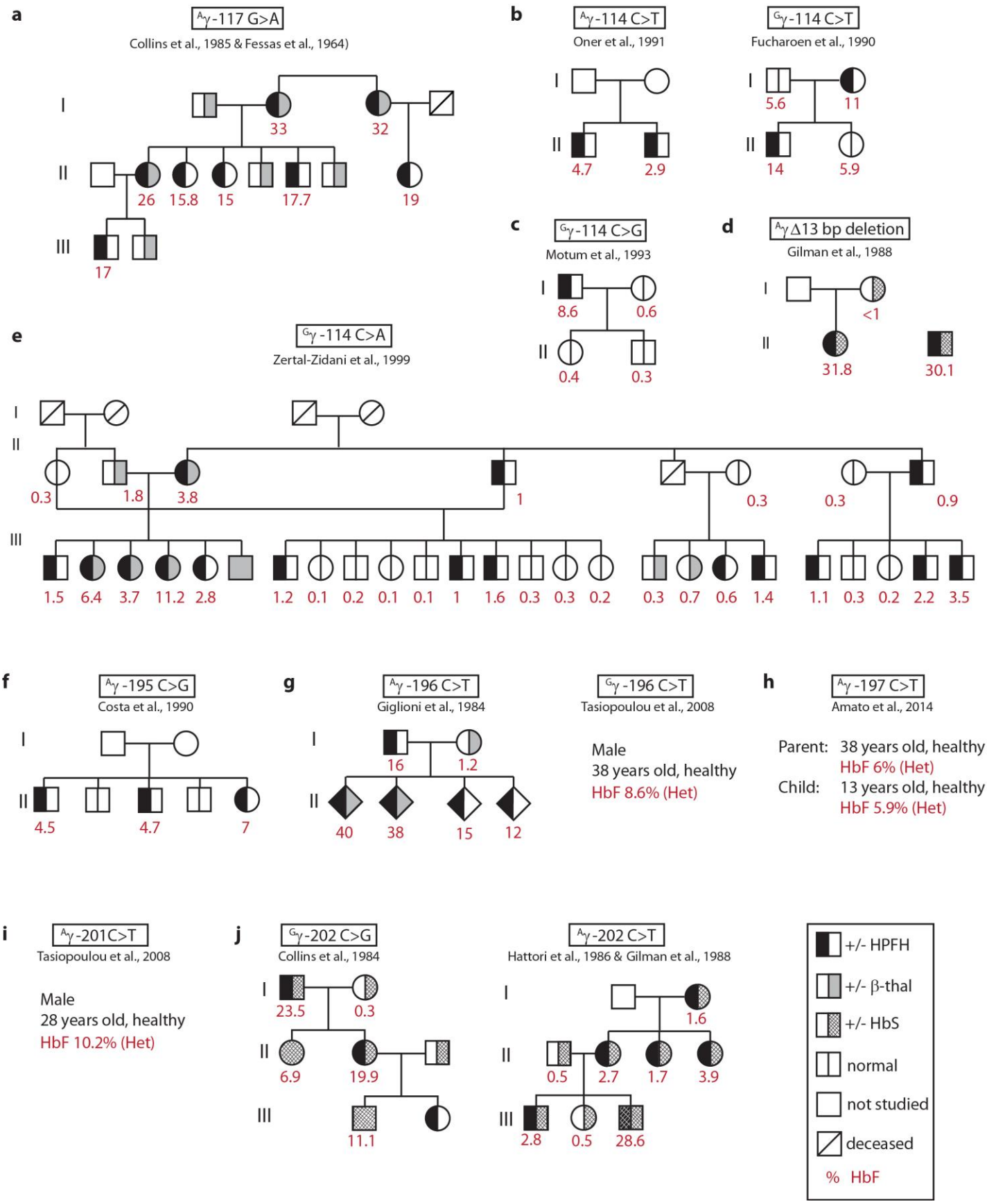
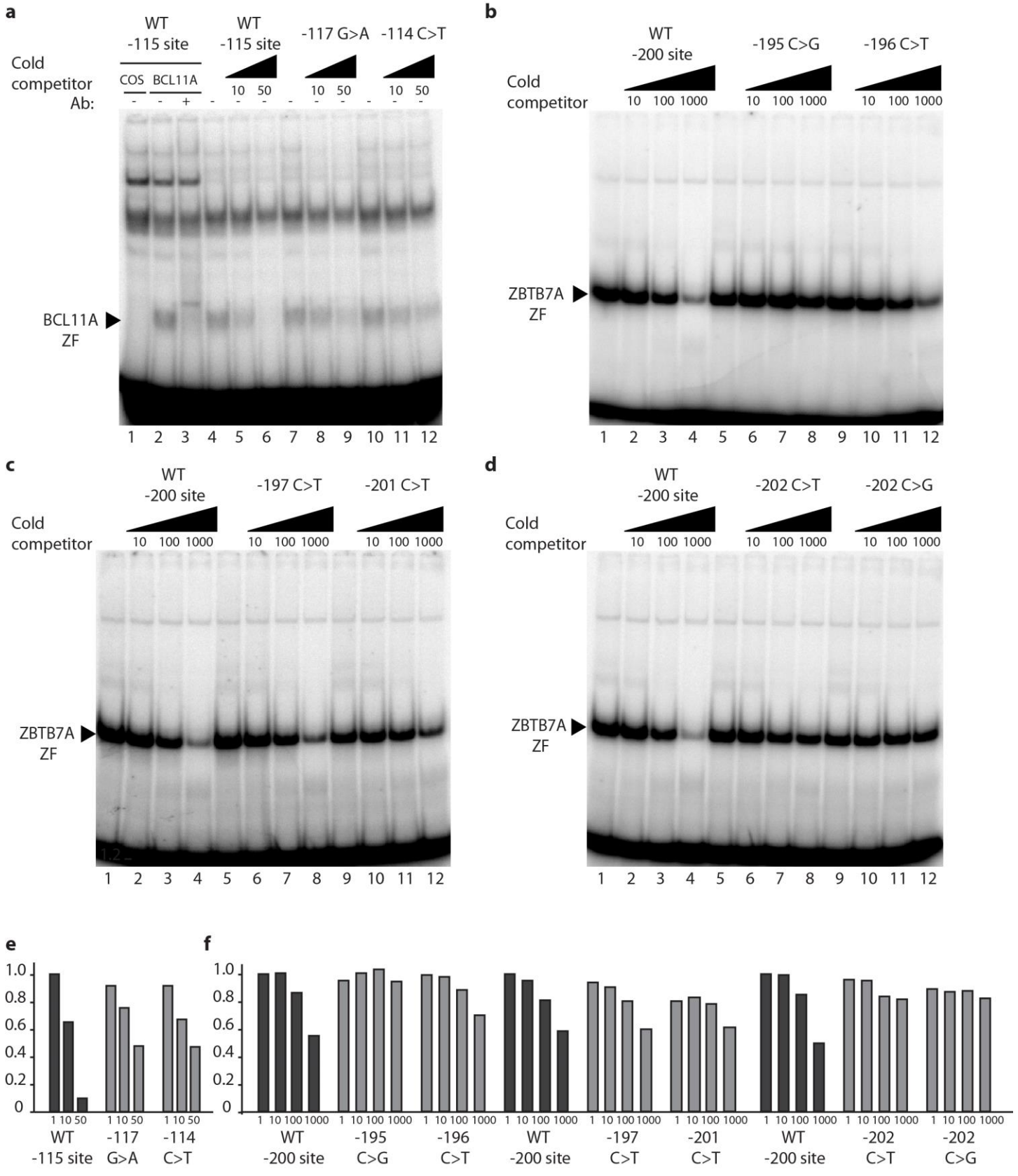

