Materials and methods

Reagents and Antibodies

Antibodies were purchased from companies: histone H3 and acetylated histone H3, Upstate Biotechnology (Buckingham, UK); p300, HDAC1, PLZF and IRP-1 Santa Cruz (Heidelberg, Germany); DNMT1, MeCP2, MBD1, β-actin (ChIP grade antibodies from Abcam, (Cambridge, UK); horseradish peroxidase-conjugated antibodies were from DakoCytomation (Saxvägen, Sweden). The LightShift® Chemiluminescent EMSA Kit (Pierce) was employed according to manufacturer's instructions for detection of immobilized nucleic acids and biotinylated proteins.

Immunoblotting. Nuclear extracts were prepared from transfected 293T, KG1 cells or from mouse tissues (*1* and described in Chromatin immunoprecipitation section), analyzed by SDS-PAGE, and visualized by Western blotting using monoclonal Anti-PLZF or beta-actin for detection. Blots were developed with the ECL kit (Amersham Pharmacia).

Expression and reporter plasmids. PLZF, $PLZF^{MUT}$ (PLZF lacking the ability to bind DNA), cDNA expression vectors, have been previously described (*2* and references within).

The tk-luc reporter plasmids were derived from the pT109luc plasmid by inserting the L1 PLZF DNA binding sequences upstream of the minimal herpes simplex virus thymidine kinase promoter. The PCR inserts were cloned into the *BamH*I and *Sal*I sites of the polylinker in the pT109 plasmid or into the *Sac*I and *Sal*I sites of the polylinker in the pt109 plasmid containing five GAL4 binding sites cloned into the *BamH*I and *Sal*I of the polylinker (described in MCB). Mutations within the PLZF binding site were generated by PCR amplification in conjunction with the Quickchange site-directed mutagenesis kit (Stratagene) using primers (derived from sequences from the L1 genomic region) 5'-ctgtttatatgctgctgtattccccttattgatttgcgtatattgaa-3' (forward) and 5'-ttcaatatacgcaaatcaataaggggaatacagcatataaacag-3' (reverse).

Cell culture and transient transfection. Transient transfections using 293T cells (*1*), maintained in Dulbecco's modified Eagle's medium with 100 U/ml penicillin, 100 μg/ml streptomycin, 20 mM glutamine, and 10% fetal calf serum, were performed using the calcium phosphate precipitation method (Promega) as previously described (*1,2*). KG1 cells were grown in RPMI 1640 media (Life Technologies Ltd, Paisley, UK) containing 100 U/ml penicillin, 100 μg/ml streptomycin, 20 mM glutamine, and 10% fetal calf serum, in a humidified atmosphere of 95% O_2 and 5% CO_2 at 37°C. BlueScribe M+ DNA was used as a carrier to equalize the total amount of transfected DNA. In all co-transfection experiments, the total amount of transfected mammalian expression vector was kept constant. All transfections were performed at least three times.

L1-EGFP transposition assay. Transient transfections using 293T cells were performed using the calcium phosphate precipitation method. On day 0, $1x10^4$ 293T cells were transfected with 0.350 μg of L1-EGFP (EF06R) with or without expression vectors (0.1 μg) and with carrier DNA to a total of 0.5 μg in total. Heat shock was carried out on day 7 for 20 minutes at 42°C and cells were returned to culture to recover overnight. On day 8, cells were harvested and the percentage of EFGP+ cells analyzed by flow cytometry (BD FACSCaliber), gating on live cells by forward/side scatter and propidium iodide/AnnexinV exclusion (as described in the Annexin V apopototic detection Kit APC, eBioscience). All transfections were performed at least three times.

Colony-forming assays

Mouse bone marrows from wild type and PLZF knock-in mice were plated in methylcellulose media (Methocult M3434; StemCell Technologies) according to the manufacturer's directions, and colonies were scored at 12 to 14 days. Images of colonies were obtained using a Nikon TE200 inverted microscope (Nikon Instruments Inc). For each replating, 10,000 cells were replated in methycellulose and total number of colonies was determined after 10 days.

Chromatin immunoprecipitation (ChIP) and sequential ChIP assays (Primers list).

L1_F2 5'-acaaatggaaatcctggaggt-3' (forward) and 5'-ctttctagaccctctcttagt-3' (reverse),

L1_F10 5'-tttgagcctatgtgtgtctct-3' (forward) and 5'-ctttctggctactgacctata-3' (reverse),
L1_F15 5'-taaggagattttgggctgaga-3' (forward) and 5'-attcctcgacacatacactct-3' (reverse),
L1 PCR1 5'-gccaagatggccgaataggaa-3' (forward) and 5'-gaggttactgctgtctttttg-3' (reverse),
L1 PCR2 5'-caaaaagacagcagtaacctc-3' (forward) and 5'-ttggtcttttcacatagtcc-3' (reverse),
L1 PCR3 5'-gggactatgtgaaaagaccaa-3' (forward) and 5'-ggatagttagctcctcttgtt-3' (reverse),
L1 PCR4 5'-aacaagaggagctaactatcc-3' (forward) and 5'-ctgttattggtcgattcagag-3' (reverse),
L1 PCR5 5'-ctctgaatcgaccaataacag-3' (forward) and 5'-cttaggattgacttggcaatg-3' (reverse),
L1 PCR6 5'-cattgccaagtcaatcctaag-3' (forward) and 5'-tctcattgttcaattcccacc-3' (reverse),
and mouse L1 PCR5 5'-ccattccttctgaaactattc-3' (forward) and 5'-tgaataccctttatttccttc-3' (reverse).

Methylation-specific PCR

A total of approximately 250ng of KG1 genomic DNA with sufficient purity was digested with 100 U of the CpG-methylation blocked restriction enzyme *Scrf*I and the insensitive *Xba*I (New England Biolabs, Beverly, MA, USA) at 37°C for 2 hours before being precipitated. Samples were resuspended with distilled H_2O at a concentration of 50ng/μl. For subsequent PCR analysis, aliquots containing 100ng of native or predigested DNA were used as template for each reaction. The primer pairs chosen (5'- gccaagatggccgaataggaa -3' and 5'-gaggttactgctgtctttttg -3') flanked *Scrf*I and *Xba*I restriction sites within the L1 5'UTR-CpG island region. PCR was performed with the Expand Long Template PCR System (Roche, Germany) in a final volume of 50μl containing, 30 pmoL of primers, 200 mM nucleotides, and 3.5U of genomic polymerase mix. Reactions were performed according to the manufacturer's protocol. Subsequently, the 559-bp-long PCR product was analyzed on an ethidium bromide-stained 1% agarose gel. Specificity and sensitivity of the methylation-specific PCR were determined in control samples with a defined methylation status. Undigested genomic DNA, which resembles a fully methylated status, served as a positive control. For the corresponding controls, genomic DNA was predigested with the CpG-methylation insensitive *Xba*I or a non-cutting XhoI endonucleases, which mimics an unmethylated or methylated promoter status respectively and control of successful enzymatic digestion. As a control for a hypomethylated sequence, we used genomic DNA derived from untransfected 293T cells (No PLZF lane).

