Comprehensive Tumor Panel (270 genes)

Test Description

The MolQ's Comprehensive Tumor Panel assay to detect cancer causing genomic alterations (SNVs, Indels, and Fusions) that may provide treatment benefit to the patient via is high throughput next-generation sequencing in solid tumor malignancies. It comprises of 271 key cancer genes for the assessment of various biomarkers. Genomic alterations such as base substitutions, insertions, deletions in the coding exons (±10 bp flanking intronic region) are screened in 231 genes and gene fusions in 91 oncogenes in patient's tumor DNA and RNA respectively.

Patient Demographic

Name: Bharti Agarwal
Sex: Female **Date of Birth/Age**: 47 years
Disease: Gastrointestinal Stromal Tumors (GIST)

Clinician

Clinician Name: Dr Sandeep K. Jasuja
Medical Facility: SMS Hospital, Jaipur
Pathologist: Not Provided

Specimen

Booking ID: 012203020059
Site: Gastric
Sample Type: FFPE block H22-2438
Date of Collection: 02-03-2022
Date of Booking: 02-03-2022

CLINICAL SYNOPSIS

Gastric mass biopsy showing gastrointestinal stromal tumor [as per the histopathology report dated 10th February 2022]. The tumor was identifiable in the block [H22-2438] and it was adequate (>10%) for further analysis.

TEST RESULT SUMMARY

Potential Treatment Impacts			Prognostic and Diagnostic Findings		Clinical Trials
11 Effective	0 Ineffective	0 Safety	1 Prognostic	0 Diagnostic	10 Trials

Genomic Variants

Genomic Alterations	Variant Allele Frequency %	AMP Classification^	Evidence Level^	Treatment	Treatment Benefit	Drug Approval^$	Clinical Trials
KIT p.Trp557_Lys558del (INFRAME-DEL)	74.4	Tier IB	6 Clinical	Prognostic			0
KIT p.Trp557_Lys558del (INFRAME-DEL)	74.4	Tier IB	6 Clinical	Imatinib	Effective	Approved	2
KIT p.Trp557_Lys558del (INFRAME-DEL)	74.4	Tier IB	6 Clinical	Sunitinib	Effective	Approved	3
KIT p.Trp557_Lys558del (INFRAME-DEL)	74.4	Tier IID	3 Preclinical	Midostaurin	Effective	Off-label	0
KIT p.Trp557_Lys558del (INFRAME-DEL)	74.4	Tier IID	3 Preclinical	Regorafenib	Effective	Approved	3

KIT p.Trp557_Lys558del (INFRAME-DEL)	74.4	Tier IID	3 Preclinical	Avapritinib	Effective	Approved	1
KIT p.Trp557_Lys558del (INFRAME-DEL)	74.4	Tier IID	3 Preclinical	Ripretinib	Effective	Approved	1
KIT p.Trp557_Lys558del (INFRAME-DEL)	74.4	Tier IID	3 Preclinical	Dasatinib	Effective	Off-label	2
KIT p.Trp557_Lys558del (INFRAME-DEL)	74.4	Tier IID	3 Preclinical	Sorafenib	Effective	Off-label	2
KIT p.Trp557_Lys558del (INFRAME-DEL)	74.4	Tier IID	3 Preclinical	Cabozantinib	Effective	Off-label	2

No clinically significant fusion has been detected in this sample

^Refer to Glossary section for the classification criteria details.

$Drug Approvals are based on US-FDA Guidelines. Kindly refer to local guidelines if required.

Note: Decisions regarding treatment action plan should not be solely based on this test results. These findings are highly recommended to be correlated with the patient's clinical, pathological, and family history for decisions on diagnosis, prognosis or therapeutics.

GLOSSARY

AMP Classification Criteria

Displays the classification of a biomarker according to the recommendations of the Association for Molecular Pathology (AMP) [PMID: 27993330].

Tier	Criteria
Tier IA	Variants of strong clinical significance. FDA-approved therapy or biomarkers included in professional guidelines
Tier IB	Variants of strong clinical significance. Well-powered studies with consensus from experts in the field.
Tier IIC	Variants of potential clinical significance. FDA-approved therapies for different cancer types or investigational therapies. Multiple small published studies with some consensus.
Tier IID	Variants of potential clinical significance. Preclinical trials or a few case reports without consensus.
Tier III	Variants of unknown clinical significance.
Tier IV	Benign or likely benign variants.

