Suppl. Table 1. Relationship of *DLEC1* methylation and clinicopathological parameters of esophageal cancer patients from TCGA cohort

Clinicopathological features	Esophageal cancer cohort (TCGA)			
	Number (n=185）	methylated	unmethylated	*p*-values
Age				
≤50	28	27 (96.4%)	1 (3.60%)	
>50<65	80	72 (90.0%)	8 (10.0%)	**≤0.001**
≥65	77	66 (85.7%)	11 (14.3%)	
Gender				
Female	27	26 (96.3%)	1 (3.70%)	0.68
Male	158	139 (88.0%)	19 (12.0%)	
Histologic_grade				
g1	19	18 (94.7%)	1 (5.30%)	
g2	77	71 (92.2%)	6 (7.80%)	**0.03**
g3	49	41 (83.7%)	8 (16.3%)	
gx	40	35 (87.5%)	5 (12.5%)	
Pathologic_stage				
Stage i	19	15 (78.9%)	4 (21.1%)	
Stage ii	79	76 (96.2%)	3 (3.80%)	**0.001**
Stage iii	56	49 (87.5%)	7 (12.5%)	
Stage iv	9	9 (100%)	0	
Pathologic_m				
0	136	125 (91.9%)	11 (8.10%)	0.42
1	9	9 (100%)	0	
Pathologic_n				
0	77	73 (94.8%)	4 (5.20%)	
1	69	59 (85.5%)	10 (14.5%)	0.39
2	12	12 (100%)	0	
3	8	7 (87.5%)	1 (12.5%)	
Pathologic_t				
0	1	1 (100%)	0	
1	31	25 (80.6%)	6 (19.4%)	
2	43	42 (97.7%)	1 (2.30%)	0.3
3	88	80 (90.9%)	8 (9.10%)	
4	5	5 (100%)	0	

Suppl. Table 2. Relationship of *DLEC1* expression and clinicopathological parameters of lung adenocarcinoma patients from TCGA cohort

Clinicopathological features	Number (n=576）	High expression	Low expression	*p*-values
Age				
≤50	41	12 (29.3%)	23 (56.1%)	
>50<65	201	74 (36.8%)	83 (41.3%)	**0.02**
≥65	296	130 (43.9%)	109 (36.8%)	
Gender				
Female	311	135 (43.4%)	108 (34.7%)	**0.008**
Male	265	95 (35.8%)	122 (46.0%)	
Residual_tumor				
r0	371	150 (40.4%)	150 (40.4%)	
r1	15	4 (26.7%)	7 (46.7%)	**0.046**
r2	4	2 (50.0%)	2 (50.0%)	
rx	27	16 (59.3%)	4 (14.8%)	
Pathologic_stage				
Stage i	307	136 (44.3%)	105 (34.2%)	
Stage ii	135	48 (35.6%)	60 (44.4%)	**<0.001**
Stage iii	97	33 (34.0%)	45 (46.4%)	
Stage iv	28	10 (35.7%)	17 (60.7%)	
Pathologic_m				
0	387	149 (38.5%)	161 (41.6%)	0.09
1	27	9 (33.3%)	17 (63.0%)	
Pathologic_n				
0	363	154 (42.4%)	136 (37.5%)	
1	108	38 (35.2%)	49 (45.4%)	**0.03**
2	87	29 (33.3%)	43 (49.4%)	
3	2	0	0	
Pathologic_t				
0	0	0	0	
1	189	92 (48.7%)	57 (30.2%)	
2	315	109 (34.6%)	144 (45.7%)	**<0.001**
3	49	18 (36.7%)	19 (38.8%)	
4	20	9 (45.0%)	9 (45.0%)	

Suppl. Table 3. Methylation status of 3p21-22 TSGs (with genome positions) in ESCC

	Sample	***PLCD1*** (~38.00 Mb)	***DLEC1*** (~38.03 Mb)	***RASSF1A*** (~50.32 Mb)	***ZMYND10*** (~50.34 Mb)
ESCC cell lines	NE1	u	u	u	u
	NE3	u	u	u	u
	EC1	u	m	m+u	u
	EC18	u	m	m+u	u
	EC109	u	m	m+u	m
	HKESC1	u	u	m+u	m+u
	HKESC2	u	u	m+u	m+u
	HKESC3	m	m	u	m+u
	SLMT1	u	m	u	u
	KYSE30	m	m	m	m+(u)
	KYSE70	m+u	(m)	m+(u)	m+u
	KYSE140	u	m	u	m+(u)
	KYSE150	m	m	u	m+(u)
	KYSE180	(m)+u	m	u	u
	KYSE270	u	m	m	u
	KYSE410	m+u	u	m+u	m+u
	KYSE450	u	m	m+u	m+u
	KYSE510	m+(u)	m	m+u	m+u
	KYSE520	u	m	u	u
	Total	m: 7/17 (41%)	m: 14/17 (82%)	m: 11/17 (65%)	m: 11/17 (65%)
ESCC primary tumors (Chinese) (Cohort I)	ESCa1T	u	m	u	u
	ESCa2T	u	u	u	u
	ESCa3T	u	m	u	m
	ESCa4T	u	m	u	(m)+u
	ESCa5T	u	m	u	u
	ESCa6T	m	m	u	m
	ESCa7T	u	u	u	u
	ESCa8T	m	m	u	m
	ESCa9T	u	m	u	u
	ESCa10T	u	m	m	u
	ESCa11T	u	m	u	u
	ESCa12T	u	u	u	u
	ESCa13T	u	m	u	u
	ESCa14T	u	u	u	u
	ESCa15T	u	m	u	(m)+u
	ESCa16T	u	u	u	(m)+u
	ESCa17T	u	u	u	m
	ESCa18T	u	u	u	u
	ESCa19T	u	m	u	u
	ESCa20T	u	u	u	m
	ESCa21T	u	m	u	u
	ESCa22T	u	u	u	m
	ESCa23T	u	u	u	u
	ESCa24T	m	m	m	m
	ESCa25T	u	m	u	u
	ESCa26T	u	m	m	u
	ESCa27T	m	u	u	u
	ESCa28T	u	m	u	u
	ESCa29T	u	m	(u)	u
	ESCa30T	m	m	m	m
	ESCa31T	u	m	u	u
	ESCa32T	u	u	m	m
	ESCa33T	u	m	m	u
	ESCa34T	u	m	(m)+u	u
	ESCa35T	u	m	u	
	Total	m: 5/35 (14%)	m: 22/35 (66%)	m: 7/35 (20%)	m: 12/34 (35%)

