Table I. Survey instrument

1. Do you have a mobile phone?	N/A	Yes	No	Not sure
2. If you do have a mobile phone, does it take pictures?				
3. If you do have a mobile phone that takes pictures…				
3a. Do you USUALLY (more than 80% of the time) take it with you to doctor's appointments?	N/A	Yes	No	Not sure
3b. Do you know how to take pictures with it?	N/A	Yes	No	Not sure
3c. If you don't know how to take pictures with it, would you be able to easily find someone to show you how?	N/A	Yes	No	Not sure
3d. Would you be willing to take a picture of your biopsy site and bring it with you to your follow-up visit to help your doctor locate the site?	N/A	Yes	No	Not sure

how to use them (n = 13) felt that they would be able to find someone to show them how. Sixty-eight percent of patients had camera phones, usually take them to doctor's appointments, and know how to use the camera function. Eighty-six percent had camera phones and either knew how to use them or could get someone to show them.

Men (n = 64) had a slightly higher probability than women (n = 36) of having camera phones, bringing them to doctor's appointments, and knowing how to use them. Differences were greater when stratifying by age. More than 96% (96.4%) of respondents <70 years of age had camera phones, compared with 73.3% of patients ≥70 years of age. Only 46.7% of those ≥70 years of age had camera phones they take to the doctor and know how to use, compared with 85.5% of those <70 years of age. However, 73.3% of those ≥70 years of age had camera phones and either knew how to use them or could have someone help them; 96.4% of patients <70 years of age fell into this category. Most patients with camera phones were willing to take pictures (96.5% overall). One hundred percent of those <70 years of age were willing to take pictures, but only 90.9% of those ≥70 years of age who had camera phones were willing to take pictures.

We feel that standardized, clinic-taken photographs at the time a biopsy specimen is obtained is the criterion standard for site identification. The increasing quality and ubiquity of camera phones, however, make them a viable alternative. We envision two scenarios for their use: (1) the physician obtaining the biopsy specimen asks the patient to photograph the site at the time the specimen is obtained or at the time of result notification, or (2) the treating surgeon's office asks the patient to photograph the site when treatment appointment is made. Our results show that patients are largely able and willing to comply with this request, although our patient demographics are likely not representative of those of many surgeons, particularly those practicing in urban centers. Increasing age is a possible predictor of those who are unable to participate. As the technology becomes more ubiquitous, a higher percentage of the population will likely be able to do so.

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Mutational profile of primary dermal melanoma: A case series

To the Editor: Primary dermal melanoma (PDM) is a rare melanoma subtype confined to the dermis or subcutis[1] that histologically simulates cutaneous

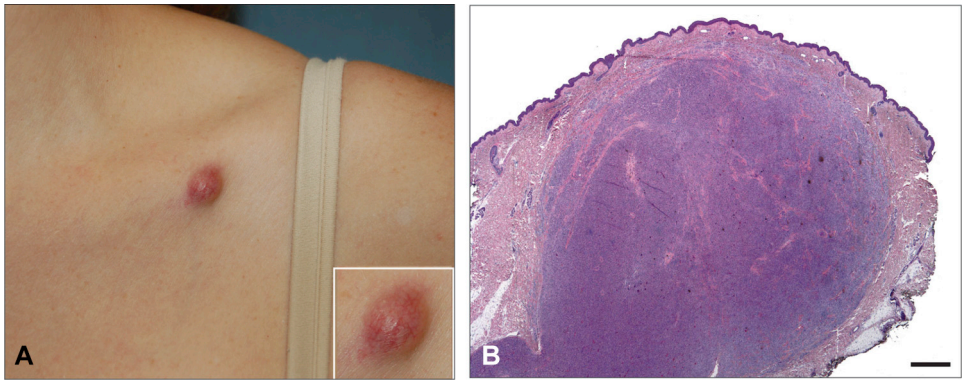

Fig 1. A, Clinical photograph of primary dermal melanoma, patient 1, with higher magnification inset. **B**, Hematoxylin–eosin staining of sample from patient 1. (Original magnification: ×10. Scale bar = 1 mm.)

