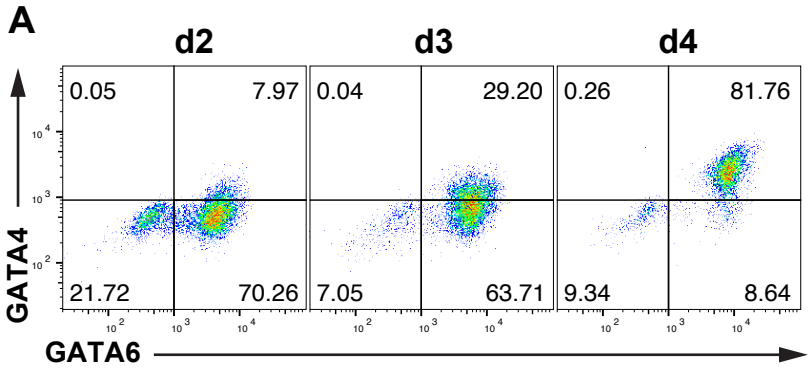
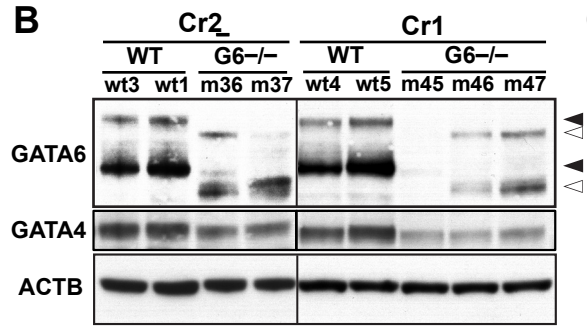
Inventory of Supplemental Information

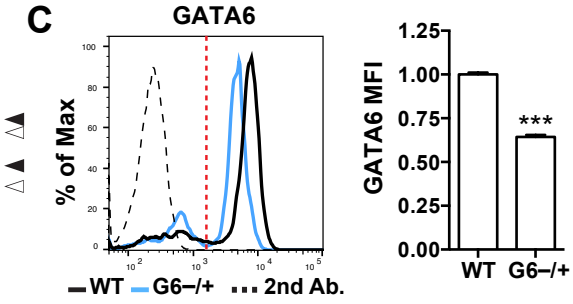
Supplementary Figures:

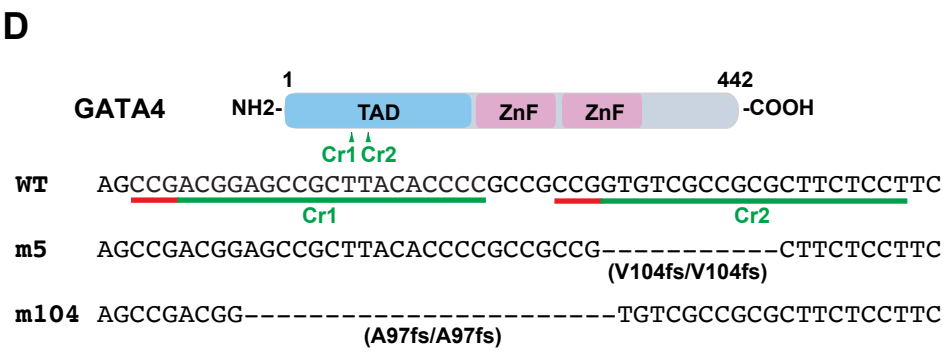
Supplementary Tables:

