differentiate into the specific trophoblast progenitor populations. PRDM1+, ASCL2+ and TPBPA+ junctional zone progenitors arise in the EPC, whereas GCM1+ and DLX3+ labyrinth trophoblast progenitors (LTPs) arise upon chorio-allantoic attachment. Finally, specialized trophoblast subtypes like trophoblast giant cells (TGCs), Spongiotrophoblast cells (Sp-T), Glycogen cells (Gly-C), and Synciotrophoblasts I. and II (SynTI, SynTII) arise to form a mature placenta

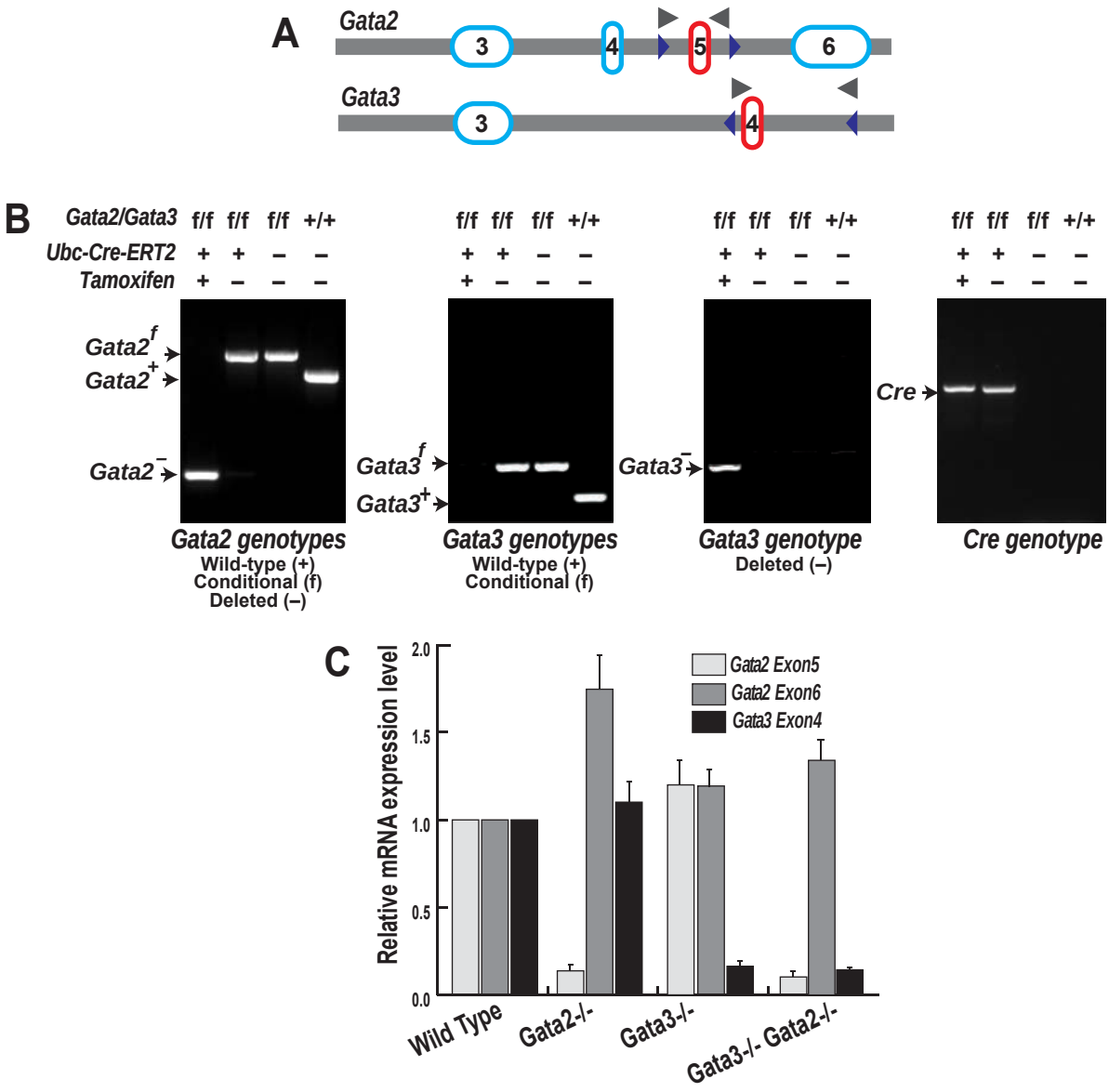

Fig. S2. (A) Schematic diagram showing part of the *Gata2* and *Gata3* loci on chromosome 6 and chromosome 2 respectively. *Gata2* exons 3, 4, 5 and 6 are shown on the diagram where exon 5 is flanked by loxP sites (blue triangle). Similarly, *Gata3* exons 3 and 4 are shown on the diagram where exon 4 is flanked by loxP sites (blue triangle). (B) Genotyping data confirming tamoxifen treatment induces deletion of floxed *Gata2* and *Gata3* alleles in the Cre expressing mouse embryos. PCR products corresponding to *Gata2 +, Gata3 +, Gata2 flox, Gata3 flox, Gata2 -, Gata3 -* are indicated. (C) Exon-specific RT-PCR analyses of the embryonic tissues showing efficient Cre-mediated excision of *Gata2* and *Gata3* floxed alleles.

