Comprehensive Panel- 500 Genes

Test Description

The MolQ Comprehensive Panel includes 500+ key solid tumor genes (for SNV, CNV, TMB, MSI and fusions) that are well characterized in the published literature and associated with oncology drugs that are FDA approved, part of NCCN guidelines, or in clinical trials.

Patient Demographic

Name: Mr. Raj Kumar Shukla
Sex: Male
Date of Birth/Age: 58 years
Disease: Prostate Cancer

Clinician

Clinician Name: Dr Amit Verma
Medical Facility: Dr AV Institute of Personalized Therapy and Cancer Research (IPTCR)
Pathologist: Not Provided

Specimen

Booking ID: 012309020063
Sample Type: FFPE Block ID- B/1370/23
Tumor Content Percentage: 10-12%
Date of Collection: 02-09-2023
Date of Booking: 02-09-2023

CLINICAL SYNOPSIS

Raj Kumar Shukla is a known case of carcinoma of prostate with a family history of cancer. His father and two brothers had carcinoma of the prostate and sister has carcinoma of breast. He has been evaluated for pathogenic variations in the genes listed in Appendix 2.

RESULTS

No clinically relevant alterations are detected.

Tumor Mutation Burden is 5.68 Mut/Mb (TMB-Intermediate).

Microsatellite Instability (MSI) is stable.

VARIANT DETECTED AS PER NCCN GUIDELINES

No clinically relevant alteration detected.

OTHER VARIANTS DETECTED

The given specimen contains *BRCA2* (p.Q3295*, VAF = 43.54%) and *ZMYM3* mutations (p.L1180Pfs*73; p.P1177Sfs*75, VAF = 12.92%;12.34%).

BRCA2 mutations have been linked to prostate cancer, according to numerous research. (PMID: 31915789). Furthermore, ***ZMYM3*** has been identified as a gene that frequently mutates in prostate cancer (PMID: 33115829).

"Important disclaimer: As a standard of care, our case selection criteria for NGS run is ≥20% tumor content. The run was performed in this case after receiving informed consent from the clinician."

RELEVANT BIOMARKERS

Gene/ Transcript (Locus)	Variant ID	Variant	Allele Frequency	#ClinVar	*Relevant Therapies (In this cancer type)	*Relevant Therapies (In other cancer type)	Tier[1]
BRCA2 NM_000059.4 (chr13:32972533)	-	c.9883C>T (p.Gln3295*) Exon 27	43.54%	Pathogenic	None	abiraterone + niraparib[i,ii] bevacizumab + olaparib[i,ii] olaparib[i,ii] olaparib + hormone therapy[i] rucaparib[i] talazoparib + hormone	IIc

			therapy[i] niraparib	

*Public data sources included in relevant therapies: FDA[i], NCCN, EMA[ii], ESMO
#Based on Clinvar version 20220709
[1]Li et al. Standards and Guidelines for the Interpretation and Reporting of Sequence Variants in Cancer: A Joint Consensus Recommendation of the Association for Molecular Pathology, American Society of Clinical Oncology, and College of American Pathologists. J Mol Diagn. 2017 Jan;19(1):4-23.

PREVALENT CANCER BIOMARKERS WITHOUT RELEVANT EVIDENCE BASED ON INCLUDED DATA SOURCES

Gene/ Transcript (Locus)	Variant	Variant Allele Frequency	Location	#ClinVar
ZMYM3 NM_201599.3 (chrX:70462824)	c.3539_3542delTCCC p.Leu1180Profs*73	12.92%	Exon 22	-
ZMYM3 NM_201599.3 (chrX:70462831)	c.3529_3535delCCCA CAC p.Pro1177Serfs*75	12.34%	Exon 22	-