Suppl. Table 4. Primers used in this study

PCR	Gene or fragment	Primer	Sequence (5'→3')	Location	Product size (bp)
RT-PCR	*DLEC1*	*DLEC1A*	ttcctccctcgcctactc	Exon 1	309
		DLEC1B	aaactcatccagccgctg	Exon 2	
Alternative splicing	*DLEC1*	*DLEC1y1*	caaagaagccagcaccgata	Exon 5	422
		DLEC1y2	gcagtaaccacagtccaaca	Exon 9	
		DLEC1N	ccgggacatgctatattagt	Exon 10	541
		DLEC1G	aggctctgggacttcctc	Exon 13	
Deletion	*DLEC1*	*DLEC1A*	ttcctccctcgcctactc	Exon 1	385
		DLEC1C	caactgcagccccagatc	Intron 1	
Cloning	Promoter	*DLEC1F1*	tgcctcttgcctctcctg	Promoter	1046 (F1/R) 313 (F2/R)
		DLEC1F2	tcagcaatcagcacagacc	Promoter	
		DLEC1R	aaccgagacgccgctaac	Exon 1	
	ORF	Fragment I	gccgccaccatggagaccagggc	Exon 1	~1.1 (kb)
			gtgaaaaacccaattggtgg	Exon 6	
		Fragment II	agtgtttctagctaagccac	Exon 6	~1.2 (kb)
			gagggcatatggctctaag	Exon 14	
		Fragment III	cttagagccatatgccctc	Exon 14	~1.4 (kb)
			gccatgtgcactgggatg	Exon 25	
		Fragment IV	catcccagtgcacatggc	Exon 25	~1.6 (kb)
			gctcgagcggagcctcaggg	Exon 36	
ChIP assay	*DLEC1*	*DLEC1S*	cttgctcaccggcgtctt	Exon 1	241 (s/c) 166 (s/b)
		DLEC1C	caactgcagccccagatc	Intron 1	
		DLEC1B	aaactcatccagccgctg	Exon 2	
		DLEC1A	ttcctccctcgcctactc	Exon 1	162
		DLEC1R2	aagacgccggtgagcaag	Exon 1	
	ACTIN	*ACTINPP*	ctgtgttggcgtacaggtc	Exon	181
		ACTININ	gtggagactgtctcccgg	Intron	
MSP	*DLEC1*	*m1*	gtttcgtagttcggtttcgtc	Exon 1	107
		m2	cgaaatatcttaaatacgcaacg	Exon 1	
		u1	tagttttgtagtttggttttgtt	Exon 1	110
		u2	acaaaatatcttaaatacacaaca	Exon 1	
BGS	*DLEC1*	*BGS1*	gaagatataaatgtttataatgatt	Promoter	597
		BGS4	aactacaaccccaaatcctaa	Intron 1	

Supplementary material and methods

Array-CGH (aCGH)

Whole-genome arrays (1-Mb resolution) and aCGH was performed and analyzed as previously [1]. Hybridized slides were scanned and analyzed with the GenePixPro 4.0 image analysis software.

Semi-quantitative RT-PCR and quantitative RT-PCR analysis

RNA was reverse-transcribed using MuLV reverse transcriptase (GeneAmp RNA PCR kit, Applied Biosystems). RT-PCR was performed as described previously using *GAPDH* as a control [2]. Primers used were listed in Suppl. Table 4.

Multiplex differential DNA-PCR

Multiplex differential genomic DNA-PCR was performed using primer pair *DLEC1*A/C for 35 cycles (annealing temperature 58 ^{o}C) with AmpliTaq Gold, using 0.1 ug of DNA per 12.5 ul PCR reaction [3]. *GAPDH* and *DLEC1* were employed to detect *DLEC1* deletion in a region spanning exon 1 and intron 1.

Promoter activity assay

Different regions of the *DLEC1* promoter were cloned by PCR from normal human placenta DNA (sigma-Aldrich, USA). PCR was carried out with a high-fidelity Platinum PfX DNA polymerase (Life Technologies, USA) with 10% DMSO. The sequences and orientations of the cloned fragments were confirmed by sequencing. The longest and shorter fragments were amplified from primer pairs *DLEC1*F1-R and *DLEC1*F2-R respectively (Suppl. Table 4). Restriction enzyme Bst XI was employed to digest the longest fragment to produce an intermediate one. These fragments were then linked to pGL2-Enhancer Vector (Promega) to generate p(-295)*DLEC1*EN, p(-685)*DLEC1*EN and p(-1021)*DLEC1*EN. Promoter activities of these fragments were assessed by transient transfection in CNE1 and CNE2 cell lines using Transfast (Promega).

Analysis of alternative splicing

*DLEC1*y1, *DLEC1*y2, *DLEC1*N and *DLEC1*G (Suppl. Table 4) were used to generate different splicing fragments. Desired PCR products were purified using QIAex II (Qiagen). Purified PCR amplicons were sequenced and aligned with *DLEC1* mRNA sequence using the “bl2seq” program (www.ncbi.nlm.nih.gov/blast).

Chromatin immunoprecipitation (ChIP) assay

ChIP assay was performed as described previously. Primers used were listed in Suppl. Table 4. Antibody to acetylated histone H4 (Upstate Biotechnology) was used to precipitate cross-linked chromatin. *ACTIN* was employed as a control for normalization of each PCR product.

Online analysis of TCGA data sets

Clinical, mRNA expression and genome-wide DNA methylation data of 185 esophageal cancer, 585 lung adenocarcinoma and 504 lung squamous cell carcinoma patients was obtained from public databases of The Cancer Genome Atlas (TCGA). Raw datasets were downloaded from TCGA Data Portal (http://gdac.broadinstitute.org/) and analyzed. "Level 3" methylation data (Illumina Infinium Human DNA Methylation 450 platform) was retrieved from Johns Hopkins University and University of Southern California. mRNAseq expression data (level 3, normalized gene expression data, Illumina HiSeq 2000 platform) was retrieved from University of North Carolina or Canada's Michael Smith Genome Sciences Centre. Genomic mutation data (Illumina Genome Analyzer platform) was retrieved from Washington University School of Medicine Proteomics (for esophageal cancer) and Broad Institute of MIT and Harvard (for lung cancer) and analyzed.

Wound-healing assay

Cell motility was assessed using a scratch wound-healing assay. Cells transient transfected with DLEC1 were cultured in 6-well plates until confluent. A single scratch was produced in the cell layer using a sterile tip. After incubation for 24 and 48 hours, cells were photographed under a phase contrast microscope. The experiments were performed in triplicates.

Immunofluorescence

Cells grown on coverslips were stained by indirect immunofluorescence as described previously [2, 4]. Briefly, cells were incubated with primary antibodies against DLEC1, E-cadherin, or Vimentin and then incubated with Alexa Fluor 594-conjugated secondary antibody against mouse IgG (A11062) (Invitrogen Molecular Probes, Carlsbad, CA), or FITC-conjugated secondary antibody against rabbit IgG (F0205) (DAKO, Denmark). To analyze the effects of DLEC1 on actin stress fiber formation, cells were serum starved for 24 h before incubation in medium-containing 5% fetal bovine serum. After 1 h, cells were fixed and stained by Rhodamine-labeled phalloidin (Invitrogen Molecular Probes). Cells were then counterstained with DAPI

and imaged with an Olympus BX51 microscope (Olympus Corporation, Tokyo, Japan).