metastasis, with no discernable overlying regression or in situ component. Clinically, however, PDM is associated with significantly increased survival rates when compared to in-transit (N2c) or cutaneous (M1a) metastasis (<70% and <30% estimated 5-year survival rates, respectively) and to similar thickness conventional cutaneous melanoma. Distinguishing PDM from similar melanocytic proliferations can be challenging. Histopathologic features, including lower p53, Ki-67, cyclin D1, and D2-40 expression, can help discern PDM from histologic mimics.[2] However, the cause of the relatively indolent behavior of PDM is unknown.

Melanocytic proliferations can be classified into biologic subtypes with distinct and characteristic genetic mutations.[3] We considered several possibilities: first, PDM may have mutations characteristic of dermal melanocytic proliferations, such as blue nevi (ie, activating mutations in *GNAQ* or *GNA11*).[4] Alternatively, PDMs may share a mutational profile with conventional melanomas but behave differently because of alternate cell origin or microenvironment. As a final possibility, PDMs may display their own unique characteristic mutations.

To discern between these possibilities, we performed deep sequencing of 3 PDM cases along with patient-matched normal control skin. Cases showed classical PDM characteristics on histopathologic grounds (Fig 1 and Supplemental Information, available online at http://www.jaad.org), with a mean tumor thickness of 15.4 mm and clinical outcome showing long-term disease-free survival (mean, 78.3 months). Sequencing of all exons of 1000 cancer-related genes was performed (Supplemental Information), which included *BRAF, NRAS, TP53, PTEN, CDKN2A, KIT, RAC1*, *BAP1*, *GNA11*, and *GNAQ*.

Table I. Genetic mutations in a series of 3 primary dermal melanomas

Patient	Gene	Mutation (coding NT)	Mutation (AA)
PDM1	*BCL11B*	c.2287G>A	p.Gly763Ser
	NF1	c.4159G>A	p.Glu1387Lys
PDM2	*BRAF*	c.1799_1800TG>AA	p.Val600Glu
	DAPK3	c.1147C>T	p.Arg382Trp
	EPHA1	c.2917G>A	p.Gly973Arg
	FGFR2	c.1634A>G	p.Lys545Arg
	JAZF1	c.130C>T	p.Arg43Trp
	MARK1	c.556G>A	p.Glu186Lys
	MLLT3	c.254T>A	p.Ile85Asn
	NPM1	c.769G>A	p.Glu256Lys
	TIAM1	c.1858G>A	p.Arg620Cys
PDM3	*BRAF*	c.1799T>A	p.Val600Glu
	CAMK1D	c.88G>A	p.Gly30Arg
	CLP1	c.904C>A	p.Arg302Ser
	EPHA3	c.488A>T	p.Lys163Met
	HCK	c.765_766GG>AA	p.Trp255*; Gly256Lys
	MAPK15	c.199G>A	p.Glu66Lys
	MEN1	c.17C>T	p.Ala6Val
	NF1	c.4159G>A	p.Glu1387Lys
	SLK	c.575G>A	p.Gly192Asp

Deep sequencing of 1000 cancer-related genes revealed tumor-specific genetic mutations associated with PDM. Variants with Combined Annotation-Dependent Depletion score[5] >20 and variant allele frequency >0.15 are presented.
AA, Amino acid; *NT*, nucleotide; *PDM*, primary dermal melanoma.

Sequencing revealed mutations seen in conventional melanomas, including *BRAF* (p.Val600Glu; two-thirds of samples), *EPHA1*, *TIAM1*, *DAPK3*, and *CLP1* (Table I). Novel variants were identified in *NF1* (p.Glu1387Lys; two-thirds of samples), *EPHA3*, and *MAPK15*. Mutations in genes not previously reported in melanomas were also observed, and represent candidates for further characterization. *GNAQ* and *GNA11* mutations, characteristic of blue nevi and

nevus of Ota, were absent. We also performed computational copy number variation prediction, followed by confirmatory immunohistochemical staining. This identified p16/CDKN2A loss—a sentinel genetic event in melanoma—in all PDM samples (Supplemental Information).