#Based on Clinvar version 20220709

CLINICAL CORRELATION AND VARIANT INTERPRETATION

BRCA2 **p.Gln3295***

Gene description: The breast cancer early onset gene 2 (*BRCA2*) encodes one of two BRCA proteins (BRCA1 and BRCA2) initially discovered as major hereditary breast cancer genes. Although structurally unrelated, both BRCA1 and BRCA2 exhibit tumor suppressor function and are integrally involved in the homologous recombination repair (HRR) pathway, a pathway critical in the repair of damaged DNA[1,2]. Specifically, BRCA1/2 are required for repair of chromosomal double strand breaks (DSBs) which are highly unstable and compromise genome integrity[1,2]. Inherited pathogenic mutations in BRCA1/2 are known to confer increased risk in women for breast and ovarian cancer and in men for breast and prostate cancer[3,4,5]. For individuals diagnosed with inherited pathogenic or likely pathogenic BRCA1/2 variants, the cumulative risk of breast cancer by 80 years of age was 69-72% and the cumulative risk of ovarian cancer by 70 years was 20-48%[3,6].

Alterations and prevalence: Inherited *BRCA1/2* mutations occur in 1:400 to 1:500 individuals and are observed in 10-15% of ovarian cancer, 5-10% of breast cancer, and 1-4% of prostate cancer[7,8,9,10,11,12,13,14]. Somatic alterations in *BRCA2* are observed in 5-15% of uterine corpus endometrial carcinoma, cutaneous melanoma, bladder urothelial carcinoma, stomach adenocarcinoma, colorectal adenocarcinoma, lung squamous cell carcinoma, lung adenocarcinoma, and uterine carcinosarcoma, 3-4% of cervical squamous cell carcinoma, head and neck squamous cell carcinoma, esophageal adenocarcinoma, ovarian serous cystadenocarcinoma, cholangiocarcinoma, breast invasive carcinoma, renal papillary cell carcinoma, and 2% of renal clear cell carcinoma, hepatocellular carcinoma, thymoma, prostate adenocarcinoma, sarcoma, and glioblastoma multiforme[15,16].

Potential relevance: Individuals possessing *BRCA1/2* pathogenic germline or somatic mutations are shown to exhibit sensitivity to platinum-based chemotherapy as well as treatment with poly (ADP-ribose) polymerase inhibitors (PARPi)[17]. Inhibitors targeting PARP induce synthetic lethality in recombination deficient *BRCA1/2* mutant cells[18,19]. Consequently, several PARP inhibitors have been FDA approved for *BRCA1/2*-mutated cancers. Olaparib[20] (2014) was the first PARPi to be approved by the FDA for *BRCA1/2* aberrations. Originally approved for the treatment of germline variants, olaparib is now indicated (2018) for the maintenance treatment of both germline *BRCA1/2*-mutated (gBRCAm) and somatic *BRCA1/2*-mutated (sBRCAm) epithelial ovarian, fallopian tube, or primary peritoneal cancers that are responsive to platinum-based chemotherapy. Olaparib is also indicated for the treatment of patients with gBRCAm HER2-negative metastatic breast cancer and metastatic pancreatic adenocarcinoma. Additionally, olaparib[20] is approved (2020) for metastatic castration-resistant prostate cancer (mCRPC) with deleterious or suspected deleterious, germline or somatic mutations in HRR genes that includes BRCA2. Rucaparib[21] is also

approved (2020) for deleterious gBRCAm or sBRCAm mCRPC. Talazoparib[22] (2018) is indicated for the treatment of gBRCAm HER2-negative locally advanced or metastatic breast cancer. Additionally, talazoparib[22] in combination with enzalutamide is approved (2023) for metastatic castration-resistant prostate cancer (mCRPC) with mutations in HRR genes that includes BRCA2. Niraparib[23] (2017) is another PARPi approved for the treatment of epithelial ovarian, fallopian tube, or primary peritoneal cancers with a deleterious or suspected deleterious BRCA mutation. Niraparib in combination with abiraterone acetate[24] received FDA approval (2023) for the treatment of deleterious or suspected deleterious BRCA-mutated (BRCAm) mCRPC. Despite tolerability and efficacy, acquired resistance to PARP inhibition has been clinically reported[25]. One of the most common mechanisms of resistance includes secondary intragenic mutations that restore BRCA1/2 functionality[26]. In addition to PARP inhibitors, other drugs which promote synthetic lethality have been investigated for BRCA mutations. In 2022, the FDA granted fast track designation to the small molecule inhibitor, pidnarulex[27], for BRCA1/2, PALB2, or other homologous recombination deficiency (HRD) mutations in breast and ovarian cancers. Like PARPi, pidnarulex promotes synthetic lethality but through an alternative mechanism which involves stabilization of G-quadruplexes at the replication fork leading to DNA breaks and genomic instability.