a
Aγ-117 G>A
Collins et al., 1985 & Fessas et al., 1964)
I
II
III
33
32
26
15.8
15
17.7
19
17
b
Aγ-114 C>T
Oner et al., 1991
4.7
2.9
Gγ-114 C>T
Fucharoen et al., 1990
5.6
11
14
5.9
c
Gγ-114 C>G
Motum et al., 1993
8.6
0.6
0.4
0.3
d
AγΔ13 bp deletion
Gilman et al., 1988
<1
31.8
30.1
e
Gγ-114 C>A
Zertal-Zidani et al., 1999
0.3
1.8
3.8
1
0.3
0.3
0.9
1.5
6.4
3.7
11.2
2.8
1.2
0.1
0.2
0.1
0.1
1
1.6
0.3
0.3
0.2
0.3
0.7
0.6
1.4
1.1
0.3
0.2
2.2
3.5
f
Aγ-195 C>G
Costa et al., 1990
4.5
4.7
7
g
Aγ-196 C>T
Giglioni et al., 1984
16
1.2
40
38
15
12
Gγ-196 C>T
Tasiopoulou et al., 2008
Male
38 years old, healthy
HbF 8.6% (Het)
h
Aγ-197 C>T
Amato et al., 2014
Parent: 38 years old, healthy
HbF 6% (Het)
Child: 13 years old, healthy
HbF 5.9% (Het)
i
Aγ-201C>T
Tasiopoulou et al., 2008
Male
28 years old, healthy
HbF 10.2% (Het)
j
Gγ-202 C>G
Collins et al., 1984
23.5
0.3
6.9
19.9
11.1
Aγ-202 C>T
Hattori et al., 1986 & Gilman et al., 1988
1.6
0.5
2.7
1.7
3.9
2.8
0.5
28.6
+/- HPFH
+/- β-thal
+/- HbS
normal
not studied
deceased
% HbF

Supplementary Figure 1

Pedigrees for HPFH families.

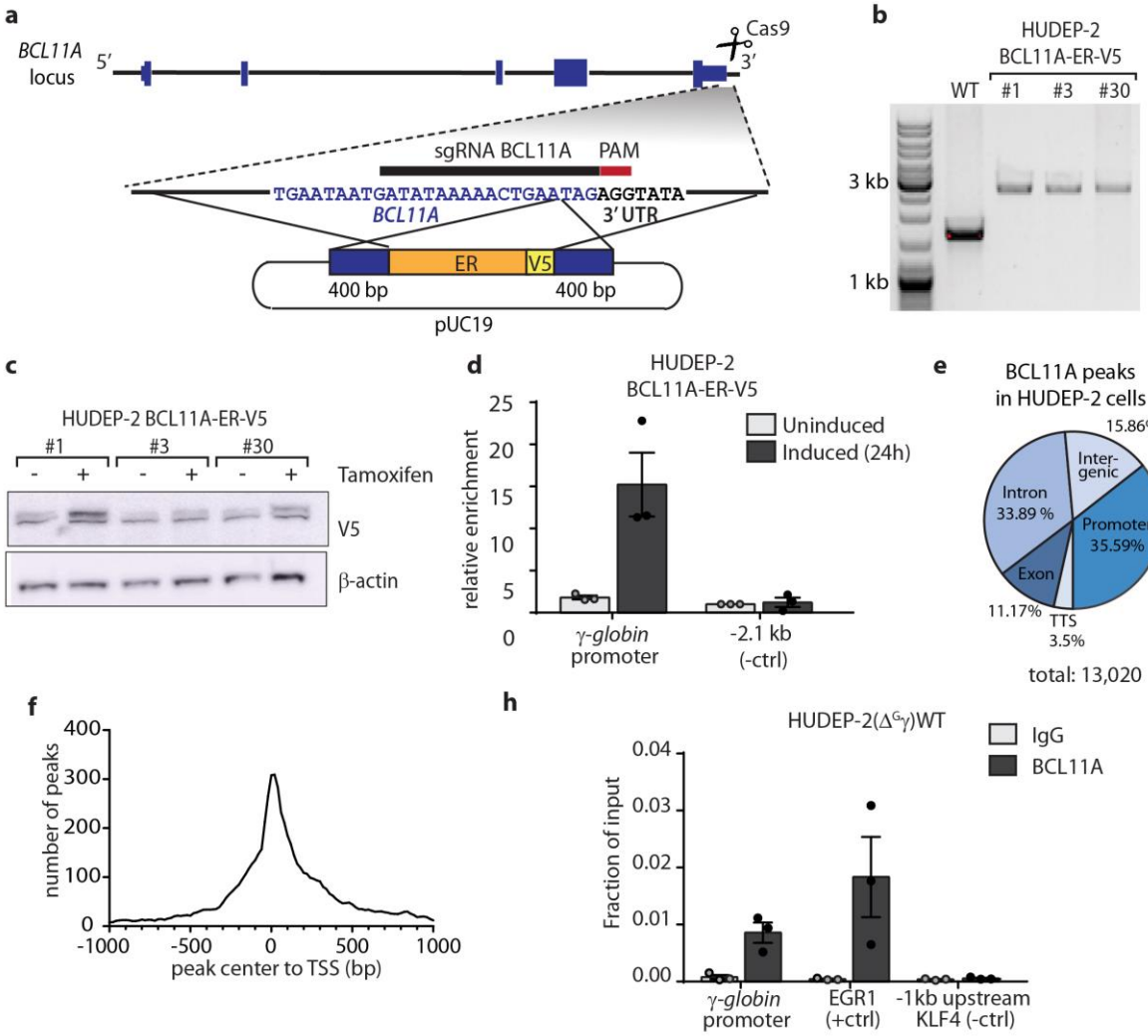
A compiled summary of previously published data is presented here describing the various HPFH mutations identified in each family, HbF levels and the genotypes of patients with HPFH mutations, β-thalassaemia (± β-thal) or sickle cell trait (± HbS). **a**–**e**, Pedigrees of families with HPFH mutations in the site at –115 bp in γ-globin. **f**–**j**, Pedigrees of families with HPFH mutations in the site at –200 bp in γ-globin.