Real-time PCR

Total RNA was isolated from tissues and cell lines using the Trixol reagent (Gibco BRL). Cells were homogenised in Trizol with a dounce homogenizer and RNA was extracted following the manufacturer's suggested protocol. RNA was resuspended in 10mM Tris (pH 7.5) and the concentration was determined by absorbance at 260 nm. One microgram of total RNA was reverse-transcribed using Moloney Murine Leukemia reverse transcriptase (M-MLV-RT; Gibco BRL) and random hexamers primers (Amersham), as suggested by the manfacturer's instructions.

Real-time PCR was performed using an ABI 7700 Sequence Detection System (PE Applied-Biosystems) in the presence of SYBR-green. The optimisation of the real-time PCR reaction was performed according to manufacturer's instructions (PE Applied-Biosystems, User Bulletin 2 applied to the SYBR-Green I core reagent protocol) but scaled down to 25 μl per reaction. The PCR conditions were standard (SYBR-Green I core reagent protocol) and all reagents were provided in the *FullVelocity™ SYBR®* Green QPCR Master Mix (Stratagene).

cDNA produced from the equivalent of 50 ng of RNA was analyzed in triplicate. After optimisation, nucleotides were used at various concentrations for the detection and quantification of *PLZF* (5'-ttactggctcattcagcgggt-3' (forward), 5'-cactcaaagggcttctcacct-3' (reverse)), *ORF1* (5'-acttcccaaatcttaaa-3' (forward), 5'-aaaagtctggtgtaatt-3' (reverse)), (reverse)) and *ABL* (5'-caaatccaagaaggggctctct-3' (forward), 5'-tcgagctgcttcgctgaga-3' (reverse)). *PLZF* and *ORF1* expression was normalized to the expression of *ABL* as the housekeeping genes.

Murine nucleotides primers were used at various concentrations for the detection and quantification of GAPDH, signal and coding of PLZF, c-kit, CRABP1, myc, and L1 ORF2:
GAPDH 5'-GCACAGTCAAGGCCGAGAAT-3' and 5'-GCCTTCTCCATGGTGGTGAA-3'
PLZF 5′-ACCAGTGTACCATCTGCACG and 5′-CTGCTCTACCATGTGTTGGG
C-kit 5′-AGAAGCAGATCTCGGACAGC and 5′-CATCACAGAAGCCAGAAGGAC
CRABP1 5'-CAA CTT CAA GGT CGG AGA GG and 5'- CAA GGG TAC ACA AGG CAA CA
Myc 5′-TCACCAGCACAACTACGCCG and 5′-CAGGATGTAGGCGGTGGCTT
ORF2 5'-ACTTCCCAAATCTTAAA-3' and 5'-AAAAGTCTGGTGTAATT-3'

Production of RNA templates
To create short (96bp) or long (188bp) RNA templates forward primers containing the T7 promoter were used in a PCR amplification on the L1 EF06R plasmid, short forward 5'-cgccgtaatacgactcactataggatgcaaggctggttcaatata-3', short reverse 5'-catctattgagataatcatgtggtttt-3', long forward tttccgtaatacgactcactagggcttatccaccatgatcaagtgggc-3', long reverse 5'-taatttattgagagtttttagcat-3'. *In vitro* transcription was done according to the manufacturer's protocol (mMESSAGE mMACHINE® kit, Ambion, Applied-Biosystems) and RNA oligonucleotides were purified with Ambion NucAway Spin columns. For biotin labelling 2µl of Biotin RNA labelling mix were added to the reaction (Roche). Successful *in vitro* transcription was monitored by running an aliquot of the reaction on 10% denaturating gel, staining with ethidium bromide, and subsequent UV detection. RNA concentration was assessed by A280 absorbance on a Nanodrop (Peqlab).
RNA EMSA was performed according to the LightShift® Chemiluminescent EMSA Kit (Pierce) with modification of the PLZF binding reaction described in (*2*).

RNA Pull-Down (RIP-Chip), primers list.
Human LINE1 primers: first round, 5'-ccattccttctgaaactattc-3' (forward) and 5'-tgaataccctttatttccttc-3' (reverse); second round, 5'-cagagacacaacaaaaaaaga-3' (forward) and 5'-aaagggaatgcttccagttttt-3' (reverse).
Mouse LINE1 primers: first round, 5'-ccagttcttcacaaactattc-3' (forward) and 5'-tgtatcccctttgatctccttt-3' (reverse); second round, 5'-aaaagatccaacaaagataga-3' (forward) and 5'-agtgggattgcttccagcttc-3' (reverse).
Human CDC14 primers: first round, 5'-taccctgagctcaacaataat-3' (forward) and 5'-acttcccaaaagaatgaactg-3' (reverse); second round, 5'-atctctgccttcttacctta-3' (forward) and 5'-gtttctcccttacttcttctt-3' (reverse).
To ensure that RNA samples had no genomic contamination, DpnI endoculease pre-digested samples were assessed by PCR. Furthermore, pre-treatment with RNase A (Roche) of the immunoprecipitated purified RNA particles, was carried out to assess that amplified products were only amplified from RNA template.

***In vitro* translation protocol**
Long ORF2 RNA templates (500 ng) were used as a template to translate ORF2 peptides *in vitro* using the T7 TNT coupled transcription-translation system (Promega), following the supplier's directions and addition of Transcend™ tRNA (Promega) in the reaction buffer to order to biotinylate lysine residues for detection (see Reagents and Antibodies). Furthermore, the translation reaction was complemented with either unprogrammed rabbit reticulocyte lysate or PLZF protein generated using the *in vitro* translation system. Biotinylated peptides were detected by Western blotting followed by chemiluminescence.