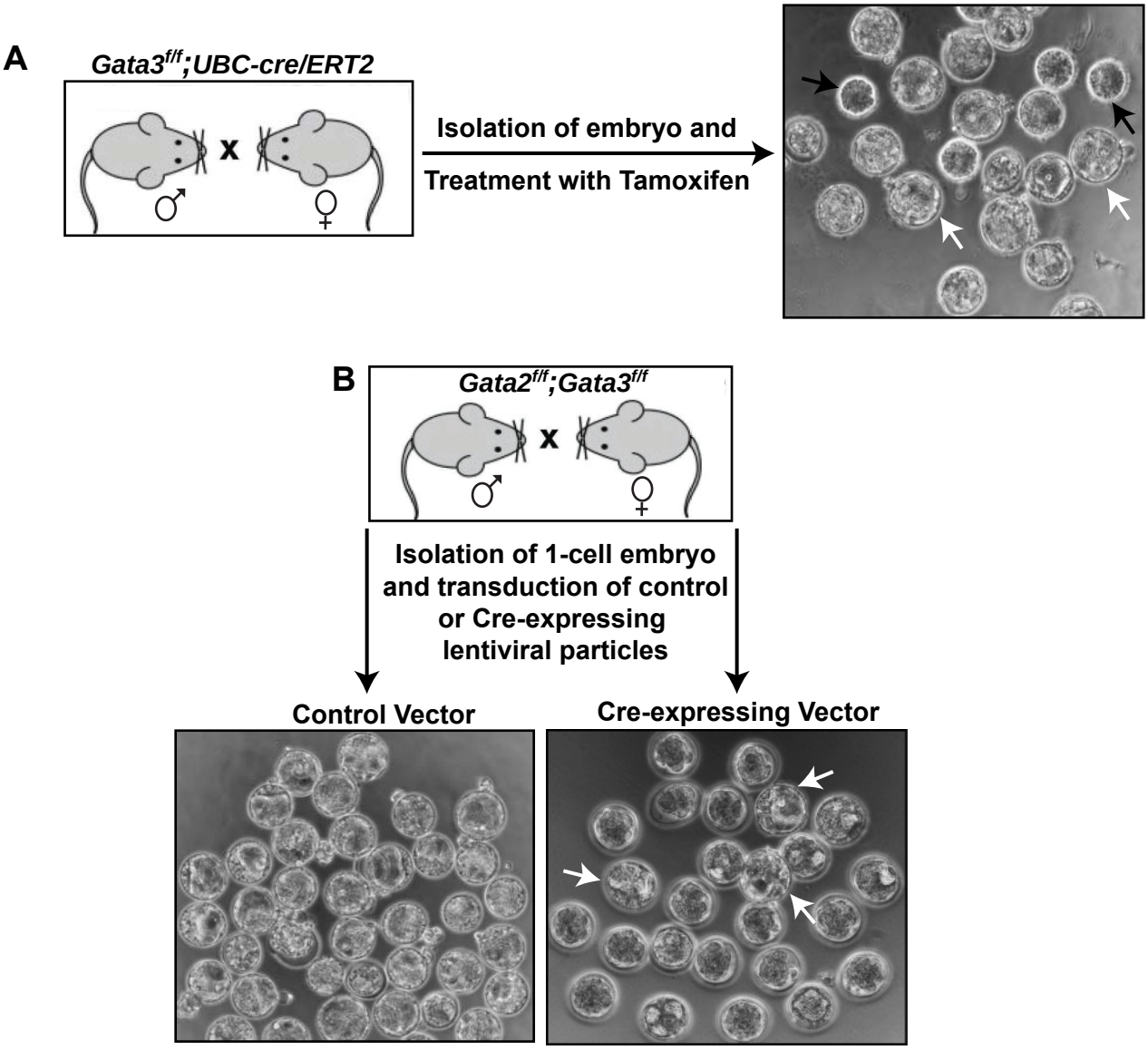

Fig. S3. (A) Schematic presentation of isolation and culture of one-cell stage *Gata3$^{f/f}$;UBC-cre/ERT2* mouse zygotes in the presence of tamoxifen results in the partial impairment of trophectoderm development. While several embryos did not develop beyond morula (black arrow), other embryos matured to the blastocyst stage (white arrows). (B) Transduction of *Gata2$^{f/f}$;Gata3f^{f}* one-cell stage mouse embryos with Cre recombinase expressing lentiviral particle results in the partial impairment of trophectoderm development but several of them matured to the blastocyst stage (white arrows). Almost all control embryos, transduced with lentiviral particles containing empty vector, matured to the blastocyst stage.