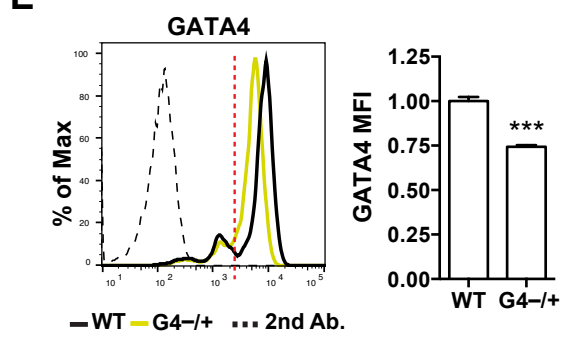
Shi_Supplementary Figure 1

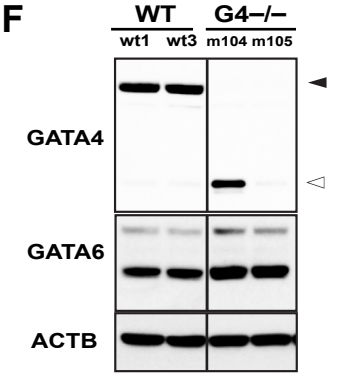


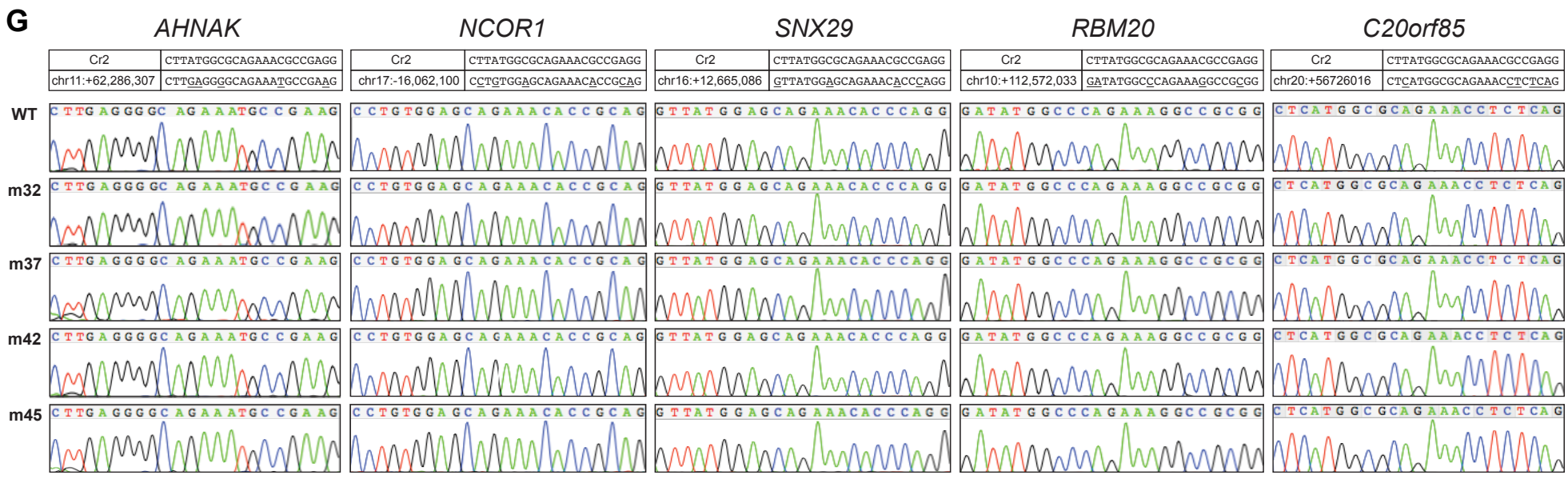


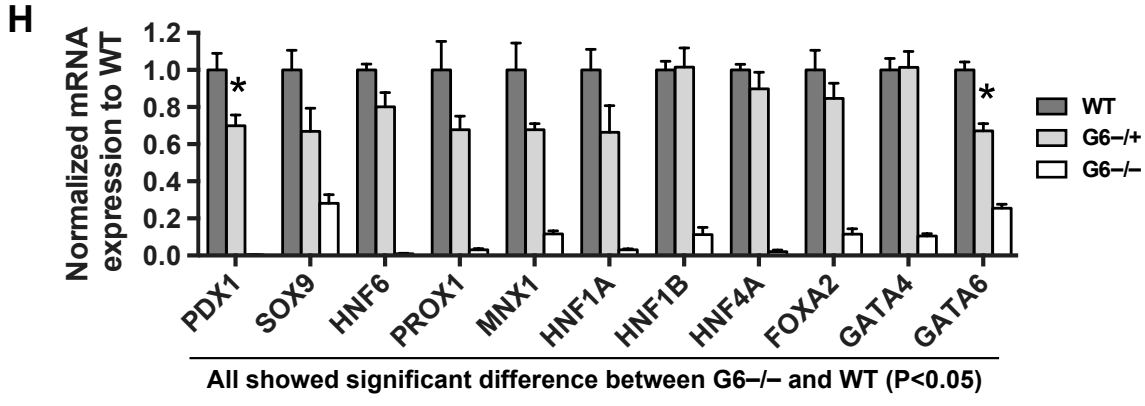


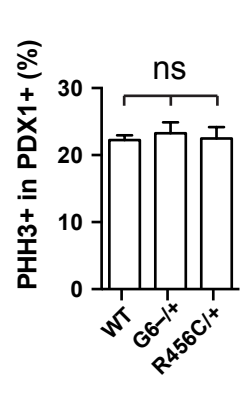


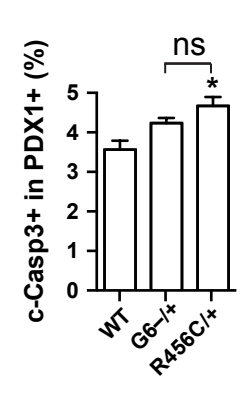


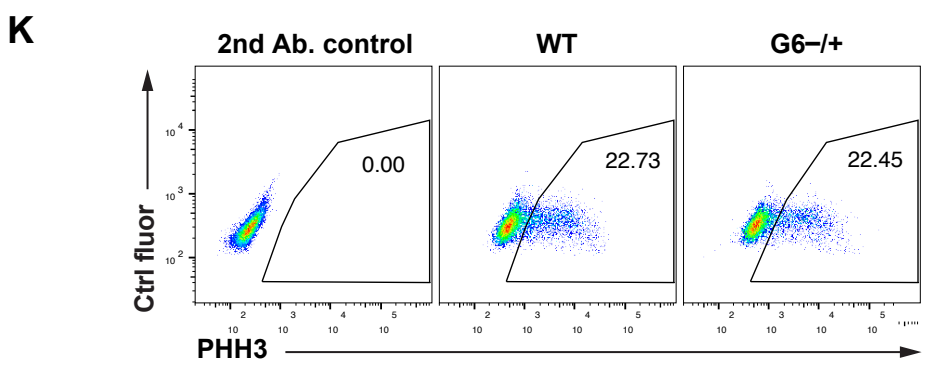












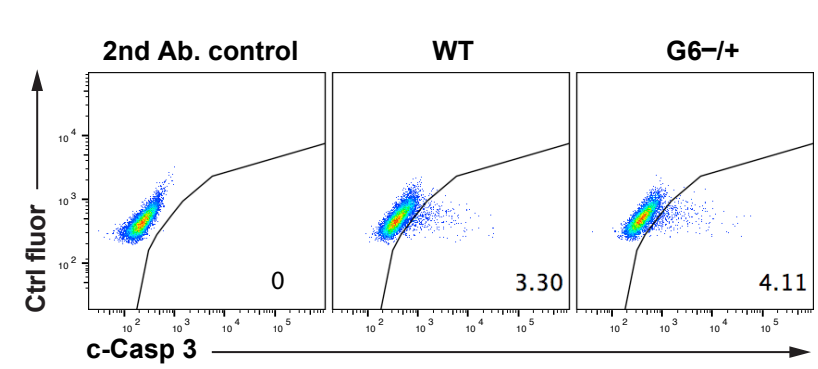


Figure S1. *GATA6* and *GATA4* mutant hPSC clones, pancreatic progenitor marker analysis, and *GATA6* mutant characterization. Related to Figures 2, 3 and 6.

(A) Time course study for emergence of GATA6+ cells and GATA4+ cells during DE differentiation. GATA6+ cells appeared earlier than GATA4+ cells and GATA4+ cells always express GATA6.

(B) GATA6 mutant cells were differentiated to DE stage and collected for Western blotting to detect GATA6 and GATA4 proteins. GATA4 expression was reduced in *GATA6*$^{-/-}$ cells. Solid arrowheads indicate the wild-type GATA6 protein isoforms. Unfilled arrowheads indicate truncated GATA6 proteins from mutant cells.

(C) Representative FACS histograms and median fluorescence intensity (MFI) quantification for *GATA6*$^{-/+}$ mutants at the DE stage stained with the GATA6 antibody (n=12).

(D) Generation of GATA4 mutants. Two CRISPR gRNAs (GATA4-Cr1 and Cr2) were used for generating GATA4 mutant clones. Sequences for two representative homozygous mutants (m5 and m104) with frameshift mutations were shown.

(E) The cells were differentiated to DE stage and processed for FACS analysis. Representative FACS histograms MFI quantification for *GATA4*$^{-/+}$ mutants stained with the GATA4 antibody (n=7).

(F) The cells were differentiated to DE stage and collected for Western blotting to detect GATA4 and GATA6 proteins. GATA6 expression was slightly increased in *GATA4*$^{-/-}$ lines. The solid arrowhead indicates WT GATA4 protein, and the unfilled arrowhead indicates the truncated GATA4 proteins from the mutant allele.

(G) Potential off-target analysis for *GATA6* mutant clones. Sequencing results from the top 5 potential off-target exomic regions identified using an online tool (crispr.mit.edu).

(H) RT-qPCR measurement of expression of PP1 pancreatic progenitor markers. The level of mRNA expression of each gene was normalized to its expression in the WT controls (n=4).

(I) Quantification of FACS analysis of PHH3+ cells for WT, *GATA6*$^{-/+}$ and *GATA6*$^{R456C/+}$ lines (n=6). Note that ns indicates not statistically significant throughout.