MeDIP and second generation sequencing
Control and target gene primers to test MeDIP:

Actin 5'-AGCCAACTTTACGCCTAGCGT and 5'-TCTCAAGATGGACCTAATACGGC

GAPDH 5'-CTCTGCTCCTCCCTGTTCC and 5'-TCCCTAGACCCGTACAGTGC

H19-ICR 5'-GCATGGTCCTCAAATTCTGCA and 5'-GCATCTGAACGCCCCAATTA

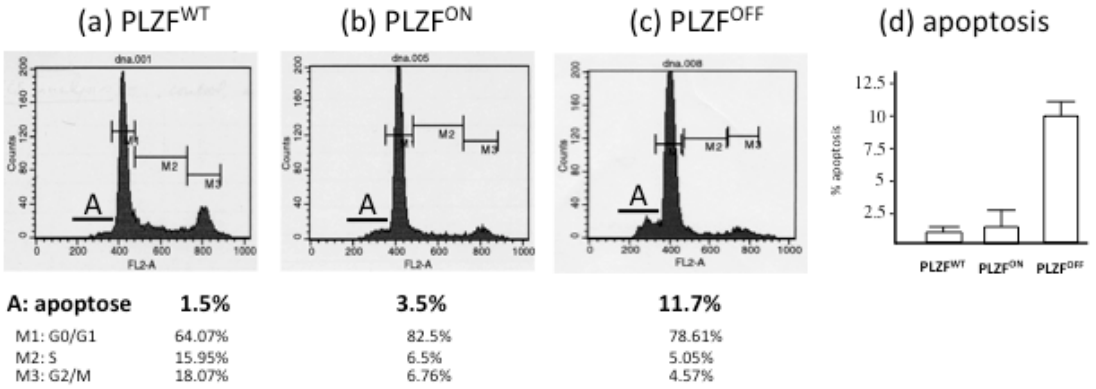
IAP (repeat) 5'-CTCCATGTGCTCTGCCTTCC and 5'-CCCCGTCCCTTTTTTAGGAGA

L1 5'LTR 5'-AAAGATCCAACAAAGATAGA and 5'-AGTGGGATTGCTTCCAGCTTC

myc 5'-CCGAACAACCGTACAGAAAG and 5'-CCGGGGTGTAAACAGTAATAG

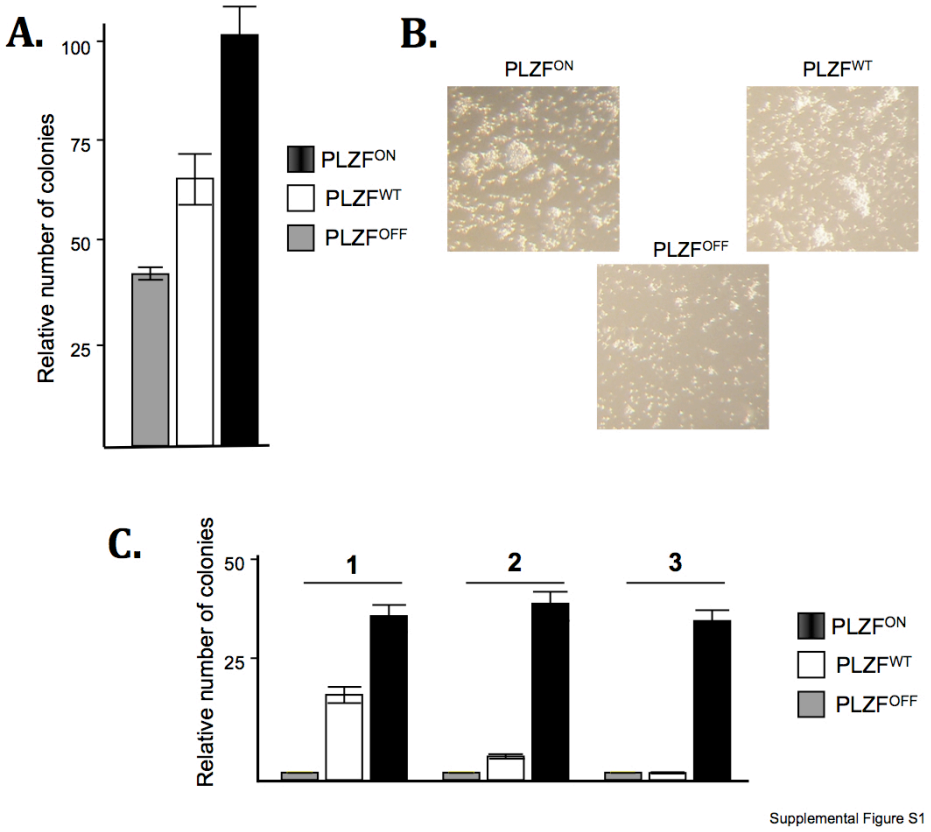
CRABP1 5'- AACGCCAGTCTCTCTGCAAC and 5'- CTCTGTACCAGCTTACCCAAC
c-kit 5'-AAAGAGCGGCAGACAAGAGGAC and 5'-CGGCACAAAAGCATCACCAAAC