Together, these results suggest that PDM mutations and copy number variations overlap with cutaneous melanomas but also display distinct variations. We hypothesize that the indolent behavior of PDM may reflect both a unique mutational profile and a distinct tumor microenvironment and cell origin. These results also indicate that patients with PDMs that metastasize can be screened for *BRAF* mutation status for consideration of therapy with *BRAF* and *MEK* inhibitors.

This study was limited by its small size given the rarity of PDM, which we intend to expand to define a more comprehensive PDM mutational profile. In addition, mutations in noncoding regions (eg, human telomerase reverse transcriptase promoter) were not captured. Nonetheless, these initial results provide insight to the genetic variations associated with PDM, contributing to the promise of accurately diagnosing this rare variant and understanding its place in the taxonomy of melanocytic neoplasms.

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RightOn Sequencing is a proprietary reagent of Elim Biopharmaceuticals.

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Increased cutaneous stimulation is required for C-fiber sensory perception in alopecia areata: A double-blind study

To the Editor: Alopecia areata (AA) is an autoimmune disease targeting hair follicles, resulting in nonscarring hair loss. The suggestion of nervous system dysfunction in AA dates to 1886 when Max Joseph[1] sectioned cat cervical ganglions, resulting in characteristic AA. Clinically, patients report burning, pruritus, tingling, or pain.[2] Neurogenic inflammation has also been implicated.[3,4] To investigate sensory perception in AA, we hypothesized a difference in sensory perception in AA scalp when compared with healthy scalp. Using transcutaneous electrical stimulation, we studied Aβ nerves, which transmit tactile sensation; Aδ nerves, which transmit short and fast pain; and unmyelinated C-fibers, which transmit delayed pain.[4]

The University of Minnesota Institutional Review Board approved the study (approval no. 0610M95566). Patients with patchy AA and healthy individuals aged 18 years and older participated. Exclusion criteria included: other diagnoses of alopecia, anti-inflammatory medications, pregnancy, radiation or chemotherapy, corticosteroid use, site trauma, recent hormone-containing medications, unstable thyroid function, nervous system dysfunction, heavy metal exposure, pain medication use, and altered cutaneous sensation. A Neurometer diagnostic stimulator (Neurotron Inc, Baltimore, MD) was used for double-blind testing.[5] We generated constant alternating current sinusoid waveform stimuli at 2000, 250, and 5 Hz, corresponding to Aβ, Aδ, and C- fibers, respectively.[5]

SUPPLEMENTAL INFORMATION

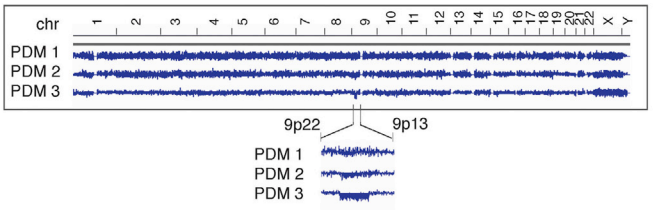
A

Patient characteristics

Subject Number	Age at diagnosis, sex, location	Breslow depth (mm), AJCC stage	Primary Tx	Disease free survival (months)
1	37 F, clavicle	13, IIB / T4aN0M0	WLE, SLNB	104
2	74 F, lower leg	30, IIB / T4aN0M0	WLE, SLNB	87
3	84 M, lower back	3.2, IIA / T3aN0M0	WLE, SLNB	44

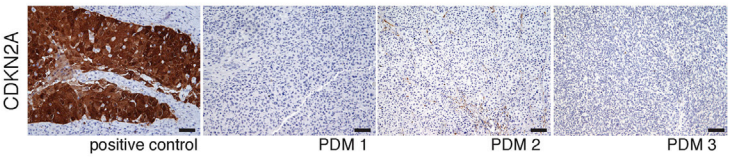
B

PDM copy number variation analysis



C

Immunohistochemical staining for CDKN2A



Supplemental File

(A) **Patient characteristics**. Patients 1 and 3 are alive and well. Patient 2 passed away from unrelated causes.