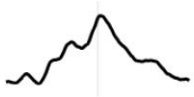
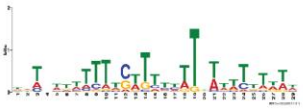
Supplementary Figure 2

Probes with HPFH mutations at –115 and –200 bp do not compete as well as wild-type probes for BCL11A and ZBTB7A binding.

a, BCL11A cold competition assays show that the c.–117G>A and c.–114C>T HPFH mutant probes do not compete as well for BCL11A binding as the WT probe, confirming specificity of binding. Increasing concentrations of cold probe were added in excess as indicated (10× and 50×). Lanes 1–3 show FLAG-tagged BCL11A ZF binding to the WT probe and a super-shift with anti-FLAG antibody as a control. Probes span –128 to –100 of the γ-globin promoter **b**–**d**, ZBTB7A cold competition EMSAs using HPFH mutant probes. Lane 1 contains ZBTB7A ZF bound to the hot WT probe. Increasing concentrations of cold probe were added in excess as indicated (10×, 100× and 1,000×). Probes span –209 to –187 of the γ-globin promoter. These data represent *n* = 1 biologically independent experiment. **e**,**f**, Densitometry analysis of the cold competition assays in **a**–**d**. Binding of BCL11A and ZBTB7A to the WT probe was used as a standard to normalize binding for the densitometry.



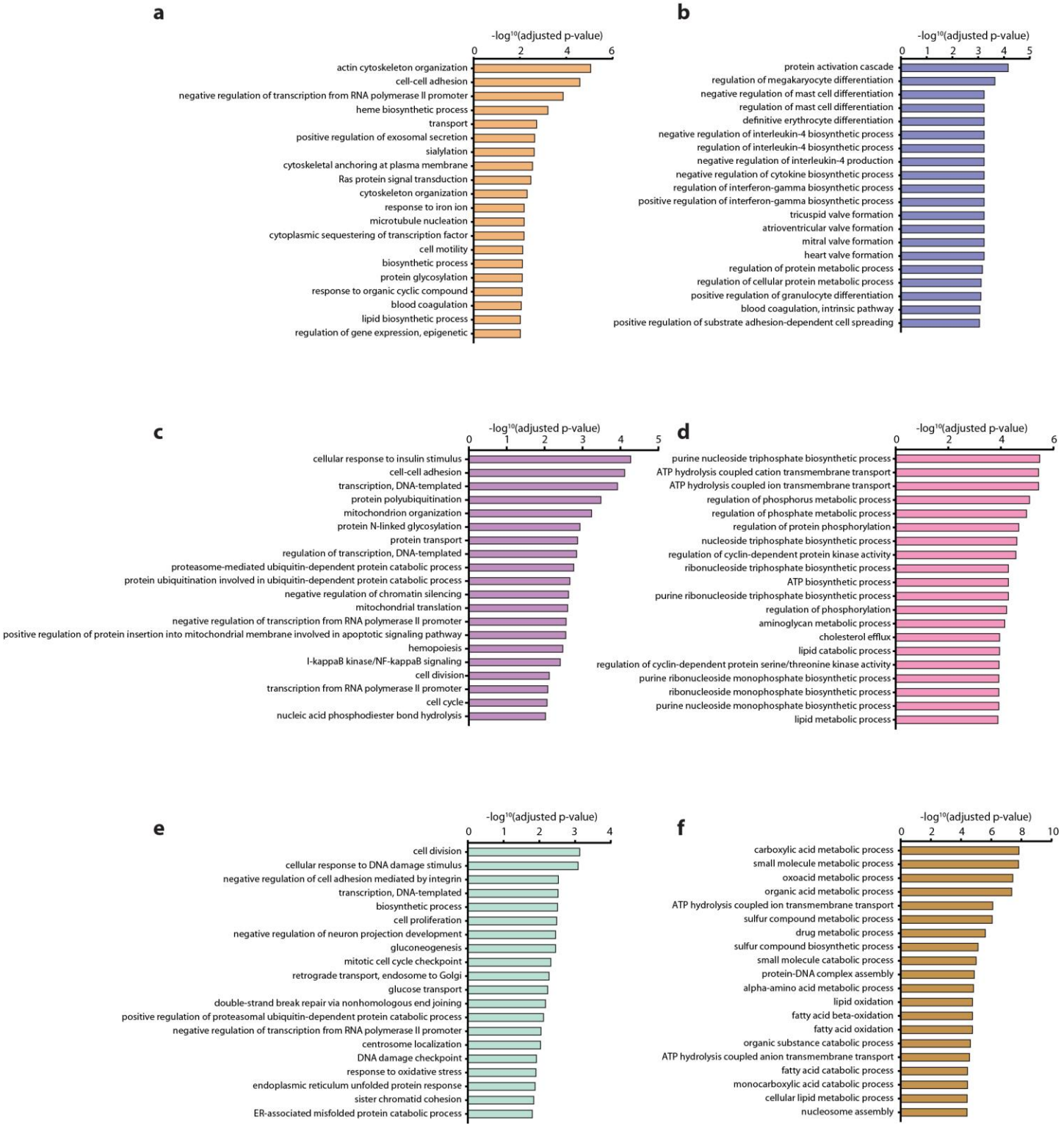
g

Motif Found	Discovery/Enrichment Program	E-value	Known or Similar Motifs	Distribution
	MEME	5.1e-227	Gata1 (MA0035.3) GATA2 (MA0036.2) Gata5_primary (UP00080_1)	Not Centrally Enriched
	MEME	1.3e-094		
	MEME	3.0e-072	SOC1 (MA0554.1) SVP (MA0555.1) FLC (MA0558.1)	Not Centrally Enriched
	DREME	2.6e-011	Klf4 (MA0039.2) KLF5 (MA0599.1) Klf7_primary (UP00093_1)	Not Centrally Enriched
	DREME	2.2e-007	ELK3 (MA0759.1) ELF1 (MA0473.2) EHF (MA0598.2)	Not Centrally Enriched

Supplementary Figure 3

Engineering the inducible BCL11A system with an ER and V5 tag for ChIP–seq.