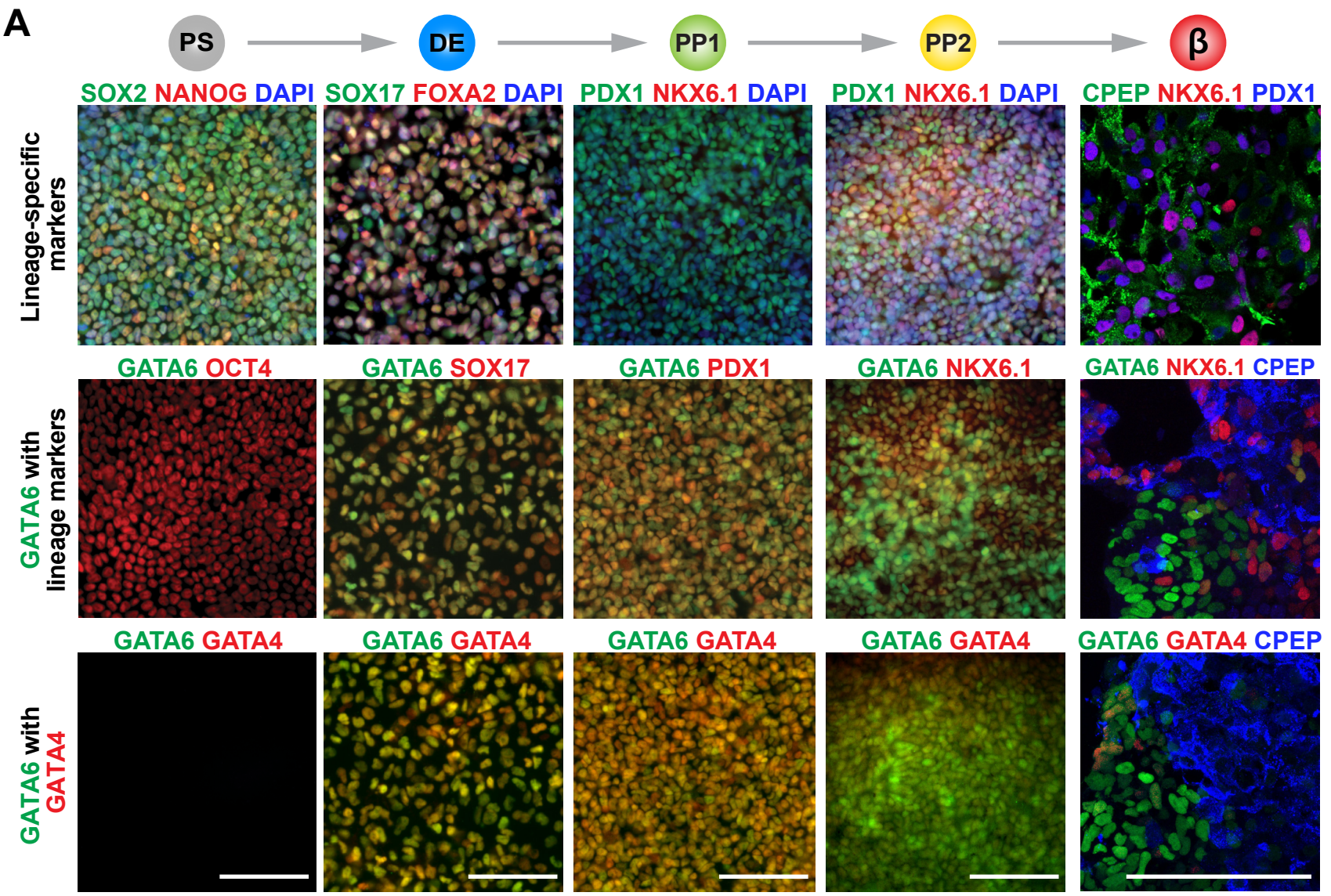
(J) Quantification of FACS analysis of cleaved caspase3+ cells for WT, *GATA6*$^{-/+}$ and *GATA6*$^{R456C/+}$ lines (n=4).

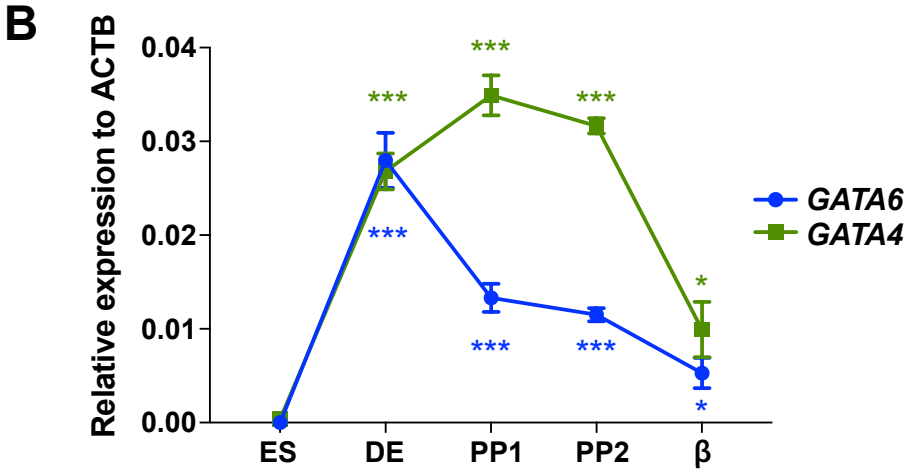
(K) Representative FACS dot plots for PHH3+ cells in the PDX1+ cell population. PHH3: Phospho-Histone H3, a proliferation marker. 2nd Ab.: controls stained with only the secondary antibody. The PDX1+ cell population was first gated and then PHH3+ cell population was gated within the PDX1+ cell population.

(L) Representative FACS dot plots for cleaved Caspase-3+ (c-Casp3+) cells in the PDX1+ cell population. The analysis was done in the same way as for PHH3.

All data in this figure were generated from HUES8 lines using the 1st-generation differentiation protocol.

Shi_Supplementary Figure 2





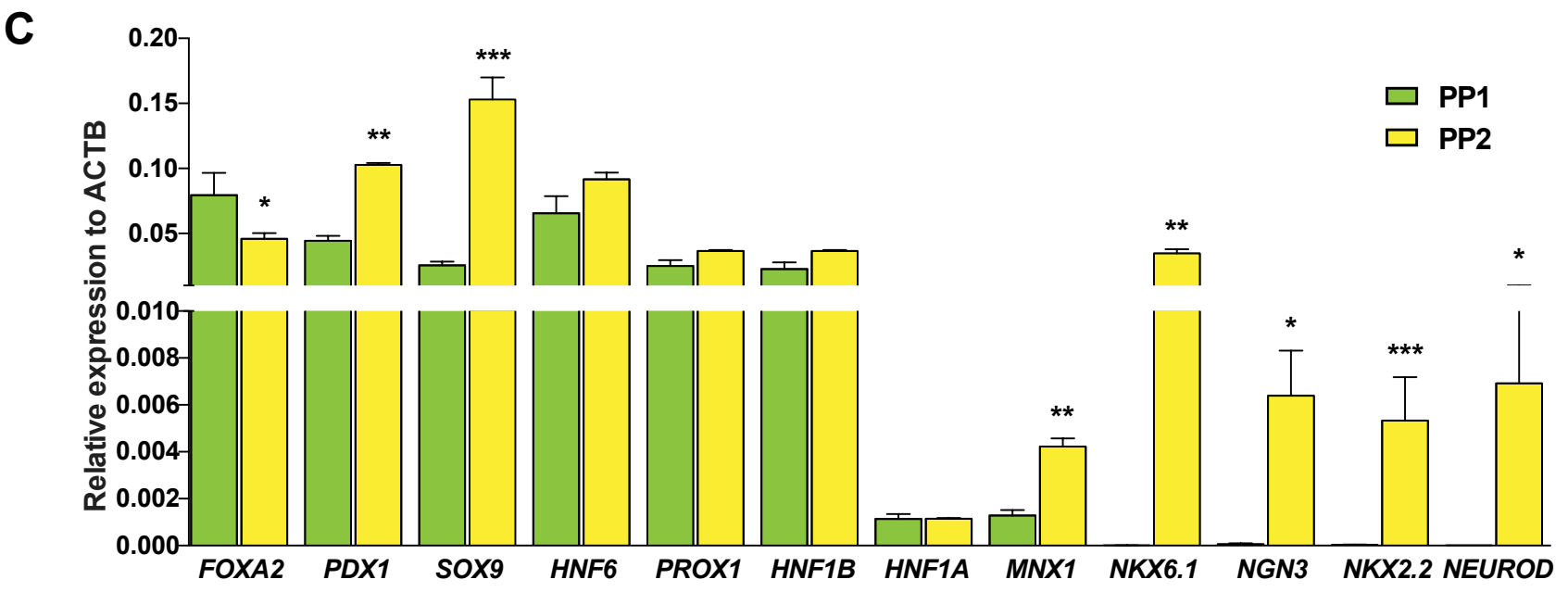


Figure S2. GATA6 and GATA4 expression during hPSC differentiation using the 2nd-generation protocol. Related to Figure 4.