(B) **Copy number analysis of PDM**. Copy number variation was inferred using standard parameters of SeqGene analysis of targeted sequencing data. Evidence of consistent genomic loss was identified in the interval between chr 9p22 and 9p13 in PDM #2 and 3. This interval contains the p16/CDKN2A locus.

(C) **Immunohistochemical staining for p16/CDKN2A**. To confirm results seen by sequencing CNV analysis, immunohistochemical staining for p16 was performed in PDM sampels and a positive control, demonstrating broad lack of p16 expression in all 3 PDM samples.

(D) **Targeted gene capture list** (following pages). Exons of the 1,000 genes listed were captured and sequenced in the PDM series.

RightOn™ Cancer Sequencing
1000 Cancer Genes, One Single Test

ELIM BIOPHARM

1000 Cancer Gene List, v1.0

AAK1	AATK	ABI1	ABL1	ABL2	ACSL3
ACSL6	ACTR2	ACVR1	ACVR1B	ACVR1C	ACVR2A
ACVR2B	ACVRL1	ADAMTSL3	ADCK1	ADCK3	ADCK4
ADCK5	ADRBK1	ADRBK2	AFF1	AFF3	AFF4
AGK	AKAP9	AKT1	AKT2	AKT3	ALDH2
ALK	ALPK1	ALPK2	ALPK3	AMHR2	ANGPT2
ANKK1	APC	AR	ARAF	ARHGAP26	ARHGEF12
ARID1A	ARID5B	ARNT	ASPSCR1	ASXL1	ATF1
ATIC	ATM	ATP8B1	ATR	ATRX	AURKA
AURKB	AURKC	AXIN1	AXL	BAP1	BCKDK
BCL10	BCL11A	BCL11B	BCL2	BCL3	BCL6
BCL7A	BCL9	BCR	BIRC3	BLK	BLM
BMI1	BMP2K	BMPR1A	BMPR1B	BMPR2	BMX
BRAF	BRCA1	BRCA2	BRD2	BRD3	BRD4
BRDT	BRIP1	BRSK1	BRSK2	BTG1	BTK
BUB1	BUB1B	C15ORF55	C9orf96	CADM1	CAMK1
CAMK1D	CAMK1G	CAMK2A	CAMK2B	CAMK2D	CAMK2G
CAMK4	CAMKK1	CAMKK2	CAMKV	CANT1	CARD11
CARS	CASC5	CASK	CBFA2T3	CBFB	CBL
CBLB	CBLC	CCDC6	CCNB1IP1	CCND1	CCND2
CCND3	CCNE1	CD274	CD74	CD79A	CD79B
CDC42BPA	CDC42BPB	CDC42BPG	CDC6	CDC7	CDC73
CDH1	CDH11	CDK1	CDK10	CDK11B	CDK12
CDK13	CDK14	CDK15	CDK16	CDK17	CDK18
CDK19	CDK2	CDK20	CDK3	CDK4	CDK5
CDK6	CDK7	CDK8	CDK9	CDKL1	CDKL2
CDKL3	CDKL4	CDKL5	CDKN2A	CDKN2B	CDKN2C
CDX2	CEBPA	CERK	CHCHD7	CHD1	CHD1L
CHD3	CHD5	CHEK1	CHEK2	CHIC2	CHN1
CHUK	CIC	CIITA	CIT	CLK1	CLK2
CLK3	CLK4	CLP1	CLTC	CLTCL1	CNBP
CNKSR2	CNTRL	COL1A1	COX6C	CREB1	CREB3L1
CREB3L2	CREBBP	CRK	CRLF2	CRP	CRTC1
CRTC3	CSF1R	CSK	CSMD3	CSNK1A1	CSNK1A1L
CSNK1D	CSNK1E	CSNK1G1	CSNK1G2	CSNK1G3	CSNK2A1
CSNK2A2	CTNNB1	CUBN	CXCL1	CXCR7	CYLD
DAPK1	DAPK2	DAPK3	DAXX	DBN1	DCLK1
DCLK2	DCLK3	DDB2	DDIT3	DDR1	DDR2
DDX10	DDX5	DDX6	DEK	DGKA	DGKB
DGKD	DGKE	DGKG	DGKH	DGKI	DGKQ
DGKZ	DHFR	DICER1	DMPK	DNMT3A	DSTYK
DUX4	DYRK1A	DYRK1B	DYRK2	DYRK3	DYRK4
EBF1	EDNRB	EEF2K	EFNA1	EGFR	EHMT1