Dual-luciferase reporter assay

The promoter activities were determined by luciferase reporter assays. Luciferase reporters of several key signaling pathways, including NF-κB-luc, AP-1-luc, SRE-luc, STATs-bs-luc, TopFlash-luc, and PAI-luc were used to examine signaling pathway regulated by DLEC1. Cells were transiently co-transfected with DLEC1 expression vector and phRL-TK (the luciferase reporters). *DLEC1*-promoter luciferase reporters were co-transfected with expression vector encoding wild-type p53. After 48 h, cells were lysed and luciferase activities were measured using Dual-Luciferase® Reporter Assay System (Promega, Madison, WI). To normalize transfection efficiency, phRL-TK luciferase activities were measured as an internal control. At least three independent experiments were performed, with each repeated in triplicates.

Supplementary figure legends

Suppl. figure 1. (A) *DLEC1* resides at 3p21-22, a locus with hemizygous deletion detected by 1-Mb array-CGH in ESCC cell lines. (B) The abundance of *DLEC1* gene copies relative to GAPDH was determined by multiplex differential genomic DNA-PCR in ESCC cell lines, with homozygous deletion of *DLEC1* detected in one. NE1, NE3 and normal PBMCs were used as normal controls. The position of *DLEC1* primers used for deletion examination is shown. ESCC, esophageal squamous cell carcinoma.

Suppl. figure 2. Alterations of *DLEC1* DNA copy numbers in ESCC and lung carcinomas, analyzed through Oncomine database. ESCC, esophageal squamous cell; Ca, carcinoma; SCLC, squamous cell lung carcinoma; Lung adeno, lung adenocarcinoma.

Suppl. figure 3. Profiling of *DLEC1* mutations in esophageal and other carcinomas. Mutation profile was analyzed using TCGA dataset. No mutation was detected in ESCC. ESCC, esophageal squamous cell; ESAD, esophageal adenocarcinoma; Lung adeno, lung adenocarcinoma; LSCC, lung squamous cell carcinoma; NSCLC, non-small cell lung cancer; SCLC, squamous cell lung carcinoma; NPC, nasopharyngeal carcinoma.

Suppl. figure 4. Defining the functional *DLEC1* promoter. (A) Diagram of *DLEC1* promoter and its CpG island. CpG sites are numbered according to the order marked in BGS, with MSP primer sites and BGS region indicated. Potential binding sites for p53, c-Myb, HSF, E2F and Sp1 are labeled. (B) Luciferase activity assay of different promoter constructs in carcinoma cell lines. Luc, luciferase gene; enh, enhancer. The shortest fragment (+18 to -295, located in the CGI) could function as a core promoter to drive gene expression. (C) Promoter activities after co-transfection of different promoter constructs with p53 plasmids. Wild-type p53 upregulated *DLEC1* promoter activity. (D-E) Box plot graphs for the distribution of *DLEC1* methylation and expression levels in esophageal and lung cancer patients from TCGA database.

Suppl. figure 5. Promoter methylation and loss of expression of *DLEC1* in NPC. (A) *DLEC1* is methylated and repressed in NPC cell lines. M: methylated; U: unmethylated. (B) Expression of *DLEC1* was restored or increased by 5-aza-dC (Aza) treatment in NPC cells, along with increased unmethylated promoter alleles. (C) Detailed BGS analysis of *DLEC1* methylation in representative NPC cell lines and primary tumors. NPx: normal nasopharynx.

Suppl. figure 6. Regulation of *DLEC1* expression by histone H4 acetylation. (A) Locations of primers used in ChIP assay in the *DLEC1* CGI. (B) TSA-treated Lung carcinoma cell line H1299 showed moderately induced *DLEC1* expression and concomitantly increased acetylation of histone H4 at the endogenous *DLEC1* CGI; while the transfected carcinoma cells showed high level of *DLEC1* expression but no increment of H4 acetylation at the endogenous *DLEC1* CGI at all.

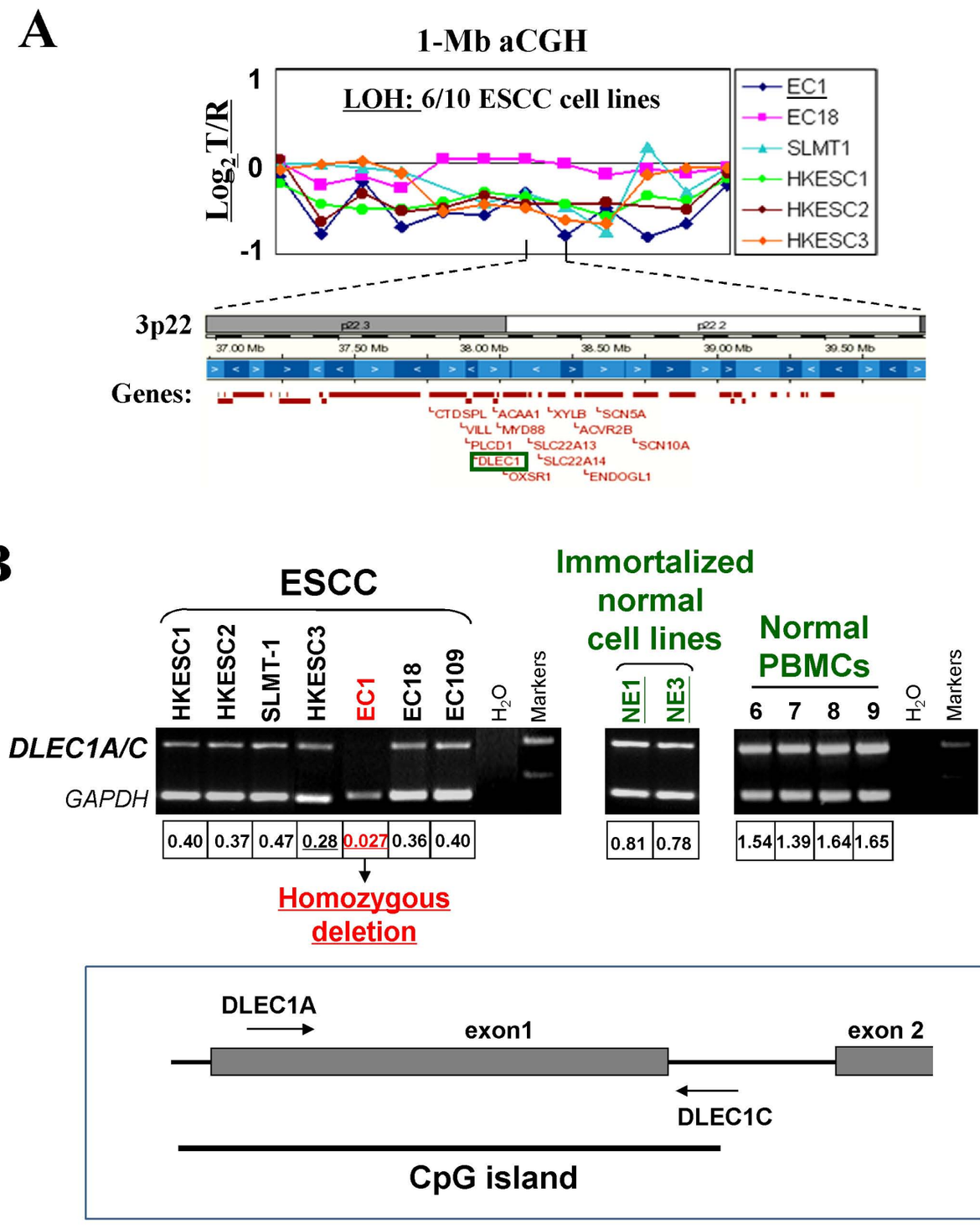
Suppl. figure 7 Analysis of *DLEC1* alternative splicings. (A) Detailed diagrams of splicing forms. (B) Different splicings of *DLEC1* were detected in multiple cell lines, normal adult and fetal tissues, as well as PBMCs.