(A) Representative immunostaining of key markers at each stage (first row), GATA6 co-staining with lineage markers (second row), or GATA4 (third row). Scale bar is 100 μm for all images. The immunostaining showed co-expression of GATA4 and GATA6 at both PP1 and PP2 stages, with a significant decrease in GATA4 expression level at the PP2 compared to the PP1 stage.

(B) GATA6/4 mRNA expression at each differentiation stage. The expression levels were normalized to the internal control *ACTB*. t-test with two-tailed distribution was used to determine the significance (n=3).

(C) Pancreatic progenitor marker expression at PP1 and PP2 stages. The expression levels were normalized to the internal control *ACTB*. Paired T-test with two-tailed distribution was used to determine the significance (n=3).

All data in this figure were generated from H1 lines using the 2nd-generation differentiation protocol.

Shi_Supplementary Figure 3

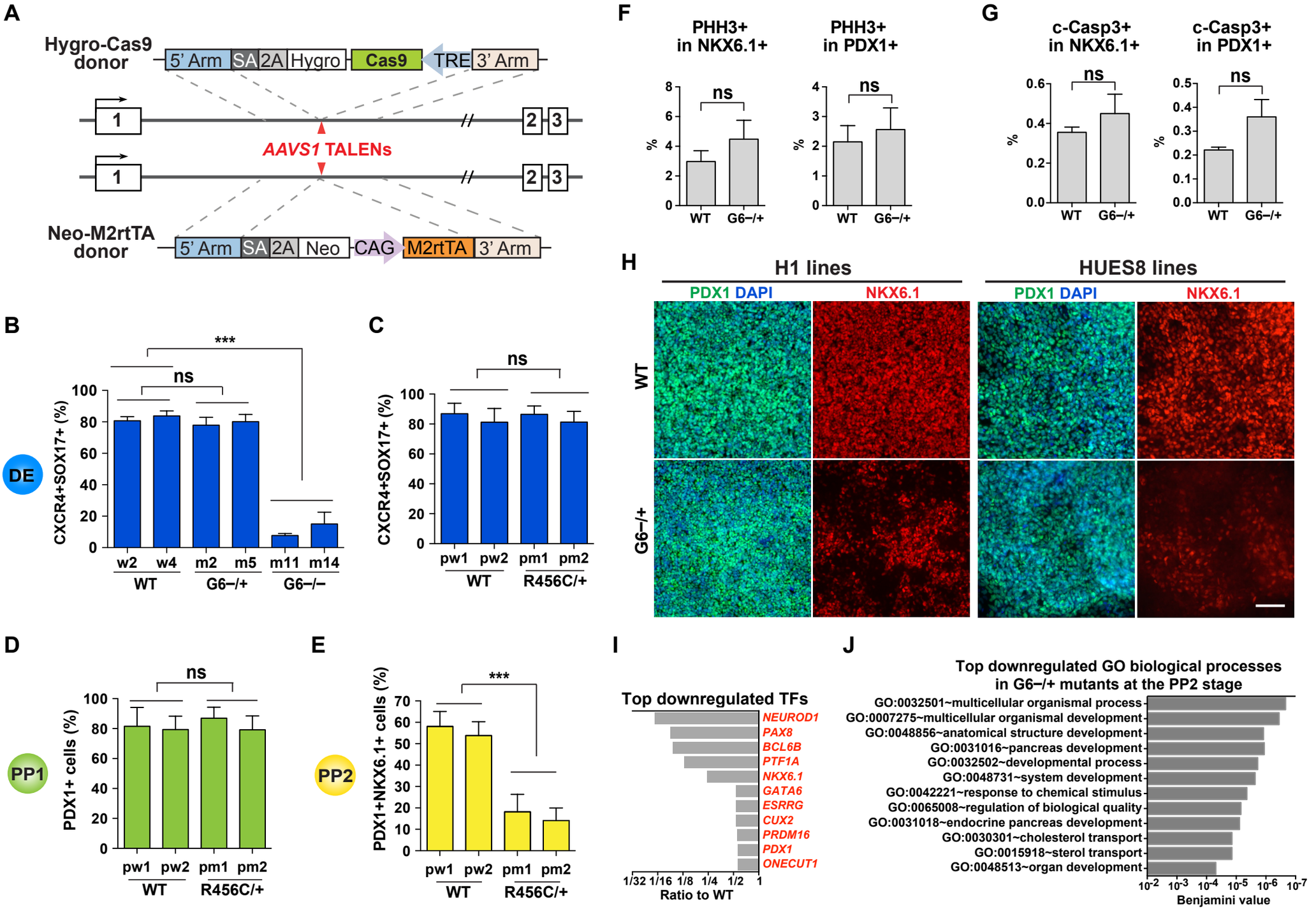


Figure S3. GATA6 haploinsufficiency in PP2 specification. Related to Figure 4.

(A) Generation of H1 iCas9 hPSC lines through TALEN-mediated gene targeting at the *AAVS1* locus. SA, splice acceptor; 2A, self-cleaving 2A peptide; Neo, neomycin resistance gene; CAG, constitutive synthetic promoter; M2rtTA, reverse tetracycline-controlled transactivator sequence; Hygro, hygromycin resistance gene; TRE, tetracycline response element. Hygro-Cas9 plasmid was established by cloning Cas9 into the Hygro-iDEST vector (Addgene #75339). Information for the AAVS1 TALENs and Neo-M2rtTA plasmids can be found in Addgene (#59025, 59026 and 60843).

(B) FACS quantification of CXCR4+SOX17+ cell percentage at DE stage. WT lines are w2 and w4, *GATA6*$^{-/+}$ mutant lines are m2 and m5, and *GATA6*$^{-/-}$ lines are m11 and m14, in which w2, m2 and m11 were picked from GATA6-Cr1 targeting and w4, m5 and m14 were picked from GATA6-Cr2 targeting. *GATA6*$^{-/+}$ mutant group is not significantly (ns) different compared to the WT group but the *GATA6*$^{-/-}$ mutant group is significantly different from the WT group (n=4-12).

(C) FACS quantification of CXCR4+SOX17+ cell percentage at DE stage for disease-mimicking H1 hPSC lines. WT lines are pw1 and pw2, *GATA6*$^{R456C/+}$ mutant lines are pm1 and pm2 (ns, not significant. n=6).

(D) FACS quantification of PDX1+ cell percentage at PP1 stage for disease-mimicking H1 hPSC lines (ns, not significant. n=6).

(E) Quantification and analysis of PDX1+NKX6.1+ cells stage for disease-mimicking lines based on FACS analysis from 3 independent experiments. The lines with the same genotypes were treated as one group (n=6).

(F) Percentage of PHH3+ cells within NKX6.1+ cells or PDX1+ cells (n=6).

(G) Percentage of cleaved caspase3 (c-Casp3)+ cells within NKX6.1+ cells or PDX1+ cells (n=6).

(H) Immunostaining shows *GATA6*$^{-/+}$ lines (both H1 and HUES8 lines) showed impaired specification of PDX1+NKX6.1+ PP2 cells.