Evidence Level

The Evidence Level, values 7-1 indicate the reliability of a biomarker to predict a specific patient outcome. This can include predictive treatment effects; in this case, the scores 7-1 apply for biomarkers associated with a single drug or drug combination. The Evidence levels are defined as follows:

Score	Definition
7, Clinically approved	Variant approved by a regulatory agency to predict a specific effect (i.e., response, resistance, or toxicity) in the patient's disease or cancer type.
6, Clinical	Variant has not yet been approved by a regulatory agency for the patient's disease but has been observed in at least one large cohort study to predict a specific effect of the drug (i.e., to be effective, resistance) in the patient's disease. The biomarker has been approved by a regulatory agency to predict a specific effect of the drug (response, resistance) with other diseases.
5, Clinical	Variant has not yet been approved by a regulatory agency for the patient's disease but has been observed to predict a specific effect of the drug (i.e., response, resistance) on patients with other diseases or conditions. For variants predicting a drug to be effective or resistant, there is evidence from some patients in several cohort studies and preclinical evidence.
4, Clinical	The variant has not yet been approved by a regulatory agency for the patient's disease. However, this variant has been observed to predict a specific effect of the drug (i.e., response, resistance) on patients with other diseases or conditions. For variants predicting a drug to be effective or resistant, there is evidence from a few clinical case reports and additional preclinical evidence. For variants predicting a drug to be toxic, there is evidence from a prospective study, >1 retrospective studies, or >1 cohort studies.
3, Preclinical	Variant has not yet been observed/tested in patients to predict a specific effect but has been observed in preclinical experiments. There is experimental evidence from cell lines or mouse models, for example.
2, Preclinical	Variant has not yet been observed/tested in patients or preclinical models to predict a specific effect. However, this effect can be inferred when drug-sensitivity data are available for another variant and if the two variants have the identical functional impact on the same downstream pathway
1, Preclinical	Variant has not yet been observed/tested in patients or preclinical models to predict a specific effect. However, this effect can be inferred when drug-sensitivity data are available for another variant and if both variants have the identical functional impact on the protein.

Drug Approval

The development stage of the treatment for the patient's indication as per US-FDA guidelines.

Stage	Definition
Approved	This drug is launched for the primary or a secondary patient disease
Off-Label	This drug is launched for a disease other than the primary or secondary patient diseases
Investigational	This drug is currently under clinical development in the patient disease.
Other	None of the other stages are applicable. The drug or drug class is, for example, suspended, discontinued, or withdrawn.

Medications with potential for adverse reaction or ineffectiveness:

Refers to the identified treatments that are predicted to be associated with negative physiological responses to a drug therapy (i.e., drug resistance and toxicity) based on the biomedical evidence.

Potential impact

The specific drug effect predicted by the identified mutation (i.e. response, resistance, or toxicity).

Treatment

The generic name of the therapeutic agent listed on the report

ACTIONABLE BIOMARKER DETAILS

KIT (p.Trp557_Lys558del)

Gene: *KIT*

Exon: 11

Nucleotide Change: chr4:g.54727435_54727440del

cDNA change: c.1667_1672del

Transcript ID: ENST00000288135.6

Protein Change: p.Trp557_Lys558del

Variant Allele Depth: 122x

Variant Allele Frequency: 74.4%

Variant Type: INFRAME-DEL

Population MAF:

***In-silico* Predictions**: NA(SIFT); NA(LRT); NA(Polyphen2)

Gene Function: Oncogene

Gene Summary

The proto-oncogene *KIT* encodes a type 3 transmembrane receptor tyrosine kinase that is activated through dimerization and autophosphorylation upon binding by its ligand, stem cell factor (SCF) also known as mast cell growth factor (MGF) [PMID: 9438854]. KIT activation results in increased intracellular signaling through several pathways including PI3K, MAPK and STAT, ultimately leading to cell proliferation and survival [PMID: 22089421]. Activating *KIT* mutations occur in 80 - 90% of GISTs and are distributed over multiple exons with different frequencies (exons 11 (66.1%), exon 9 (13%), exon 13 (1.2%), and exon 17 (0.6%)) [PMID: 11719439]. There are at least eight small molecule tyrosine kinase inhibitors (TKIs) targeting KIT that have been approved by the US Food and Drug Administration with the efficacy of each TKI strongly depending on the location of the activating *KIT* mutation [PMID: 19164557].