Suppl. figure 8 (A) Representative colony formation assay of *DLEC1*-transfected carcinoma cells. DLEC1 expression was measured by Western blot. (B) Wound-healing assay of carcinoma cells transfected with either vector or DLEC1. Pictures were taken at 0, 24 or 36 h. Right panel: width of remaining open wound measured in relation to time 0 h separation.

References:

1. Ying J, Li H, Seng TJ, Langford C, Srivastava G, Tsao SW, et al. Functional epigenetics identifies a protocadherin PCDH10 as a candidate tumor suppressor for nasopharyngeal, esophageal and multiple other carcinomas with frequent methylation. Oncogene. 2006; 25: 1070-80.
2. Li L, Ying J, Tong X, Zhong L, Su X, Xiang T, et al. Epigenetic identification of receptor tyrosine kinase-like orphan receptor 2 as a functional tumor suppressor inhibiting beta-catenin and AKT signaling but frequently methylated in common carcinomas. Cell Mol Life Sci. 2014; 71: 2179-92.
3. Qiu GH, Tan LK, Loh KS, Lim CY, Srivastava G, Tsai ST, et al. The candidate tumor suppressor gene BLU, located at the commonly deleted region 3p21.3, is an E2F-regulated, stress-responsive gene and inactivated by both epigenetic and genetic mechanisms in nasopharyngeal carcinoma. Oncogene. 2004; 23: 4793-806.
4. Hu XT, Zhang FB, Fan YC, Shu XS, Wong AH, Zhou W, et al. Phospholipase C delta 1 is a novel 3p22.3 tumor suppressor involved in cytoskeleton organization, with its epigenetic silencing correlated with high-stage gastric cancer. Oncogene. 2009; 28: 2466-75.

Suppl. Fig1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Log2 T/R
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-1
EC1
EC18
SLMT1
HKESC1
HKESC2
HKESC3
3p22
p22.3
p22.2
37.00 Mb
37.50 Mb
38.00 Mb
38.50 Mb
39.00 Mb
39.50 Mb
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VILL MYD88 ACVR2B
PLCD1 SLC22A13 SCN10A
DLEC1 SLC22A14
OXSR1 ENDOGL1
B
ESCC
Immortalized normal cell lines
Normal PBMCs
HKESC1
HKESC2
SLMT-1
HKESC3
EC1
EC18
EC109
H2O
Markers
NE1
NE3
6 7 8 9
H2O
Markers
DLEC1A/C
GAPDH
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0.81 0.78
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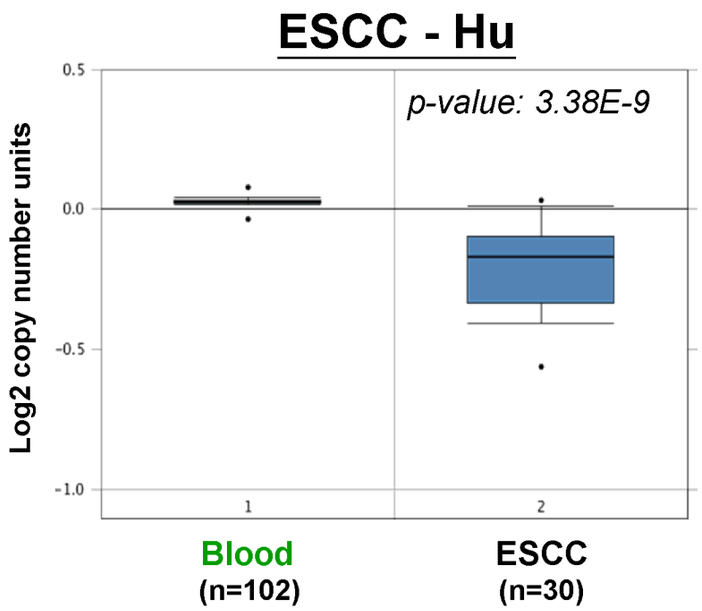
exon1
exon 2
DLEC1C
CpG island

