

Liquid Trace Solid Tumor

Patient Name:		Ordering Physician:	
Date of Birth:		Accession #:	
Gender (M/F):		Specimen Type:	Peripheral Blood
Client:		Specimen ID:	
Case #:			
Body Site:	PERIPHERAL BLOOD		

MRN:	
Collected Date:	
Received Date:	
Reported Date:	

Detected Genomic Alterations				
Level 1 (FDA-Approved)	Level 2 (Standard of Care)	Level 3 (Clinical Evidence)	Level 4 (Biological Evidence)	Other
KRAS (G12D)	Tumor Mutation Burden Intermediate: 7 Mut/Mb	TP53, KDM6A, ELANE, RAD51D, MEN1	TERT	Autosomal chromosomal structural analysis shows 4q-, 8q+ (distal), 14q+

Results Summary

- **-Mutations in KRAS, TP53, KDM6A, ELANE, RAD51D, TERT, and MEN1 genes**
- Tumor Mutation Burden Intermediate: 7 Mut/Mb**
- Autosomal chromosomal structural analysis shows 4q-, 8q+ (distal), 14q+**
- The findings suggest presence of circulating solid tumor DNA.
- Intermediate TMB suggests response to immune checkpoint inhibitors.
- KRAS mutation suggests possible response to pan-RAS inhibitors (daraxonrasib etc).
- RAD51D mutation suggests response to PARP inhibitors.
- TP53 mutation suggests possible response to eprenetapopt (APR-246), Aurora kinase A and Wee1 inhibitors.

Note: Per client request, RNA sequencing was not performed, and results are based on DNA testing only. RNA sequencing is necessary for evaluating fusion genes, the presence of HPV and EBV, detecting clonality and alternative splicing (MET exon 14, EGFRvIII and AR-v7).

See additional report information at the end of the report.

Tumor Heterogeneity

There are abnormal clones with KRAS, TP53, KDM6A, ELANE, RAD51D, TERT, and MEN1 mutations.

Diagnostic Implications

KRAS, TP53, KDM6A, ELANE, RAD51D, TERT, MEN1	The findings suggest presence of circulating solid tumor DNA (see results summary).
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FDA-Approved Therapeutics

KRAS (G12D)	Daraxonrasib
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FDA-Approved Therapeutics in Other Tumor Types

RAD51D	Olaparib
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Relevant Alteration Associated with Resistance

KRAS mutations suggest resistance to targeted anti-EGFR therapy

TP53 mutation is associated with resistance to therapy.

Levels 2, 3 & 4 (Standard of Care and Clinical/Biological Evidence)

KRAS (G12D)	pan-RAS inhibitors
TP53	Aurora kinase A inhibitors, Wee1 inhibitors, Chk1 inhibitors, kevetrin, APR-246, nutlins, gene therapy
KDM6A	KDM6A inhibitors (GSK-J4)
ELANE	ELANE inhibitors
RAD51D	PARP inhibitors
MEN1	PARP inhibitors
TMB-Intermediate	Immunotherapy with checkpoint inhibitor

Relevant Genes with NO Alteration

No evidence of mutation in NRAS, EGFR, BRAF, or BRCA 1/2

Test Description:

This is a comprehensive molecular profile which uses next generation sequencing (NGS) to identify molecular abnormalities, including single nucleotide variants (SNVs), insertions/deletions (indels), copy number variants (CNVs), tumor mutation burden (TMB), fusions, B- and T-cell clonality, and viruses (HPV, EBV, HTLV1, and TTV), in cell-free (cf) DNA of 318 genes and cfrNA in greater than 1600 genes implicated in solid tumors. Whenever possible, clinical relevance and implications of detected abnormalities are described below.