(I) Top downregulated transcription factors (TFs) in GATA6−/+ mutants at the PP2 stage based on RNA-seq analysis.

(J) Top downregulated biological process gene ontology (GO) analysis in GATA6−/+ mutants at the PP2 stage.

All data in this figure were generated using the 2nd-generation differentiation protocol from H1 lines, except that S3H contains data from both H1 and HUES8 lines as indicated.

Shi_Supplementary Figure 4

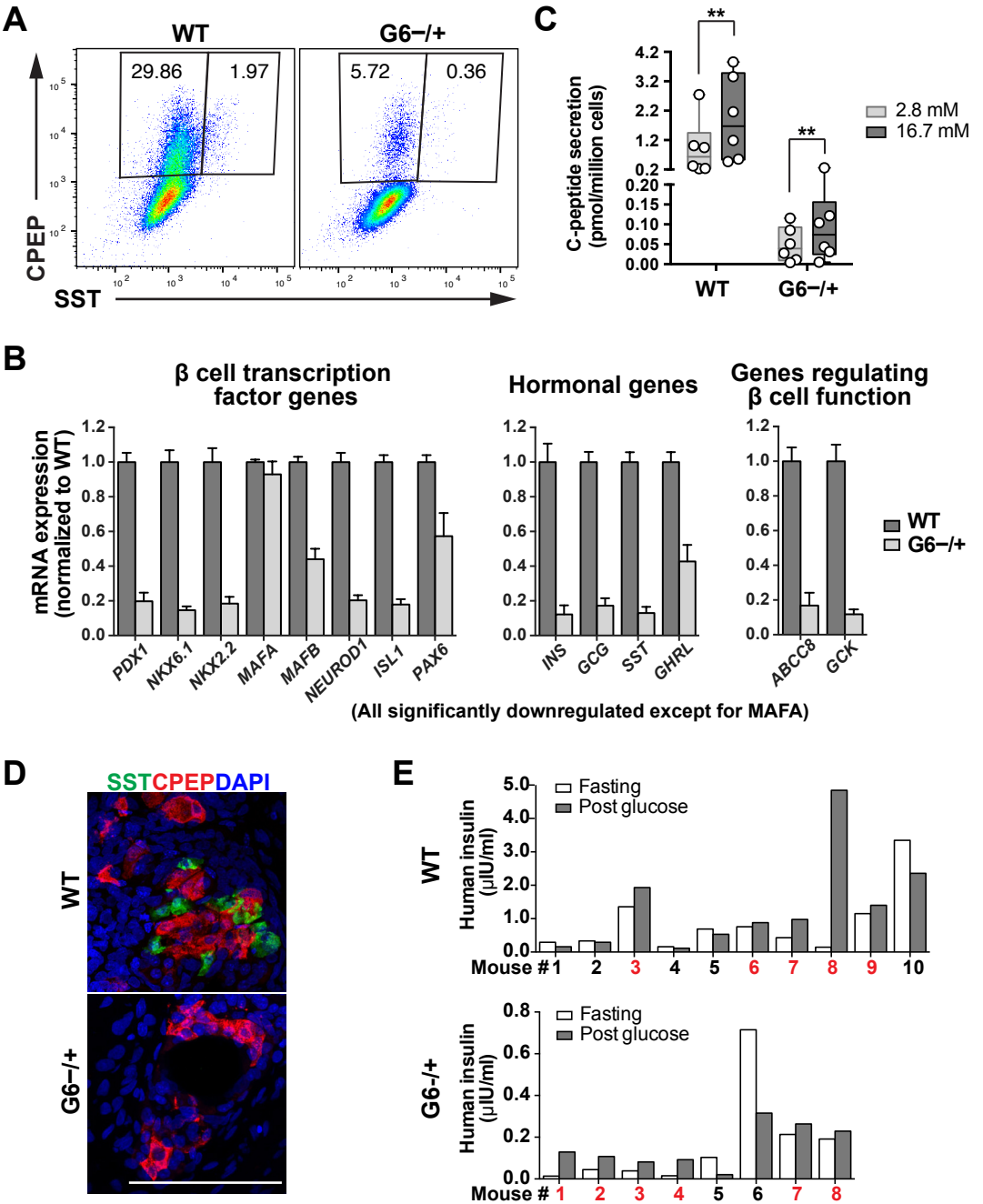


Figure S4. GATA6 haploinsufficiency in the formation of β-like cell. Related to Figure 5.

(A) Representative FACS plots for CPEP and SST (somatostatin) intracellular staining at β-like stage.

(B) RT-qPCR analysis of expression of β cell transcription factors, hormonal markers, and β cell functional markers at the β-like stage (n=4).

(C) Box and whisker plots of in vitro GSIS at β cell stage. Two-tailed ratio paired t test was used to calculate the significance of C-peptide secretion between 16.7 mM glucose and 2.8 mM glucose (n=6).

(D) Immunostaining of CPEP and SST in grafts collected from mouse kidney capsules at 4 months after transplantation.

(E) *In vivo* GSIS at one month after transplantation. Blood samples were collected after an overnight fasting and 30 min post glucose injection, respectively. Human insulin was measured for each mouse transplanted with either WT or *GATA6*$^{-/+}$ cells.

All data in this figure were generated from H1 lines using the 2nd-generation differentiation protocol.

