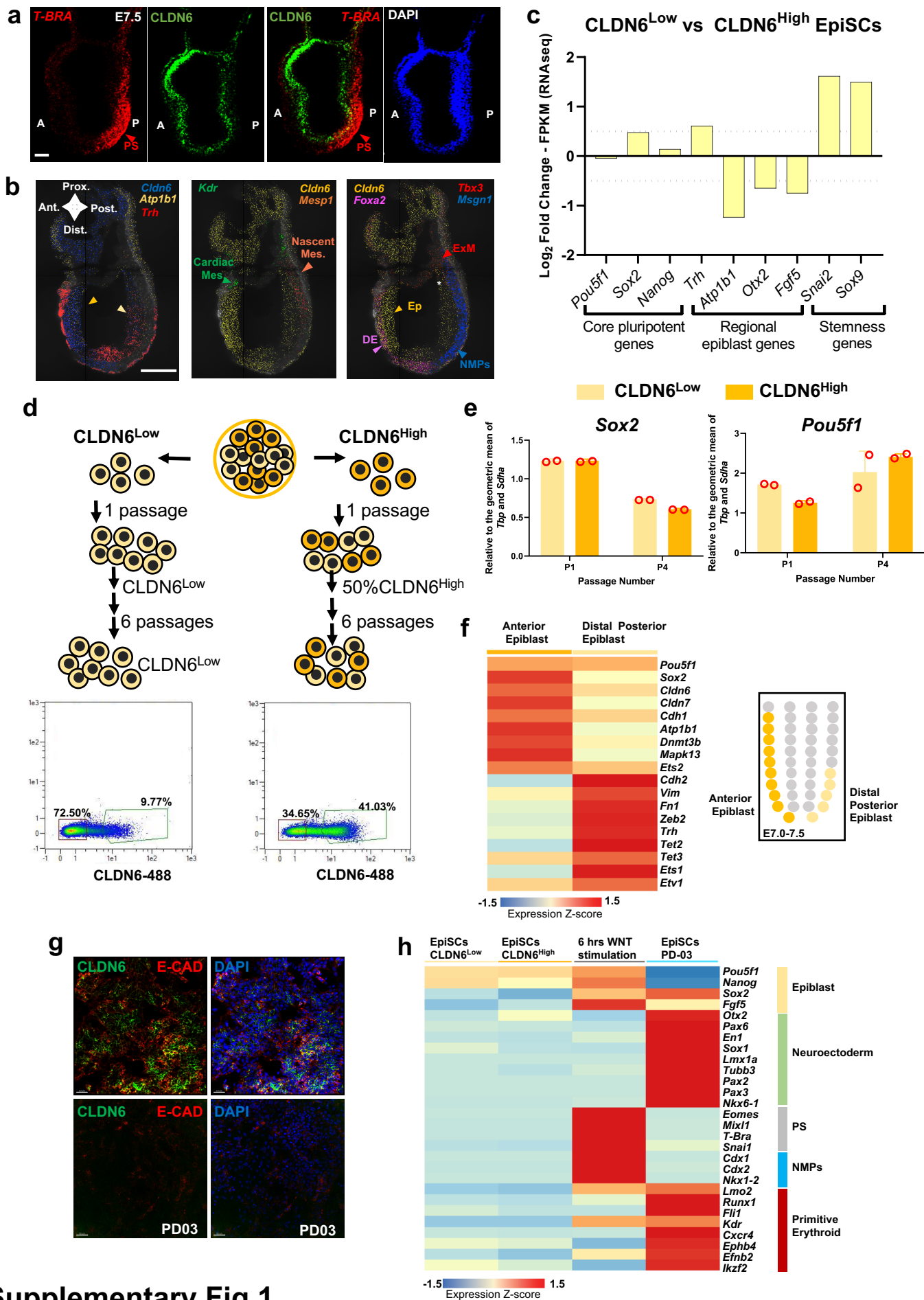


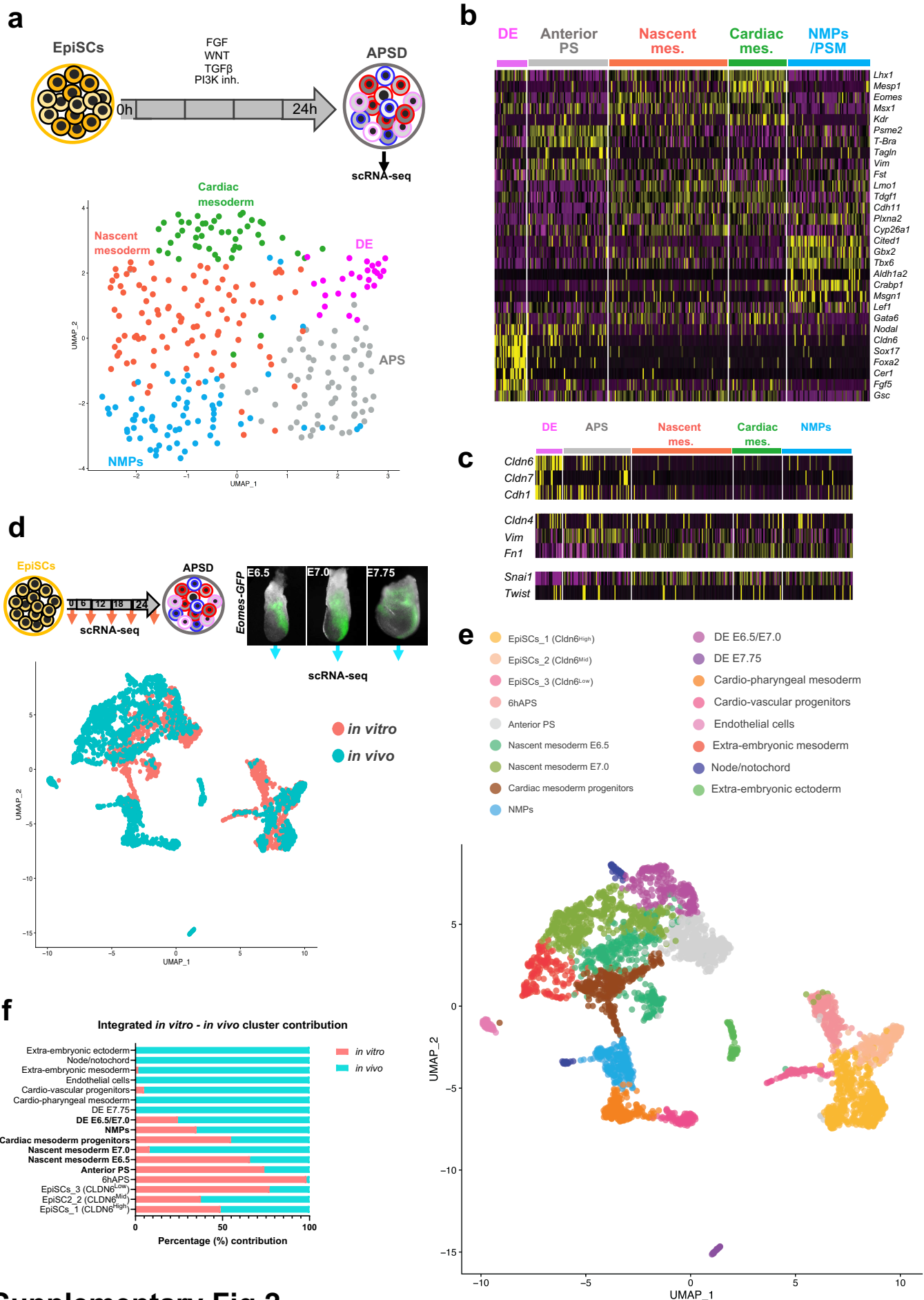


Supplementary Fig.1

Supplementary Fig. 1. CLDN6^{Low} and CLDN6^{High} EpiSCs are two distinct populations of pluripotent cells that can be propagated *in vitro*.