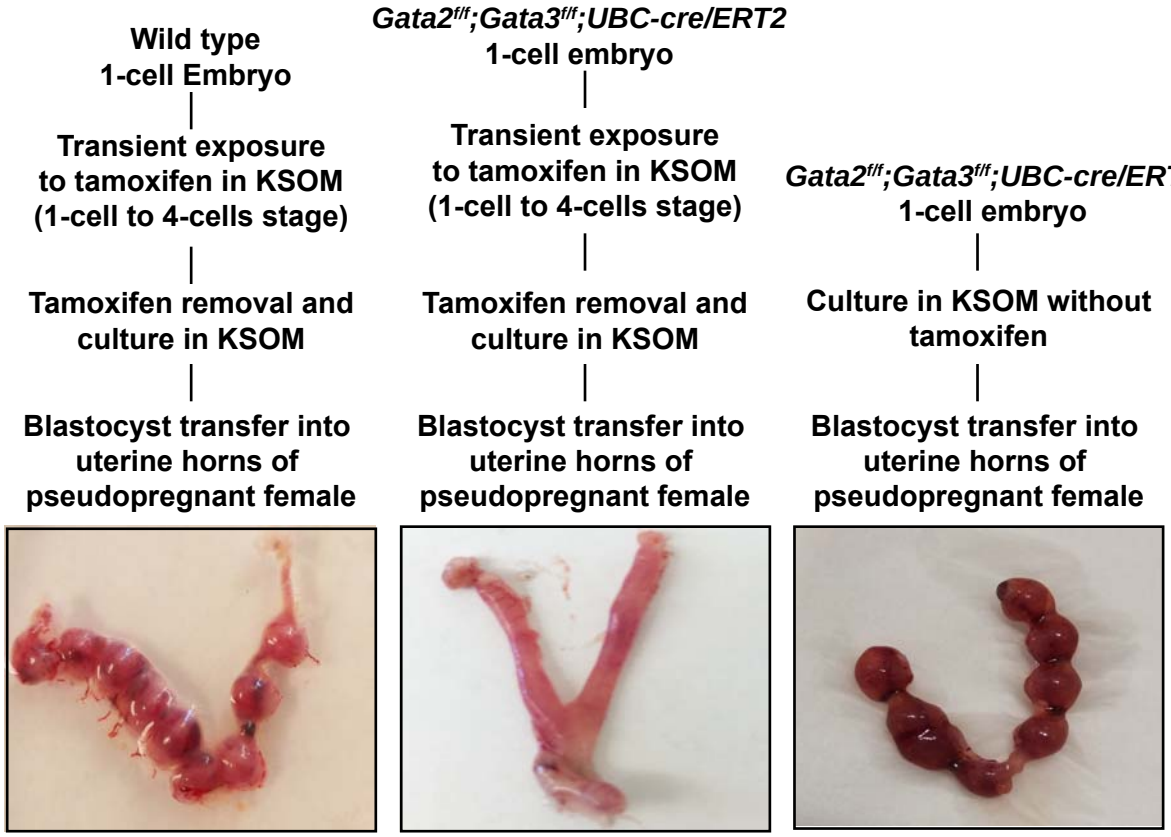

Fig. S4. Experimental strategy showing the importance of GATA2 and GATA3 function in blastocyst implantation. Wild type and *Gata2*$^{f/f}$*;Gata3*$^{f/f}$*;UBC-cre/ERT2* 1-cell embryos were treated with tamoxifen. At 4-cells stage, embryos were transferred to KSOM medium without tamoxifen and allowed to develop *ex vivo for* blastocyst maturation. In addition, *Gata2*$^{f/f}$*;Gata3*$^{f/f}$*;UBC-cre/ERT2* embryos without tamoxifen exposure were used as control. Although, transient exposure to tamoxifen impaired blastocyst development in the majority of *Gata2*$^{f/f}$*;Gata3*$^{f/f}$*;UBC-cre/ERT2* embryos, several of them developed to the blastocyst stage. Blastocyst developments were unaffected in wild type embryos upon transient tamoxifen exposure. Finally, matured blastocysts from each experimental condition were transferred into uterine horns of pseudopregnant mice. Images show impaired implantation efficiency of *Gata2*$^{f/f}$*;Gata3*$^{f/f}$*;UBC-cre/ERT2* blastocysts that were developed after tamoxifen exposure. In contrast, blastocysts from tamoxifen exposed wild type embryos and *Gata2*$^{f/f}$*;Gata3*$^{f/f}$*;UBC-cre/ERT2* embryos without tamoxifen exposure readily implanted.