EIF2AK1	EIF2AK2	EIF2AK3	EIF2AK4	EIF4A2	ELF4
ELK4	ELL	ELN	EML4	EP300	EPHA1
EPHA10	EPHA2	EPHA3	EPHA4	EPHA5	EPHA6
EPHA7	EPHA8	EPHB1	EPHB2	EPHB3	EPHB4
EPHB6	EPS15	ERBB2	ERBB3	ERBB4	ERC1
ERCC2	ERCC3	ERCC4	ERCC5	ERG	ERN1
ERN2	ESR1	ESR2	ETV1	ETV4	ETV5
ETV6	EWSR1	EXT1	EXT2	EZH2	FAM123A
FAM123B	FAM123C	FANCA	FANCC	FANCD2	FANCE
FANCF	FANCG	FAS	FASTK	FAT3	FBXW7
FCGR2B	FCRL4	FER	FES	FEV	FGF2
FGFR1	FGFR1OP	FGFR2	FGFR3	FGFR4	FGR
FH	FHIT	FIP1L1	FKBP8	FLCN	FLI1
FLNB	FLT1	FLT3	FLT4	FNBP1	FOXL2
FOXO1	FOXO3	FOXO4	FOXP1	FRK	FRS2
FSCB	FSTL3	FUS	FYN	GAB1	GABRA6
GAK	GAS7	GATA1	GATA2	GATA3	GCK
GMPS	GNA11	GNAQ	GNAS	GOLGA5	GOPC
GPC3	GPHN	GRB2	GRK1	GRK4	GRK5
GRK6	GRK7	GSG2	GSK3A	GSK3B	GUCY1A2
GUCY2C	GUCY2D	GUCY2F	HAUS3	HCK	HDAC9
HERPUD1	HGF	HIF1A	HIP1	HIPK1	HIPK2
HIPK3	HIPK4	HIST1H4I	HLF	HMGA1	HMGA2
HMGN2P46	HNF1A	HNRNPA2B1	HOOK3	HOXA11	HOXA13
HOXA9	HOXC11	HOXC13	HOXD11	HOXD13	HRAS
HSP90AA1	HSP90AB1	HSPB8	HUNK	ICAM1	ICK
IDH1	IDH2	IGF1R	IGF2R	IKBKB	IKBKE
IKZF1	IL2	IL21R	IL6ST	IL7R	ILK
INPP4A	INSR	INSRR	IP6K1	IP6K2	IP6K3
IPMK	IPPK	IRAK1	IRAK2	IRAK3	IRAK4
IRF4	IRS2	IRS4	ITGA9	ITGAV	ITGB3
ITGB5	ITK	ITPK1	ITPKA	ITPKB	ITPKC
JAK1	JAK2	JAK3	JAZF1	JUN	JUNB
KALRN	KAT6A	KAT6B	KDM5A	KDM5C	KDM6A
KDM6B	KDR	KDSR	KIAA1468	KIAA1549	KIAA1804
KIT	KLF6	KLHL4	KLK2	KRAS	KSR1
KSR2	KTN1	L3MBTL2	L3MBTL3	LASP1	LATS1
LATS2	LCK	LCP1	LEF1	LHFP	LIFR
LIMK1	LIMK2	LMO1	LMO2	LMTK2	LMTK3
LPP	LRP1B	LRRK1	LRRK2	LTK	LYL1
LYN	MAF	MAFB	MAK	MALAT1	MALT1
MAML2	MAP2K1	MAP2K2	MAP2K3	MAP2K4	MAP2K5
MAP2K6	MAP2K7	MAP3K1	MAP3K10	MAP3K11	MAP3K12
MAP3K13	MAP3K14	MAP3K15	MAP3K2	MAP3K3	MAP3K4
MAP3K5	MAP3K6	MAP3K7	MAP3K8	MAP3K9	MAP4K1
MAP4K2	MAP4K3	MAP4K4	MAP4K5	MAPK1	MAPK10
MAPK11	MAPK12	MAPK13	MAPK14	MAPK15	MAPK3
MAPK4	MAPK6	MAPK7	MAPK8	MAPK9	MAPKAPK2
MAPKAPK3	MAPKAPK5	MARK1	MARK2	MARK3	MARK4
MAST1	MAST2	MAST3	MAST4	MASTL	MATK
MDM2	MDM4	MDS2	MECOM	MEF2B	MELK
MEN1	MERTK	MET	MINK1	MITF	MKL1
MKNK1	MKNK2	MLF1	MLH1	MLKL	MLL
MLL2	MLL3	MLLT1	MLLT10	MLLT11	MLLT3
MLLT4	MLLT6	MN1	MNX1	MOS	MPL