Supplementary figures

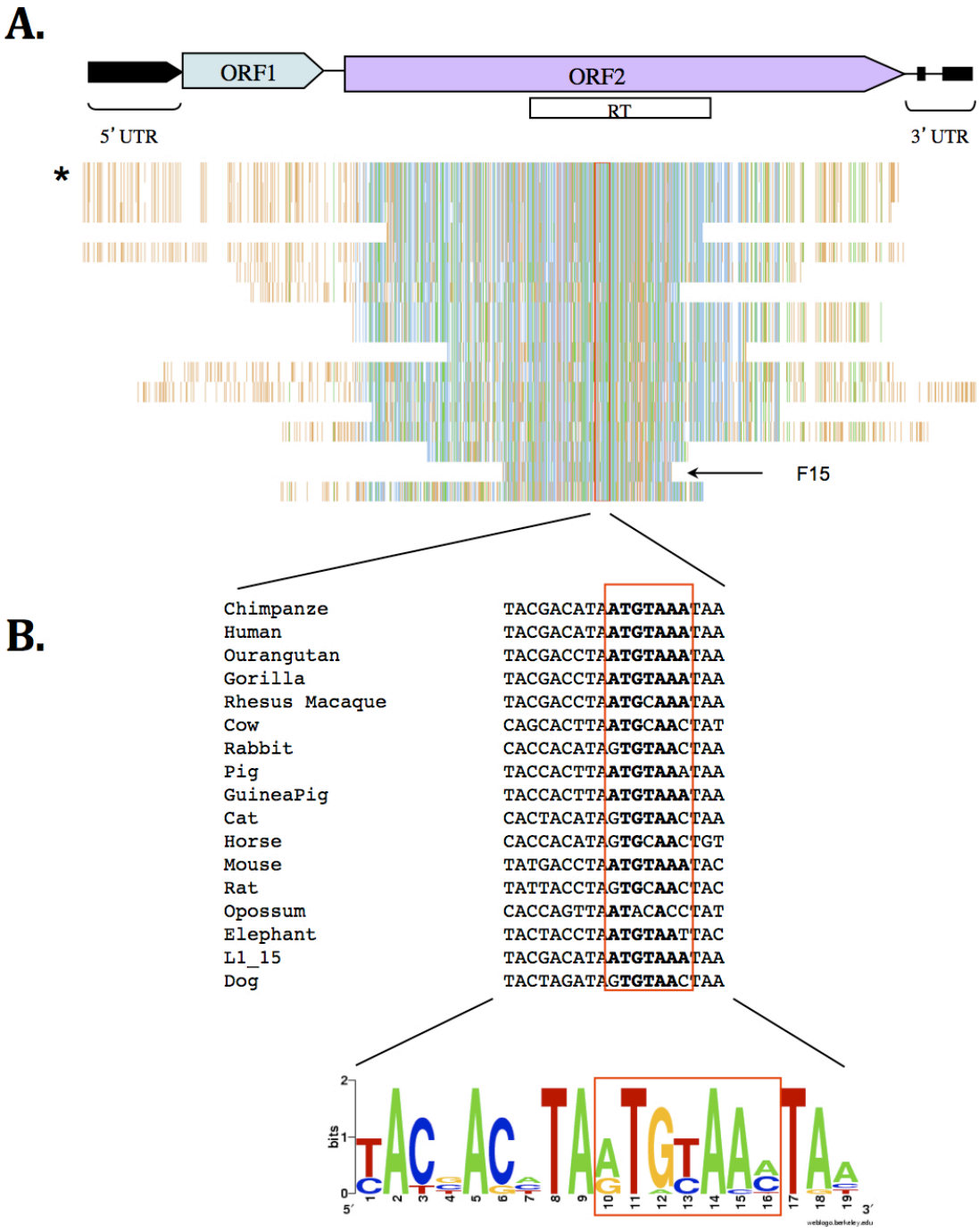


A: apoptose	1.5%	3.5%	11.7%
M1: G0/G1	64.07%	82.5%	78.61%
M2: S	15.95%	6.5%	5.05%
M3: G2/M	18.07%	6.76%	4.57%

Supplementary Figure S1-1: PLZF^{ON} acetylation mutant expression is associated with increased spontaneous apoptosis in purified testicular cells. Flow cytometric analysis of percentage apoptosis in testicular cells purified from (a) PLZF^{WT}, (b) PLZF^{ON} and (c) PLZF^{OFF} mice. DNA content of cells was estimated by flow cytometery following the propidium iodide staining protocol kit (eBiosciences). DNA histograms sub G0/G1 fraction indicates apoptotic cells (A), G0/G1, S and G2 by M1, M2 and M3 gating respectively. Percentage of cells are indicated under each histogram. (d) The results shown in the bar diagram are means±SD of 2 individual mice. The PLZF^{OFF} expression, associated with loss of PLZF epigenetic function, correlates with the loss of PLZF expression (PLZF knock-out mouse model, Costoya *et al.* 2004) associated with an increased spontaneous apoptosis in the testicular tissue.



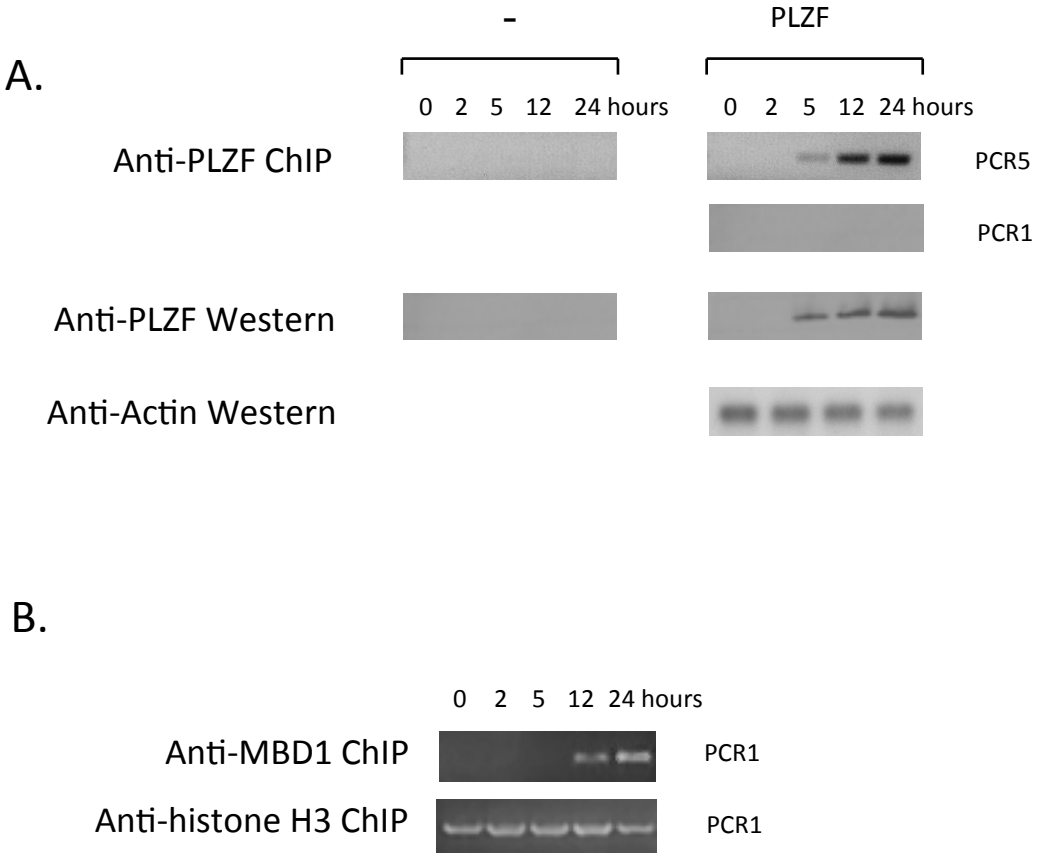
Supplementary Figure S1-2: PLZF acetylation mutants impair self-renewal of bone marrow progenitors. (A) Whole bone marrow cells from PLZF^{ON}, PLZF^{OFF} and PLZF^{WT} mice were plated in methylcellulose supplemented with cytokines (Methocult M3434). Ten thousand cells were plated per well, in triplicate. Results are mean ± SEM from 3 different subtype of mice. Colonies were scored at 12 to 14 days. As seen, bone marrow cells from PLZF^{OFF} mice raised fewer colonies than PLZF^{ON} or PLZF^{WT} bone marrow cells, note that the number of PLZF^{ON} mutant colonies have an increase of 25% compared to the PLZF^{WT} cells. Number under the histograms are absolute numbers of colonies of a representative experiment. (B) Photomicrograph of CFUs grown in Methocult M3434, magnification x40. (C) For serial replating assay, cells were extracted from methylcellulose, washed and identical cell numbers were replated in Methocult M3434 and the total number of colonies was determined after 10 days. PLZF^{WT} bone marrow cells can still induce colonies after 2 rounds of replating, mutant cells from PLZF^{OFF} background are not able to form any colony, while PLZF^{ON} cells have a self-renewal capacity increased with equal number of colonies after 3 rounds of replating. Number under the histograms are absolute numbers of colonies of a representative experiment