a) Immunofluorescence of E7.5 embryo showing a CLDN6 anterior-posterior gradient. CLDN6 (green), T-BRA (red), DAPI (blue). The images are representative of least three IFs. Scale bar: 30 μ m. **b)** Spatial transcriptomics analysis of E7.5 embryos showing anterior epiblast expression of *Atp1b1* and higher *Cldn6*, with *Trh* and lower *Cldn6* expression in distal posterior epiblast cells. *Cldn6* is absent in nascent mesoderm progenitors labelled by *Mesp1* (orange arrowhead) and cardiac mesoderm by *Kdr* (green arrowhead). *Cldn6* colocalizes with DE markers *Foxa2* (pink arrowhead) but not NMPs marked by *Msgn1* (blue arrowhead). *Cldn6* is expressed in *Tbx3^{pos}* PPS cells differentiation towards ExM (red arrowhead), but not in ExM cells. Ep: epiblast, DE: definitive endoderm, NMPs: neuromesodermal progenitors, ExM: extraembryonic mesoderm. Representative image taken from n = 4 (E7.5 embryos). Scale bar: 100 μ m. **c)** Histogram representing the differential expression of core pluripotent genes (*Pou5f1*, *Nanog*, *Sox2*), regional epiblast genes (*Trh*, *Atp1b1*, *Fgf5*) and stemness genes (*Snai2*, *Sox9*) in CLDN6^{Low} versus CLDN6^{High} EpiSCs ($P < 0.05$, biological replicates (n) = 2, Wald Test with Benjamini–Hochberg adjusted P-values on DESeq2). Based on RNAseq (FPKM - fragments per kilobase of exon per million mapped fragments). Dotted lines from the X-axis represent 1.5x fold change over gene expression. **d)** Schematics illustrating the self-renewal and expansion properties of CLDN6^{Low} and CLDN6^{High} EpiSCs. While CLDN6^{Low} cells maintain their mesenchymal characteristics, CLDN6^{High} cells revert to the unsorted salt-and-pepper state after one passage. **e)** Relative mRNA expression of the pluripotent markers *Pou5f1* and *Sox2* measured by RT-qPCR along sequential passages of CLDN6^{Low} and CLDN6^{High} EpiSCs, confirming the maintenance of the pluripotency state. Data are mean $\pm$ s.e.m. (n = 2 biological replicates). **f)** Heatmap showing specific gene expression in anterior epiblast (dark yellow) and distal posterior epiblast (light yellow). Graphical cornplot depicts anterior epiblast (dark yellow) and distal-posterior epiblast (light yellow) positional identity. FPKM values from positions highlighted in the cornplot were merged for anterior epiblast (dark yellow) and distal-posterior epiblast (light yellow), and represented in the heatmap based on published E7.0-E7.5 epiblast GEO-sequencing data^{28,29}. **g)** Immunofluorescence of control and PD-03 stimulated EpiSCs showing loss

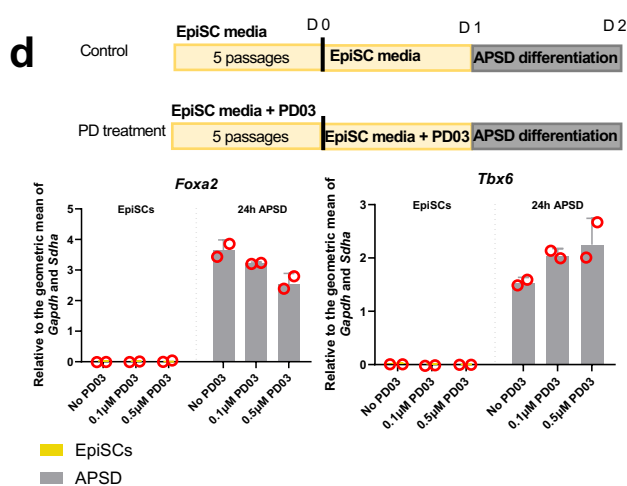
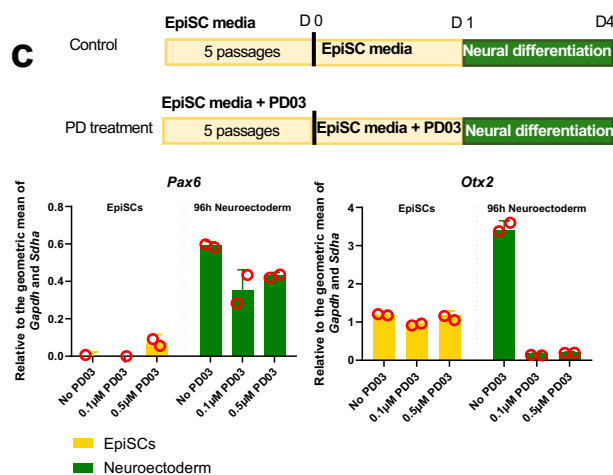
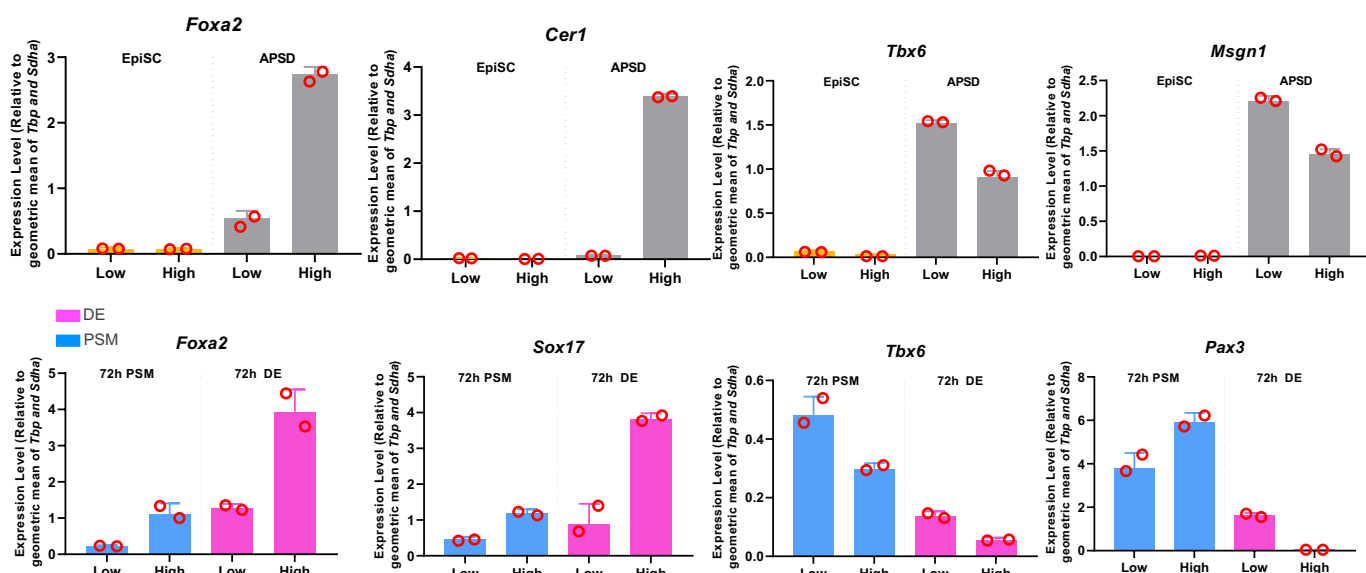
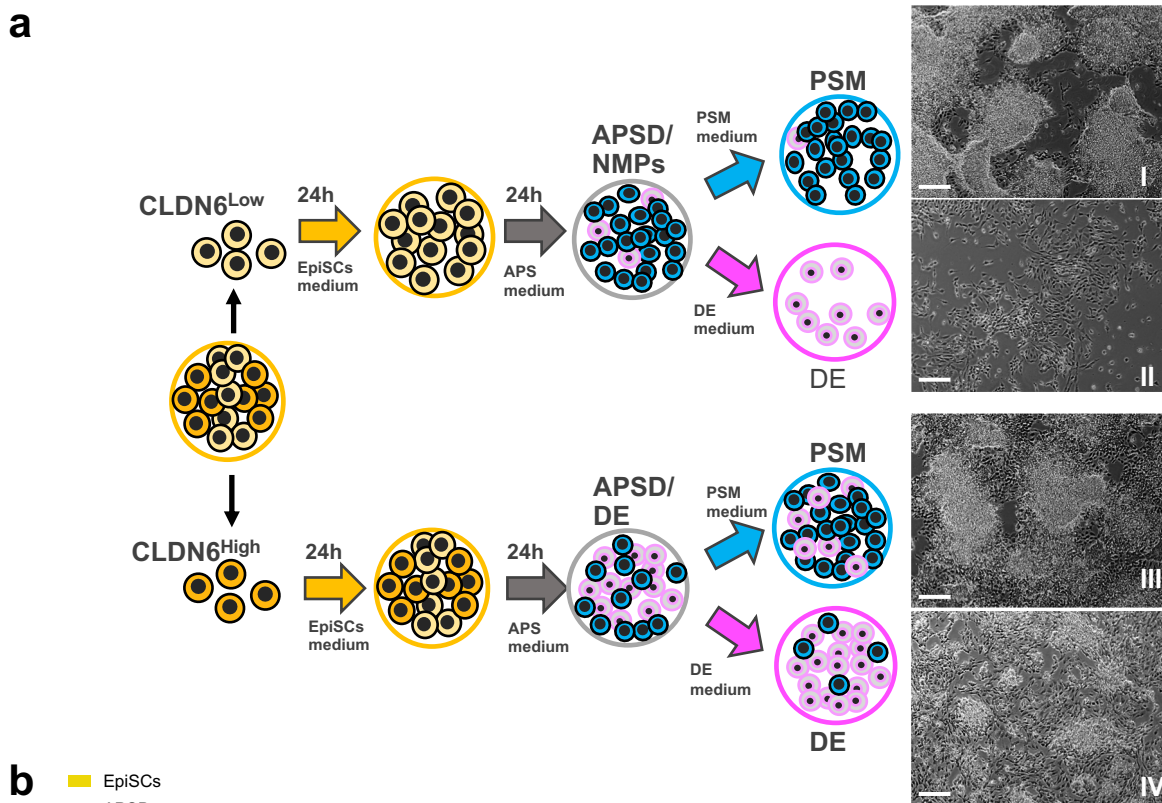
of epithelial signature markers E-CADHERIN (red) and CLDN6 (green) upon ERK inhibition with PD03. Each PD03 treatment was performed independently more than three times, and multiple immunofluorescence images were acquired for each experiment. Scale bars: 50 μ m. **h)** Heatmap showing differential expression analysis of CLDN6^{Low}, CLDN6^{High}, 6h WNT-induced and PD03-treated EpiSCs, showing increased primitive streak (PS, grey) and NMP (blue) markers upon 6h WNT-induction; and neuroectodermal (green) and hemato-endothelial (red) differentiation markers in PD03-treated EpiSCs. RNAseq data: $P \leq 0.05$, fold-change ≥ 1.5 , $n = 2$ biological replicates. Significance was assessed by DESeq2 on the basis of two-sided Wald test with Benjamini–Hochberg adjusted P-values.