Shi_Supplementary Figure 5

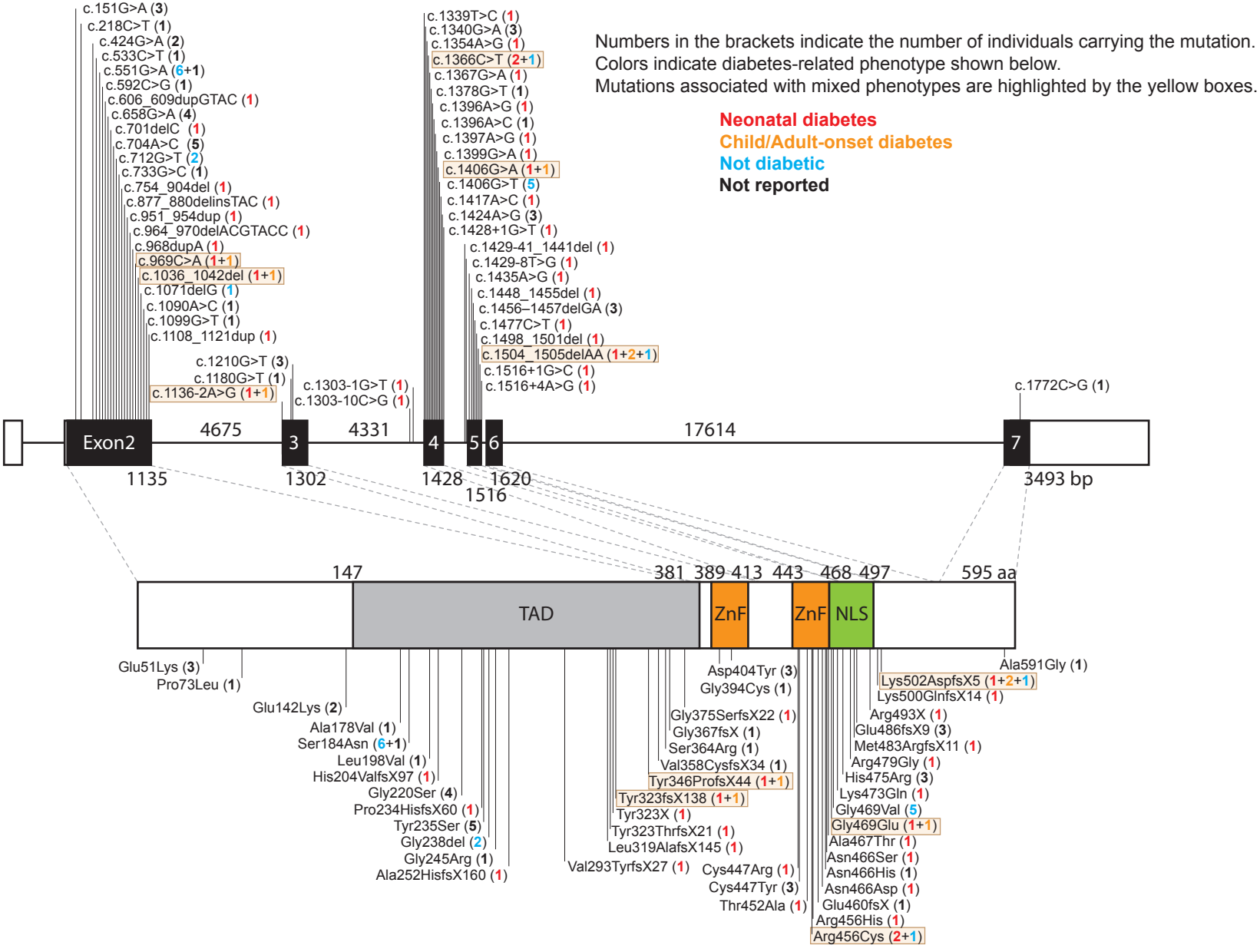


Figure S5. *GATA6* heterozygous mutations in humans. Related to Figure 6.

Both positions of gene and protein (if available) of the mutations are shown. Color-coded numbers in the brackets indicate number of cases with certain diabetes phenotypes. Red indicates neonatal diabetes; Orange indicates child- or adult-onset diabetes; Blue indicates not diabetic; and Black indicates pancreatic or diabetes-related phenotype was not reported. Mutations in the *GATA6* promoter region were excluded. Cases with mosaic mutation were also excluded. Yellow boxes indicate mixed phenotypes associated with the mutation.

Shi_Supplementary Figure 6

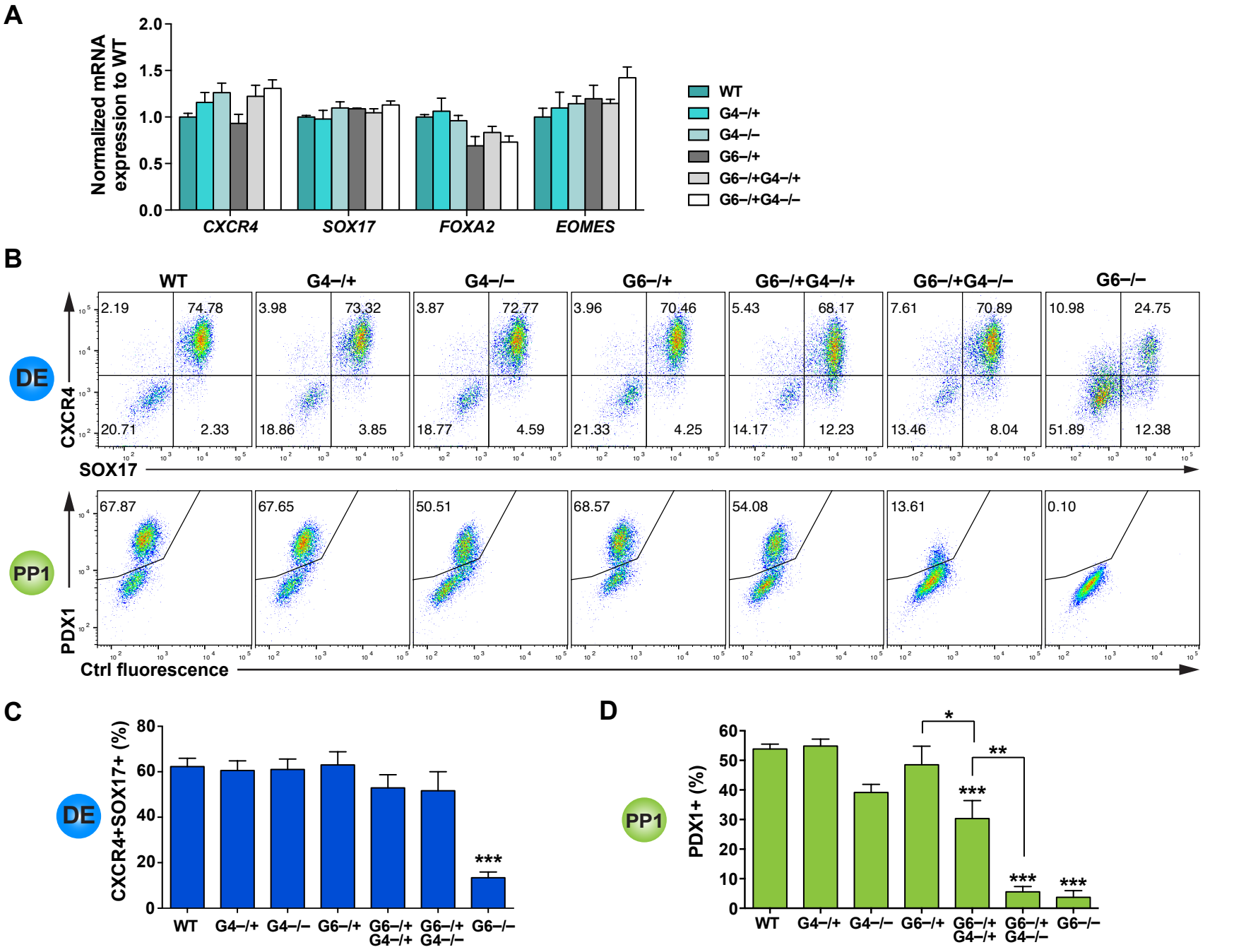


Figure S6. Genetic interaction of *GATA4* and *GATA6* in the specification of definitive endoderm cells and pancreatic progenitors. Related to Figure 6.

(A) RT-qPCR for DE marker mRNA expression in *GATA6/4* mutants (n=4) using the 2nd-generation protocol.

(B) Representative FACS dot plots of various GATA mutants at DE stage (d5) stained for CXCR4 and SOX17 and PP1 stage stained for PDX1 using the 1st-generation protocol.

(C) FACS quantification of DE differentiation efficiency for individual GATA mutant lines using the 1st-generation protocol (n=6-12).

(D) FACS quantification of PP1 differentiation efficiency for individual GATA mutant lines using the 1st-generation protocol (n=6-12).

All data in this figure were generated from HUES8 lines.

Table S1. Summary of CRISPR targeting sequences and oligonucleotides used for targeting experiments. Related to Figure 2.

