

**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**

**Pathogenic variant was detected in *BRCA2* gene.**  
**Tumor Mutation Burden is 5.68 Mut/Mb (TMB-Intermediate).**  
**Microsatellite Instability (MSI) is stable.**

**VARIANT DETECTED AS PER NCCN GUIDELINES**

The given specimen contains ***BRCA2*** (p.Q3295\*, VAF = 43.54%). *BRCA2* mutations have been linked to prostate cancer, according to numerous research. (PMID: 31915789).

**OTHER VARIANTS DETECTED**

***ZMYM3*** mutations (p.L1180Pfs\*73; p.P1177Sfs\*75, VAF = 12.92%;12.34%). ***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)	(In other cancer type)	Tier <sup>1</sup>
<i>BRCA2</i> NM_000059.4 (chr13:32972533)	-	c.9883C>T (p.Gln3295*) Exon 27	43.54%	Pathogenic	abiraterone + niraparib <sup>i,ii</sup> abiraterone + olaparib <sup>i,ii</sup>	abiraterone + niraparib <sup>i,ii</sup> bevacizumab + olaparib <sup>i,ii</sup> olaparib <sup>i,ii</sup> olaparib + hormone therapy <sup>i</sup> rucaparib <sup>i</sup> talazoparib + hormone therapy <sup>i</sup> niraparib	IIc

\*Public data sources included in relevant therapies: FDA<sup>1</sup>, NCCN, EMA<sup>1</sup>, ESMO

#Based on Clinvar version 20220709

<sup>1</sup>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_3535delCCCCA CAC p.Pro1177Serfs*75	12.34%	Exon 22	-

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**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<sup>1,2</sup>. Specifically, BRCA1/2 are required for repair of chromosomal double strand breaks (DSBs) which are highly unstable and compromise genome integrity<sup>1,2</sup>. 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<sup>3,4,5</sup>. 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%<sup>3,6</sup>.

**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<sup>7,8,9,10,11,12,13,14</sup>. 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<sup>15,16</sup>.

**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)<sup>17</sup>. Inhibitors targeting PARP induce synthetic lethality in recombination deficient *BRCA1/2* mutant cells<sup>18,19</sup>. Consequently, several PARP inhibitors have been FDA approved for *BRCA1/2*-mutated cancers. Olaparib<sup>20</sup> (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<sup>20</sup> 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<sup>21</sup> is also approved (2020) for deleterious gBRCAm or sBRCAm mCRPC. Talazoparib<sup>22</sup> (2018) is indicated for the treatment of gBRCAm HER2-negative locally advanced or metastatic breast cancer. Additionally, talazoparib<sup>22</sup> in combination with enzalutamide is approved (2023) for metastatic castration-resistant prostate cancer (mCRPC) with mutations in HRR genes that includes BRCA2. Niraparib<sup>23</sup> (2017) is another PARPi approved for the treatment of epithelial ovarian, fallopian tube, or primary peritoneal

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cancers with a deleterious or suspected deleterious BRCA mutation. Niraparib in combination with abiraterone acetate<sup>24</sup> 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<sup>25</sup>. One of the most common mechanisms of resistance includes secondary intragenic mutations that restore BRCA1/2 functionality<sup>26</sup>. 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<sup>27</sup>, 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<sup>28</sup>. 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)<sup>29</sup>.

**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<sup>15,16</sup>. 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<sup>30</sup>. Biallelic deletion of *ZMYM3* is observed in 3% of cholangiocarcinoma and 2% of sarcoma and kidney chromophobe<sup>15,16</sup>.

**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 TaqMan™ 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**

<b>Tier I:</b> 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 <b>professional guidelines</b> as <b>therapeutic, diagnostic, and/or prognostic biomarkers</b> 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 <b>well- powered studies with expert consensus</b> .
<b>Tier II:</b> Variants of Potential Clinical Significance	2C	Biomarkers that predict response or resistance to therapies approved by FDA or professional <b>societies for a different tumor type (ie, off-label use of a drug)</b> , 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 <b>small studies or multiple case reports</b> with no consensus.
<b>Tier III:</b> 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.
<b>Tier IV:</b> 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.
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  - 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).
  - In a very few cases genetic test may not show the correct results, e.g. because of the quality of the material provided to MolQ Laboratory. In case where any test provided by MolQ Laboratory fails for unforeseeable or unknown reasons that cannot be influenced by MolQ Laboratory in advance, MolQ Laboratory shall not be responsible for the incomplete, potentially misleading or even wrong result of any testing if such could not be recognized by MolQ Laboratory in advance.
  - 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.

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- 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



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