Supplementary Fig.2

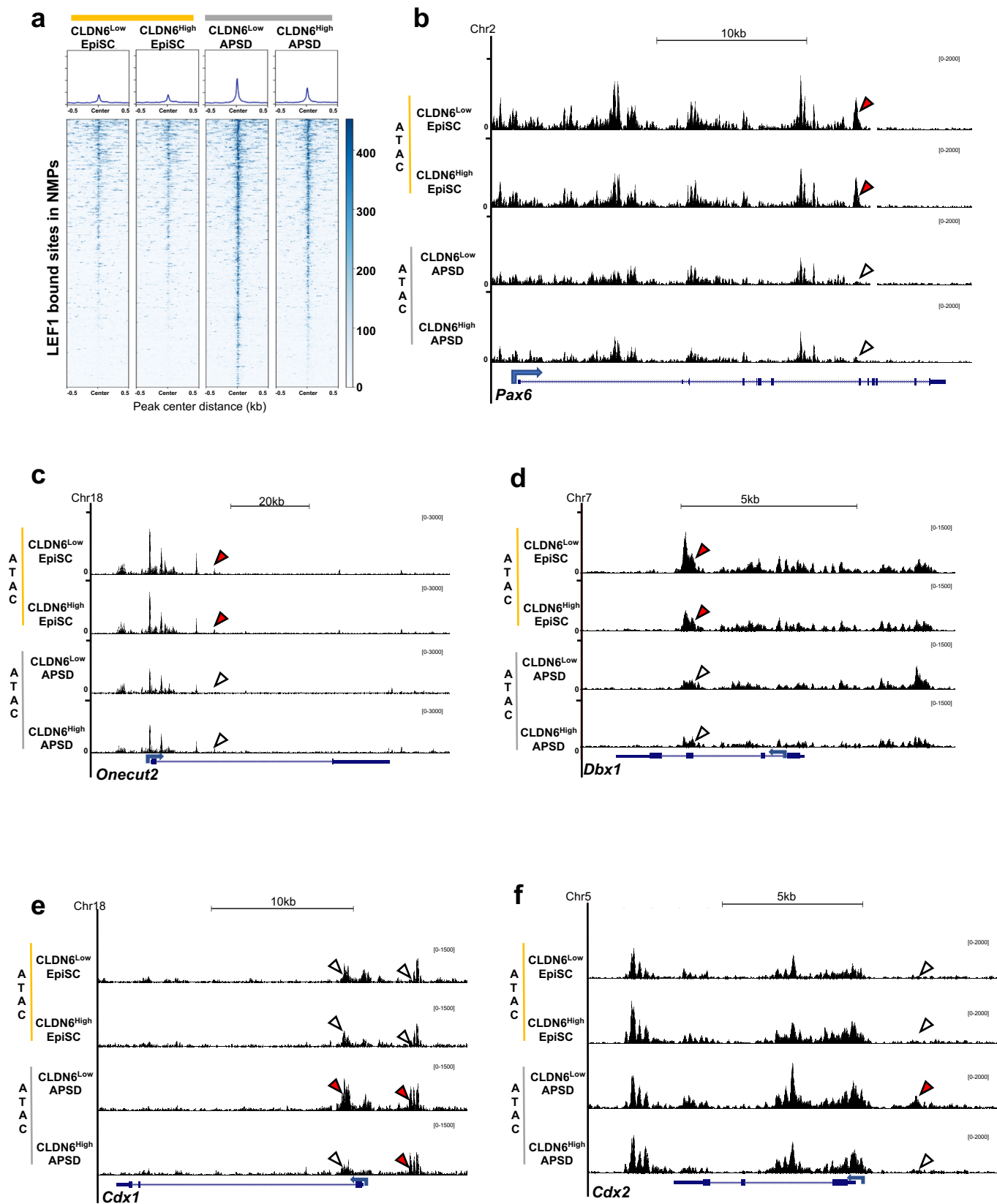
Supplementary Figure 2. *In vitro* differentiation of EpiSCs towards APSD gives rise to neuromesodermal, cardiac mesoderm, and definitive endoderm progenitors. a)

Upper panel. Cartoon summarizing the protocol used to differentiate EpiSCs towards anterior primitive streak derivatives (APSD). Lower panel. Unbiased gene clustering via highly variable gene expression identified five clusters. Cell identity is established by using lineage-annotated markers^{30,51}. **b)** Heatmap showing expression levels of selected markers across differentiation. DE progenitors (pink) exhibit higher *Sox17*, *Gata6*, *Cldn6*, and *Foxa2* expression levels. Anterior primitive streak cells (APS) (grey) express *T-Bra*, and EMT markers like *Vim*, *Tagln*, and *Fst*. Cardiac mesoderm progenitors (green) are enriched in *Lhx1*, *Mesp1*, and *Eomes*. Nascent mesoderm (orange) expresses mixed mesoderm markers like *Lmo1* and *Tdgf1*, and NMPs (blue) show enhanced levels of *Cited1*, *Gbx2*, *Msgn1*, and *Tbx6*. **c)** Heatmap showing higher expression of epithelial marker (*Cadherins* and *Claudins*) in DE and mesenchymal markers (*Fn1* and *Snai1*) in mesodermal progenitors. **d)** Mapping *in vitro* generated progenitor populations to the mouse embryos. Schematic representation of the strategy to integrate *in vitro* differentiated cells and EOMES^{pos} cells isolated from mouse embryos at E6.5, E7.0, and E7.75. EpiSCs were spiked as a control on all *in vitro* and *in vivo* scRNAseq plates. No epiblast cells were detected amongst EOMES^{pos} cells. Lower panel: UMAP plots showing the distribution of the *in vitro* generated cells (light pink) and the EOMES^{pos} cells (turquoise). **e)** Seurat integration of the two scRNAseq datasets indicates strong similarity between *in vitro* population at 24 h and cells isolated from E6.5/E7.0 embryos: anterior PS (grey), nascent mesoderm (green), DE (pink). The NMPs/PSM cells are similar to the E7.75 anterior presomitic mesoderm cluster (light blue). **f)** Bar plot comparing the percentage of *in vitro* (light pink) and *in vivo* (turquoise) cell contribution within the different clusters, showing that our *in vitro* APSD protocol recapitulates *in vivo* anterior mesoderm and DE progenitors.