ZMYM3 **p.Leu1180Profs*73; p.Pro1177Serfs*75**

Gene description: The *ZMYM3* gene encodes the zinc finger MYM-type containing 3 protein[28]. While the function is not fully understood, ZMYM3 is capable of binding histones and DNA, and may facilitate the repair of double-strand breaks (DSBs)[29].

Alterations and prevalence: Somatic mutations in *ZMYM3* are observed in 12% of uterine corpus endometrial carcinoma, 5% of skin cutaneous melanoma, 4% of colorectal adenocarcinoma, 3% of lung adenocarcinoma, lung squamous cell carcinoma, cervical squamous cell carcinoma, esophageal adenocarcinoma, and bladder urothelial carcinoma[15,16]. In prostate cancer, *ZMYM3* mutations have been observed to be enriched in African American men compared to white men with one study demonstrating occurrence in 11.7% vs. 2.7% of patients, respectively[30]. Biallelic deletion of *ZMYM3* is observed in 3% of cholangiocarcinoma and 2% of sarcoma and kidney chromophobe[15,16].

Potential relevance: Currently, no therapies are approved for *ZMYM3* aberrations.

RECOMMENDATIONS

- Genetic counselling is advised for interpretation on the consequences of the variant(s).

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APPENDIX 1: TEST METHODOLOGY

METHOD

Pathology Assessment

The FFPE block is reviewed for presence of tumor cells and tumor percentage by histopathologists through screening of H & E staining slides.

Assay Methods

The test was performed using the Oncomine Comprehensive Assay Plus targeted, amplicon based next-generation sequencing assay that analyses 500+ unique genes for SNV, CNV, TMB, MSI and fusions. The minimum of 20ng of DNA isolated by Qiagen nucleic acid isolation kit is amplified using Oncomine Comprehensive assay plus as per the instruction manual. The amplicon libraries are prepared from 4 pools of primer which includes 2 pools of DNA based targets. The amplified primer pools are enzyme fragmented and Ion adapter barcodes are added. Amplified library is purified followed by quantitation using Ion Library TaqManTM Quantitation Kit. The quality of amplified libraries having 150-200bp sizes are confirmed by Agilent TapeStation. The quantified pooled library is loaded on Ion 550 Chip using Ion Chef and sequencing is performed on the Ion GeneStudio S5 prime system. For the current report RNA was not included.

Secondary Analysis Methods

The sequence data is processed using Ion Torrent server and the Ion reporter software 5.20.2.0. TMB is reported as High (>10 mutations/Mb), Intermediate (>3 to 10 mutations/Mb) and Low (<3 mutations/Mb). All the reported alterations are manually curated using Integrative Genomics Viewer (IGV). The Final report is generated using oncomine knowledgebase which includes contextual investigations of sample-specific variants with respect to labels, guidelines (AMP, ASCO, CAP), current clinical trials and peer-reviewed literature which is frequently updated.

Genes Assayed

The panel covers 1.50M bases of DNA region, including 1.06M bases of exonic regions. It includes a total of 500+ genes covering 165 hotspot genes, 333 genes with focal CNV gains and loss, 227 genes with full coding sequence (CDS), >1 Mb exonic regions for TMB evaluation and 76 MSI markers for Microsatellite Instability (MSI) and Microsatellite stable (MSS). It also covers 46 genes (SNVs, Indels, CNVs) for homologous recombination deficiency (HRD) including *BRCA1* and *BRCA2*. A subset of these (20 genes) were assessed for determining Loss of Heterozygosity (LOH) at gene level. Details available on request.