Clinical and Therapeutic Relevance

The mast/stem cell growth factor receptor KIT (SCFR, CD117) is a receptor tyrosine kinase that activates RAS/MAPK, PI3K/AKT, and JAK/STAT signaling pathways to promote cell proliferation and migration. Small in-frame mutations in exon 11 at codons 550-561 likely abolish the autoinhibitory fragment Y553-I563 for KIT kinase activity. Mutations in *KIT* exon 11 are frequently seen in gastrointestinal stromal tumors (GIST) and are associated with improved treatment outcomes to imatinib as compared to patients with *KIT* exon 9 mutations. Patients with a deletion in KIT exon 11 had a worse 5-year RFS rate than those with any other *KIT* exon 11 mutation. Tumors of GIST patients with in-frame mutations in this region were reported to respond to imatinib and sunitinib. Preclinical models with such mutations are sensitive to regorafenib, sorafenib, nilotinib, dasatinib, ponatinib, cabozantinib, midostaurin, ripretinib, and avapritinib.

PubMed References: 33212994, 21527588, 32350132, 31205508, 30792533, 30684595, 30274985, 29093181, 28334365, 27600498

ADDITIONAL BIOMARKERS DETECTED

This section provides information about variants that do not have any therapeutic value. However, these variants may or may not have a likely oncogenic effect.

Gene	Exon	Nucleotide change	Protein change	Alternate allele Depth (x)	Allele Burden (%)	Functional predictions	Population MAF (%)
APC	16	ENST00000257430.9 c.5611G>T chr5:112841205: G>T	p.Asp1871Tyr	196x	45.8%	D(SIFT); D(LRT); PrD(Polyphen2)	0.019968% (1000G); 0 (gnomAD)
XPC	6	ENST00000285021.12 c.637C>G chr3:14165570: G>C	p.Leu213Val	491x	49.8%	D(SIFT); D(LRT); PrD(Polyphen2)	0 (1000G); 0 (gnomAD)
APC	12	ENST00000257430.9 c.1483A>G chr5:112827182: A>G	p.Ile495Val	239x	43.4%	T(SIFT); N(LRT); BN(Polyphen2)	0 (1000G); 0.001395% (gnomAD)

No clinically significant fusion has been detected in this sample

DISCLAIMER

- **Decisions regarding treatment action plan should not be solely based on these test results. These findings are highly recommended to be correlated with the patient's clinical, pathological, radiological and family history for decisions on diagnosis, prognosis, or therapeutics.**
- The therapy information provided in this report is based on FDA approved drugs data, NCCN guidelines, peer-reviewed published literature, standard clinical databases, and strength of biomarker results. These therapies may or may not be suitable/beneficial to a particular patient. This clinical report summarizes potentially effective medications, potentially ineffective medications, and medications that may pose a higher risk of adverse reactions by mapping the patient's genetic alterations to the biomedical reference information. The report may also provide prognostic and diagnostic biomarkers detected or shown for the given disease context.
- The clinical trials information provided in this report is compiled from www.clinicaltrials.gov as per currently available data, however completeness of information provided herein cannot be guaranteed. This information should only be used as a guide and specific eligibility criteria should be reviewed thoroughly for the concerned patient. MolQ Labs does not guarantee or promise an enrolment in any clinical trials.
- The identification of a genomic biomarker does not necessarily imply pharmacological effectiveness or ineffectiveness. The medications identified by the treating physician may or may not be suitable for use on a particular patient. Thus, the clinical report does not guarantee that any particular agent will be effective in the treatment of any particular condition. Also, the absence of a treatment option does not determine the effectiveness or predict an ineffective or safety-relevant effect of a medication selected by the treating physician.
- The classification and clinically relevant information for the reported variants is based on peer-reviewed publications, public clinical databases, medical guidelines (NCCN, ASCO, AMP) or other publicly available information and it has been ensured that the information provided is up to date at the time of report generated, however continuous updates may happen in public domains. Also, the classification of variants can change based on the updated literature evidence. Re-analysis of the results can be requested at additional cost.
- This test is performed on the patient's tumor sample without a paired blood sample; therefore, it may include variations which may be of germline origin. However, this test is designed and validated for the detection and reporting of somatic genomic variants only and does not discriminate between germline and somatic variants. If clinically warranted, appropriate germline testing and genetic counselling for the patient should be considered for further evaluation.
- Due to poor quality of FFPE tissue blocks, the QC parameters for extracted DNA and RNA may not pass to proceed further with the testing, therefore there is a possibility of assay failure at various steps (DNA/RNA QC, Library QC, Bioinformatics QC) or compromised results that include low gene coverage and low variant depth. However, sample status in such scenarios shall be sent through mail to the ordering clinician.
- This test has been validated at Reference Laboratory and the limit of detection (LOD) of allele fraction for SNVs is ≥5%, short Indels is ≥10% and for fusions is ≥10 spanning reads. However, the report may include, at the discretion of laboratory director, the variants with lower allele burden (3-5%) having strong or potential clinical significance or those have been reported earlier in the patient. Variants with <1% allele fraction and variants of uncertain significance with <5% allele fraction are not routinely reported. However, possibility of false negative or false positive below the limit of detection of this assay cannot be ruled out.
- Detection of large insertions, deletions, copy number variations and deep intronic variations are beyond the scope of this test.
- **Additional case specific disclaimer: None**