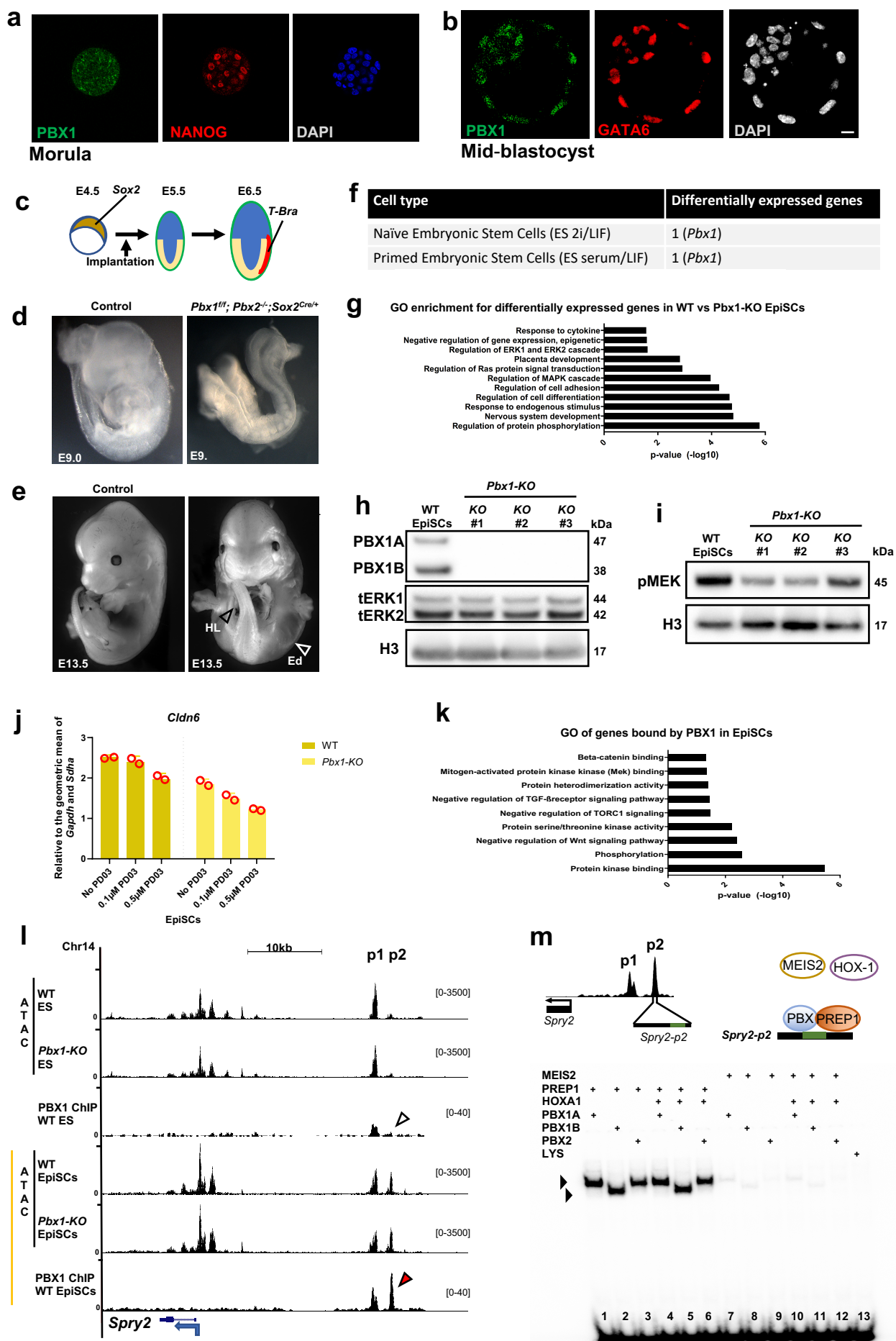
Supplementary Fig. 3

Supplementary Fig. 3. CLDN6^{Low} EpiSCs are multipotent, while CLDN6^{High} EpiSCs retain pluripotent capacities upon self-renewal. **a)** Illustration summarizing the strategy to assess APSD differentiation potency of CLDN6^{Low} and CLDN6^{High} EpiSCs, reseeded and maintained for 24 h in EpiSC medium prior to differentiation. CLDN6^{Low} EpiSCs have restricted potency and preferentially differentiate towards NMPs (panel I) instead of DE (panel II). CLDN6^{High} EpiSCs revert towards mixed CLDN6^{Low} and CLDN6^{High} populations and maintain the same differentiation potential as unsorted EpiSCs (panels III and IV). The images are representative of at least three independent differentiations. Scale bars: 100 μ m. **b)** Relative mRNA expression of DE and NMPs/PSM genes along 24 h EpiSC (yellow), 24 h APSD (grey) that contains NMP and DE progenitors, as well as 72 h NMPs/PSM (blue), and 72h DE (pink) differentiation, showing preferential DE differentiation (*Foxa2*, *Cer1*, *Sox17*) for CLDN6^{High} and NMP bias (*Msgn1*, *Tbx6*, *Pax3*) for CLDN6^{Low} EpiSCs, although CLDN6^{High} EpiSCs possess NMP differentiation potential. **c)** ERK dosage regulates neural differentiation potential of EpiSCs. (Upper panel) A diagram illustrates the protocol used to evaluate the priming effects of the ERK inhibitor PD03 dosage on EpiSCs (yellow), with the differentiation outcome assessed towards neuroectoderm (green). The relative mRNA expression analysis shows that EpiSCs treated with increasing concentrations of PD03, exhibited reduced levels of anterior neural marker *Pax6* and *Otx2* in neuroectoderm compared to untreated neural EpiSCs. **d)** ERK inhibitor PD03 differentially modulates the anterior primitive streak differentiation (APSD), including definitive endoderm (DE) and neuromesodermal progenitors (NMP). (Upper panel) A diagram illustrates the protocol used to assess the effects of PD03 treatment on EpiSCs (yellow) and their differentiation into APSD (grey). Relative mRNA expression analysis indicates that EpiSCs treated with increasing concentrations of PD03 show reduced expression of the DE marker *Foxa2* and increased expression of the NMP marker *Tbx6*, compared to untreated EpiSCs. For RT-qPCR in **b)** **c)** and **d)**, data are mean $\pm$ s.e.m. (n = 2 biological replicates).



Supplementary Fig. 4

Supplementary Fig. 4. NMP lineage decisions rely on a permissive chromatin environment regulated by WNT signalling upon differentiation. **a)** Average density plots (top) and heatmaps (bottom) of LEF1 ChIP-seq signals of CLDN6^{Low} and CLDN6^{High} EpiSCs and APSD reveals LEF1 recruitment only after WNT stimulation (in APSD)³³. **b-d)** ATACseq visualization in CLDN6^{Low} and CLDN6^{High} EpiSCs and APSD. The neuroectoderm/anterior neural genes *Pax6* (b), *Onecut2* (c) and *Dbx1* (d) shows similar accessibility signatures in CLDN6^{Low} and CLDN6^{High} EpiSCs and reduced accessibility upon APSD differentiation. Reduced chromatin accessibility (white arrowheads), increased accessibility (red arrowheads). **e-f)** The posterior neural genes *Cdx1* (e) and *Cdx2* (f) displays specific openings of loci in CLDN6^{Low} APSD upon WNT-mediated APSD differentiation. The threshold of the vertical viewing range of data based on RPKM values is noted. Accessible CREs are shown with red arrowheads, while inaccessible CREs marked with white arrowheads.