AMP/ASCO/CAP Classification

Tier I: Variants of Strong Clinical Significance	1A	Biomarkers that predict response or resistance to US FDA-approved therapies for a specific type of tumor or have been included in **professional guidelines** as **therapeutic, diagnostic, and/or prognostic biomarkers** for specific types of tumors.
	1B	Biomarkers that predict response or resistance to a therapy based on well-powered studies with consensus from experts in the field, or have diagnostic and/or prognostic significance of certain diseases based on **well- powered studies with expert consensus**.
Tier II: Variants of Potential Clinical Significance	2C	Biomarkers that predict response or resistance to therapies approved by FDA or professional **societies for a different tumor type (ie, off-label use of a drug)**, serve as inclusion criteria for clinical trials, or have diagnostic and/or prognostic significance based on the results of multiple small studies.
	2D	Biomarkers that show plausible therapeutic significance based on preclinical studies, or may assist disease diagnosis and/or prognosis themselves or along with other biomarkers based on **small studies or multiple case reports** with no consensus.
Tier III: Variants of Unknown Clinical Significance		Not observed at a significant allele frequency in the general or specific subpopulation databases, or pan-cancer or tumor-specific variant databases No convincing published evidence of cancer association.
Tier IV: Benign or Likely Benign Variants		Observed at significant allele frequency in the general or specific subpopulation databases.

DISCLAIMER

- This report was generated using the materials and methods as recommended which required the use of quality reagents, protocols, instruments, software, databases and other items, some of which were provided or made accessible by third

parties. A defect or malfunction in any such reagents, protocols, instruments, software, databases and/or other items may compromise the quality or accuracy of the report.

- The report has been created based on, or incorporated inferences to, various scientific manuscripts, references, and other sources of information, including without limitation manuscripts, references, and other sources of information that were prepared by third parties that describe correlations between certain genetic mutations and particular diseases (and/or certain therapeutics that may be useful in ameliorating the effects of such diseases). Such information and correlations are subject to change over time in response to future scientific and medical findings. MolQ Laboratory makes no representation or warranty of any kind, expressed or implied, regarding the accuracy of the information provided by or contained in such manuscripts, references, and other sources is later determined to be inaccurate, the accuracy and quality of the Report may be adversely impacted. MolQ Laboratory is not obligated to notify you of any of the impact that future scientific or medical findings may have on the report.
- The report must always be interpreted and considered within the clinical context, and a physician should always consider the report along with all other pertinent information and data that a physician would prudently consider prior to providing a diagnosis or developing and implementing a plan of care for the patient. The report should never be considered or relied upon alone in making any diagnosis or prognosis. The manifestations of many diseases are caused by more than one gene variant, a single gene variant may be relevant to more than one disease, and certain relevant gene variants may not have been considered in the report. In addition, many diseases are caused or influenced by modifier genes, epigenetic factors, environmental factors, and other variables that are not addressed by the report. This report is based on a Next Generation Assay which does not distinguish between a somatic and a germline variant. If germline variant is in question, further testing is recommended. The report provided by MolQ Laboratory is on a "as is" basis. MolQ Laboratory makes no representation or warranty of any kind, expressed or implied, regarding the report. In no event will MolQ Laboratory be liable for any actual damages, indirect damages, and/or special or consequential damages arising out of or in any way connected with the Report, your use of the report, your reliance on the report, or any defect or inaccurate information included within the report.
- Medical knowledge and annotation are constantly updated and reflects the current knowledge at the time.
- Due to inherent technology limitations of the assay, not all bases of the exome can be covered by this test. Accordingly, variants in regions of insufficient coverage may not be identified and/or interpreted. Therefore, it is possible that certain variants are present in one or more of the genes analyzed, but have not been detected. The variants not detected by the assay that was performed may/ may not impact the phenotype.
- It is also possible that a pathogenic variant is present in a gene that was not selected for analysis and/or interpretation in cases where insufficient phenotypic information is available.
- The report shall be generated within turnaround time (TAT), however, such TAT may vary depending upon the complexity of test(s) requested. MolQ Laboratory under no circumstances will be liable for any delay beyond afore mentioned TAT.
- It is hereby clarified that the report(s) generated from the test(s) do not provide any diagnosis or opinion or recommends any cure in any manner. MolQ Laboratory hereby recommends the patient and/or the guardians of the patients, as the case may be, to take assistance of the clinician or a certified physician or doctor, to interpret the report(s) thus generated. MolQ Laboratory hereby disclaims all liability arising in connection with the report(s).
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- This is a laboratory developed test and the development and the performance characteristics of this test was determined by reference laboratory as required by the CLIA 1988 regulations. The report, and the tests used to generate the Report have not been cleared or approved by the US Food and Drug Administration (FDA). The FDA has determined that such clearance or approval is not necessary. The test results have scientifically shown to be clinically useful.