a, CRISPR–Cas-9 system used to tag the endogenous *BCL11A* gene. The sgRNA target site is indicated. The donor plasmid contains 400 bp of homology on either side of the ER-V5 construct. **b**, PCR screening BCL11A-ER-V5 clones in HUDEP-2 cells. Shown are three clonal populations with the desired modification (n = 3). **c**, Western blot of nuclear extracts from HUDEP-2 BCL11A-ER-V5 cells in the uninduced and induced (tamoxifen for 24 h) state. Shown are three BCL11A-ER-V5 clonal populations in the uninduced and induced (tamoxifen) state (n = 3). **d**, HUDEP-2 ChIP–qPCR, using an antibody specific to the V5 tag, demonstrating BCL11A-ER-V5 binding to the γ-globin promoter (n = 3). Shown is the mean ± s.e.m. **e**, Genomic distribution of BCL11A-ER-V5 ChIP–seq peaks in HUDEP-2 cells. TTS, transcription termination site. **f**, Distribution of BCL11A-ER-V5 ChIP–seq peaks relative to the transcription start site (TSS). **g**, Motifs identified within BCL11A binding sites from BCL11A ChIP–seq in HUDEP-2 cells. Shown are the top five motifs as identified using MEME-ChIP[46], which incorporates multiple motif discovery programs such as MEME and DREME. The specific program used to identify the motif, the statistical significance (E value) and motif distribution within the peak list are shown. Similar known motifs are also shown. These data were generated from n = 2 biologically independent replicates. **h**, ChIP–qPCR of BCL11A, using an antibody specific to BCL11A, in HUDEP-2($\Delta^{G}\gamma$) WT (n = 3), with the *EGR1* gene as a positive control and the 1-kb region upstream of *KLF4* as a negative control. Means ± s.e.m. are shown.

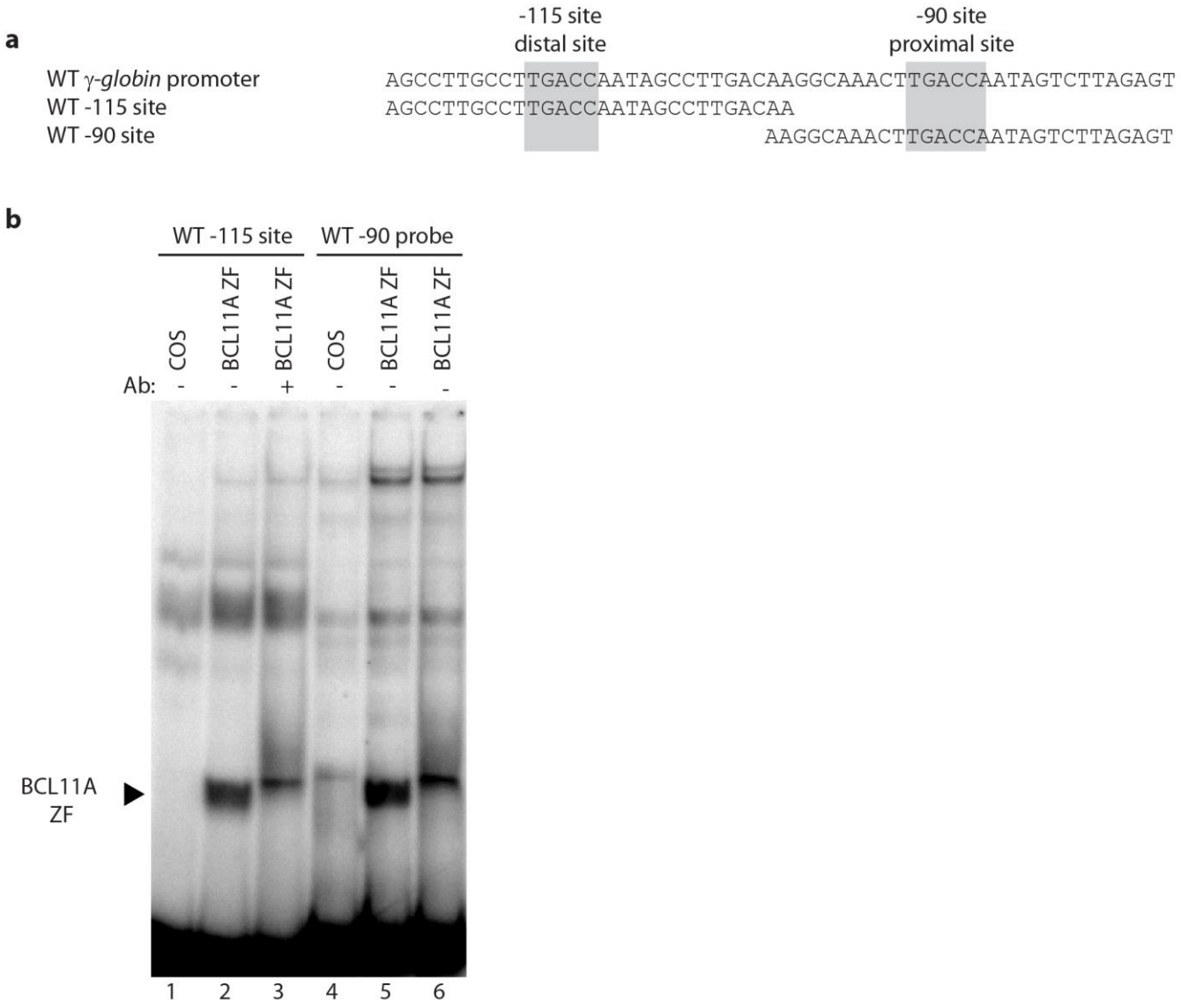


Supplementary Figure 4

Gene ontology analysis from BCL11A ChIP–seq in HUDEP-2 cells.

a,**b**, Gene ontology for the top 3,000 BCL11A ChIP–seq peaks analyzed with DAVID Bioinformatics Resources 6.7 and GOrilla, respectively. **c**,**d**, Gene ontology for the top 3,000 BCL11A ChIP–seq peaks located in promoters (–1,000 to +100 bp from the TSS) that do not contain the TGNCCA motif, analyzed by DAVID Bioinformatics Resources 6.7 and GOrilla, respectively. **e**,**f**, Gene ontology

for the top 3,000 BCL11A ChIP–seq peaks located in promoters (–1,000 to +100 bp from the TSS) that do contain the TGNCCA motif, analyzed by DAVID Bioinformatics Resources 6.7 and GOrilla, respectively. Shown are the top 20 most significant GO terms for each analysis. The DAVID Bioinformatics Resources 6.7 and GOrilla analyses were performed on the peaks generated from $n = 2$ biologically independent replicates.