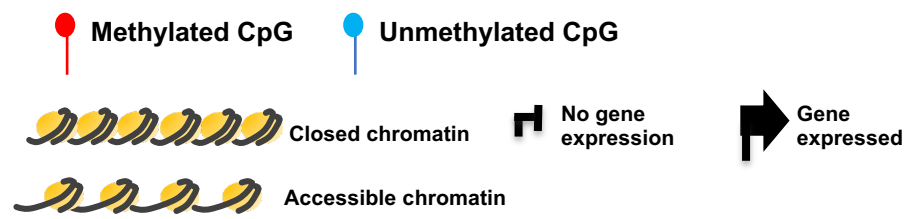
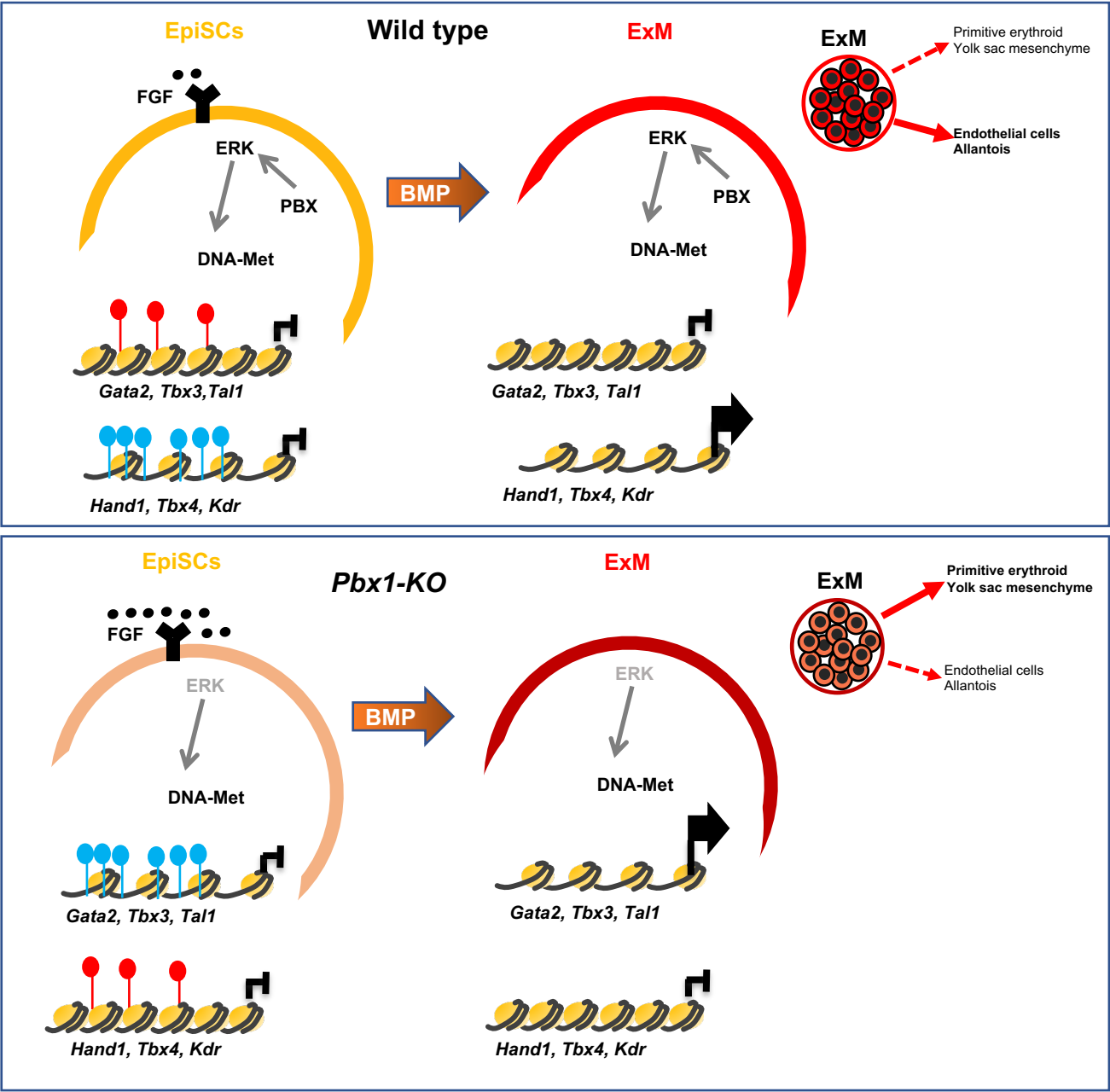


Supplementary Fig. 5

Supplementary Fig 5. Epiblast-specific ERK feedback regulation by a PBX1/PREP1 complex.

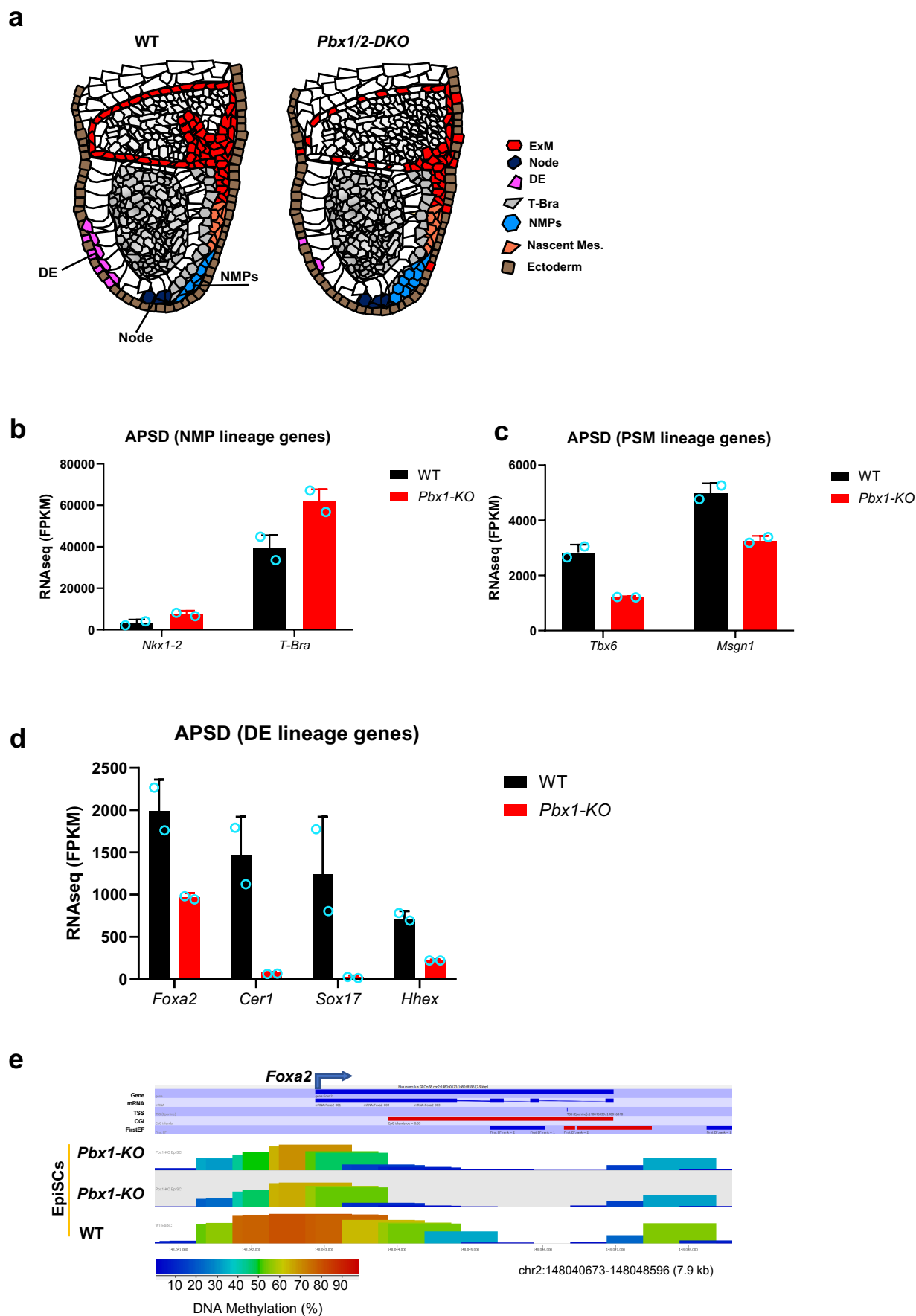
a) IF of E3.5 blastocyst probed with PBX1 (green) and NANOG (red) antibodies and nuclear DAPI counterstaining (blue), showing the absence of PBX1. **b)** IF of E4.5 blastocyst showing expression of PBX1 (green), co-stained with GATA6 (red) and DAPI (grey). The images are representative of at least three independent embryo IFs. Scale bars, 15 μ m. **c)** Schematic illustration of *Pbx1* inactivation at different developmental times. *Pbx1* is inactivated in the epiblast via *Sox2*^{Cre/+} (*Pbx1/2-DKO*) and in the PS using *T-Bra*^{Cre/+} alleles. **d)** Gross morphology of E9.0 *Pbx1/2-DKO* embryos show severe abnormal embryonic development. **e)** Deletion of *Pbx1* in the PS with *T-Bra*^{Cre/+} allele in *Pbx2*-deficient background (*Pbx1*^{ff};*Pbx2*^{-/-};*T-Bra*^{Cre/+}) exhibits a milder phenotype that includes edema (Ed) and abnormal limb formation (HL). **f)** No differentially expressed genes are detected comparing RNAseq data of WT and *Pbx1*-KO ES cells cultured in 2i/LIF or serum/LIF conditions. **g)** GO term enrichment analysis of differentially expressed genes in WT versus *Pbx1*-KO EpiSCs displays significant overrepresentation of terms associated with MAPK/ERK cascade (Binomial test with Bonferroni correction). **h-i)** Western blot analyses showing **h)** absence of PBX1 in *Pbx1*-KO and unchanged levels of total ERK1/2 proteins, and **i)** reduced levels of pMEK in three *Pbx1*-KO EpiSC cell lines compared with WT. **j)** Relative mRNA expression analysis of WT EpiSCs (dark yellow) and *Pbx1*-KO EpiSCs (light yellow) revealed that both cell types exhibit similar downregulating trends in *Cldn6* expression when treated with increasing concentrations of PD03. CLDN6 levels in untreated *Pbx1*-KO EpiSCs appear to be similar to 0.5 μ M PD-treated WT EpiSCs. Data are mean $\pm$ s.e.m. (n = 2 biological replicates). **k)** GO-term enrichment analysis of genes bound by PBX1 in EpiSCs displays a significant overrepresentation of terms associated with FGF/ERK activity (Binomial test with Bonferroni correction). **l)** UCSC visualization track of WT and *Pbx1*-KO ES (2i/LIF) and EpiSCs showing chromatin accessibility on *Spry2* putative CREs bound by PBX1 (ChIP-seq). p1 indicated a peak accessible in ES and EpiSCs bound by PBX1, p2 is a peak specific to the EpiSC state and bound by PBX1. Red arrowhead marks epiblast-specific PBX1 binding on p2. **m)** Top: schematics of *Spry2* locus, the accessible p1 and p2 regions in EpiSCs, and the formation of the PBX1/PREP1 complex on the *Spry2*-p2 oligonucleotide. Bottom: EMSA with *in vitro* translated TALE and HOX proteins and