Suppl. Fig2


ESCC - Hu
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-1.0
1
2
Blood
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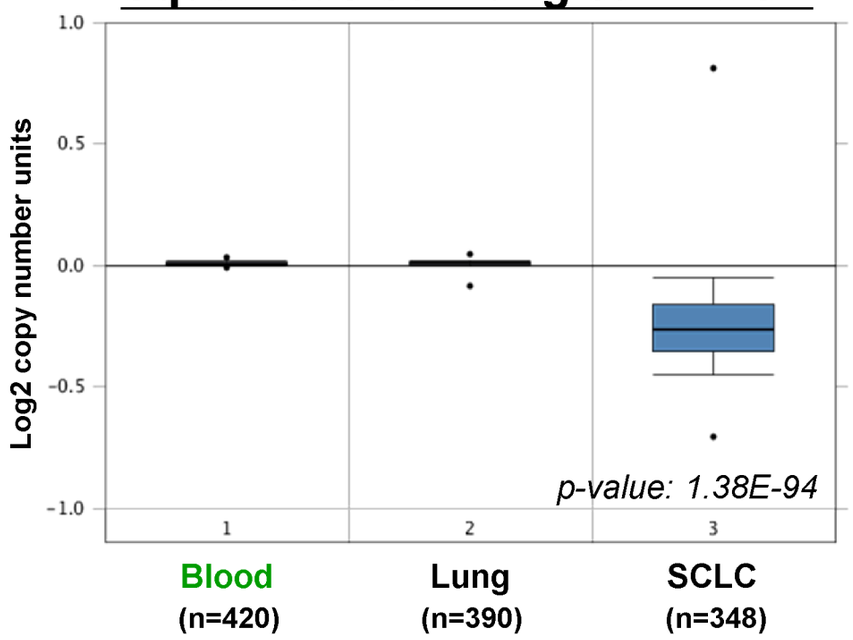
ESCC
(n=30)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2
3
Blood
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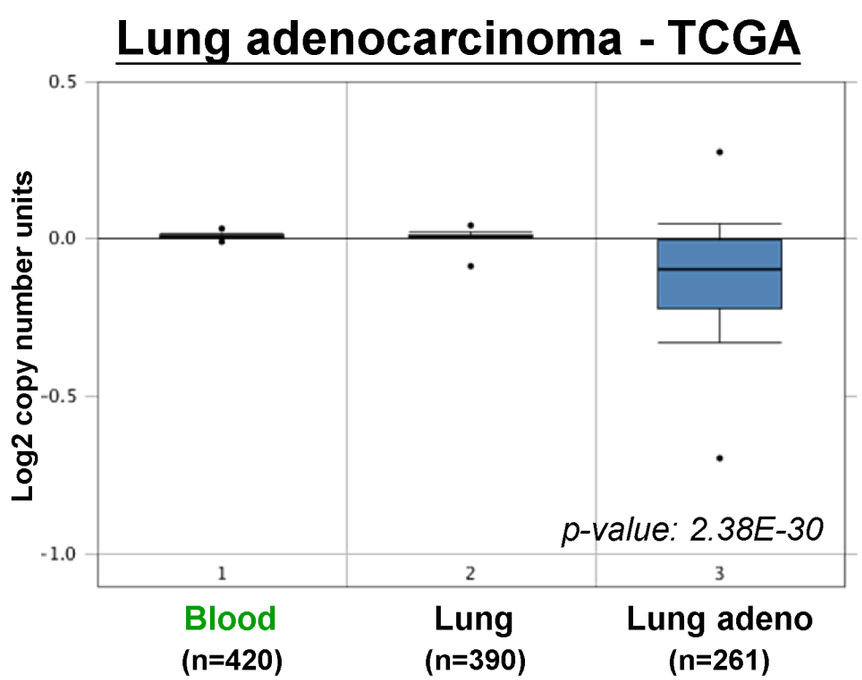
Lung
(n=390)
SCLC
(n=348)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-1.0
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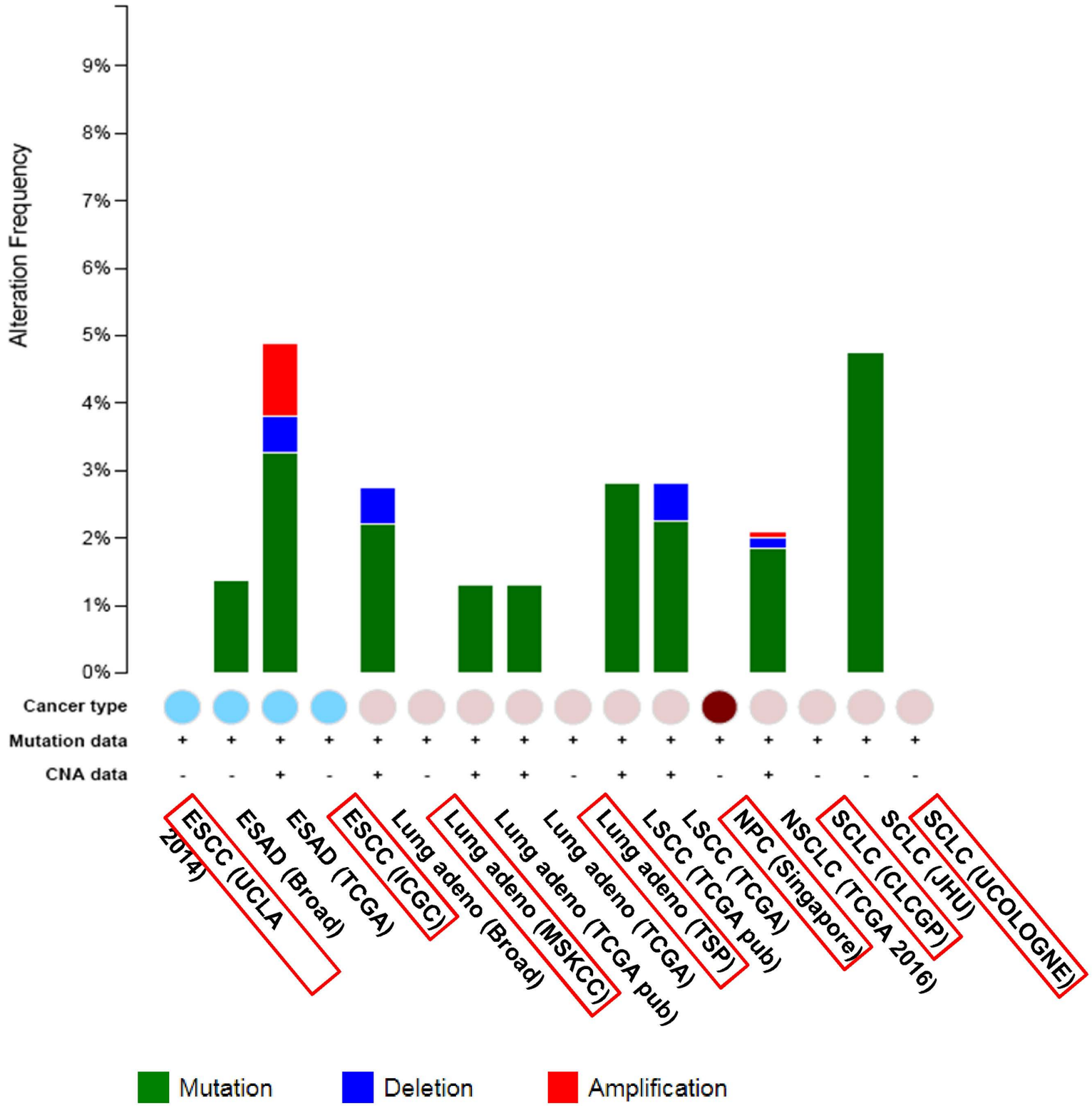
2
3
Blood
(n=420)
Lung
(n=390)
Lung adeno
(n=261)