Supplemental Figure S2: Mammalian L1 elements alignment
(A) **Schematic representation of the genomic structure of the human L1 element**. Black boxes represent the 5' and 3' untranslated regions (5'UTR and 3' UTR); coloured boxes represent the two open reading frames (ORF1, ORF2). The white box represents the coding sequence of the reverse transcriptase (RT).

Sequence relationships between the chimpanzee (*pan troglodytes*), human (*Homo sapiens**), ourangutan (*Pongo pygmaeus*), gorilla (*Gorilla gorilla)*, macaque (*Macaca mulatta*), cow (*Bos taurus*), rabbit (*Oryctolagus cuniculus*), guinea pig (*Cavia*

porcellus), pig (*Sus scrofa*), cat (*Felis catus*), horse (*Equus caballus*), mouse (*mus musculus*), rat (*rattus norvegicus*), opossum (*Monodelphis domestica*), elephant (*Loxodonta africana*), L1_15 purified sequence and dog (*Canis familiaris*) L1-related sequences. The genomic sequences of the *L1 element* for the respective species were aligned using http//:ebi.ac.uk/clustalW.
(B) **Alignment of the L1 central region** show that the deduced consensus sequence (using http:weblogo.berkeley.edu/logo.cgi) for the PLZF-BS (ATGTAAA, red box) nucleotides are conserved to a lesser degree between mammalian species.

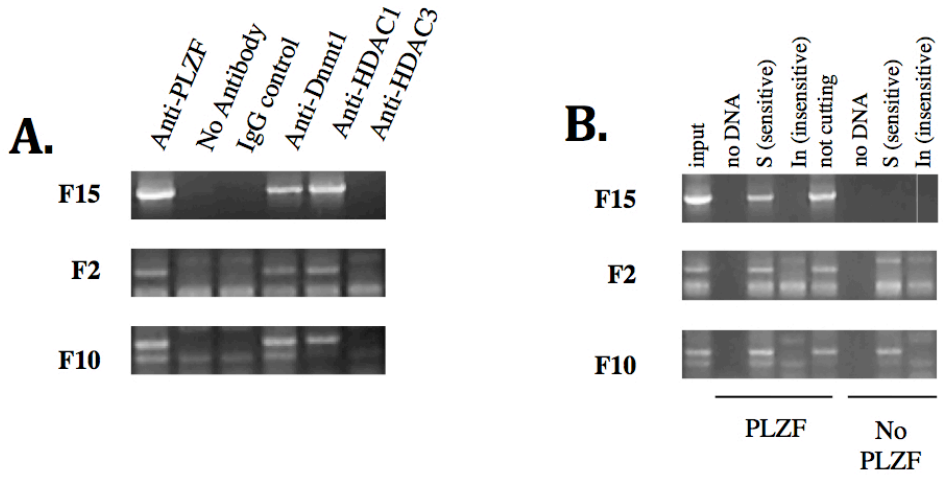


Supplemental Figure S3: PLZF and MBD1 recruitment to L1 sequences.

A. **Ectopic expression of PLZF protein is correlated to its L1 DNA binding activity.**

Expression vector for PLZF was transfected into non PLZF-expressing 293T cells. Levels of transiently expressed PLZF were monitored at various time points (0,2,5,12 and 24h) by Western blot analysis using an anti-PLZF (Anti-PLZF Western) antibody and compared to Actin expression (Anti-Actin Western). PLZF DNA binding activity was monitored by ChIP assay using the same antibody (Anti-PLZF ChIP) at the same time points (Anti-PLZF ChIP). As shown in the experiment, when PLZF proteins are detected by Western blot analysis (from 5h onward), PLZF DNA binding is also detected at the site of the PLZF BS localization (PCR5, L1 ORF2) and not at the L1 5'UTR (PCR1) indicating that PLZF interaction with L1 sequences is only through the PLZF BS characterized.

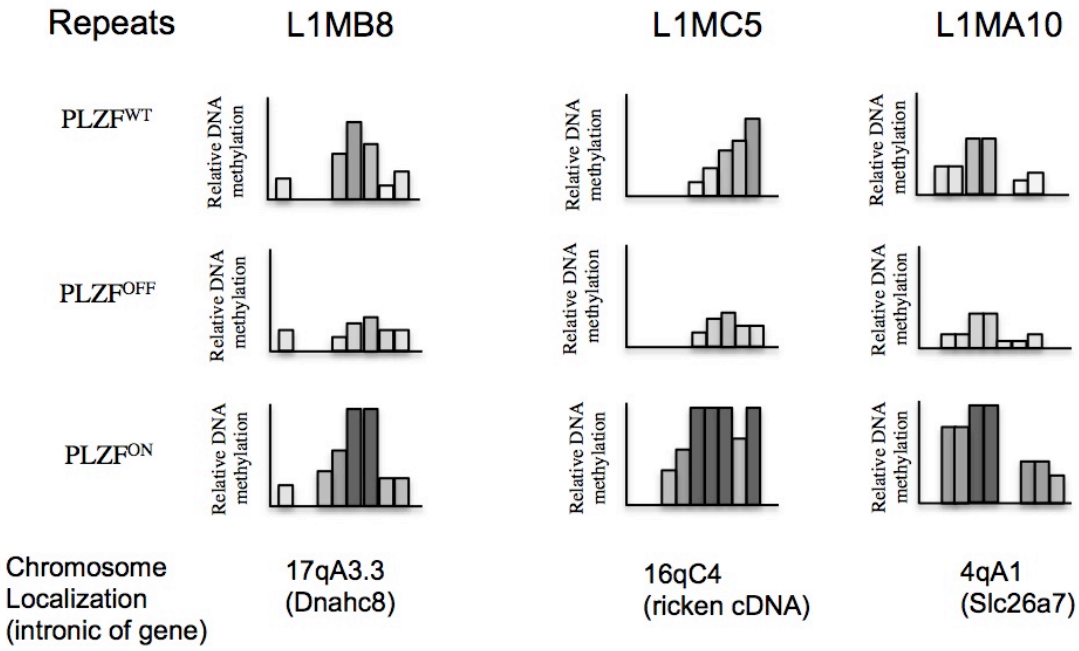
B. **MBD1 recruitment to L1 CpG island.** An antibody against MBD1 (Anti-MBD1) was used to assess the abundance of MBD1 proteins on L1 genomic DNA, in particular within the 5' UTR region containing a CpG island (PCR1, 5'UTR-CpG).