LIMITATIONS

- Testing has been performed assuming that the sample received belongs to the above-named individual(s) and any stated relationships between individuals are accepted as true.
- Due to inherent technology limitations, coverage is not uniform across all regions. Hence pathogenic variants present in areas of insufficient coverage may not be analyzed/ reported.
- The classification and interpretation of all the variants in this assay reflects the current state of scientific understanding at the time this report was issued. In some instances, the classification and interpretation of such variants may change as new scientific information comes to light.
- Test results should be interpreted in context of clinical findings, tumor sampling, histopathology, and other laboratory data.

- If results obtained do not match other clinical laboratory findings, please contact the laboratory for possible. Misinterpretation of results may occur if the information provided is inaccurate or incomplete.
- Genetic testing is highly accurate. Rarely, inaccurate results may occur for various reasons. These reasons include, but are not limited to mislabelled samples, inaccurate reporting of clinical/medical information, rare technical errors or unusual circumstances such as bone marrow transplantation, blood transfusion; or the presence of change(s) in such a small percentage of cells that may not be detectable by the test (mosaicism).

APPENDIX 2: GENE LIST

Gene	Gene	Gene	Gene	Gene	Gene	Gene
ABL1	*CDKN2A*	*FANCF*	*HIST3H3*	*MEN1*	*PIK3R3*	*SMAD3*
ABL2	*CDKN2B*	*FANCG*	*HLA-A*	*MET*	*PIM1*	*SMAD4*
ACVR1	*CDKN2C*	*FANCI*	*HLA-B*	*MGA*	*PLCG2*	*SMARCA4*
ACVR1B	*CEBPA*	*FANCL*	*HLA-C*	*MITF*	*PLK2*	*SMARCB1*
AKT1	*CENPA*	*FAS*	*HNF1A*	*MLH1*	*PMAIP1*	*SMARCD1*
AKT2	*CHD2*	*FAT1*	*HNRNPK*	*MLL*	*PMS1*	*SMC1A*
AKT3	*CHD4*	*FBXW7*	*HOXB13*	*MLLT3*	*PMS2*	*SMC3*
ALK	*CHEK1*	*FGF1*	*HRAS*	*MPL*	*PNRC1*	*SMO*
ALOX12B	*CHEK2*	*FGF10*	*HSD3B1*	*MRE11A*	*POLD1*	*SNCAIP*