Suppl. Fig3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8%
7%
6%
5%
4%
3%
2%
1%
0%
Cancer type
Mutation data
CNA data
ESCC (UCLA 2014)
ESAD (Broad)
ESAD (TCGA)
ESCC (ICGC)
Lung adeno (Broad)
Lung adeno (MSKCC)
Lung adeno (TCGA pub)
Lung adeno (TCGA)
LSCC (TCGA pub)
LSCC (TSP)
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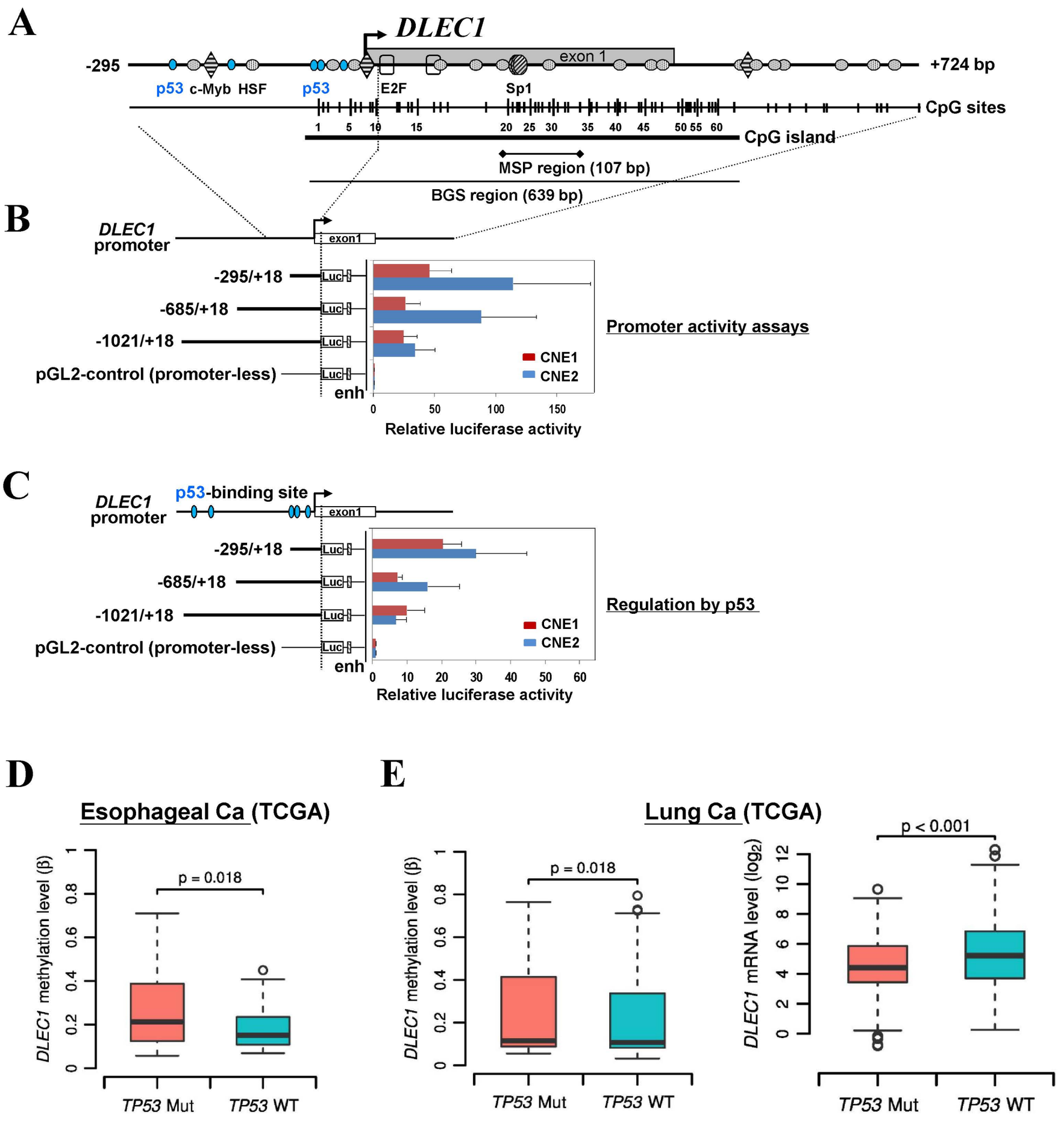
SCLC (UCOLOGNE)
Mutation
Deletion
Amplification

Suppl. Fig4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p53 c-Myb HSF
p53
E2F
Sp1
CpG sites
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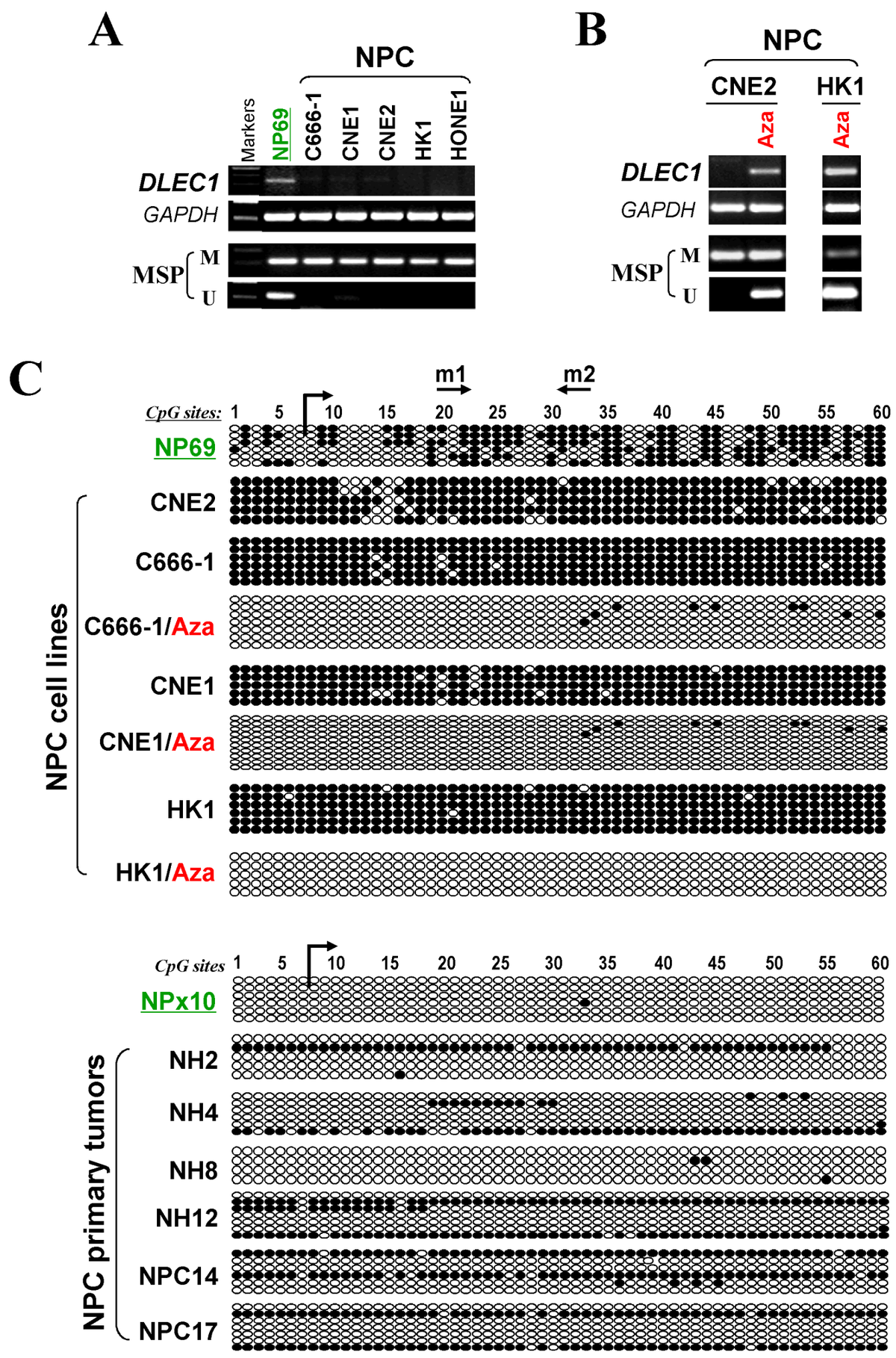
CpG island
MSP region (107 bp)
BGS region (639 bp)
B
DLEC1 promoter
exon1
-295/+18
-685/+18
-1021/+18
pGL2-control (promoter-less)
Luc
enh
CNE1
CNE2
0 50 100 150
Relative luciferase activity
Promoter activity assays
C
DLEC1 promoter
p53-binding site
exon1
-295/+18
-685/+18
-1021/+18
pGL2-control (promoter-less)
Luc
enh
CNE1
CNE2
0 10 20 30 40 50 60
Relative luciferase activity
Regulation by p53
D
Esophageal Ca (TCGA)
DLEC1 methylation level (β)
p = 0.018
TP53 Mut
TP53 WT
E
Lung Ca (TCGA)
DLEC1 methylation level (β)
p = 0.018
TP53 Mut
TP53 WT
DLEC1 mRNA level (log2)
p < 0.001
TP53 Mut
TP53 WT