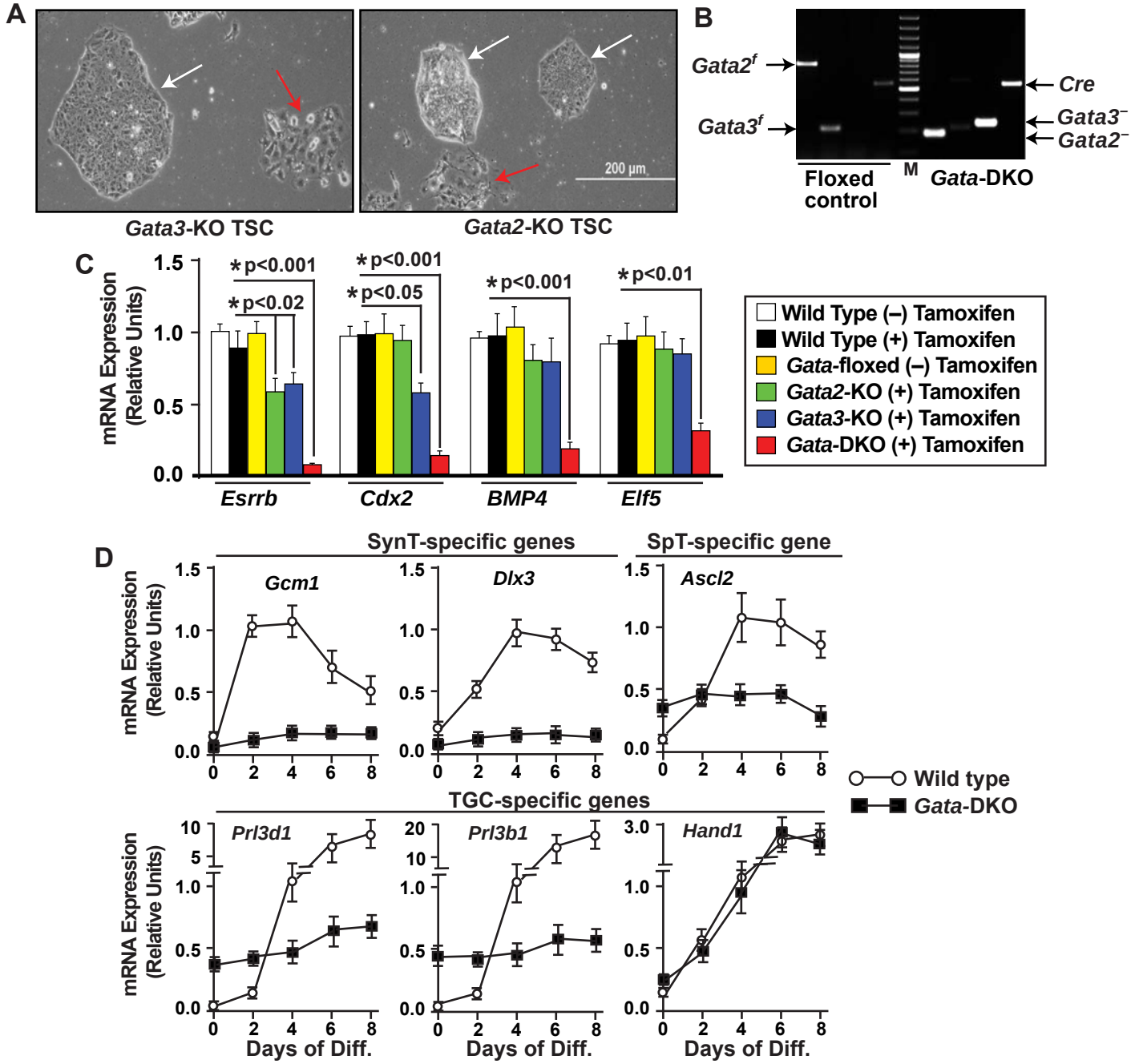

Fig. S5. (A) Micrographs show colony morphologies of *Gata2*-KO and *Gata3*-KO TSCs that were maintained in standard TSC culture condition. Both *Gata2*-KO and *Gata3*-KO TSCs largely maintains stem-state colony morphology (white arrows). However, both have higher propensities for spontaneous differentiation as revealed by the presence of colonies with differentiated cells (red arrows). (B) Genotyping shows *Gata* genes deletion in TSCs. (C) qRT-PCR analyses shows that mRNA expression of trophoblast stem state genes are strongly repressed in *Gata*-DKO TSCs. The plot shows that tamoxifen treatment does not alter gene expression in wild type control TSCs (black bars). The starting TSC population for *Gata*-DKO TSCs [*Gata* floxed TSCs (–) tamoxifen (yellow bars)] and wild type TSCs (white bars) have similar levels of gene expression. mRNA expression of *Esrrb*, which is a target gene of both GATA2 and GATA3, are repressed by ~50% in both *Gata2*-KO (green bars) and *Gata3*-KO (blue bars) TSCs. However, *Esrrb,* as well as other stem state genes were more strongly repressed in *Gata*-DKO TSCs (red bars). The expression level of a gene in wild type TSCs was considered as 1 (mean ± SE; n = 3). (D) qRT-PCR analyses showing mRNA expressions of trophoblast differentiation markers in wild-type and *Gata*-DKO TSCs when they were cultured in TSC differentiation culture condition different time interval. Representative genes of syncytiotrophoblast (SynT), spongiotrophoblast (SpT) and trophoblast giant cells (TGC) were analyzed. The expression level of a gene in wild type TSCs at day 4 of differentiation was considered as 1 (mean ± SE; n = 3).