ANKRD11	*CIC*	*FGF14*	*HSP90AA1*	*MSH2*	*POLE*	*SOCS1*
ANKRD26	*CREBBP*	*FGF19*	*ICOSLG*	*MSH3*	*PPARG*	*SOX10*
APC	*CRKL*	*FGF2*	*ID3*	*MSH6*	*PPM1D*	*SOX17*
AR	*CRLF2*	*FGF23*	*IDH1*	*MST1*	*PPP2R1A*	*SOX2*
ARAF	*CSF1R*	*FGF3*	*IDH2*	*MST1R*	*PPP2R2A*	*SOX9*
ARFRP1	*CSF3R*	*FGF4*	*IFNGR1*	*MTOR*	*PPP6C*	*SPEN*
ARID1A	*CSNK1A1*	*FGF5*	*IGF1*	*MUTYH*	*PRDM1*	*SPOP*
ARID1B	*CTCF*	*FGF6*	*IGF1R*	*MYB*	*PREX2*	*SPTA1*
ARID2	*CTLA4*	*FGF7*	*IGF2*	*MYC*	*PRKAR1A*	*SRC*
ARID5B	*CTNNA1*	*FGF8*	*IKBKE*	*MYCL1*	*PRKCI*	*SRSF2*
ASXL1	*CTNNB1*	*FGF9*	*IKZF1*	*MYCN*	*PRKDC*	*STAG1*
ASXL2	*CUL3*	*FGFR1*	*IL10*	*MYD88*	*PRSS8*	*STAG2*
ATM	*CUX1*	*FGFR2*	*IL7R*	*MYOD1*	*PTCH1*	*STAT3*
ATR	*CXCR4*	*FGFR3*	*INHA*	*NAB2*	*PTEN*	*STAT4*
ATRX	*CYLD*	*FGFR4*	*INHBA*	*NBN*	*PTPN11*	*STAT5A*
AURKA	*DAXX*	*FH*	*INPP4A*	*NCOA3*	*PTPRD*	*STAT5B*
AURKB	*DCUN1D1*	*FLCN*	*INPP4B*	*NCOR1*	*PTPRS*	*STK11*
AXIN1	*DDR2*	*FLI1*	*INSR*	*NEGR1*	*PTPRT*	*STK40*
AXIN2	*DDX41*	*FLT1*	*IRF2*	*NF1*	*QKI*	*SUFU*
AXL	*DHX15*	*FLT3*	*IRF4*	*NF2*	*RAB35*	*SUZ12*
B2M	*DICER1*	*FLT4*	*IRS1*	*NFE2L2*	*RAC1*	*SYK*
BAP1	*DIS3*	*FOXA1*	*IRS2*	*NFKBIA*	*RAD21*	*TAF1*
BARD1	*DNAJB1*	*FOXL2*	*JAK1*	*NKX2-1*	*RAD50*	*TBX3*
BBC3	*DNMT1*	*FOXO1*	*JAK2*	*NKX3-1*	*RAD51*	*TCEB1*
BCL10	*DNMT3A*	*FOXP1*	*JAK3*	*NOTCH1*	*RAD51B*	*TCF3*
BCL2	*DNMT3B*	*FRS2*	*JUN*	*NOTCH2*	*RAD51C*	*TCF7L2*
BCL2L1	*DOT1L*	*FUBP1*	*KAT6A*	*NOTCH3*	*RAD51D*	*TERC*
BCL2L11	*E2F3*	*FYN*	*KDM5A*	*NOTCH4*	*RAD52*	*TERT*
BCL2L2	*EED*	*GABRA6*	*KDM5C*	*NPM1*	*RAD54L*	*TET1*
BCL6	*EGFL7*	*GATA1*	*KDM6A*	*NRAS*	*RAF1*	*TET2*
BCOR	*EGFR*	*GATA2*	*KDR*	*NRG1*	*RANBP2*	*TFE3*
BCORL1	*EIF1AX*	*GATA3*	*KEAP1*	*NSD1*	*RARA*	*TFRC*
BCR	*EIF4A2*	*GATA4*	*KEL*	*NTRK1*	*RASA1*	*TGFBR1*
BIRC3	*EIF4E*	*GATA6*	*KIF5B*	*NTRK2*	*RB1*	*TGFBR2*
BLM	*EML4*	*GEN1*	*KIT*	*NTRK3*	*RBM10*	*TMEM127*
BMPR1A	*EP300*	*GID4*	*KLF4*	*NUP93*	*RECQL4*	*TMPRSS2*
BRAF	*EPCAM*	*GLI1*	*KLHL6*	*NUTM1*	*REL*	*TNFAIP3*