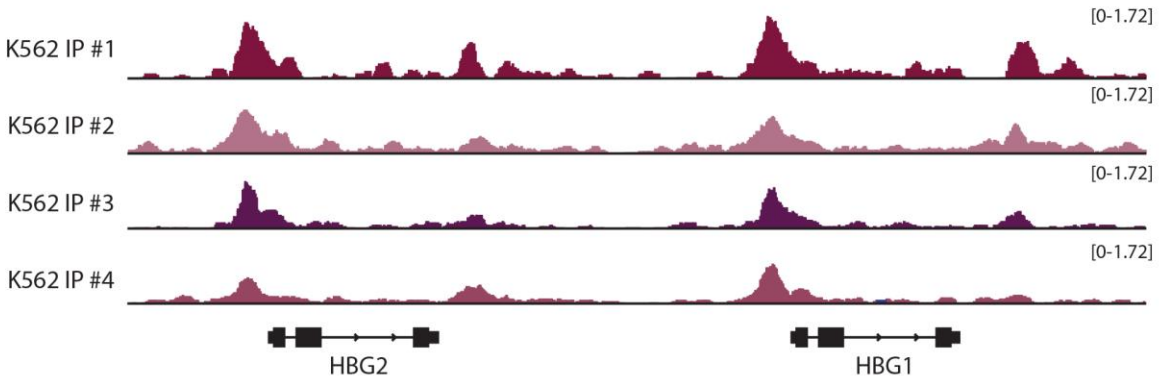


Supplementary Figure 5

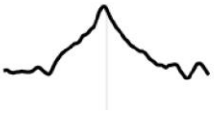
BCL11A binds to both the proximal and distal TGACC sites in the γ-globin promoter.

a, There are two TGACC motifs within the proximal promoter of the γ-globin gene, located at approximately –115 and –90 bp with respect to the TSS. **b**, The BCL11A ZF can bind to both the proximal and distal TGACC sites in the γ-globin promoter via EMSA in vitro. These data represent *n* = 1 independent experiment.

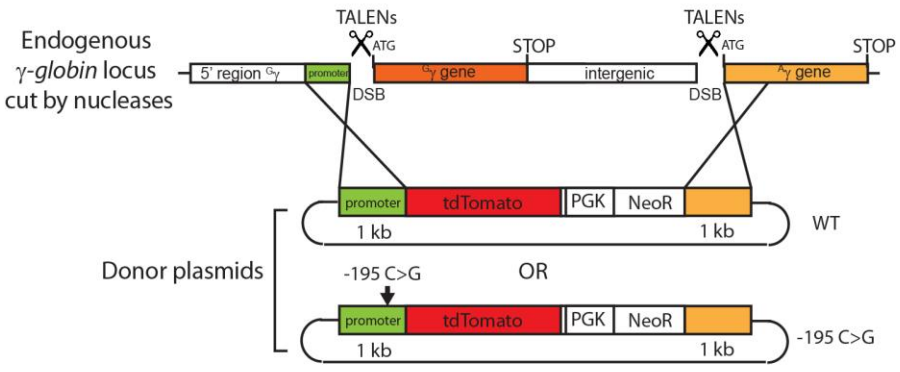
a



b

Motif Found	Discovery/Enrichment Program	E-value	Known or Similar Motifs	Distribution
	MEME	5.7e-187	SOC1 (MA0554.1) Nhp6b (UP00325_1) Mtf1_secondary (UP00097_2)	Not Centrally Enriched
	MEME	3.3e-133	Gata4 (MA0482.1) Gata1 (MA0035.3) GATA2 (MA0036.2)	Not Centrally Enriched
	MEME	2.2e-124		Not Centrally Enriched
	DREME	3.8e-031	RXRB (MA0855.1) usp (MA0016.1) Hnf4a (MA0114.3)	
	DREME	2.7e-015	Ascl2_secondary (UP00099_2) KLF5 (MA0599.1) EGR1 (MA0162.2)	Not Centrally Enriched

c

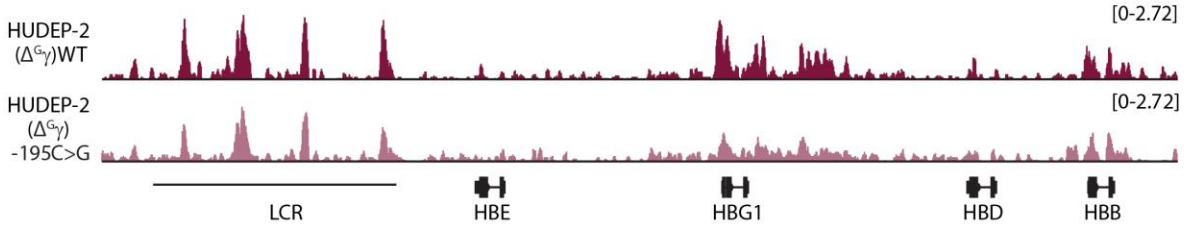


Supplementary Figure 6

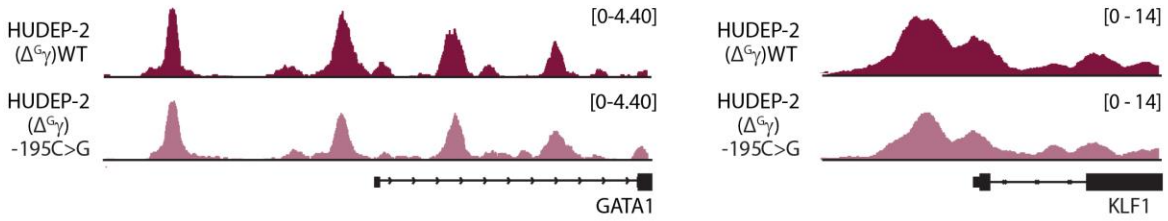
ZBTB7A ChIP–seq in K562 cells.

a, ZBTB7A binding across the γ-globin genes in four biologically independent replicates (*n* = 4) of ZBTB7A ChIP–seq in K562 cells. **b**, Shown are the top five motifs as identified using MEME-ChIP. The analysis was performed on the peaks generated from *n* = 4 biologically independent replicates. **c**, TALENs cut at the ATG of both endogenous γ-globin genes. TdTomato is integrated by homologous recombination from a donor vector with 1-kb arms of homology. The donor plasmid contains either the WT or –195C>G γ-promoter.

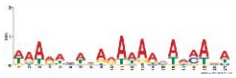
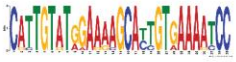
a



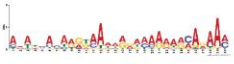
b



c

Motif Found	Discovery/Enrichment Program	E-value	Known or Similar Motifs	Distribution
	MEME	2.5e-172	SOC1 (MA0554.1) Mtf1_secondary (UP00097_2) FOXP1 (MA0481.1)	Not Centrally Enriched
	MEME	1.1e-136	Gata1 (MA0035.3) Gata5_primary (UP00080_1) Gata4 (MA0482.1)	Not Centrally Enriched
	MEME	2.2e-073	NFATC2 (MA0152.1)	Not Centrally Enriched
	DREME	1.4e-025	Glis2_primary (UP00024_1) GLIS1 (MA0735.1) ZIC1 (MA0696.1)	
	DREME	4.5e-014	KLF5 (MA0599.1) SP1 (MA0079.3) EGR1 (MA0162.2)	Not Centrally Enriched