CRISPR gRNA target sequences for targeting *GATA6* and *GATA4*

Gene	CRISPR	gRNA target sequence (5' of PAM)
GATA6	G6-Cr1	GGCGTTTCTGCGCCATAAGG
	G6-Cr2	TTATGGCGCAGAAACGCCG
GATA4	G4-Cr1	GGGGTGTAAGCGGCTCCGT
	G4-Cr2	AGGAGAAGCGCGGCGACAC

PCR and sequencing primers used for genotyping and sequencing

Gene	Primer	Sequence (5' to 3')
GATA6	Forward	ACATACTTGTTGATGACAGGGACA
	Reverse	CGTTTGCAATAGTTCAACTGG
	Sequencing	CGTTTGCAATAGTTCAACTGG
GATA4	Forward	TCGTTGTTGCCGTCGTTTTC
	Reverse	CGTCGGCCATGTAAGC
	Sequencing	GCCAGTCTACGTGCCCACACC

PCR and sequencing primers used for off-target analysis

Gene	Primer	Sequence (5' to 3')
NCOR1	Forward	AACACCAAGCTTCAGTCAGC
	Reverse	TGAGCTCTTGGTCCATGAGAC
	Sequencing	TGAGCTCTTGGTCCATGAGAC
SNX29	Forward	GCACAGACCGTAGAGTCGAG
	Reverse	GTGGTCCCAGGAATCCAAGG
	Sequencing	ATGACAGCAACTCCCCGAAG
AHNAK	Forward	CTCCCCCACCCTCCATTTTC
	Reverse	GTGAACTCCAGGCACCTGAT
	Sequencing	GACCTGAAAGGCCCCAAAGT
RBM20	Forward	AAGCTGGAACCGAGCCAAAT
	Reverse	GTCATCTCCGTTGTCCCTCC
	Sequencing	CTGGCATAGGGAGAGTGCTC
C20orf85	Forward	GTCTGGAGGTTTCCCCAAGG
	Reverse	GGACATGAGAGCGACAGGAG
	Sequencing	CCCAGACGTGAGCAAAGGAT

Sequences of ssDNA donor for HDR

CRISPR	Genotype designation	Sequence (5' to 3')
GATA6-Cr2	WT reference	GGATTGTCCTGTGCCAACTGTCACACCACAACTACCACCTTATGGCGCAGAAACG CCGAGGGTGAACCCGTGTGCAATGCTTGTGGACTCTACATGAAACTCCATGGGGT
	c.1366C>T donor	GGATTGTCCTGTGCCAACTGTCACACCACAACTACCACCTTATGG**T**GCAGAAACG CCGA**A**GGTGAACCCGTGTGCAATGCTTGTGGACTCTACATGAAACTCCATGGGGT

The underlined sequence indicates CRISPR target sequence; nucleotide base **T** in red color is the c.1366C>T mutation and orange **A** is a silent mutation in the PAM sequence.

Table S2. Summary of *GATA6/4* mutant lines investigated. Related to Figures 2-6.

HUES8 hESC clonal lines					
Gene	**Clone name**	**Genotype designation**	**CRISPR**	**Predicted protein**	
GATA6		**G6**		***GATA6* alleles**	
	wt1	WT	G6-Cr2	+/+	
	wt3	WT	G6-Cr2	+/+	
	m32	−/+	G6-Cr2	E460fs/+	
	m33	−/+	G6-Cr2	N458fs/+	
	m23	−/−	G6-Cr2	E460fs/E460fs	
	m36	−/−	G6-Cr2	N458fs/N458fs	
	m37	−/−	G6-Cr2	N458fs/N458fs	
	m38	R456C/+	G6-Cr2	R456C/+	
	m40	R456C/+	G6-Cr2	R456C/+	
	m34	R456C/R456C	G6-Cr2	R456C/R456C	
	m35	R456C/R456C	G6-Cr2	R456C/R456C	
	wt4	WT	G6-Cr1	+/+	
	wt5	WT	G6-Cr1	+/+	
	m42	−/+	G6-Cr1	L454fs/+	
	m44	−/−	G6-Cr1	L454fs/+	
	m45	−/−	G6-Cr1	T452fs/T452fs	
	m46	−/−	G6-Cr1	T451fs/T451fs	
	m47	−/−	G6-Cr1	L454fs/L454fs	
GATA4		**G4**		***GATA4* alleles**	
	wt8	WT	G4-Cr1	+/+	
	wt9	WT	G4-Cr1	+/+	
	wt10	WT	G4-Cr1	+/+	
	m96	−/+	G4-Cr1	D95fs/+	
	m97	−/+	G4-Cr1	A97fs/+	
	m98	−/+	G4-Cr1	A97fs/+	
	m99	−/+	G4-Cr1	D95fs/+	
	m104	−/−	G4-Cr1	A97fs/A97fs	
	m105	−/−	G4-Cr1	P87fs/P87fs	
	m5	−/−	G4-Cr2	V104fs/V104fs	
	m6	−/−	G4-Cr2	G96fs/G96fs	
GATA6*; *GATA4		**G6;G4**		***GATA6* alleles**	***GATA4* alleles**
	m106	−/+;−/+	G6-Cr2; G4-Cr1	N458fs/+	D95fs/+
	m107	−/+;−/+	G6-Cr2; G4-Cr1	N458fs/+	A94fs/+
	m108	−/+;−/+	G6-Cr2; G4-Cr1	N458fs/+	Q91fs/+
	m109	−/+;−/+	G6-Cr2; G4-Cr1	N458fs/+	G96fs/+
	m110	−/+;−/−	G6-Cr2; G4-Cr1	N458fs/+	D95fs/G96fs
	m111	−/+;−/−	G6-Cr2; G4-Cr1	N458fs/+	G93fs/Q91fs

H1 hESC clonal lines				
Gene	**Clone name**	**Genotype designation**	**CRISPR**	**Predicted protein**
GATA6	w2	WT	G6-Cr1	+/+
	w4	WT	G6-Cr2	+/+
	m2	−/+	G6-Cr1	L454fs/+
	m5	−/+	G6-Cr2	R457fs/+
	m11	−/−	G6-Cr1	L454fs/ L454fs
	m14	−/−	G6-Cr2	N458fs/N458fs
	pw1	WT	G6-Cr2	+/+
	pw2	WT	G6-Cr2	+/+
	pm1	R456C/+	G6-Cr2	R456C/+
	pm2	R456C/+	G6-Cr2	R456C/+

Note: Mutant alleles are described according to the predicted changes at the protein level following the Human Genome Variation Society (HGVS) guidelines (http://www.hgvs.org/mutnomen). In brief, the first amino acid affected and its position is described. Following that, “fs” indicates a frame-shift change; and an amino acid code indicates a substitution. Some frameshift mutant alleles share the same description, but the length of the amino acids from the frame-shift to the new stop codon may be different.

Table S3. hPSC differentiation into glucose-responsive β-like cells. Related to Figures 4-6.