Spry2-p2 oligonucleotide displaying the formation of dimeric PREP1-PBX complexes (lines 1-6, black arrowheads). The composition of each binding reaction is indicated on the top. LYS indicates control.



Supplementary Fig. 6

Supplementary Figure 6. DNA methylation and chromatin states predetermine primed state of proximal posterior epiblast cell populations towards diverse ExM tissues. In WT EpiSCs, PBX1 regulates pERK levels, which multiple studies have shown regulate DNA methylation status of primed pluripotent cells^{21,40}. These WT EpiSCs display a hypermethylated methylome and closed chromatin status over yolk sac and primitive erythroid genes (*Gata2*, *Tbx3*, *Tal1*), but a hypomethylated methylome and open chromatin nature over allantois and endothelial genes (*Hand1*, *Tbx4*, *Kdr*). Upon BMP-mediated extraembryonic mesoderm (ExM) differentiation, WT EpiSCs readily differentiate towards allantois and endothelial lineages. In contrast, *Pbx1*-KO EpiSCs have lower pERK but higher expression of *Fgf* ligands, pointing to a possible compensatory mechanism. These *Pbx1*-KO EpiSCs have a closed chromatin environment and hypermethylated methylome over allantois and endothelial genes (*Hand1*, *Tbx4*, *Kdr*), but a permissive chromatin landscape over yolk sac and primitive erythroid genes (*Tbx3*, *Gata2*, *Tal1*), which are also hypomethylated in nature. Upon BMP-mediated ExM differentiation, *Pbx1*-KO EpiSCs differentiate towards yolk sac and primitive erythroid lineages, but not towards allantois or endothelial lineages. These results provide context to previous work that suggested priming of ExM lineages in the epiblast stages^{6,8}.



Supplementary Fig. 7

Supplementary Figure 7.

a) Schematic illustration depicting the phenotype of E7.5 WT (left) and *Pbx1*-KO (right) mouse embryos, showing reduced DE (pink) formation and increased NMPs (light blue). Extraembryonic mesoderm (ExM), definitive endoderm (DE), Neural Mesodermal progenitors (NMPs), wild type (WT). **b-c)** RNAseq analyses assessing PSM of WT and *Pbx1*-KO EpiSCs showing expected increased differentiation of NMPs (*Nkx1-2*, *T-Bra*) in *Pbx1*-KO cells, and decreased expression of Presomitic mesodermal (PSM) markers (*Tbx6*, *Mgn1*)³³. **d)** RNAseq analyses of DE markers (*Foxa2*, *Cer1*, *Sox17* and *Hhex*) showed reduced DE differentiation in *Pbx1*-KO cells. $P < 0.05$, biological replicates (n) = 2, Wald Test with Benjamini–Hochberg adjusted P-values on DESeq2. Based on RNAseq (FPKM - fragments per kilobase of exon per million mapped fragments), for b), c) and d). **e)** Visualization of WGBS (SeqMonk) indicating differentially methylated regions (DMR) of the *Foxa2* locus in WT and *Pbx1*-KO EpiSCs showing hypomethylation in the *Pbx1*-KO EpiSCs. DNA methylation (%) is indicated in the bar.

Supplementary Table 1. Summary of cytokines and signalling pathway inhibitors used in this study.

Item name	Description	Company	Catalog No.
A-83-01	TGF- β inhibitor	Tocris	2939
Activin A	TGF- β agonist	Peprotech	120-14E
BMP4	BMP agonist	Peprotech	120-05ET
C59	WNT inhibitor	Cellagen Technology	C7641
CHIR-99021	WNT agonist	Axon MedChem	1386
bFGF	FGF agonist	Peprotech	450-33
LDN193189	BMP inhibitor	Sigma	SML0559
LIF	Made in house		
PD-0325901	MEK/ERK inhibitor	Axon MedChem	1408
PIK-90	PI 3-K inhibitor	EMD Millipore	528117
XAV939	WNT inhibitor	Sigma	X3004

Supplementary Table 2. Antibodies used for immunofluorescence staining of adherent cells.

Primary Antibody (clonality, host)	Company	Catalog	Dilution
CLDN6 (monoclonal, Rabbit)	CST	62831	1:100
FOXA2 (monoclonal, Rabbit)	CST	8186S	1:100
TBX6 (polyclonal, Goat)	R&D Systems	AF4744	1:50
E-CADHERIN (polyclonal, Goat)	R&D Systems	AF748	1:1000
Secondary Antibody (host)	Company	Catalog	Dilution
Goat Alexa Fluor 568 (Donkey)	Invitrogen	A-11057	1:200
Rabbit Alexa Fluor 488 (Donkey)	Invitrogen	A-21206	1:200

Supplementary Table 3. Antibodies used for embryo immunostainings.

Primary Antibody (clonality, host)	Company	Catalog	Dilution
CLDN6 (monoclonal, Rabbit)	CST	62831	1:100
FOXA2 (monoclonal, Rabbit)	CST	8186S	1:100
GATA6 (polyclonal, Goat)	R&D Systems	AF1700	1:100
HAND1 (polyclonal, Goat)	R&D Systems	AF3168	1:100
PBX1 (polyclonal, Rabbit)	CST	2230	1:100
T-BRA (polyclonal, Goat)	R&D Systems	AF2085	1:100
SOX17 polyclonal, Goat)	R&D Systems	NL1924R	1:200
NANOG (monoclonal, Rat)	eBioscience	14-5761	1:100
Secondary Antibody (host)	Company	Catalog	Dilution
Goat Alexa Fluor 568 (Donkey)	Invitrogen	A-11057	1:500
Mouse Alexa Fluor 647 (Donkey)	Invitrogen	A-31571	1:500
Rabbit Alexa Fluor 488 (Donkey)	Invitrogen	A-21206	1:500
Rat Alexa Fluor 647 (Donkey)	Invitrogen	A78947	1:500

Supplementary Table 4. Primers and probes used for RT-qPCR.