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APPENDIX 1: TEST METHODOLOGY

SNVs, Indels and Fusions

The comprehensive tumor panel§ testing was performed on DNA and RNA extracted from patient's FFPE tissue blocks. A histopathologic review was performed to determine the tumor content in the FFPE block. Tumor genomic DNA extracted from FFPE tissue block was used to perform targeted gene capture using a custom capture kit. The QC passed libraries were sequenced on Illumina sequencing platform. The DNA sequences obtained were aligned to human reference genome (GRCh38) using BWA program [PMID: 20080505, PMID: 23155063] and mutation calling was performed using Lofreq pipeline version 2 [PMID: 23066108, PMID: 19505943]. Only non- synonymous, splice site and other disease-causing mutations found in the target regions were used for clinical interpretation. The mutations were annotated using reference laboratory in-house annotation pipeline (VariMAT). Gene annotation of the variants was performed using VeP program [PMID: 27268795] against the Ensembl release 90 human gene Model# [PMID: 31691826]. Clinically relevant mutations were annotated using peer- reviewed publications, public clinical databases (ClinVar, HGMD, CiViC) medical guidelines (NCCN, ASCO, AMP). The common variants were filtered out based on the minor allele frequency (MAF) in various population databases (1000G, ExAC, gnomAD, GAsP, dbSNP, OncoCrDb (Reference Laboratory in- house curated database)) and only variants with MAF <0.01% were considered for reporting [PMID: 26432245, PMID: 27535533, https://esp.gs.washington.edu/drupal/]. Tumor RNA was extracted from FFPE tissue block and targeted sequencing was performed using Reference Laboratory custom design. The reads obtained from sequencer are pre-processed and then aligned to the Human transcriptome model (Genome Version: hg19) using STAR aligner [PMID: 23104886]. Gene fusions at the RNA level were determined using multiple published fusion detection programs [https://www.biorxiv.org/content/10.1101/011650v1, https://www.biorxiv.org/content/10.1101/120295v1, PMID: 21593131]. These tools have been validated and fine-tuned to Reference Laboratory assays. The unique fusion coordinates predicted by different programs are checked for their specificity for confirmation. The fusion events are filtered out based on internal controls and fusions identified in healthy individuals. The fusions were reported based on the total read depth (number of spanning reads and split reads) supporting the finding [PMID: 26019724]. Reportable alterations in DNA and fusions in RNA are prioritized, classified, and reported based on AMP-ASCO-CAP guidelines [PMID: 27993330] and NCCN guidelines.

#The transcript used for clinical reporting generally represents the canonical transcript (according to Ensembl release 90 human gene model), which is usually the longest coding transcript with strong/multiple supporting evidence. However, clinically relevant variants annotated in alternate complete coding transcripts could also be reported. Variants annotated on incomplete, and nonsense mediated decay transcripts are not reported.

"This test is developed, and its performance characteristics is determined by Reference Laboratory".

Accuracy	Tumor Cellularity %	Variant Allele Ratio	Performance
Sensitivity: Base substitutions	≥10%	≥15%	99.5%
Sensitivity: Indels	≥10%	≥15%	>98%
Average Coverage across all targets		>250X	

Reproducibility	Concordance
Intra-batch precision	100%
Inter-batch precision	99%

APPENDIX 2: CLINICAL TRIALS

The following trials are potentially best suited for your patient's indication, considering all reported treatment recommendations. See https://clinicaltrials.gov (clinical trials from NCT) or https://trialsearch.who.int (clinical trials from other registries) for more information.