The 2nd generation differentiation protocol

Stage	Day	Media	Supplement							
S0 (2d)	d-2	Seeded 75,000 hPSCs/cm^2 on vitronectin-coated plates in E8 media with 5 uM Y-27632.								
	d-1	Changed media.								
S1 (3d)	d0	Began differentiation when the cells reached ~75% confluency (~48 hr after seeding). Rinsed cells with DPBS (w/o Ca^{2+}, Mg^{2+}) for 5 min and added differentiation media (see below).								
	d0	S1-2	Activin A 100 ng/ml	CHIR-99021 5 uM						
	d1	S1-2	Activin A 100 ng/ml	CHIR-99021 0.3 uM						
	d2	S1-2	Activin A 100 ng/ml							
	d3	>80% DE cells expressing endoderm markers including CXCR4, SOX17 and FOXA2.								
S2 (2d)	d3-4	Briefly rinsed cells with 1X DPBS (w/o Ca^{2+}, Mg^{2+}).								
		S1-2	Vitamin C 0.25 mM	FGF7 50 ng/ml	IWP-2 1.25 uM					
S3 (2d)	d5-6	S3-4	Vitamin C 0.25 mM	FGF7 50 ng/ml	SANT-1 0.25 uM	RA 1 uM	LDN 100 nM	ITS-X 1:200	TPB 200 nM	
S4 (3d)	d7-9	S3-4	Vitamin C 0.25 mM	FGF7 2 ng/ml	SANT-1 0.25 uM	RA 0.1 uM	LDN 200 nM	ITS-X 1:200	TPB 100 nM	
	d10	Cells were examined for expression of pancreatic progenitor markers including PDX1 and NKX6.1.								
S5 (3d)	d10	S4 cells were treated with 10 uM Y-27632 for 4 hr, collected using TrypLE and washed with basal MCDB 131 media. Cell pellets were re-suspended in S5-7 media with S5 chemical supplements at ~50 million/ml and spotted onto transwell insert filters (6-well plate: Corning 3414; 10cm plate: Corning 3420) for culture in air-liquid interface at ~7.5 ul/spot and ~10 spots per 6-well or ~100 spots per 10cm plate. S5-7 media with supplements were added to the bottom of each insert: ~1.5 ml per 6-well or ~8 ml per 10cm plate.								
	d10-12	S5-7	T3 1 uM	Alk5i II 10 uM	SANT-1 0.25 uM	RA 0.05 uM	LDN 100 nM	ITS-X 1:200	ZnSO4 10 uM	Heparin 10 ug/ml
S6 (7d)	d13-19	S5-7	T3 1 uM	Alk5i II 10 uM	GSiXX 100 nM		LDN 100 nM	ITS-X 1:200	ZnSO4 10 uM	Heparin 10 ug/ml
S7 (2w)	d20-33	S5-7	T3 1 uM	Alk5i II 10 uM	N-Cys 1 mM	Trolox 10 uM	R428 2 uM	ITS-X 1:200	ZnSO4 10 uM	Heparin 10 ug/ml

Change media every day as indicated.

Media

Media Stage	Media component (add corresponding supplements listed above)				
S1-2	MCDB 131	GlutaMAX 1X	BSA 0.5%	NaHCO3 1.5 g/L	Glucose 10 mM
S3-4	MCDB 131	GlutaMAX 1X	BSA 2%	NaHCO3 2.5 g/L	Glucose 10 mM
S5-7	MCDB 131	GlutaMAX 1X	BSA 2%	NaHCO3 1.5 g/L	Glucose 20 mM

Chemicals and media components

Components	Vendor	Cat. No.
MCDB 131	GIBCO	10372-019
GlutaMAX	GIBCO	35050-061
NaHCO3	Fisher Scientific	144-55-8
D-Glucose	Sigma-Aldrich	G8769
BSA (bovine serum albumin)	LAMPIRE Biological Laboratories	7500855
Activin A	PeproTech	120-14E
CHIR-99021, GSK-3 inhibitor	Stemgent	04-0004
L-Ascorbic acid (vitamin C)	Sigma-Aldrich	A4544
FGF7 (KGF)	R&D	251-KG
SANT-1, Hedgehog inhibitor	Sigma	S4572
RA (retinoic acid)	Sigma	R2625
LDN, BMP inhibitor	Stemgent	04-0019
ITS-X (insulin-transferrin-selenium-ethanolamine)	GIBCO	51500-056
TPB, Protein kinase C (PKC) activator	EMD Millipore	565740
T3 (3,3',5-Triiodo-L-thyronine)	Sigma-Aldrich	T6397
ALK5i II (ALK5 inhibitor II)	Enzo Life Sciences	ALX-270-445
ZnSO4	Sigma-Aldrich	Z0251
Heparin	Sigma-Aldrich	H3149
GSiXX (gamma secretase inhibitor XX)	EMD Millipore	565789
N-Cys (N-acetyl cysteine)	Sigma-Aldrich	A9165
Trolox, vitamin E analogue	EMD Millipore	648471
R428, AXL receptor tyrosine kinase inhibitor	Selleck Chemicals	S2841
IWP-2, Wnt antagonist	Tocris Bioscience	3533

Table S4. *GATA6* heterozygous mutations reported in humans. Related to Figure 6.

Available as a separate spreadsheet.

Table S5. Primers for qRT-PCR. Related to Figures 1, 4.

Gene	Forward (5' to 3')	Reverse (5' to 3')
ACTB	CATGTACGTTGCTATCCAGGC	CTCCTTAATGTCACGCACGAT
EOMES	CGGCCTCTGTGGCTCAAA	AAGGAAACATGCGCCTGC
GATA6	TCTCCATGTGCATTGGGGAC	AAGGAAATCGCCCTGTTCGT
GATA4	AAAGAGGGGATCCAAACCAG	TTGCTGGAGTTGCTGGAAG
FOXA2	GGGAGCGGTGAAGATGGA	TCATGTTGCTCACGGAGGAGTA
SOX17	GGCGCAGCAGAATCCAGA	CCACGACTTGCCCAGCAT
CXCR4	CACCGCATCTGGAGAACCA	GCCCATTTCCTCGGTGTAGTT
PDX1	TGGAGCTGGCTGTCATGTTGA	CGCTTCTTGTCCTCCTCCTTTT
SOX9	AGCTCTGGAGACTTCTGAACGAGAG	CGTTCTTCACCGACTTCCTCCGC
HNF6	CGCTCCGCTTAGCAGCAT	GTGTTGCCTCTATCCTTCCCAT
HNF1A	GGTCCTACGTTCACCAACACA	CTCTGGGTCACATGGCTCT
HNF1B	TCACAGATACCAGCAGCATCAGT	GGGCATCACCAGGCTTGTA
HNF4A	CATGGCCAAGATTGACAACCT	TTCCCATATGTTCCTGCATCAG
MNX1	TCCACCGCGGGCATGATCCT	GCGCTTGGGCCGCGACAGGTA
PROX1	AAAGCAAAGCTCATGTTTTTTTATACC	GTAAAACTCACGGAAATTGCTAAACC
PTF1A	CCAGAAGGTCATCATCTGCC	AGAGAGTGTCCTGCTAGGGG
NKX6.1	CTGGCCTGTACCCCTCATCA	CTTCCCGTCTTTGTCCAACAA
NGN3	CTATTCTTTTGCGCCGGTAG	ACTTCGTCTTCCGAGGCTCT
NEUROD1	GGATGACGATCAAAAGCCCAA	GCGTCTTAGAATAGCAAGGCA
NKX2.2	ATGTAAACGTTCTGACAACT	TTCCATATTTGAGAAATGTTTGC
MAFA	TTCAGCAAGGAGGAGGTCAT	CGCCAGCTTCTCGTATTTCT
MAFB	TCAAGTTCGACGTGAAGAAGG	GTTCATCTGCTGGTAGTTGCT
ISL1	TTCCCTATGTGTTGGTTGCGGC	CGCATTTGATCCCGTACAACCTGA
PAX6	AGACATCCGAGATTTCAGAGCCCCA	GGCCGCCCGTTGACAAAGAC
INS	CCCTGCAGAAGCGTGGCATT	CCATCTCTCTCGGTGCAGGA
GCG	AAGCATTTACTTTGTGGCTGGATT	TGATCTGGATTTCTCCTCTGTGTCT
SST	GATGCTGTCCTGCCGCCTCC	TGCCATAGCCGGGTTTGA
GHRL	TGAACACCAGAGAGTCCAGCA	GCTTGGCTGGTGGCTTCTT
ABCC8	CTGCTAAACCGGATCATCCTAGCC	CGAGGAACACAGGTGTGACATAGG
GCK	TGCAGATGCTGGACGACAG	GAACTCTGCCAGGATCTGCTCTA