Gene	Forward	Reverse	Probe
<i>Sox17</i>	CGCTTCTCTGCCAAGGTC	CACAACGCAGAGCTAAGCAA	97
<i>Foxa2</i>	GAGCAGCAACATCACAG	CGTAGGCCTTGAGGTCCAT	77
<i>Hhex</i>	CTACACGCACGCCCTACTC	CAGAGGTCGCTGGAGGAA	50
<i>Cer1</i>	GACTGTGCCCTTCAACCAG	AGCAGTGGGAGCAGAAGC	105
<i>Sox2</i>	GGCAGAGAAGAGAGTGTTTGC	TCTTCTTTCTCCCAGCCCTA	34
<i>Msgn1</i>	AATTACCTGCCGCCTGTCT	TGAGTGTCTGGATCTTGGTGA	106
<i>Pax3</i>	GCGAGAAAAAGGCTAAACACA	CGGAGCCTTCATCTGACTG	110
<i>Pgk1</i>	TACCTGCTGGCTGGATGG	CACAGCCTCGGCATATTTCT	108
<i>Sdha</i>	TGTTCAAGTTCCACCCCA	TCTCCACGACACCCTTCTG	71
<i>Tbp</i>	GGGGAGCTGTGATGTGAAGT	CCAGGAAATAATTCTGGCTCA	95
<i>T-Bra</i>	CAGCCCACCTACTGGCTCTA	GAGCCTGGGGTGATGGTA	100
<i>Tbx6</i>	TGACTGATACTCGGCAAGCA	CGCTGTGGGGACAGAGAT	9
<i>Pou5f1</i>	GTTGGAGAAGGTGGAACCAA	CTCCTTCTGCAGGGCTTTC	95

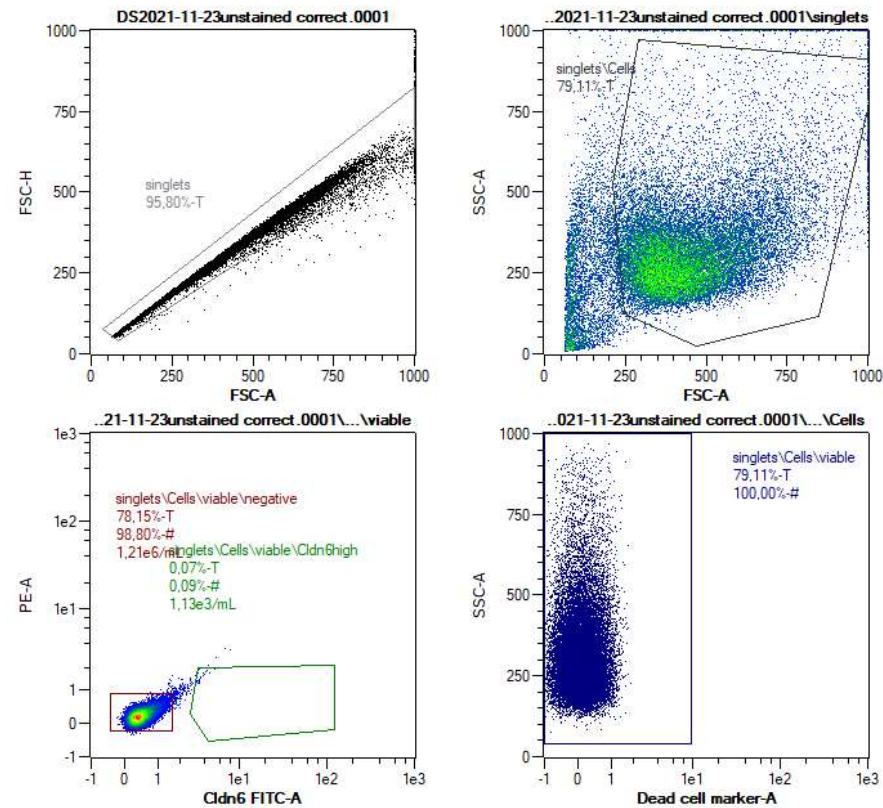
Supplementary Table 5. Gene names and probes created by Resolve Biosciences.

Gene ID	Gene Name
ENSMUST00000027863	<i>Atp1b1</i>
ENSMUSG00000037025	<i>Foxa2</i>
ENSMUST00000074667	<i>T-Bra</i>
ENSMUST00000006046	<i>Trh</i>
ENSMUSG00000023906	<i>Cldn6</i>
ENSMUSG00000074637	<i>Sox2</i>
ENSMUSG00000030699	<i>Tbx6</i>
ENSMUSG00000037335	<i>Hand1</i>
ENSMUSG00000025902	<i>Sox17</i>
ENSMUSG00000062960	<i>Kdr</i>
ENSMUSG00000030544	<i>Mesp1</i>
ENSMUSG00000018604	<i>Tbx3</i>
ENSMUSG00000047002	<i>Msgn1</i>

Raw FACS plots showing example of the sorting and purifying steps to get 90% positive CLDN6^{High} and CLDN6^{Low} sorts.

November 23rd

Unstained



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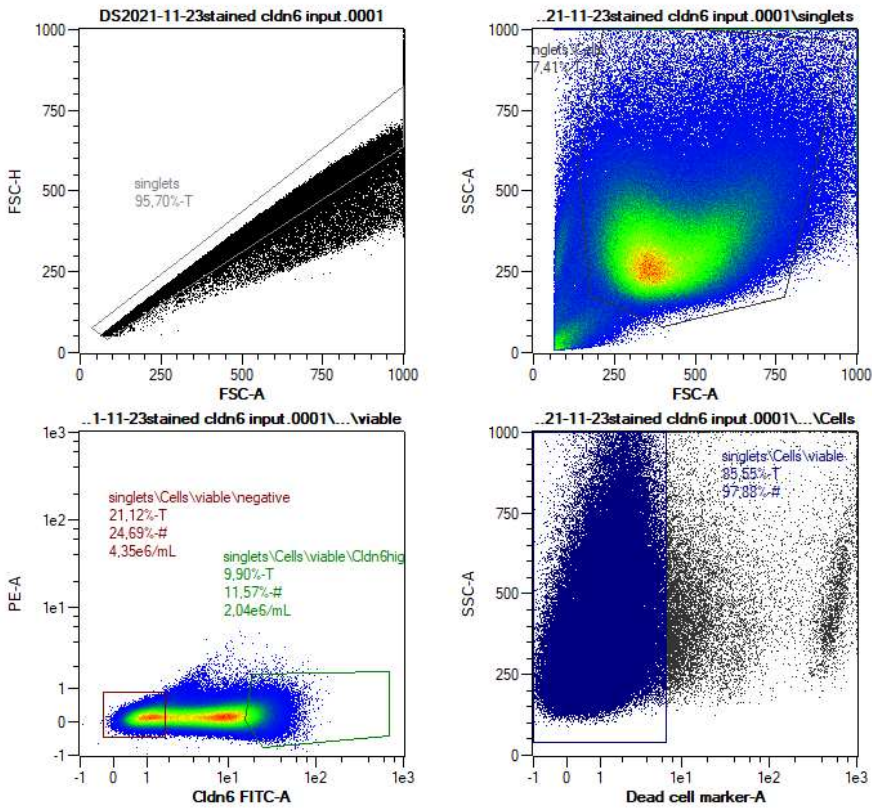
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singlets\Cells\viable	79.11	100.00	1.22e6
singlets\Cells\viable\Cldn6high	0.07	0.09	1.13e3
singlets\Cells\viable\negative	78.15	98.80	1.21e6

November 23rd

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(Cldn6+)

Vol: 2 mL (added 8mL, i.e. sorted in 10mL)

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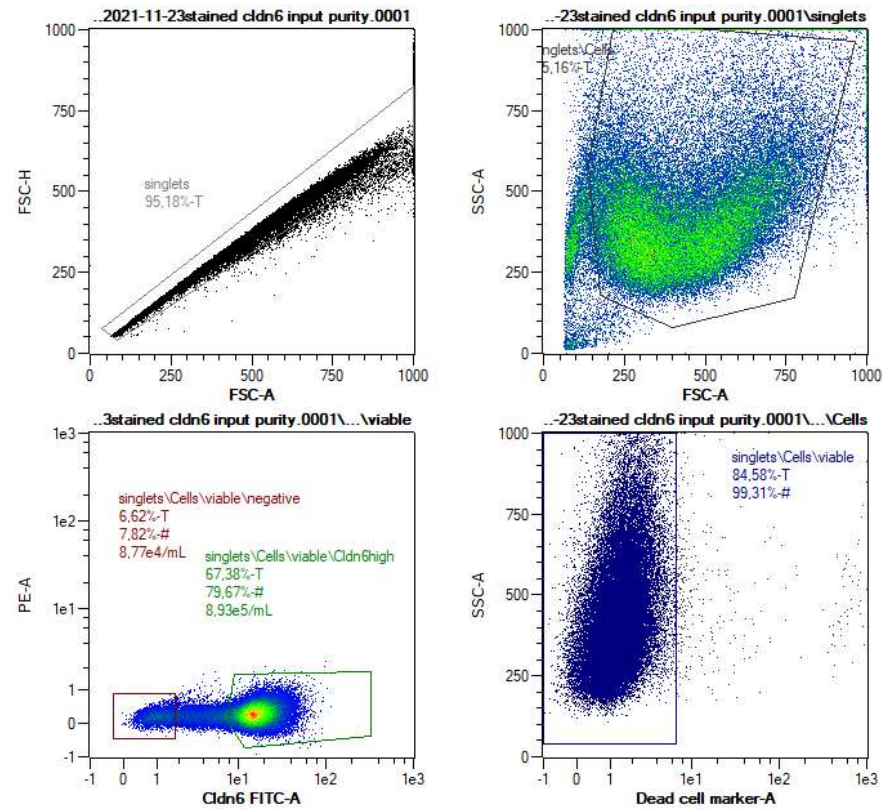
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singlets\Cells\viable\Cldn6high	9.90	11.57	2.04e6
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November 23rd