Comprehensive Panel- 500 Genes

BRCA1	*EPHA3*	*GNA11*	*KMT2B*	*PAK1*	*RET*	*TNFRSF14*
BRCA2	*EPHA5*	*GNA13*	*KMT2C*	*PAK3*	*RFWD2*	*TOP1*
BRD4	*EPHA7*	*GNAQ*	*KMT2D*	*PAK7*	*RHEB*	*TOP2A*
BRIP1	*EPHB1*	*GNAS*	*KRAS*	*PALB2*	*RHOA*	*TP53*
BTG1	*ERBB2*	*GPR124*	*LAMP1*	*PARK2*	*RICTOR*	*TP63*
BTK	*ERBB3*	*GPS2*	*LATS1*	*PARP1*	*RIT1*	*TRAF2*
C11orf30	*ERBB4*	*GREM1*	*LATS2*	*PAX3*	*RNF43*	*TRAF7*
CALR	*ERCC1*	*GRIN2A*	*LMO1*	*PAX5*	*ROS1*	*TSC1*
CARD11	*ERCC2*	*GRM3*	*LRP1B*	*PAX7*	*RPS6KA4*	*TSC2*
CASP8	*ERCC3*	*GSK3B*	*LYN*	*PAX8*	*RPS6KB1*	*TSHR*
CBFB	*ERCC4*	*H3F3A*	*LZTR1*	*PBRM1*	*RPS6KB2*	*U2AF1*
CBL	*ERCC5*	*H3F3B*	*MAGI2*	*PDCD1*	*RPTOR*	*VEGFA*
CCND1	*ERG*	*H3F3C*	*MALT1*	*PDCD1LG2*	*RUNX1*	*VHL*
CCND2	*ERRFI1*	*HGF*	*MAP2K1*	*PDGFRA*	*RUNX1T1*	*VTCN1*
CCND3	*ESR1*	*HIST1H1C*	*MAP2K2*	*PDGFRB*	*RYBP*	*WISP3*
CCNE1	*ETS1*	*HIST1H2BD*	*MAP2K4*	*PDK1*	*SDHA*	*WT1*
CD274	*ETV1*	*HIST1H3A*	*MAP3K1*	*PDPK1*	*SDHAF2*	*XIAP*
CD276	*ETV4*	*HIST1H3B*	*MAP3K13*	*PGR*	*SDHB*	*XPO1*
CD74	*ETV5*	*HIST1H3C*	*MAP3K14*	*PHF6*	*SDHC*	*XRCC2*
CD79A	*ETV6*	*HIST1H3D*	*MAP3K4*	*PHOX2B*	*SDHD*	*YAP1*
CD79B	*EWSR1*	*HIST1H3E*	*MAPK1*	*PIK3C2B*	*SETBP1*	*YES1*
CDC73	*EZH2*	*HIST1H3F*	*MAPK3*	*PIK3C2G*	*SETD2*	*ZBTB2*
CDH1	*FAM123B*	*HIST1H3G*	*MAX*	*PIK3C3*	*SF3B1*	*ZBTB7A*
CDK12	*FAM175A*	*HIST1H3H*	*MCL1*	*PIK3CA*	*SH2B3*	*ZFHX3*
CDK4	*FAM46C*	*HIST1H3I*	*MDC1*	*PIK3CB*	*SH2D1A*	*ZNF217*
CDK6	*FANCA*	*HIST1H3J*	*MDM2*	*PIK3CD*	*SHQ1*	*ZNF703*
CDK8	*FANCC*	*HIST2H3A*	*MDM4*	*PIK3CG*	*SLIT2*	*ZRSR2*
CDKN1A	*FANCD2*	*HIST2H3C*	*MED12*	*PIK3R1*	*SLX4*	
CDKN1B	*FANCE*	*HIST2H3D*	*MEF2B*	*PIK3R2*	*SMAD2*	