Supplementary Materials and Methods

Cell culture and reagents

Mouse trophoblast stem cells (TSCs) were cultured with FGF4, Heparin and MEF-conditioned medium (CM) according to the protocol (Tanaka et al., 1998). To induce differentiation, Fibroblast Growth Factor 4 (FGF4), Heparin, as well as CM, were withdrawn from the culture and cells were allowed to grow. ExE/ EPCs were harvested from ~E7.0-E7.5 pregnant female mouse and were grown in the presence of FGF4, Heparin, and CM (to maintain undifferentiated state) or in mouse TSC media only (for differentiation) at the normoxic condition. For time course analysis differentiation was induced, and cells were harvested at different day point for RNA analysis.

Gene deletions in preimplantation embryos

For lentiviral-mediated Cre expression, 1-cell stage embryos were harvested from superovulated females. These embryos were subjected to perivitelline space microinjection, (Home et al., 2012), with concentrated lentiviral particles prepared from Puro.Cre empty vector expressing Cre recombinase. Injected embryos were allowed to grow in KSOM as described above. PCR confirmed successful expression of Cre recombinase and subsequent gene deletions.

For implantation efficiency experiments, wild type and *Gata2*$^{f/+}$*;Gata3*$^{f/+}$*;UBC-cre/ERT2* 1-cell embryos were treated with tamoxifen. At 4-cells stage, embryos were transferred to KSOM medium without tamoxifen and allowed to develop ex vivo for blastocyst maturation. In addition, *Gata2*$^{f/+}$*;Gata3*$^{f/+}$*;UBC-cre/ERT2* embryos without tamoxifen exposure were used as control. Matured blastocysts were transferred to surrogate females following earlier described procedures (Saha et al., 2013)

Tissue collection from postimplantation embryos

Injected animals were euthanized on at desired day points as indicated in the main text. Uterine horn and conceptuses were photographed. Conceptuses were dissected to isolate embryos, yolk sacs, and placentae. All embryos, yolk sacs, and placentae were photographed at equal magnification for comparison purposes. Uteri containing placentation sites were dissected from pregnant female mice on E7.5, E9.5, E11.5, E13.5, and E18.5 and frozen in dry ice-cooled heptane and stored at −80°C until used for histological analysis. Tissues were subsequently embedded in optimum cutting temperature (OCT) (Tissue-Tek) and were cryosectioned (10μm thick) for immunohistochemistry (IHC) studies using Leica CM-3050-S cryostat.

Flowcytometry

96 hours explant cultures of mouse ectoplacental cones were trypsinized. Single cell suspension was formaldehyde fixed and permeabilized using BD Cytofix/Cytoperm Fixation and Permeabilization solution (BD Biosciences, #554722) according to the manufacturer's protocol. All washings were done using saponin containing wash buffer. A standard protocol for doing FACS staining & analysis was followed using anti-wide spectrum Cytokeratin antibody (Abcam) to analyze Cytokeratin-positive placental trophoblast populations in a BD LSR II flow cytometer.

Genotyping

Genomic DNA was prepared using tail tissue from mouse using REDExtract-N-Amp Tissue PCR kit (Sigma-Aldrich). Genotyping was done using REDExtract-N-Amp PCR ReadyMix (Sigma-Aldrich) and respective primers. Genomic DNA from individual blastocysts was prepared

by the following technique using REDExtract-N-Amp Tissue PCR kit (Sigma-Aldrich). Each blastocyst was collected into separate PCR tubes and was lysed with 4 μl of Extraction buffer and 1μl of Tissue Prep buffer. Briefly, they were incubated at 42°C for 10 mins followed by heat inactivation at 98°C for 3 mins. and neutralization with 4μl of Neutralization buffer. 4μl of this genomic DNA was used for a 20μl PCR reaction. For genotyping in embryos, part of the yolk sac or embryo proper from each conceptus was used to prepare genomic DNA as described above. Respective primers are listed in the Table S5.

Quantitative RT-PCR

Total RNA from cells was extracted with RNeasy Mini Kit (Qiagen) with on-column DNaseI digestion. Purified RNA was used to prepare cDNA using cDNA preparation kit. All these samples were analyzed by qRT-PCR following procedures described earlier (Dutta et al., 2008). For expression analysis in preimplantation embryos, total RNA was isolated from embryos using PicoPure RNA isolation kit (Thermo Fisher Scientific) and processed as described earlier (Home et al., 2012). Primers, used for qRT-PCR analysis, are listed in the Table S5.