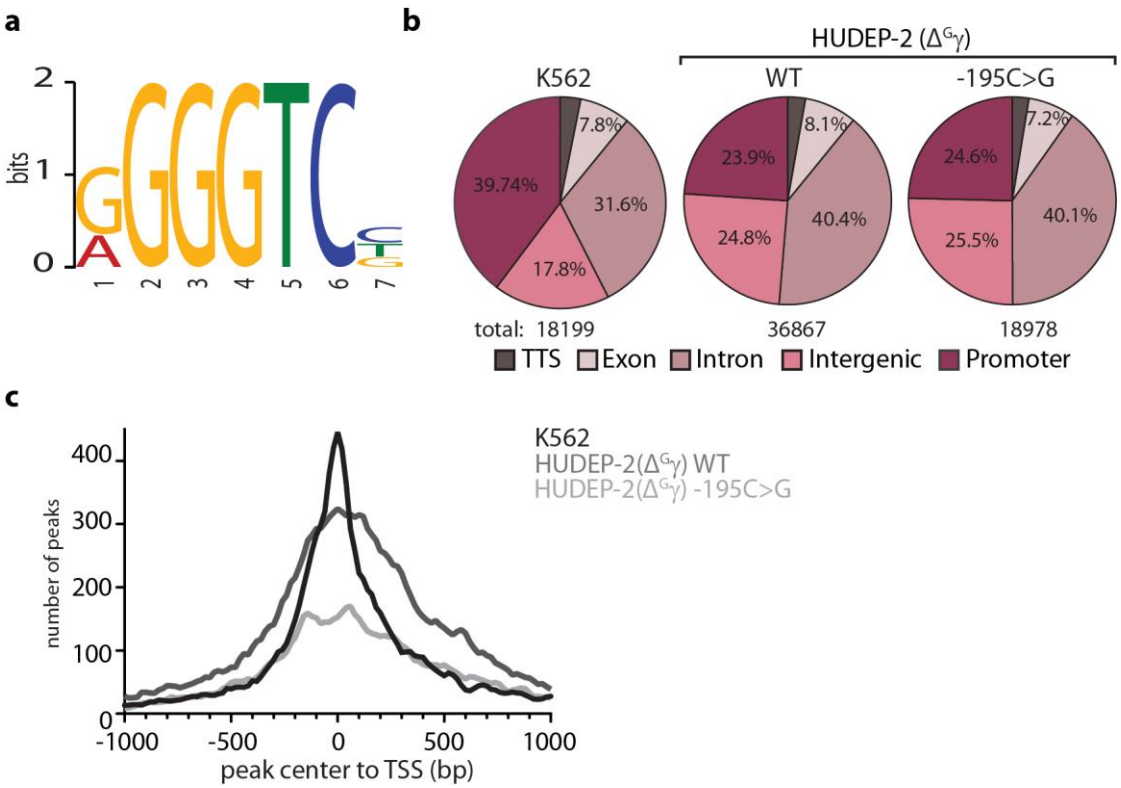
d

Motif Found	Discovery/Enrichment Program	E-value	Known or Similar Motifs	Distribution
	MEME	1.4e-272	Gata1 (MA0035.3) GATA2 (MA0036.2) Gata5_primary (UP00080_1)	Not Centrally Enriched
	MEME	6.1e-191		Not Centrally Enriched
	MEME	2.4e-179	SOC1 (MA0554.1) FOXP1 (MA0481.1)	Not Centrally Enriched
	DREME	4.5e-022	Glis2_primary (UP00024_1) GLIS1 (MA0735.1) ZIC1 (MA0696.1)	
	DREME	2.1e-012	SP1 (MA0079.3) KLF5 (MA0599.1) EGR1 (MA0162.2)	Not Centrally Enriched

Supplementary Figure 7

ZBTB7A ChIP–seq in HUDEP-2 cells.

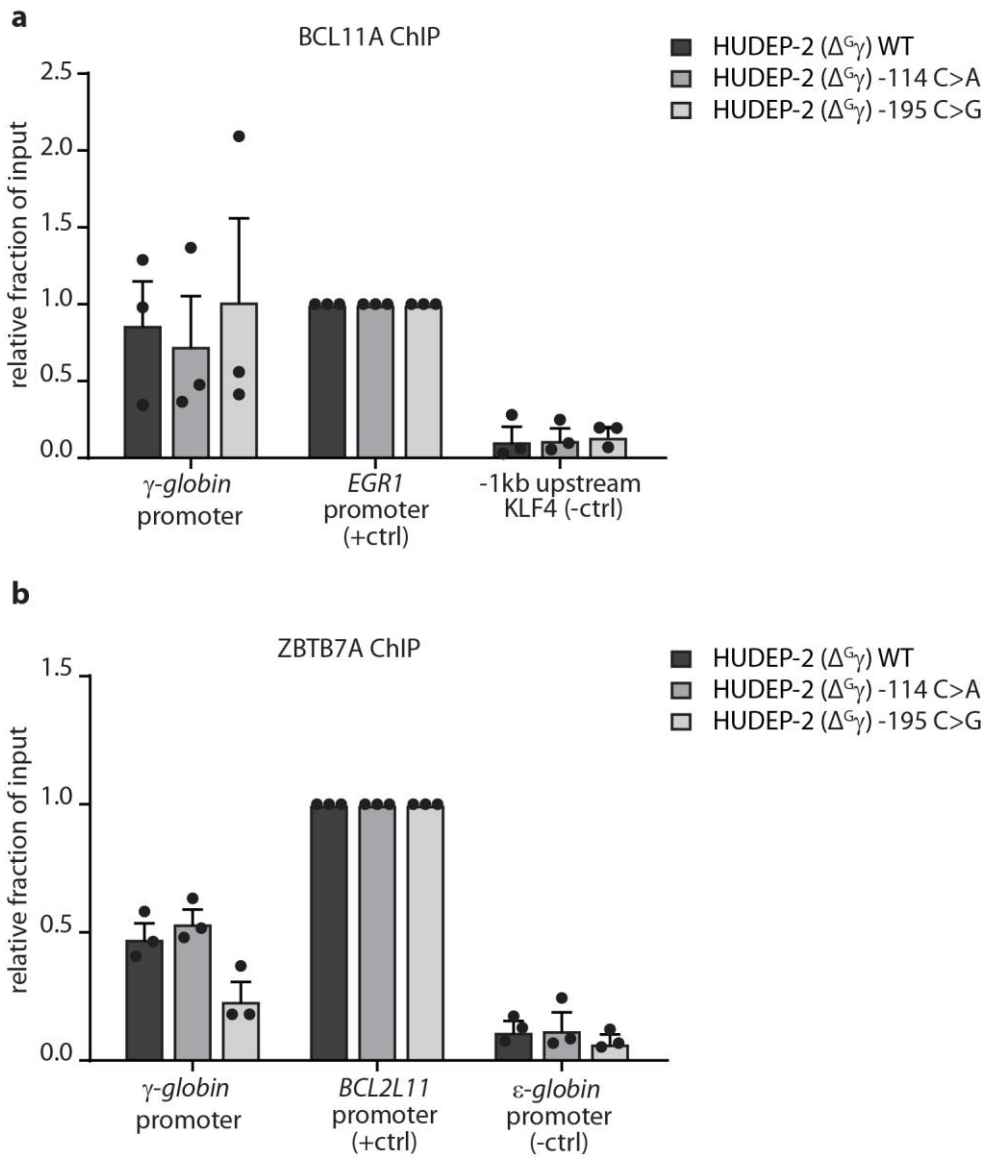
a, ZBTB7A ChIP–seq tracks across the β-globin locus in HUDEP-2($\Delta^{G}\gamma$) WT and –195C>G cells. LCR, locus control region. These data represent *n* = 1 independent experiment. **b**, ZBTB7A binding at control promoter regions of *GATA1* and *KLF1* in HUDEP-2($\Delta^{G}\gamma$) cells. These data represent *n* = 1 independent experiment. **c**,**d**, ZBTB7A binding motifs from ChIP–seq in HUDEP-2 cells. Shown are the top five motifs as identified using MEME-ChIP, known similar motifs and their distribution within the peak in HUDEP-2($\Delta^{G}\gamma$) WT (**c**) and –195C>G (**d**) cells. The analysis was performed on the peaks generated from *n* = 1 independent experiment.