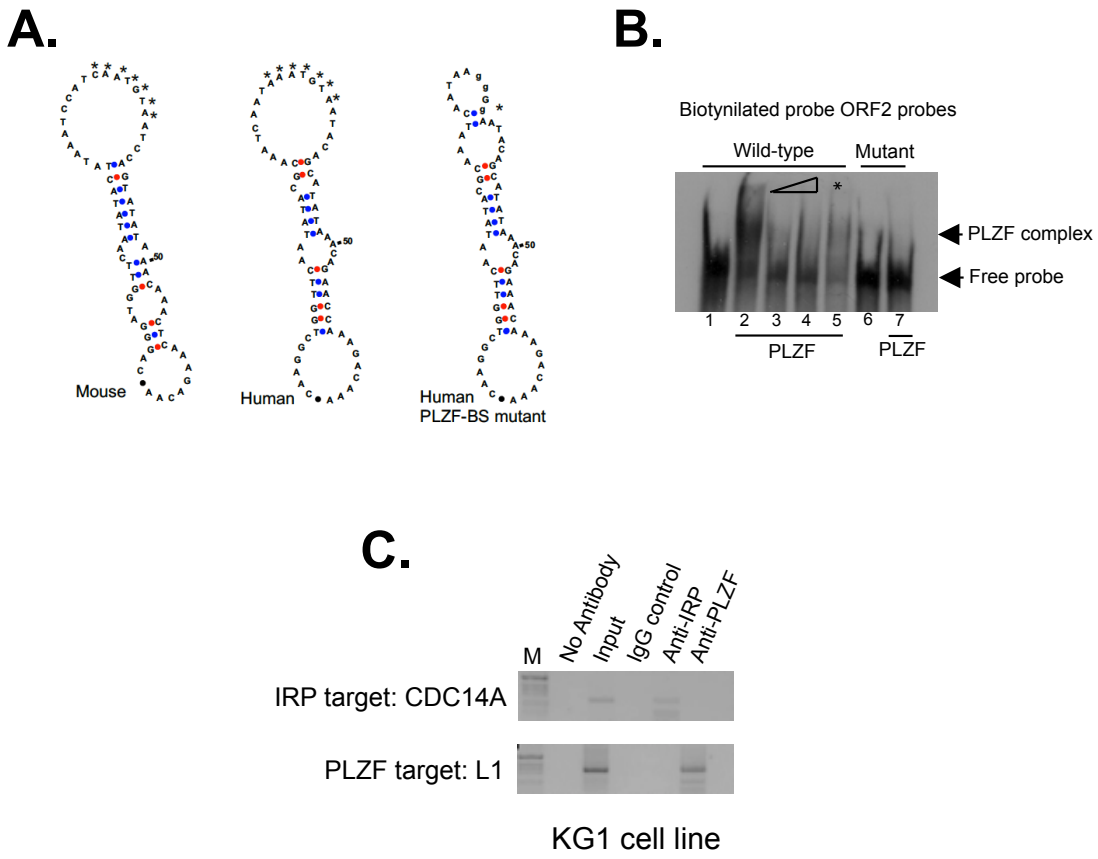
Supplemental Figure S4: Recruitment of PLZF protein complex to F2, F10 and F15 L1 ChIP-purified fragments.

A. **PLZF recruits Histone deacetylase and DNA methylase complex** to L1 genomic sequences while interacting with F2, F10 and F15 ChIP fragments. Chromatin prepared from PLZF-expressing haematopoietic KG1 cells was subjected to the ChIP procedure with anti-PLZF and were again precipitated using antibodies shown at the top of the image.

B. **Local methylation status of the L1 PLZF-target sequences** (F2, F10 and F15 fragments). Methylation status was determined by digesting the genomic DNA from 293T cells transfected or not with PLZF expression vector by sensitive (S) and insensitive (In) endonucleases (see Material and methods) behaving differently to DNA methylation. Non cutting endonuclease (XhoI) was also used (not cutting lane) as negative control.



Supplemental Figure S5: Differential DNA methylation of L1-containing DMRs. Examples of found DMRs. 1000bp genomic regions show differences in methylation status using the pipistrelle analysis tool. Hypermethylation (size and darker color of the histograms) is associated with $PLZF^{WT}$ and $PLZF^{ON}$ mutants in contrast to a decrease of DNA methylation when $PLZF^{OFF}$ mutant is expressed.