Clinical trials in total: 10 **Trial Countries:** IN-India, US-United States

S. No	Title	Phase and ID	Intervention	Disease	Age & Sex
1	Avapritinib for the Treatment of CKIT or PDGFRA Mutation-Positive Locally Advanced or Metastatic Malignant Solid Tumors	Phase 2 NCT04771520	Avapritinib		Age: 18 Sex: Both
	Status: Recruiting				
	Stratification: KIT mutation: activating mutation | PDGFRA mutation: activating mutation				
	Exclusion: KIT SNV: p.T670I|KIT SNV: p.V654A				
2	A Trial to Learn Whether Regorafenib in Combination with Nivolumab Can Improve Tumor Responses and How Safe it is for Participants with Solid Tumors	Phase 2 NCT04704154	Regorafenib	Solid Tumor	Age: 18 Sex: Both
	Status: Recruiting				
	Stratification: CDKN2A protein expression: expression				
	Exclusion: MSI: MSI-H|NTRK1 fusion gene: any| MSI: dMMR				
3	Study to Assess the Long-term Safety of Lenvatinib Monotherapy, a Lenvatinib Combination Regimen, or a Comparator Treatment Arm to Cancer Participants in Eisai Sponsored Lenvatinib Trials	Phase 2 NCT03477175	Sorafenib	Solid Tumor	Age: 18 Sex: Both
	Status: Recruiting				
4	TAPUR: Testing the Use of Food and Drug Administration (FDA) Approved Drugs That Target a Specific Abnormality in a Tumor Gene in People with Advanced Stage Cancer (TAPUR)	Phase 2 NCT02693535	Regorafenib Sunitinib	Solid Tumor	Age: 12 Sex: Both
	Status: Recruiting				
	Stratification: BRCA1 mutation: germline mutation | BRCA2 mutation: inactivating mutation | FLT4 mutation: mutations | PDGFRB SCNA: amp | SRC mutation: mutation | EPHA2 mutation: mutations | PDGFRB mutation: mutations | RAF1 SCNA: amp | CDK4 SCNA: amp | PDGFRA mutation: mutation | BRCA1 mutation: inactivating mutation | FLT4 protein expression: overexpression | FLT1 SCNA: amp | MTOR mutation: mutations | ABL1 fusion gene: BCR | KDR mutation: mutations | KIT mutation: mutation | BRAF SNV: p.V600E | CSF1R mutation: mutations | TSC2 mutation: mutation | ERBB2 SCNA: amp | LYN mutation: mutations | LCK mutation: mutations | FYN mutation: mutations | KRAS wild type: wildtype | BCR fusion gene: ABL1 | ATM mutation: mutation | RAF1 mutation: mutations | POLE mutation: alteration | BRAF mutation: mutation | FLT1 mutation: mutations | MET mutation: mutations | ROS1 mutation: mutations | KDR protein expression: overexpression | NRAS wild type: wildtype | KDR SCNA: amp | ATM SCNA: loss | FLT1 protein expression: overexpression | RET SCNA: amp | TSC1 mutation: mutation | BRAF wild type: wildtype | KIT SCNA: amp | ALK mutation: mutations | BRAF SCNA: amp | CDKN2A SCNA: loss | BRCA2 mutation: germline mutation | POLD1 mutation: alteration | FLT4 SCNA: amp | RET mutation: mutations | CDK6 SCNA: amp | YES1 mutation: mutations				
5	Targeted Therapy Directed by Genetic Testing in Treating Patients with Advanced Refractory Solid Tumors, Lymphomas, or Multiple Myeloma (The MATCH Screening Trial)	Phase 2 NCT02465060	Dasatinib Sunitinib		Age: 18 Sex: Both
	Status: Recruiting				
	Stratification: ALK fusion gene: any | RB1 expression | ERBB2 mutation: activating mutation | KIT mutation: mutation | FGFR1 mutation: mutation | DDR2 SNV: p.L239R | PIK3CA mutation: mutation | EGFR SNV: p.T790M | PTCH1 mutation: mutation | BRAF fusion gene: any | MET mutation: exon 14 skipping | PTEN mutation: mutation | CCND3 SCNA: amp | BRAF SNV: p.V600E | KRAS mutation: mutation | BRAF SNV: p.V600D | NF2 mutation: inactivating mutation | SMO mutation: mutation | GNAQ mutation: mutation | DDR2 SNV: p.I638F | MSH2 protein expression: no expression | FGFR2 mutation: mutation | FGFR2 fusion gene: any | FGFR3 mutation: mutation | BRAF SNV: p.V600R | MLH1 protein expression: no expression | PIK3CA SCNA: amp | NF1 mutation: mutation | ERBB2 SCNA: amp | MET SCNA: amp | PTEN expression | NRAS SNV: p.G13X | BRAF SNV: p.V600K | NRAS SNV: p.Q61X				