Suppl. Fig5


A
NPC
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NP69
CNE2
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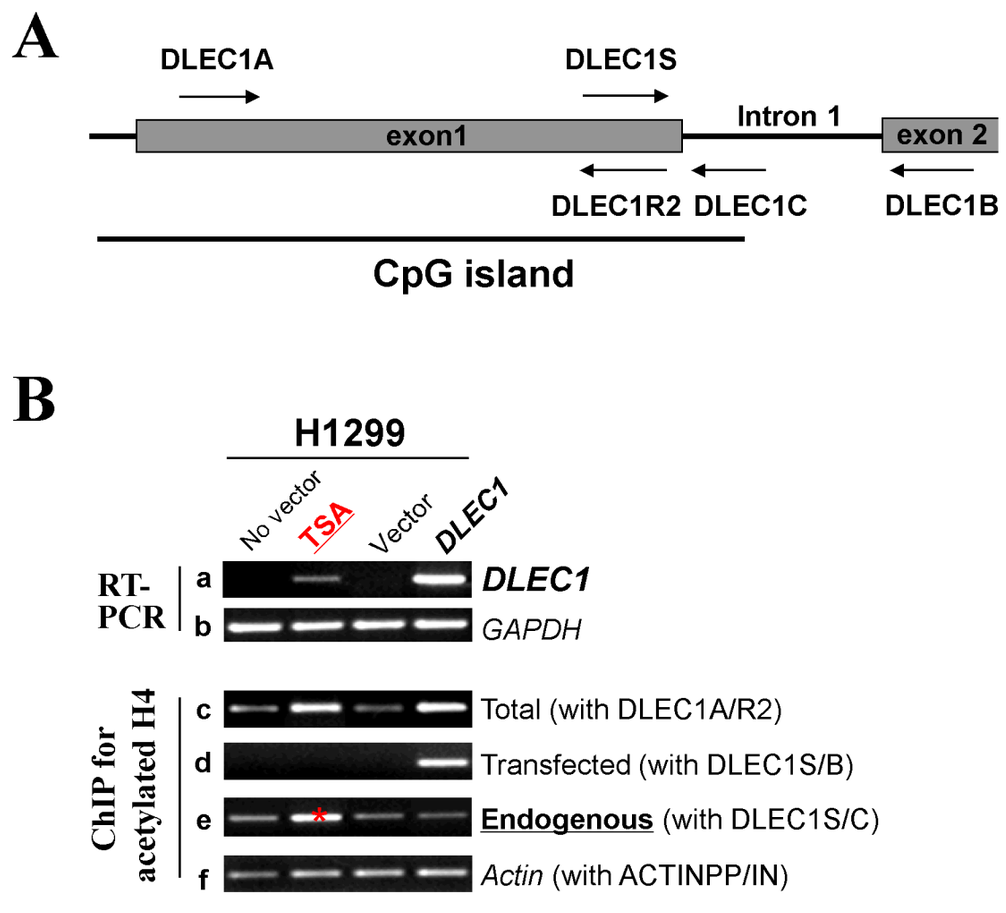
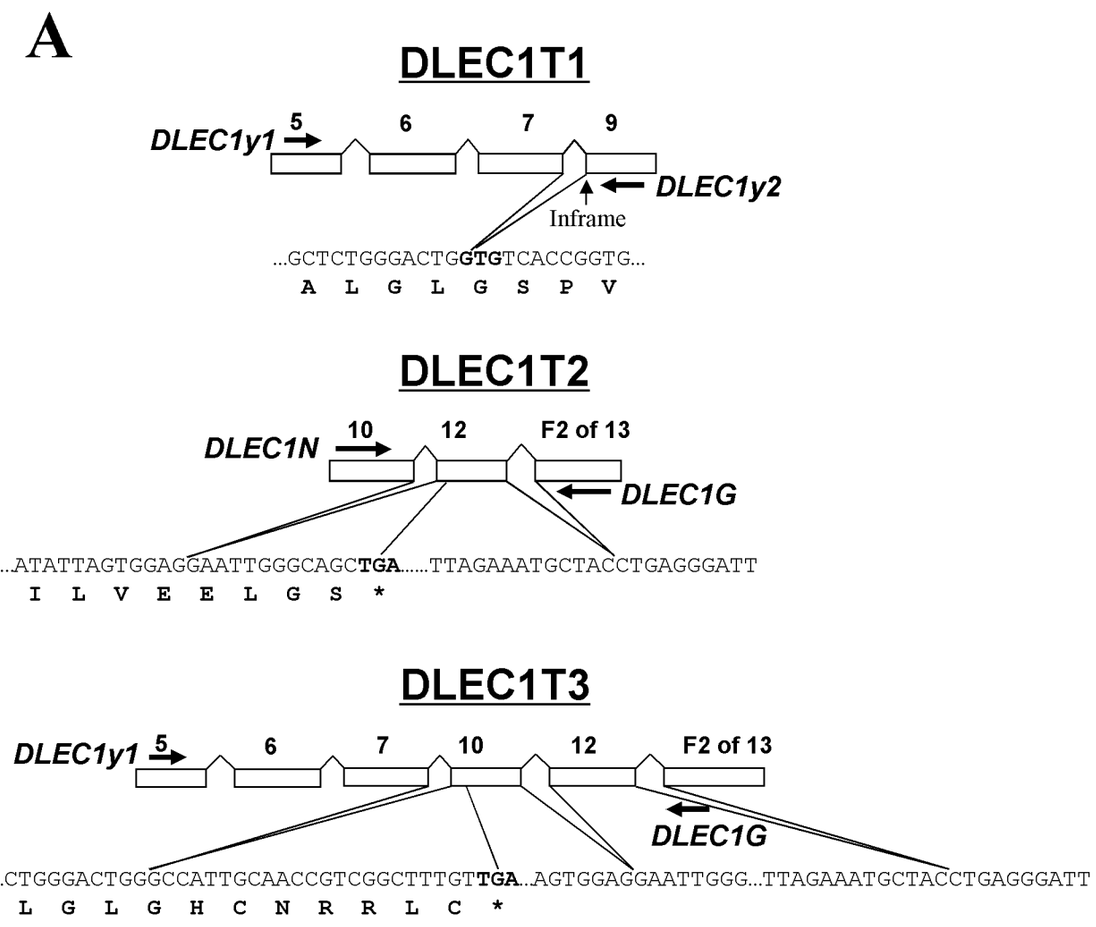
C666-1/Aza
CNE1
CNE1/Aza
HK1
HK1/Aza
NPC cell lines
CpG sites 1 5 10 15 20 25 30 35 40 45 50 55 60
NPx10
NH2
NH4
NH8
NH12
NPC14
NPC17
NPC primary tumors