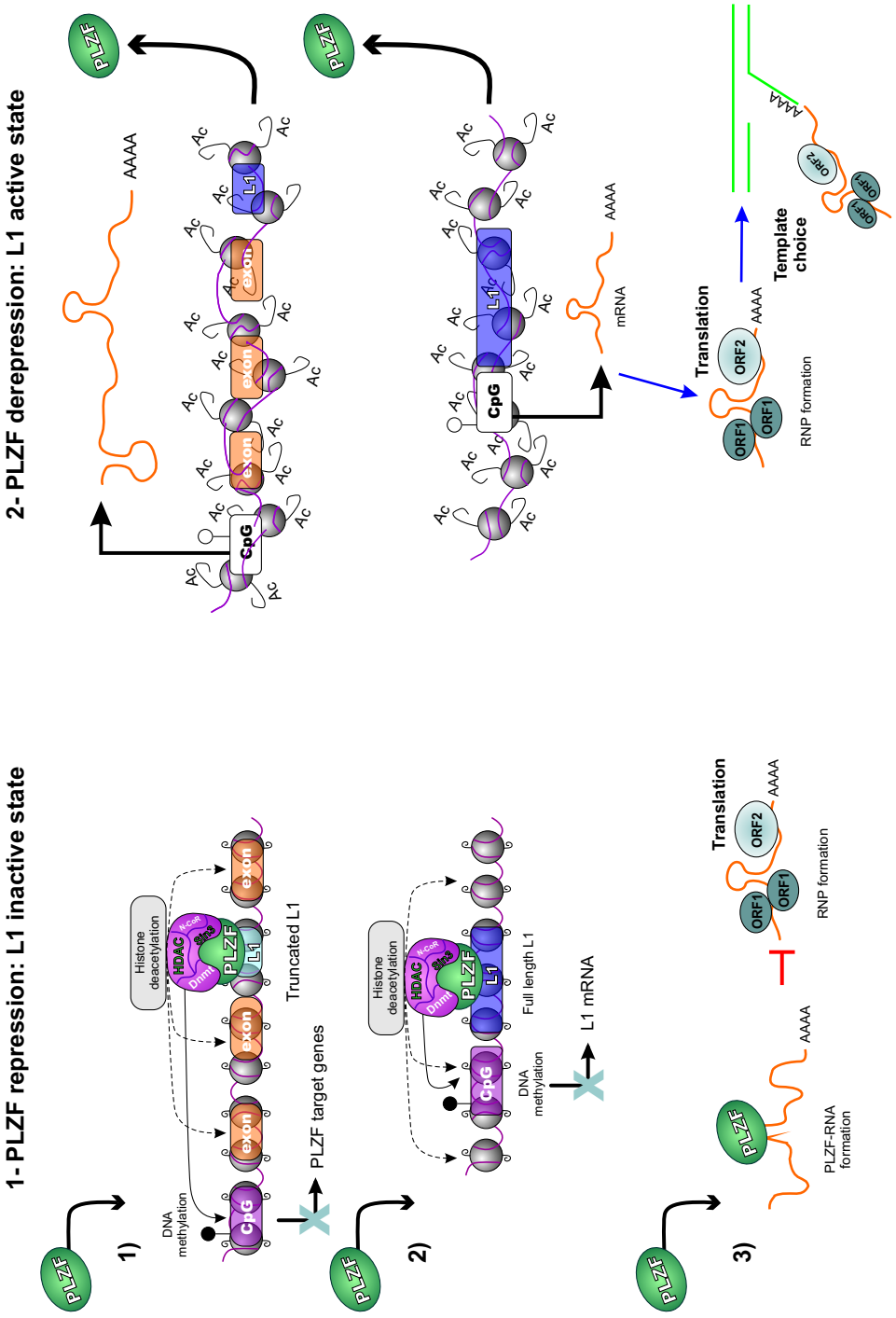
Supplemental Figure S6: PLZF interacts with L1 mRNAs and regulates their translation

A. **Schematic structure of the mRNA stemloops associated with the PLZF-BS found in human and mouse sequences**. The sequences were processed with mfold software (The Burnet institute, http://mfold.burnet.edu.au) and secondary structure are shown. Nucleotides marked with * are part of the PLZF-BS.

B. **L1 RNA shift assay**. RNA EMSA of wild type and mutant L1 probes were carried out in presence or absence of PLZF protein. Experiments were performed at least 3 times with a representative blot shown. L1 probes were biotinylated and incubated in binding buffer buffer with unprogrammed rabbit reticulocyte (lanes 1 and 6 for wild type or mutant probes) or with rabbit reticulocyte *in vitro* translated PLZF protein (lanes 2 and 7). Competition experiments with increasing amount of unlabelled L1 probes are shown in lanes 3 and 4 and supershift experiment with an anti-PLZF antibody (lane 5 *) show a loss of PLZF binding to L1 biotinylated probes compared to the PLZF/L1 probe interaction detected in lane 2. No PLZF binding was detected using a L1 mutated probe (lane 7).

C. **Validation of the RNA Pull Down in KG1 cell model (RNA-IP).**
Immunoprecipitation of mRNA protein complexes was used to confirm that PLZF was able to bind ORF2 transcript in cell line. As positive control of RNA pull down, IRP protein was used to pull down one of its specific mRNA target CDC14. mRNAs was pull down from KG1 cells according to (*3* and described in materials and methods) and RT-PCR was performed on the immunoprecipitated material using CDC14 specific primers. Un-related IgG and anti-PLZF antibodies were used as

negative control. The same procedure was used for ORF2 mRNA pull down by PLZF using anti-IRP and IgG as control antibodies and PCR was performed with specific ORF2 primers. The presence of an amplicon revealed an interaction between the protein of interest and its mRNA target.



Supplemental Figure S7: Dual regulation of L1 retrotransposon by PLZF

Interaction of PLZF protein to truncated (1) or full length (2) L1 retrotransposon. Under PLZF interaction with DNA genomic sequences, PLZF recruits histone deacetylase (HDAC) and DNA methylase (DNMT) activities in a protein complex including co-repressors (N-CoR and SIN3). Heterochromatin is formed and transcriptional repression occurs unabling expression of L1 mRNAs or surrounding genes (1-PLZF repression: L1 inactive state). Histones are deacetylated and DNA is methylated at CpG islands. PLZF can also interact with L1 mRNAs through a hairpin loop and block the translation of L1 mRNAs to inhibit the formation of L1 RNA particle (RNP), thus stop new L1 insertion events (3). When PLZF is, via specific deacetylation or inactivated in response to cell stress, released from heterochromatin, histone acetylation (Ac) and DNA demethylation (White CpG box) occur, leading to formation of open chromatin, allowing active transcription (2- PLZF derepression: L1 active state) and production of mRNAs from nearby genes and L1 retrotransposon.

Supplemental tables:

Table I: DMR containing L1 sequences

DMR	**Chromosome**	**L1**	**gene position**	**L1 (size bp)**	**other repeats**
2	1 (qB)	L1M2	mKIAA112 (intronic)	379	-
3	1 (qC1.1)	L1M5	MyoIB (intronic)	111	-
6	1 (qE1.2)	L1Md_F2	mEST AA674321 (intronic)	1203	M1_Mm (Alu)
7	1 (qH2.3)	L1MB7	mEST AK080019 (intronic)	963	(TATG)n (simple repeat)
11	2 (qA3)	Lx8	mEST AY101367 (intronic)	151	RLTR20B4_MM (LTR)
15	2 (qC1.1)	L1Md_F2	intergenic	6582*	-
17	2 (qE2)	L1MB7	mEST BU842339 (intronic)	254	B2 (sine)
18	2 (qE5)	HAL1	Bub1b (intronic)	382	-
21	2 (qE5)	Lx2	mEST BU555796 (intronic)	74/1713	B1_Mus2 (Alu)
23	2 (qF1)	L1MC	Gm14005 (intronic)	396	RSINE1 (sine)
26	3 (qA1)	L1MdA	intergenic	1714	-
27	4 (qG1)	L1ME2z	intergenic	218/261	RSINE1 (sine)
28	4 (qE1)	L1_Mur2	vpsd13d (intronic)	181	MYSERV6 (LTR)
34	4 (qE1)	L1_Mus1	HMGB1 (intronic)	67/347	B2_Mm2 (sine)
35	4 (qE1)	L1_Mur2	mEST CA533858 (intronic)	1403	MT2B (LTR)
36	5 (qF)	L2	intergenic	227	B4 (sine)
44	5 (qG2)	HAL1-3A, L1M4C	sdk1 (intronic)	83/235	-
47	6 (qG3)	L1MA4A	Sox5 (3')	879/410	-
48	7 (qA3)	MusHAL1	Plaur (intronic)	1754	-
53	7 (qA3)	Lx4	CYP2b13 (intronic)	1697	-
56	7 (qA3)	L1_Mus2, Lx4B	CYP2b13 (3')	518,794	-