| HRAS mutation: mutation | ROS1 fusion gene: any | NRAS SNV: p.G12X | AKT1 mutation: mutation | GNA11 mutation: mutation | DDR2 SNV: p.S768R | FGFR1 fusion gene: any | CCND1 SCNA: amp | PTEN SCNA: loss | BRAF mutation: mutation | CCND2 SCNA: amp | NRAS mutation: mutation | FGFR3 fusion gene: any | EGFR mutation: activating mutation

6	A Drug-Drug Interaction Study to Evaluate the Effect of Ripretinib on the Pharmacokinetics of a CYP2C8 Probe Substrate in Patients with Advanced GIST	Phase 1 NCT04530981	Ripretinib	Gastrointestinal Stromal Tumors	Age: 18 Sex: Both
	Status: Recruiting				
7	Testing the Combination of the Anti-cancer Drugs XL184 (Cabozantinib) and Nivolumab in Patients with Advanced Cancer and HIV	Phase 1 NCT04514484	Cabozantinib		Age: 18 Sex: Both
	Status: Recruiting				
8	Serial Measurements of Molecular and Architectural Responses to Therapy (SMMART) PRIME Trial	Phase 1 NCT03878524	Cabozantinib Dasatinib Imatinib Ponatinib Regorafenib Sorafenib Sunitinib		Age: 21 Sex: Both
	Status: Recruiting **Stratification:** ERBB2 protein expression: expression | BCR fusion gene: ABL1				
9	Combination Nilotinib and Paclitaxel in Adults with Relapsed Solid Tumors	Phase 1 NCT02379416	Nilotinib	Solid tumor	Age: 12 Sex: Both
	Status: Recruiting				
10	Ipilimumab and Imatinib Mesylate in Advanced Cancer	Phase 1 NCT01738139	Imatinib	Gastrointestinal Stromal Tumors Solid Tumor	Age: 15 Sex: Both
	Status: Recruiting **Stratification:** KIT mutation: mutations | KIT SNV: p.D816X|KIT SNV: p.T670I|KIT protein expression: overexpression | KIT mutation: exon_18.X|KIT SNV: p.V654X				