Supplementary Figure 8

Comparison between ZBTB7A ChIP–seq in K562 and HUDEP-2($\Delta^{G}\gamma$) cells.

a, In vivo consensus motif of ZBTB7A in K562 and HUDEP-2($\Delta^{G}\gamma$) cells. **b**, Distribution of ZBTB7A peaks within the genome of K562 and HUDEP-2($\Delta^{G}\gamma$) cells. **c**, Location of ZBTB7A ChIP–seq peaks relative to the TSS.



Supplementary Figure 9

BCL11A and ZBTB7A bind the proximal promoter of the γ-globin gene independently.

a, BCL11A ChIP–qPCR in HUDEP-2($\Delta^{G}\gamma$) cells with WT, –114C>A and –195C>G HPFH alleles (*n* = 3). Means ± s.e.m. **b**, ZBTB7A ChIP–qPCR in HUDEP-2($\Delta^{G}\gamma$) cells with a WT, –114C>A or –195C>G HPFH mutation (*n* = 3). Means ± s.e.m.

Supplementary Table 1: Mammalian expression plasmids

Mammalian Expression Plasmids	**Species**	**Source**
pcDNA3-empty	N/A	*Invitrogen*
pcDNA3-FLAG BCL11A ZF (4-6) 740-835aa	Human	Gabriella Martyn
pcDNA3-FLAG ZBTB7A ZF 370-500aa	Human	Dr Alister Funnell

Supplementary Table 2: EMSA probes from Figure 1c

-128 to -100 bp *γ-globin* promoter	**Fwd (F)/ Rev (R)**	**5'- Sequence – 3'**
Wild-type (WT)	F	AGCCTTGCCTTGACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTCAAGGCAAGGCT
-117G>A	F	AGCCTTGCCTTAACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTTAAGGCAAGGCT
-114C>T	F	AGCCTTGCCTTGACTAATAGCCTTGACAA
	R	TTGTCAAGGCTATTAGTCAAGGCAAGGCT
-114C>A	F	AGCCTTGCCTTGACAAATAGCCTTGACAA
	R	TTGTCAAGGCTATTTGTCAAGGCAAGGCT
-114C>G	F	AGCCTTGCCTTGACGAATAGCCTTGACAA
	R	TTGTCAAGGCTATTCGTCAAGGCAAGGCT

Supplementary Table 3: EMSA probes from Figure 1d

-128 to -100 bp *γ-globin* promoter	**Fwd (F)/ Rev (R)**	**5'- Sequence – 3'**
Wild-type (WT)	F	AGCCTTGCCTTGACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTCAAGGCAAGGCT
-114-102 deletion (Δ13pb)	F	AGCCTTGCCTTGACAA
	R	TTGTCAAGGCAAGGCT

Supplementary Table 4: EMSA probes from Figure 1e

-209 to -187 bp *γ-globin* promoter	**Fwd (F)/ Rev (R)**	**5'- Sequence – 3'**
Wild-type (WT)	F	TGGGGGCCCCTTCCCCACACTAT
	R	ATAGTGTGGGGAAGGGGCCCCCA
-195 C>G	F	TGGGGGCCCCTTCCGCACACTAT
	R	ATAGTGTGCGGAAGGGGCCCCCA
-196 C>T	F	TGGGGGCCCCTTCTCCACACTAT
	R	ATAGTGTGGAGAAGGGGCCCCCA
-197 C>T	F	TGGGGGCCCCTTTCCCACACTAT
	R	ATAGTGTGGGAAAGGGGCCCCCA
-201 C>T	F	TGGGGGCCTCTTCCCCACACTAT
	R	ATAGTGTGGGGAAGAGGCCCCCA
-202 C>T	F	TGGGGGCTCCTTCCCCACACTAT
	R	ATAGTGTGGGGAAGGAGCCCCCA
-202 C>G	F	TGGGGGCGCCTTCCCCACACTAT
	R	ATAGTGTGGGGAAGGCGCCCCCA

Supplementary Table 5: EMSA probes from Figure 2

-128 to -100 bp *γ-globin* promoter	**Fwd (F)/ Rev (R)**	**5'- Sequence – 3'**
Wild-type (WT)	F	AGCCTTGCCTTGACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTCAAGGCAAGGCT
-120C>G	F	AGCCTTGC**G**TTGACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTCAA**C**GCAAGGCT
-120C>T	F	AGCCTTGC**T**TTGACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTCAA**A**GCAAGGCT
-120C>A	F	AGCCTTGC**A**TTGACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTCAA**T**GCAAGGCT
-119T>G	F	AGCCTTGCC**G**TGACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTCA**C**GGCAAGGCT
-119T>C	F	AGCCTTGCC**C**TGACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTCA**G**GGCAAGGCT
-119T>A	F	AGCCTTGCC**A**TGACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTCA**T**GGCAAGGCT
-118T>G	F	AGCCTTGCCT**G**GACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTC**C**AGGCAAGGCT
-118T>C	F	AGCCTTGCCT**C**GACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTC**G**AGGCAAGGCT
-118T>A	F	AGCCTTGCCT**A**GACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTC**T**AGGCAAGGCT
-117G>T	F	AGCCTTGCCTT**T**ACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGT**A**AAGGCAAGGCT
-117G>C	F	AGCCTTGCCTT**C**ACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGT**G**AAGGCAAGGCT
-117G>A	F	AGCCTTGCCTT**A**ACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGT**T**AAGGCAAGGCT
-116A>G	F	AGCCTTGCCTTG**G**CCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGG**C**CAAGGCAAGGCT
-116A>T	F	AGCCTTGCCTTG**T**CCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGG**A**CAAGGCAAGGCT
-116A>C	F	AGCCTTGCCTTG**C**CCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGG**G**CAAGGCAAGGCT
-115C>G	F	AGCCTTGCCTTGA**G**CAATAGCCTTGACAA
	R	TTGTCAAGGCTATTG**C**TCAAGGCAAGGCT
-115C>T	F	AGCCTTGCCTTGA**T**CAATAGCCTTGACAA
	R	TTGTCAAGGCTATTG**A**TCAAGGCAAGGCT
-115C>A	F	AGCCTTGCCTTGA**A**CAATAGCCTTGACAA
	R	TTGTCAAGGCTATTG**T**TCAAGGCAAGGCT
-114C>G	F	AGCCTTGCCTTGAC**G**AATAGCCTTGACAA
	R	TTGTCAAGGCTATT**C**GTCAAGGCAAGGCT