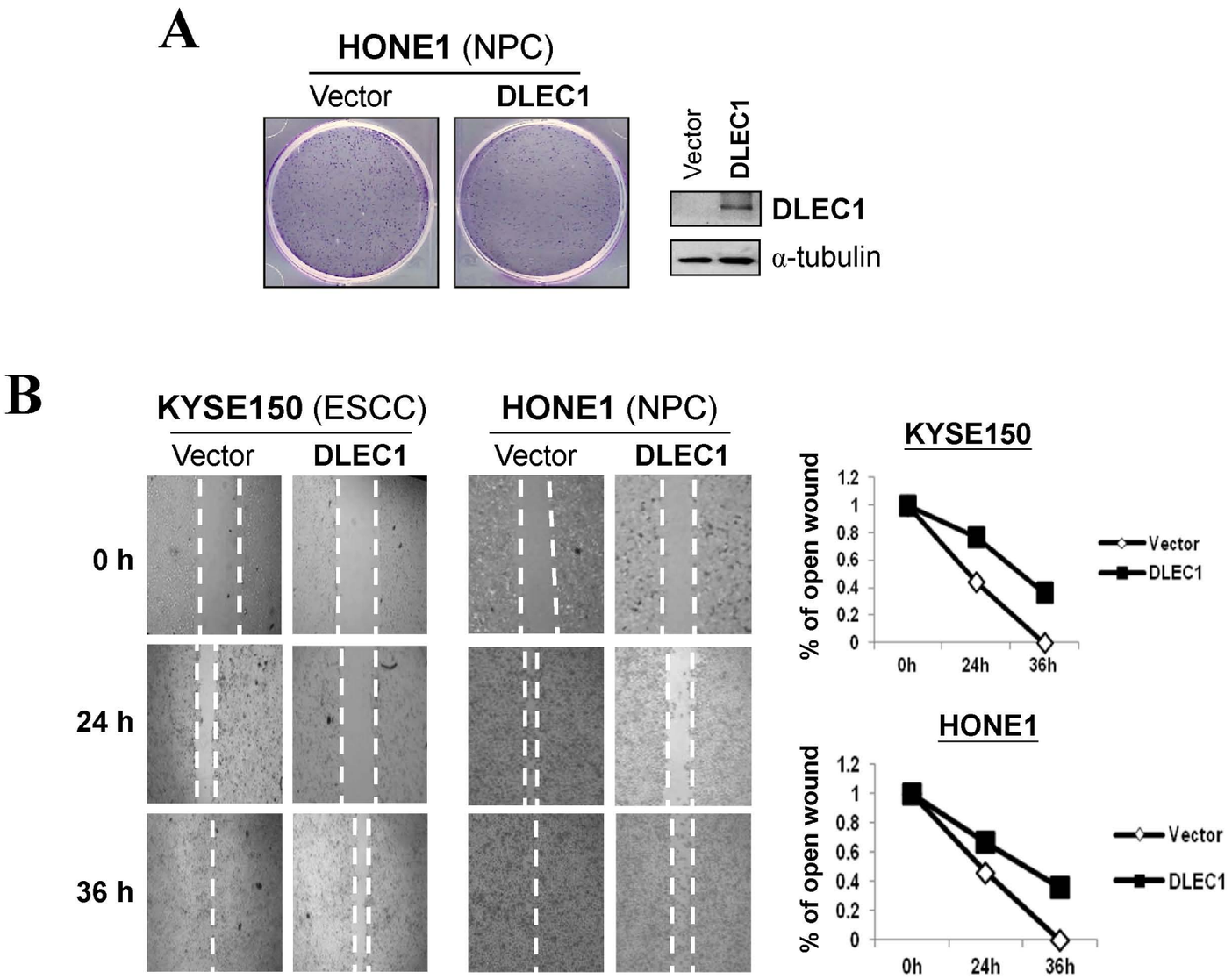
Suppl. Fig6


A
DLEC1A
DLEC1S
exon1
Intron 1
exon 2
DLEC1R2
DLEC1C
DLEC1B
CpG island
B
H1299
No vector
TSA
Vector
DLEC1
RT-PCR
a
DLEC1
b
GAPDH
ChIP for acetylated H4
c
Total (with DLEC1A/R2)
d
Transfected (with DLEC1S/B)
e
Endogenous (with DLEC1S/C)
f
Actin (with ACTINPP/IN)

Suppl. Fig7


A
DLEC1T1
DLEC1y1
5
6
7
9
DLEC1y2
Inframe
...GCTCTGGGACTGGTGTCACCGGTG...
A L G L G S P V
DLEC1T2
DLEC1N
10
12
F2 of 13
DLEC1G
...ATATTAGTGGAGGAATTGGGCAGCTGA.....TTAGAAATGCTACCTGAGGGATT
I L V E E L G S *
DLEC1T3
DLEC1y1
5
6
7
10
12
F2 of 13
DLEC1G
...CTGGGACTGGGCCATTGCAACCGTCGGCTTTGTTGA...AGTGGAGGAATTGGG...TTAGAAATGCTACCTGAGGGATT
L G L G H C N R R L C *
B
ESCC
Other Ca. cell lines
Immortalized normal cell lines
Normal PBMCs
Normal adult tissues
Normal fetal tissues
HKESC1
HKESC2
SLMT1
MCF-7
HCT116
C33A
SiHa
hUH7
Kato III
NE1
NP69
10 11 12 13 14
H2O
Markers
Heart
Sk. Muscle
Liver
Kidney
Stomach
Colon
Rectum
Larynx
Trachea
Lung
Breast
Cervix
Ovary
Placenta
Testis
B. Marrow
Prostate
Brain
Pancreas
Esophagus
S.M
Skin
Kidney
Colon
Bladder
Brain
Stomach
Lung
Liver
DLEC1 y1/y2
wt
T1
DLEC1 N/G
wt
S1
S2
T2

Suppl. Fig8


A
HONE1 (NPC)
Vector
DLEC1
Vector
DLEC1
DLEC1
α-tubulin
B
KYSE150 (ESCC)
HONE1 (NPC)
Vector
DLEC1
Vector
DLEC1
0 h
24 h
36 h
KYSE150
% of open wound
1.2
1
0.8
0.6
0.4
0.2
0
0h
24h
36h
Vector
DLEC1
HONE1
% of open wound
1.2
1
0.8
0.6
0.4
0.2
0
0h
24h
36h
Vector
DLEC1