APPENDIX 3: GENE LIST WITH COVERAGE

Genes Analyzed in Tumor DNA (231)							
ABRAXAS1	*CCNE1*	*ERBB4*	*FGFR2*	*KMT2C*	*NF2*	*RAD51B*	*SUFU*
AKT1	*CD28*	*ERCC1*	*FGFR3*	*KMT2D*	*NFE2*	*RAD51C*	*TCF3*
AKT2	*CD58*	*ERCC2*	*FGFR4*	*KRAS*	*NOTCH1*	*RAD51D*	*TERT*
AKT3	*CD79A*	*ERCC3*	*FLT1*	*LAMP1*	*NOTCH2*	*RAD54L*	*TET2*
ALK	*CD79B*	*ERCC4*	*FLT3*	*MAP2K1*	*NOTCH3*	*RAF1*	*TFRC*
APC	*CDH1*	*ERCC5*	*FOXL2*	*MAP2K2*	*NPM1*	*RB1*	*TG*
AR	*CDK12*	*ERG*	*FOXO1*	*MCL1*	*NRAS*	*RET*	*TGFBR2*
ARID1A	*CDK4*	*ESR1*	*GEN1*	*MDM2*	*NRG1*	*RHOA*	*TNFAIP3*
ARID1B	*CDK6*	*EZH2*	*GNA11*	*MDM4*	*NTRK1*	*RICTOR*	*TNFRSF14*
ARID2	*CDKN2A*	*FANCA*	*GNA13*	*MEF2B*	*NTRK3*	*ROS1*	*TP53*
ATM	*CDKN2B*	*FANCB*	*GNAQ*	*MET*	*PALB2*	*RPS6KB1*	*TRAF3*
ATR	*CEBPA*	*FANCC*	*GNAS*	*MFHAS1*	*PDGFRA*	*SDHA*	*TRAF7*
ATRX	*CHD8*	*FANCE*	*H3-3A*	*MLH1*	*PDGFRB*	*SDHAF2*	*TSC1*
B2M	*CHEK1*	*FANCI*	*H3C2*	*MLLT3*	*PIK3CA*	*SDHB*	*TSC2*
BAP1	*CHEK2*	*FANCL*	*HNF1A*	*MN1*	*PIK3CB*	*SDHC*	*TSHR*
BARD1	*CIC*	*FBXW7*	*HRAS*	*MPL*	*PIK3CD*	*SDHD*	*VHL*
BCL2	*CIITA*	*FGF1*	*ID3*	*MRE11*	*PIK3CG*	*SF3B1*	*XPC*
BCL6	*CREBBP*	*FGF10*	*IDH1*	*MSH2*	*PIK3R1*	*SLX4*	*XPO1*
BCORL1	*CSF1R*	*FGF14*	*IDH2*	*MSH3*	*PIM1*	*SMAD4*	*XRCC1*
BIRC3	*CSF3R*	*FGF19*	*INPP4B*	*MSH6*	*PMS2*	*SMARCA4*	*XRCC2*
BLM	*CTNNB1*	*FGF2*	*IRF4*	*MTOR*	*POLD1*	*SMARCB1*	*ZAP70*
BRAF	*CXCR4*	*FGF23*	*ITPKB*	*MUTYH*	*POLE*	*SMARCE1*	
BRCA1	*DDR2*	*FGF3*	*JAK2*	*MYB*	*PPARG*	*SMO*	
BRCA2	*DDX3X*	*FGF4*	*JAK3*	*MYBL1*	*PPP2R2A*	*SOCS1*	
BRIP1	*DNMT3A*	*FGF5*	*KDM6A*	*MYC*	*PRDM1*	*SOX11*	
BTK	*EGFR*	*FGF6*	*KDR*	*MYCL*	*PTCH1*	*SRC*	
CARD11	*EIF1AX*	*FGF7*	*KEAP1*	*MYCN*	*PTEN*	*STAT3*	
CCND1	*EP300*	*FGF8*	*KIT*	*MYD88*	*PTPN11*	*STAT5B*	
CCND2	*ERBB2*	*FGF9*	*KLF4*	*NBN*	*RAD50*	*STAT6*	
CCND3	*ERBB3*	*FGFR1*	*KMT2A*	*NF1*	*RAD51*	*STK11*	

Genes Analyzed in Tumor RNA (91)									
ABL1	*CCND1*	*ERG*	*FGFR4*	*HOXA3*	*KIF5B*	*MSH2*	*NTRK3*	*ROS1*	*TP63*
AKT3	*CCND2*	*ESR1*	*FLI1*	*HOXA4*	*KIT*	*MYB*	*PAX3*	*RPS5KB1*	
ALK	*CDK4*	*ETS1*	*FLT1*	*HOXA5*	*KMT2A*	*MYBL1*	*PAX7*	*RPS6KB1*	
AR	*CDKN2A*	*ETV1*	*FLT3*	*HOXA6*	*LCK*	*MYC*	*PDGFRA*	*SS18*	
AXL	*CSF1R*	*ETV4*	*FUS*	*HOXA7*	*LMO1*	*NOTCH1*	*PDGFRB*	*TCF3*	
BCL2	*CTLA4*	*ETV5*	*HOXA1*	*HOXA9*	*LMO2*	*NOTCH2*	*PIK3CA*	*TCL1A*	
BCL6	*DUSP22*	*EWSR1*	*HOXA10*	*IRF4*	*LYL1*	*NOTCH3*	*PPARG*	*TLX1*	
BRAF	*EGFR*	*FGFR1*	*HOXA11*	*ITK*	*MDM2*	*NRG1*	*RAF1*	*TLX3*	
BRCA1	*EML4*	*FGFR2*	*HOXA13*	*JAK2*	*MET*	*NTRK1*	*RELA*	*TMPRSS2*	
BRCA2	*ERBB2*	*FGFR3*	*HOXA2*	*KDR*	*MLLT3*	*NTRK2*	*RET*	*TP53*	