Immunofluorescence

For immunostaining, preimplantation embryos were fixed with 4% paraformaldehyde, permeabilized in 0.25% Triton X-100, and blocked with 10% fetal bovine serum and 0.1% Triton X-100 in phosphate-buffered saline (PBS) for 1 h at room temperature. Embryos were incubated with antibodies (1:100 dilution) overnight at 4 °C, washed with 0.1% Triton X-100 in PBS. After incubation (1:400, 1 hour, room temperature) with conjugated secondary antibodies, embryos were washed and mounted using anti-fade mounting medium (Thermo Fisher Scientific) containing DAPI and viewed in LSM 5 Laser Scanning Microscope (Carl Zeiss Microimaging). For IHC with mouse tissues, slides containing cryosections were thawed and fixed with 4% PFA

followed by permeabilization with 0.25% Triton X-100 and blocking with 10% fetal bovine serum and 0.1% Triton X-100 in PBS. Sections were incubated with primary antibodies overnight at 4 °C, washed with 0.1% Triton X-100 in PBS. After incubation (1:400, 1 h, room temperature) with conjugated secondary antibodies, sections were washed, mounted using anti-fade mounting medium (Thermo Fisher Scientific) containing DAPI and visualized using Nikon Eclipse 80i fluorescent microscope. To test expression of GATA3 in postimplantation embryos, *Gata3-LacZ* knock-in mice (Pandolfi et al., 1995) were used. Staining was done using placentation sites from pregnant *Gata3*$^{-/+}$ female mice (*lacZ* knock-in). Anti-β Galactosidase antibody was used to stain β Galactosidase in the GATA3 expressing cells. Antibodies, used for immunofluorescence analyses are mentioned in Table S6.

Immunohistochemistry

Paraffinized placental sections were processed for immunostaining according to the protocol described by Holets et al. (Holets et al., 2006). Briefly, 10-µm tissue sections were cut from paraffinized first-trimester placentas. Sections (10µm thick) were placed onto slides, rehydrated and were subjected to heat mediated antigen retrieval using citrate based Reveal buffer (BioCare Medical). Non-specific immunoglobulin binding was blocked with 10% normal goat serum (Thermo Fisher Scientific). The primary antibody or its isotype-specific control (IgG1) was incubated with the tissue sections for 4°C overnight. Secondary antibody (biotinylated goat anti-mouse/ rabbit IgG) (Vector Laboratories) incubation was followed by endogenous peroxidase depletion using 3% H_2O_2. Reactivity was detected using the streptavidin-peroxidase (Thermo Fisher Scientific) and DAB reagent kit (Dako) and tissues were counterstained with Mayer's hematoxylin (Sigma-Aldrich). Positive staining was confirmed as a brown coloration under the microscope.

Quantitation of trophoblast cell population

Tissue sections from three individual placentation sites were used to quantitate trophoblast giant cell (TGC) and spongiotrophoblast (SpT) numbers in *Floxed*-control and *Gata*-DKO embryos. Cell populations at three different areas of equal size within the junctional zone of each tissue sections were counted for TGCs and SpTs. The data was plotted as a relative percentage considering the average cell number/area in control embryos as 100%.

Statistical analyses

We used at least 3 independent cultures for the experiments with single KO or double KO analyses and indicated those numbers with “n” in the legends. Similarly, at least 3 biological replicates were used for the analyses with blastocysts, ExE/ EPC explant cultures, placentae and conceptuses.

ChIP and ChIP-Seq

Quantitative ChIP analysis was performed following published protocols (Home et al., 2009; Home et al., 2012). TSCs, homogenized EPCs, and homogenized placentae cells were cross-linked by with 0.4% formaldehyde (Sigma) for 10 mins at room temperature with gentle rotation. Chromatin crosslinking were stopped with glycine (125mM). These samples were sonicated. Chromatin fragments were immunoprecipitated with different antibodies. Quantification of the precipitated DNA was performed using qPCR amplification. A list of the primers used for ChIP analysis and the antibodies used for ChIP analysis are mentioned in the Table S5 and S6. For ChIP-seq in TSCs, immunoprecipitated chromatin fragments from three independent

experiments were pooled. Libraries were sequenced in Illumina Genome Analyzer II using TruSeq SBS kit v5-GA chemistry and in Illumina HiSeq using TruSeq SBS v2-HS chemistry (Illumina, San Diego, CA) to generate 35 bp single-end reads. Binding of the nonspecific immunoglobulin G (IgG) antibody was used as the negative control for eliminating false positive peaks.

Sequences were aligned using ELANDv2 (CASAVA 1.7) to the mouse reference genome (NCBI37/mm9) using default parameters. Peak detection was performed using the Model-based Analysis of ChIP-Seq (MACS) software (Zhang et al., 2008). MACS was run with the peak detection p-value cutoff set at 1e-5 (default). Highly enriched peaks were selected from the two experiments based on a false discovery rate (FDR) cutoff of 1% for GATA2 sites and 15% for GATA3 sites. We further searched for the GATA2 and GATA3 consensus sequence in their respective ChIP-Seq targets within a 250 bp region from either side of the peak center using a weight-matrix match with at least 80% similarity. The weight matrices were obtained from the JASPAR database (Sandelin et al., 2004). A substantial proportion of the highly enriched GATA2 and GATA3 ChIP-Seq binding sites consisted of at least one instance of the consensus motif. All raw data for ChIP-seq analyses are submitted to the GEO database (http://www.ncbi.nlm.nih.gov/gds), with accession number GSE92295.