Input purity sort
(Pos Chamber from sort 1)

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Dilution: 1:2



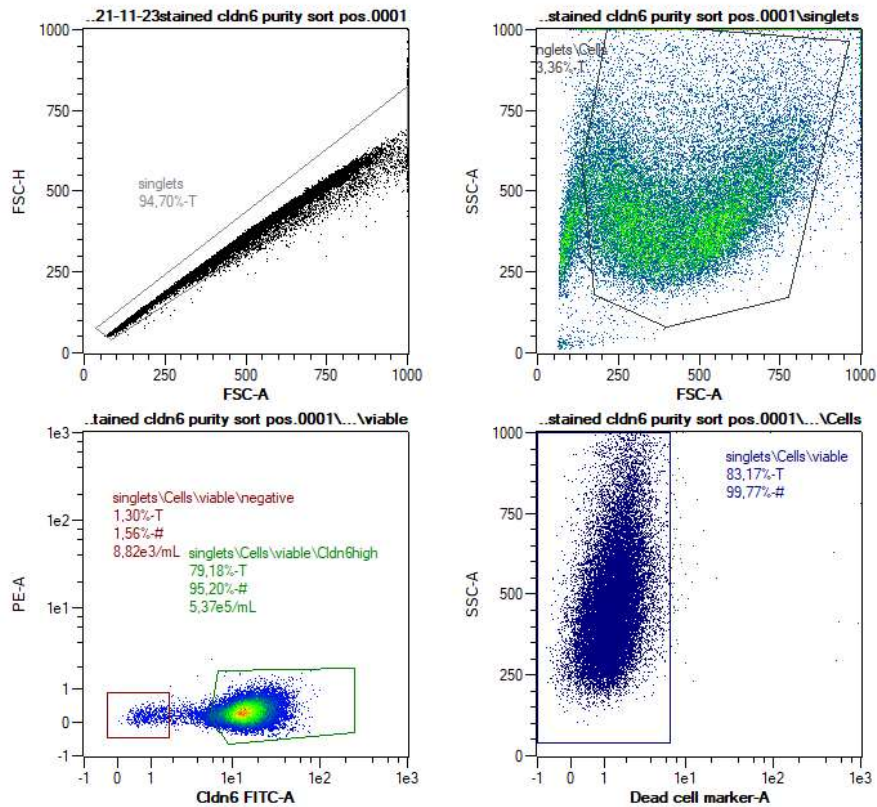
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singlets\Cells\ viable	84.58	99.31	1.12e6
singlets\Cells\ viable\Cldn6high	67.38	79.67	8.93e5
singlets\Cells\ viable\negative	6.62	7.82	8.77e4

November 23rd

Pos chamber purity sort

Vol: 300 µL

Dilution: 1:20



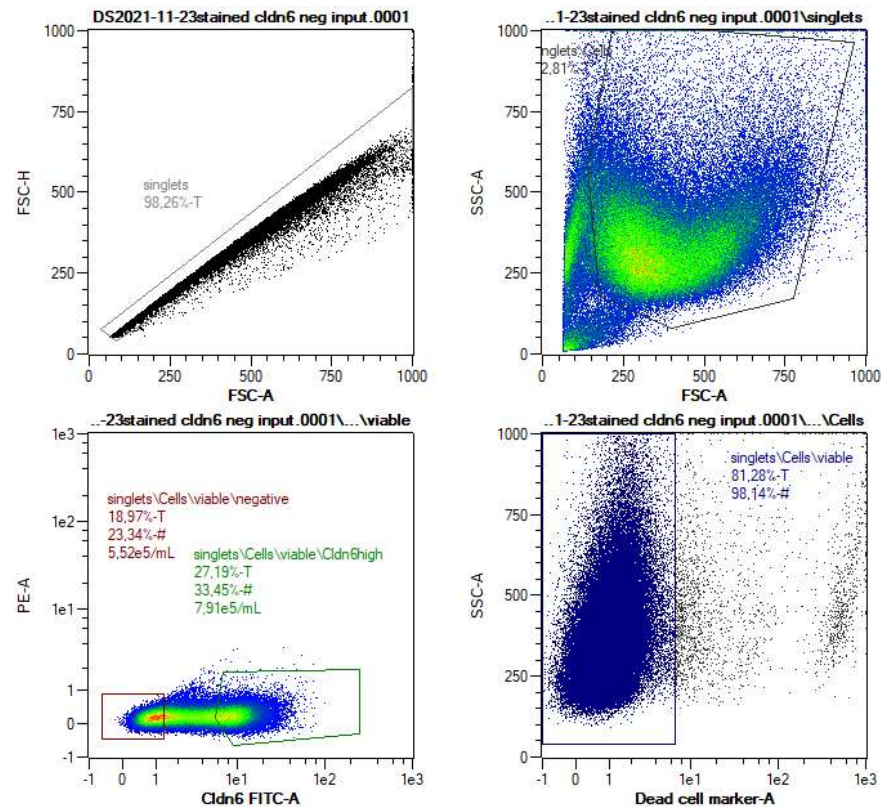
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singlets\Cells\ viable	83.17	99.77	5.65e5
singlets\Cells\ viable\Cldn6high	79.18	95.20	5.37e5
singlets\Cells\ viable\negative	1.30	1.56	8.82e3

November 23rd

Sort 3 on Cldn neg (from neg chamber sort 1)

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Dilution: 1:2



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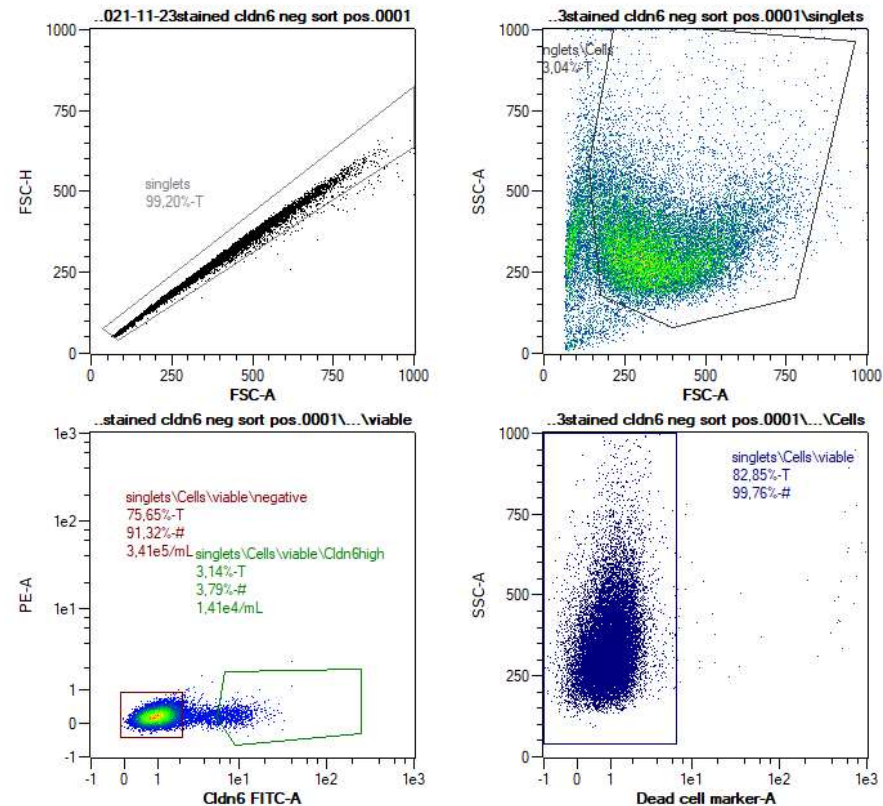
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singlets\Cells\viab\Cldn6high	27.19	33.45	7.91e5
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November 23rd

Pos chamber Sort3 (sort on Cldn6neg from Sort1)

Vol: 300 µL

Dilution: 1:20



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singlets\Cells\viable\negative	75.65	91.32	3.41e5