-114C>T	F	AGCCTTGCCTTGAC**T**AATAGCCTTGACAA
	R	TTGTCAAGGCTATT**A**GTCAAGGCAAGGCT
-114C>A	F	AGCCTTGCCTTGAC**A**AATAGCCTTGACAA
	R	TTGTCAAGGCTATT**T**GTCAAGGCAAGGCT
-113A>G	F	AGCCTTGCCTTGACC**G**ATAGCCTTGACAA
	R	TTGTCAAGGCTAT**C**GGTCAAGGCAAGGCT
-113A>T	F	AGCCTTGCCTTGACC**T**ATAGCCTTGACAA
	R	TTGTCAAGGCTAT**A**GGTCAAGGCAAGGCT
-113A>C	F	AGCCTTGCCTTGACC**C**ATAGCCTTGACAA
	R	TTGTCAAGGCTAT**G**GGTCAAGGCAAGGCT
-112A>G	F	AGCCTTGCCTTGACCA**G**TAGCCTTGACAA
	R	TTGTCAAGGCTA**C**TGGTCAAGGCAAGGCT
-112A>T	F	AGCCTTGCCTTGACCA**T**TAGCCTTGACAA
	R	TTGTCAAGGCTA**A**TGGTCAAGGCAAGGCT
-112A>	F	AGCCTTGCCTTGACCA**C**TAGCCTTGACAA
	R	TTGTCAAGGCTA**G**TGGTCAAGGCAAGGCT
-111T>G	F	AGCCTTGCCTTGACCAA**G**AGCCTTGACAA
	R	TTGTCAAGGCT**C**TTGGTCAAGGCAAGGCT
-111T>C	F	AGCCTTGCCTTGACCAA**C**AGCCTTGACAA
	R	TTGTCAAGGCT**G**TTGGTCAAGGCAAGGCT
-111T>A	F	AGCCTTGCCTTGACCAA**A**AGCCTTGACAA
	R	TTGTCAAGGCT**T**TTGGTCAAGGCAAGGCT
-110A>G	F	AGCCTTGCCTTGACCAAT**G**GCCTTGACAA
	R	TTGTCAAGGC**C**ATTGGTCAAGGCAAGGCT
-110A>T	F	AGCCTTGCCTTGACCAAT**T**GCCTTGACAA
	R	TTGTCAAGGC**A**ATTGGTCAAGGCAAGGCT
-110A>C	F	AGCCTTGCCTTGACCAAT**C**GCCTTGACAA
	R	TTGTCAAGGC**G**ATTGGTCAAGGCAAGGCT
-109G>T	F	AGCCTTGCCTTGACCAATA**T**CCTTGACAA
	R	TTGTCAAGG**A**TATTGGTCAAGGCAAGGCT
-109G>C	F	AGCCTTGCCTTGACCAATA**C**CCTTGACAA
	R	TTGTCAAGG**G**TATTGGTCAAGGCAAGGCT
-109G>A	F	AGCCTTGCCTTGACCAATA**A**CCTTGACAA
	R	TTGTCAAGG**T**TATTGGTCAAGGCAAGGCT

Supplementary Table 6: EMSA probes from Supplementary Figure 5b

-128 to -100 bp *γ-globin* promoter	**Fwd (F)/ Rev (R)**	**5'- Sequence – 3'**
-115 site wild-type (WT)	F	AGCCTTGCCTTGACCAATAGCCTTGACAA
	R	TTGTCAAGGCTATTGGTCAAGGCAAGGCT
-90 site wild-type (WT)	F	AAGGCAAACTTGACCAATAGTCTTAGAGT
	R	ACTCTAAGACTATTGGTCAAGTTTGCCTT

Supplementary Table 7: Human RT-PCR primers

Gene	**Fwd (F)/ Rev (R)**	**5'- Sequence – 3'**
18S	F	CACGGCCGGTACAGTGAAAC
	R	AGAGGAGCGAGCGACCAA
HBG	F	CCTGTCCTCTGCCTCTGCC
	R	GGATTGCCAAAACGGTCAC
HBB	F	TGTCCACTCCTGATGCTGTTATG
	R	GGCACCGAGCACTTTCTTG

Supplementary Table 8: Primers for screening successfully modified BCL11A-ER-V5 clones from Supplementary Figure 2.

Description	**Fwd (F)/ Rev (R)**	**5'- Sequence – 3'**
Human BCL11A screen	F	GGACACTTGCGACGAAGA
	R	ATTACCTCACCCAATGCT

Supplementary Table 9: Human ChIP-qPCR Primers

Gene	**Fwd (F)/ Rev (R)**	**5'- Sequence – 3'**
γ-globin promoter	F	TCAATGCAAATATCTGTCTGAAACG
	R	CAAGGCTATTGGTCAAGGCAA
-2.1 kb *γ-globin*	F	CCTGACCAGGAACCAGCAGAAAAG
	R	AAGGTGCTATAACAAAATAGCATAG
γ-globin promoter (Figure 4c)	F	TGGAATGACTGAATCGGAACAA
	R	GGACCGTTTCAGACAGATATTTGC
HS2	F	CATCACTCTAGGCTGAGAACATCTG
	R	GGCTCAAGCACAGCAATGC
β-globin promoter	F	GGAGGGCTGAGGGTTTGAAGTCC
	R	TGTCCTTGGCTCTTCTGGCACTG
BCL2L11 promoter	F	GCTTAGAAACTCAGGGCAC
	R	GTGTGAAGCCTCGGGAG
ε-globin promoter	F	CACAAACTTAGTGTCCATCCATCAC
	R	CCCTGTTCTCCATGGTACTTAAAAG
EGR1 promoter	F	CACTCCCGGTTCGCTCTC
	R	GGGTTCTATCGCTGTCATCC
1kb upstream *KLF4* promoter	F	CGTACTCACCGCCATTGTC
	R	TGGATGAGTCACGCGGATAA
γ-globin promoter (Supp. Figure 9)	F	CAAATATCTGTCTGAAACGGTCCC
	R	ACTCTAAGACTATTGGTCAAGTTTGC