RNA-Seq analysis

The changes in gene expression as a result of the double knockout of GATA2 and GATA3 was measured by whole transcriptome sequencing (RNA-Seq) of control and *Gata*-DKO TSCs. Sequencing was performed on an Illumina HiSeq 2000 sequencing machine (*Illumina, San Diego, CA*) at a 50 bp single-end resolution. Sequence reads were mapped to the mouse reference genome (GRCm38) using STAR (Dobin et al., 2013) with default parameters. Transcript abundance estimates were generated using Cufflinks (Trapnell et al., 2010) and

differential gene expression calculated using Cuffdiff (Trapnell et al., 2013) with default parameters. Approximately, 44.3 and 41.1 million reads were generated of which around 96.7% and 96.6% were mapped to the genome for the control and *Gata*-DKO TSC samples respectively. In the absence of replicate samples, Cuffdiff uses a heuristic approach to generate a significance p-value (adjusted for false discovery by the Benjamini and Hochberg method (Benjamini and Hochberg, 1995) giving a q-value) where the variance is measured across conditions under the assumption that most transcripts are not differentially expressed. While these p-values do not substitute for a p-value derived with biological replicates, they form a reasonable statistic to filter the gene list. Genes with an absolute fold change ≥ 1.5 fold and a q-value ≤ 0.05 were deemed significant for further analysis. All raw data for RNA-seq analyses are submitted to the GEO database (http://www.ncbi.nlm.nih.gov/gds), with accession number GSE92295.

Combining ChIP-Seq and RNA-Seq data

Genes with a highly enriched GATA2 and GATA3 binding site within 50,000 bp upstream from the 5 prime end or downstream from the 3 prime end or overlapping the gene were selected. These genes were further filtered to include genes that were significantly differentially expressed or remained silent in the *Gata*-DKO sample. Ingenuity Systems Pathway Analysis software (IPA, Ingenuity Systems, www.ingenuity.com) was used to identify the biological functions that are associated with significantly differentially expressed genes (from the *Gata*-DKO samples) that contained both an enriched GATA2 and GATA3 site in its vicinity. IPA performs this task with the aid of its knowledge base which has curated information from the literature of genes and gene products that interact with each other. IPA use the right-tailed Fisher's exact test to calculate a significance p-value of the overlap between these genes and genes associated with a particular biological function. A *p-value* less than or equal to 0.05 is considered significant. The IPA database contains information from the literature on the relative

direction of a gene's expression in relation to a biological function. Using this information, IPA calculates an activation z-score for a biological function in relation to a set of genes (with expression information) indicating whether the function is activated of inhibited, based on the directionality of expression of the genes (Kramer et al., 2014). A positive activation z-score signifies an increase in the biological function's activity and a negative score signifies a decrease in its activity. Biological functions with an absolute activation z-score greater than or equal to 2 were considered significant.

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Table S1A

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Table S1B

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Table S2

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Table S3

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Table S4

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Table S5. Primer list

Primers used for genotyping			
Mouse	Gata2-floxed and Gata2-null	GCCTGCGTCCTCCAACACCTCTAA	TCCGTGGGACCTGTTTCCTTAC
Mouse	Gata3-flox	CAGTCTCTGGTATTGATCTGCTTCTT	GTGCAGCAGAGCAGGAAACTCTCAC
Mouse	Gata3-null	TCAGGGCACTAAGGGTTGTTAACTT	GTGCAGCAGAGCAGGAAACTCTCAC
	Cre	AAAATTTGCCTGCATTACCG	ATTCTCCCACCGTCAGTACG
Primers used for quantitative RT-PCR analysis			
Species	**Gene**	**Forward 5'**	**Reverse 3'**
Mouse	*Gata3 Ex3/4*	CGGGTTCGGATGTAAGTCGA	GTAGAGGTTGCCCCGCAGT
Mouse	*Gata2 Ex5*	CAGACGACAACCACCACCTT	GCTTGTAGTAGAGGCCACAGG
Mouse	*Gata2 Ex6*	GGAAGATGTCCAGCAAATCC	TGGAGAGCTCCTCGAAACAT
Mouse	*Cdx2*	GCAGTCCCTAGGAAGCCAAGTGA	CTCTCGGAGAGCCCGAGTGTG
Mouse	*Hand1*	GGCAGCTACGCACATCATCA	CCTGGCATCGGGACCATAG
Mouse	*Prl3d1*	ACATTTATCTTGGCCGCAGATGTGT	TTTAGTTTCGTGGACTTCCTCTCGAT
Mouse	*Prl3b1*	GGGGCACTCCTGTTGCTGGCA	GGACTTGCTCGCTGTTTTCTGGA
Mouse	*Elf5*	ATGTTGGACTCCGTAACCCAT	GCAGGGTAGTAGTCTTCATTGCT
Mouse	*Ascl2*	AAGTGGACGTTTGCACCTTCA	AAGCACACCTTGACTGGTACG
Mouse	*Gcm1*	CTGACTGGTTCCAGGAGTGG	TGTCGTCCGAGCTGTAGATG
Mouse	*Prl2c2*	GACCATTCCTCATTGCACACA	TCCTGGATACTGCTCCTACTACT
Mouse	*Prdm1*	TCAAGCCGAGGCATCCTTAC	AGCGTGTTCCCTTCGGTATG
Mouse	*Esrrb*	AGTACAAGCGACGGCTGG	CCTAGTAGATTCGAGACGATCTTAGTCA