MSH2	MSH6	MSI2	MSN	MST1R	MST4
MTCP1	MTOR	MUC1	MUSK	MUTYH	MYB
MYC	MYCL1	MYCN	MYD88	MYH1	MYH11
MYH9	MYLK	MYLK2	MYLK3	MYLK4	MYO3A
MYO3B	NACA	NBN	NCKIPSD	NCOA1	NCOA2
NCOA4	NEK1	NEK10	NEK11	NEK2	NEK3
NEK4	NEK5	NEK6	NEK7	NEK8	NEK9
NF1	NF2	NFE2L2	NFIB	NFKB1	NFKB2
NFKBIA	NFKBIE	NIM1	NIN	NKX2-1	NLK
NONO	NOS3	NOTCH1	NOTCH2	NOTCH3	NPM1
NPR1	NPR2	NR4A3	NRAS	NRBP1	NRBP2
NRK	NRP1	NSD1	NTRK1	NTRK2	NTRK3
NUAK1	NUAK2	NUMA1	NUP214	NUP98	OBSCN
OLIG2	OMD	OXSR1	P2RY8	PAFAH1B2	PAK1
PAK2	PAK3	PAK4	PAK6	PAK7	PALB2
PASK	PATZ1	PAX3	PAX5	PAX7	PAX8
PBK	PBRM1	PBX1	PCM1	PCSK7	PDCD1LG2
PDE4DIP	PDGFB	PDGFRA	PDGFRB	PDIK1L	PDK1
PDK2	PDK3	PDK4	PDPK1	PEAK1	PER1
PGF	PHF6	PHKG1	PHKG2	PHOX2B	PI4K2A
PI4K2B	PI4KA	PI4KB	PICALM	PIK3C2A	PIK3C2B
PIK3C2G	PIK3C3	PIK3CA	PIK3CB	PIK3CD	PIK3CG
PIK3R1	PIK3R2	PIK3R3	PIK3R4	PIK3R5	PIK3R6
PIKFYVE	PIM1	PIM2	PIM3	PINK1	PIP4K2A
PIP4K2B	PIP4K2C	PIP5K1A	PIP5K1B	PIP5K1C	PIP5KL1
PIPSL	PKDCC	PKHD1	PKLR	PKMYT1	PKN1
PKN2	PKN3	PLAG1	PLCG1	PLCG2	PLK1
PLK2	PLK3	PLK4	PMEL	PML	PMS1
PMS2	PNCK	POU2AF1	POU5F1	PPARG	PPP2R1A
PRCC	PRDM1	PRDM16	PRF1	PRKAA1	PRKAA2
PRKACA	PRKACB	PRKACG	PRKAR1A	PRKCA	PRKCB
PRKCD	PRKCE	PRKCG	PRKCH	PRKCI	PRKCQ
PRKCZ	PRKD1	PRKD2	PRKD3	PRKDC	PRKG1
PRKG2	PRKX	PRKY	PRPF4B	PRRX1	PSIP1
PSKH1	PSKH2	PTCH1	PTEN	PTK2	PTK2B
PTK6	PTK7	PTPN11	PTPN2	PXK	RABEP1
RAC1	RAD51B	RAF1	RAGE	RALGDS	RANBP17
RAP1GDS1	RARA	RB1	RBM15	RECQL4	REL
RET	RHEB	RHOH	RIOK1	RIOK2	RIOK3
RIPK1	RIPK2	RIPK3	RIPK4	RMI2	RNASEL
RNF213	RNF220	ROBO1	ROBO2	ROCK1	ROCK2
ROR1	ROR2	ROS1	RPL22	RPN1	RPS6KA1
RPS6KA2	RPS6KA3	RPS6KA4	RPS6KA5	RPS6KA6	RPS6KB1
RPS6KB2	RPS6KC1	RPS6KL1	RUNDC2A	RUNX1	RUNX1T1
RYK	SBDS	SBK1	SBK2	SCML2	SCYL1
SCYL2	SCYL3	SDHAF2	SDHB	SDHC	SDHD
SEPT5	SEPT6	SEPT9	SERPINE1	SET	SETD2
SETDB1	SFPQ	SGK1	SGK196	SGK2	SGK223
SGK3	SGK494	SH3GL1	SIK1	SIK2	SIK3
SIX4	SLC45A3	SLK	SMAD2	SMAD4	SMARCA4
SMARCB1	SMG1	SMO	SMYD4	SNRK	SNX4
SOCS1	SOX2	SP1	SPECC1	SPEG	SPHK1
SPHK2	SPOP	SPP1	SPTAN1	SRC	SRGAP3
SRM	SRMS	SRPK1	SRPK2	SRPK3	SRSF3
SS18	SS18L1	SSX1	SSX2	SSX4	STIL