58	7 (qB2)	L1MA4	mRNA AK181724 (intronic)	949	-
59	7 (qB2)	L1Mus4	mEST CA464463 (intronic)	747	-
63	7 (qC)	Lx8	Lrrk1 (intronic)	533	-
67	8 (qC4)	Lx9	mESTCJ046943 (intronic)	504	-
73	8 (qA1.1)	L1M18	ATP11a (intronic)	187	RSINE1 (sine)
75	9 (qA5.3)	L1MA5	Gria4 (intronic)	1953	-
79	9 (qA5.3)	L1MB3	intergenic	662	-
80	9 (qA5.3)	L1_Mus3	intergenic	6657*	-
83	9 (qF1)	L1M3	Cpne4 (intronic)	453	-
89	9 (qF3)	Lx7	intergenic	1489	B4 (sine)
91	10 (qB2)	L1MEf	NSMUST0000009932	360	B1_Mus1 (sine)
95	10 (qC2)	L1_Mur2	Anks1b (intronic)	2654	B1_Mus1 (sine)
96	10 (qC2)	HAL1	Anks1b (intronic)	67	B1F1 (Alu)
97	10 (qC2)	Lx5	Anks1b (intronic)	349	-
98	10 (qC2)	HAL1-3A	Tmcc3 (intronic)	65	B4a (sine)
99	10 (qD1)	L1Mb7	Poc1b (intronic)	987	-
100	11 (qB1.3)	Lx7, Lx8	intergenic	485,255	(CA)n (simple repeat)
105	11 (qB1.3)	L1_Mur1, L1MC1	Slc25a21 (intronic)	96,77	B1_Mm (sine)
107	12 (qC1)	Lx9	intergenic	744	B1_Mus1 (Alu)
110	13 (qC3)	L1MC1	Rasa1 (intronic)	794	-
116	13 (qC3)	Lx5,L1MdF2	Naip 6 and Naip 7 (3')	1153,587	-
118	14 (qA)	Lx	Acox2 (intronic)	2979	B1_Mus1 (Alu)
125	14 (qC1)	L1MB3	mEST BY716586 (intronic)	315	Rsine1 (sine)
128	14 (qD1)	L1M2	Ebp1 (intronic)	1806	-
130	15 (qB3.1)	L1Md_F2, L1MC4, Lx7	Sem5a (intronic)	2366,685,6293*	-
132	16 (qB3)	L1Md_F2	intergenic	2083	-
137	17 (qA3.3)	L2A	Grm4 (intronic)	212	B1_Mur4 (sine)
150	17 (qA3.3)	Lx7	btbd9 (intronic)	471	-
151	17 (qA3.3)	L1_Mus1	Dnahc8 (intronic)	481	REMER17B (LTR)
158	17 (qA3.3)	Lx6	Dnahc8 (intronic)	653	-
160	17 (qA3.3)	Lx9	Dnahc8 (intronic)	182	-
165	17 (qA3.3)	L1Ma6	Dnahc8 (intronic)	297	-
167	17 (qA3.3)	Lx6	Dnahc8 (intronic)	161	-
169	17 (qA3.3)	L1MB5	Dnahc8 (intronic)	189	B1F (Alu)
173	17 (qB1)	L1_Mus1	H2-t3 (3')	382	-
174	17 (qB3)	L1_Mus1	Cyp39a1 (3')	366	-
177	18 (qA1)	L1_Mus1	MEST CD354501 (intronic)	313	orrD1 (LTR)
179	18 (qA2)	Lx8	Asx13 (intronic)	178	-
182	18 (qE3)	L1MA4	Loxhd1 (intronic)	6596*	-
188	19 (qD3)	L1M5,Lx9	intergenic	156,106	(TCCA)n (simple repeat)

* full length L1

Table II: ChIP-purified L1 targets

Clone	Chromsome	L1	Gene (position)	ChIP purified fragment size (bp)	Other repeats
F2	10 (q25.3)	L1MA6int*/L1ME3B	ATRNL1 (intronic)	2206	AluYb8 (sine)
F3	22 (q12.1)	L1M3	ADRBK1 (last intron)	1402	AluSz/AluY (sine), (T)n (simple repeat)
F6	6 (q21)	L1PA4	POPDC3 (last intron)	675	-
F7	8 (q24.13)	L1PA16,L1P2,L1Me3A	BC952578 (3'UTR)	3315	AluSg/AluSz6 (sine)
F10	10 (q31.1)	L1PA2,L1MA5int	BU168212 (intronic)	2171	AluSz (sine)
F11	5 (p14.3)	L1PA5	CDH18 (last intron)	3520	AluSx1/AluJo (sine)
F12	12 (q12.2)	L1MCA4	SEC14L2 (intronic)	2531	MIR3/AluJo (sine)
F13	19 (p13.11)	L2a	KCNN1 (intronic)	696	-
F14	12 (q24.21)	L1M5*,L1MA8	L13705 (intronic)	2959	HAL1b, THE1C (ERVL_MaLR)
F15	10 (q23.1)	L1PA3*, LIPB4 int	intergenic	1260	L1M2
F16	1 (p34.3)	L1ME1*	intergenic	4012	MSTB (ERVL_MaLR)
F18	13 (q22.33)	L1ME3B	MZT1 (intronic)	1387	MLT1Bint (ERVL_MaLR), Alu Sq2 (sine)
F21	3 (p13)	L1PA3	intergenic	1634	MLT1J (ERVL_MaLR)
F23	10 (q25.1)	L1ME3A	SLC9A40 (intronic)	383	-
F25	5 (q15)	L1PA13	intergenic	2263	-
F28	3 (q25.32)	L1PA13	intergenic	1338	AluSx (sine)
F29	9 (p21.1)	L2a	NDUFB6 (intronic)	788	L1MC4
F30	14 (q13.2)	L2C	PSMA6 (intronic)	649	AluS2 (Sine)

* full length L1
int: interrupted