Mouse	*Tfap2c*	ATCCCTCACCTCTCCTCTCC	CCAGATGCGAGTAATGGTCGG
Mouse	*Dlx3*	CACTGACCTGGGCTATTACAGC	GAGATTGAACTGGTGGTGGTAG
Mouse	*Foxp1*	GGTCTGAGACAAAAAGTAACGGA	CGCACTCTAGTAAGTGGTTGC
Mouse	*Bmp4*	GACTTCGAGGCGACACTTCTA	GAATGACGGCGCTCTTGCTA
Mouse	*Foxd3*	ACCACGTCGCTCATCAAGTC	GCGCCTATGATGTTCTCGAT
Mouse	*Ets2*	ACGGGCCTGGATTCTGTCT	TGAGCAAAGGCAGCTCGC
Mouse	*Pcsk3*	TCGGTGACTATTACCACTTCTGG	CTCCTGATACACGTCCCTCTT
Mouse	*Pcsk6*	CAGGCGCGAAGTGACTCTC	GACCGACAGCGACTGTTCTT
Mouse	*Porcn*	GCATGCTTCAGGTAAGACGG	CCATCTGCTTCGCCTGCC
Mouse	*18s rRNA*	AGTTCCAGCACATTTTGCGAG	TCATCCTCCGTGAGTTCTCCA
Mouse	*Gapdh*	TGCCCCCATGTTTGTGATG	TGTGGTCATGAGCCCTTCC
Primers used for quantitative ChIP analyses			
Mouse	*Prdm1 promoter*	CGAAGTACGTCGGATCCTGT	GGGGACTCCTCCTCAAAAGA
Mouse	*Gcm1 promoter*	TGATTGGACAGTTGCCAGAG	AAGTGGTCGCTGTTCCCTAA
Mouse	*Dlx3 promoter*	TCCTTCCACAAACACCCAAT	GGTGGGCTTAGGTGAGATGA
Mouse	*Ascl2 promoter*	GGAGAGCTGGCTGTAAGGTG	TTGCCCTGACCTGAGAGAAT

Table S6. Antibody list

Primary antibodies	Species raised in	Vendor	Catalog number	Batch/ Lot number	Dilutions used
anti-GATA2	Rabbit	Abcam	ab109241	GR143635-2	1:100 (IF/IHC), 6μg/10 million cells for ChIP
anti-GATA3	Mouse	BD Biosciences	558686	5288632	1:100 (IF/IHC), 6μg/10 million cells for ChIP
anti- β-Galactosidase	Mouse	Promega	Z3781	0000125058	1:200 (IF)
anti-CDX2	Rabbit	Abcam	EPR2764Y	GR133702-8	1:100 (IF)
anti-Oct-3/4	Mouse	Santa Cruz Biotechnology	sc-5279	G1610	1:100 (IF)
anti-wide spectrum Cytokeratin	Rabbit	Abcam	ab9377	GR218349-5	1:100 (IF)
anti-pan Cytokeratin	Mouse	Abcam	ab7753	GR185314-12	1:100 (IF)
anti-Vimentin	Mouse	Santa Cruz Biotechnology	sc-373717	K2713	1:100 (IF)
anti-Proliferin	Goat	Santa Cruz Biotechnology	sc-47347	K1212	1:75 (IF)
anti-trimethyl Histone H3 (Lys9)	Mouse	Millipore	05-1242	NG1698976	6μg/10 million cells for ChIP
anti-trimethyl Histone H3 (Lys27)	Rabbit	Millipore	CS200603	1987188	6μg/10 million cells for ChIP
anti-trimethyl Histone H3 (Lys4)	Rabbit	Millipore	CS200580	DAM1612220	6μg/10 million cells for ChIP
Anti- RNA Pol II (H5)	Mouse	BioLegend	MMS-129R	14862302	6μg/10 million cells for ChIP

Purified IgG1 k isotype control	Mouse	BD Biosciences	554121	4324640	6μg/10 million cells for ChIP
Purified IgG	Rabbit	BD Biosciences	550875	2139944	6μg/10 million cells for ChIP
Secondary antibodies	**Species raised in**	**Vendor**	**Catalog number**	**Batch/ Lot number**	**Dilutions used**
Alexa Fluor 568 anti-rabbit IgG	Donkey	Thermo Fisher Scientific	A10042	1606268	1:400 (IF)
Alexa Fluor 488 anti-mouse IgG	Donkey	Thermo Fisher Scientific	A21202	1562298	1:400 (IF)
Alexa Fluor 647 anti-goat IgG	Donkey	Thermo Fisher Scientific	A21447	1661244	1:400 (IF)
Biotinylated anti-mouse IgG	Goat	Vector Labs	BA-9200	W0206	10μg/ml (IHC)
Biotinylated anti-rabbit IgG	Goat	Vector Labs	BA-1000	X0212	10μg/ml (IHC)