STK10	STK11	STK16	STK17A	STK17B	STK19
STK24	STK25	STK3	STK31	STK32A	STK32B
STK32C	STK33	STK35	STK36	STK38	STK38L
STK39	STK4	STK40	STRADA	STRADB	STYK1
SUFU	SUZ12	SYK	TAF1	TAF15	TAF1L
TAL1	TAL2	TAOK1	TAOK2	TAOK3	TBCK
TBK1	TBX22	TCEA1	TCF12	TCF3	TCL1A
TCL6	TEC	TEK	TERT	TESK1	TESK2
TET1	TET2	TEX14	TFDP1	TFE3	TFEB
TFG	TFPT	TFRC	TGFBR1	TGFBR2	TGFBR3
THRAP3	TIAM1	TIE1	TIMP3	TLK1	TLK2
TLX1	TLX3	TMPRSS2	TNFAIP3	TNFRSF14	TNFRSF17
TNIK	TNK1	TNK2	TNNI3K	TOP1	TOP2A
TP53	TP53RK	TP73	TPM3	TPM4	TPR
TRIB1	TRIB2	TRIB3	TRIM24	TRIM27	TRIM28
TRIM33	TRIO	TRIP11	TRPM6	TRPM7	TRRAP
TSC1	TSC2	TSHR	TSSK1B	TSSK2	TSSK3
TSSK4	TSSK6	TTBK1	TTBK2	TTK	TTL
TTN	TXK	TYK2	TYRO3	UHMK1	ULK1
ULK2	ULK3	ULK4	USP28	USP6	VCAM1
VEGF-A	VEGF-B	VEGF-C	VHL	VPREB1	VRK1
VRK2	VRK3	WAS	WEE1	WEE2	WHSC1
WHSC1L1	WHSC2	WIF1	WNK1	WNK2	WNK3
WNK4	WRN	WT1	XPA	XPC	YES1
YSK4	ZAK	ZAP70	ZBTB16	ZMYM2	ZMYND8
ZNF331	ZNF384	ZNF521			