Biological relevance of detected Alterations

- **KRAS.** This gene, a Kirsten ras oncogene homolog from the mammalian ras gene family, encodes a protein that is a member of the small GTPase superfamily. A single amino acid substitution is responsible for an activating mutation. The transforming protein that results is implicated in various malignancies, including lung adenocarcinoma, mucinous adenoma, ductal carcinoma of the pancreas and colorectal carcinoma. Alternative splicing leads to variants encoding two isoforms that differ in the C-terminal region. [provided by RefSeq, Jul 2008]
- **TP53.** This gene encodes a tumor suppressor protein containing transcriptional activation, DNA binding, and oligomerization domains. The encoded protein responds to diverse cellular stresses to regulate expression of target genes, thereby inducing cell cycle arrest, apoptosis, senescence, DNA repair, or changes in metabolism. Mutations in this gene are associated with a variety of human cancers, including hereditary cancers such as Li-Fraumeni syndrome. Alternative splicing of this gene and the use of alternate promoters result in multiple transcript variants and isoforms. Additional isoforms have also been shown to result from the use of alternate translation initiation codons from identical transcript variants (PMIDs: 12032546, 20937277). [provided by RefSeq, Dec 2016]
- **KDM6A.** This gene is located on the X chromosome and is the corresponding locus to a Y-linked gene which encodes a tetratricopeptide repeat (TPR) protein. The encoded protein of this gene contains a JmjC-domain and catalyzes the demethylation of tri/dimethylated histone H3. Multiple alternatively spliced transcript variants have been found for this gene. [provided by RefSeq, Apr 2014]
- **ELANE.** Elastases form a subfamily of serine proteases that hydrolyze many proteins in addition to elastin. Humans have six elastase genes which encode structurally similar proteins. The encoded preproprotein is proteolytically processed to generate the active protease. Following activation, this protease hydrolyzes proteins within specialized neutrophil lysosomes, called azurophil granules, as well as proteins of the extracellular matrix. The enzyme may play a role in degenerative and inflammatory diseases through proteolysis of collagen-IV and elastin. This protein also degrades the outer membrane protein A (OmpA) of *E. coli* as well as the virulence factors of such bacteria as *Shigella*, *Salmonella* and *Yersinia*. Mutations in this gene are associated with cyclic neutropenia and severe congenital neutropenia (SCN). This gene is present in a gene cluster on chromosome 19. [provided by RefSeq, Jan 2016]
- **RAD51D.** The protein encoded by this gene is a member of the RAD51 protein family. RAD51 family members are highly similar to bacterial RecA and *Saccharomyces cerevisiae* Rad51, which are known to be involved in the homologous recombination and repair of DNA. This protein forms a complex with several other members of the RAD51 family, including RAD51L1, RAD51L2, and XRCC2. The protein complex formed with this protein has been shown to catalyze homologous pairing between single- and double-stranded DNA, and is thought to play a role in the early stage of recombinational repair of DNA. Alternative splicing results in multiple transcript variants. Read-through transcription also exists between this gene and the downstream ring finger and FYVE-like domain containing 1 (RFFL) gene. [provided by RefSeq, Jan 2011]
- **TERT.** Telomerase is a ribonucleoprotein polymerase that maintains telomere ends by addition of the telomere repeat TTAGGG. The enzyme consists of a protein component with reverse transcriptase activity, encoded by this gene, and an RNA component which serves as a template for the telomere repeat. Telomerase expression plays a role in cellular senescence, as it is normally repressed in postnatal somatic cells resulting in progressive shortening of telomeres. Deregulation of telomerase expression in somatic cells may be involved in oncogenesis. Studies in mouse suggest that telomerase also participates in chromosomal repair, since de novo synthesis of telomere repeats may occur at double-stranded breaks. Alternatively spliced variants encoding different isoforms of telomerase reverse transcriptase have been identified; the full-length sequence of some variants has not been determined. Alternative splicing at this locus is thought to be one mechanism of regulation of telomerase activity. [provided by RefSeq, Jul 2008] In addition, recurring somatic mutations at multiple spots in the proximal promoter (particularly at 124bp and 146bp upstream of the translation start site) are found in tumors of many tissue origins. These mutations are thought to affect binding of Ets family proteins and nuclear factor kappa B and alter secondary structure and long-range interactions, leading to increased promoter activity. [provided by RefSeq, May 2023]
- **MEN1.** This gene encodes menin, a tumor suppressor associated with a syndrome known as multiple endocrine neoplasia type 1. Menin is a scaffold protein that functions in histone modification and epigenetic gene regulation. It is thought to regulate several pathways and processes by altering chromatin structure through the modification of histones. [provided by RefSeq, May 2019]

Drug Information

APR-246

APR-246 is a first-in-class agent targeting mutant p53. In vitro and in vivo preclinical models have demonstrated that APR-246 has excellent efficacy in OC (both adenocarcinoma and squamous cell carcinoma) and potentially synergises with chemotherapies used in the treatment of OC, restoring sensitivity to chemotherapy-resistant tumours. An initial phase I clinical trial has shown APR-246 to be safe in humans and early results from a currently running phase Ib/II trial of APR-246 with carboplatin and liposomal doxorubicin in ovarian cancer have been promising. Together, these data provide a strong rationale for investigating the efficacy of APR-246 in OC.

APR-246 has been used in trials studying the treatment of Prostatic Neoplasms, Hematologic Neoplasms, and Platinum Sensitive Recurrent High-grade Serous Ovarian Cancer With Mutated p53.

APR-246 is an analogue of PRIMA-1, which modifies the core domain of mutant p53, resulting in restoration of wild-type p53 conformation and

reactivation of normal p53 function, leading to increased cell cycle arrest and tumor cell death (PMID: 20498645).

Olaparib

Olaparib (LYNPARZA) is an antineoplastic agent, Poly(ADP-ribose) Polymerase 1;2;3 inhibitor. (PARP 1;2;3 inhibitor).

Lynparza is a poly (ADP-ribose) polymerase (PARP) inhibitor indicated for the treatment of adult patients with deleterious or suspected deleterious germline BRCA-mutated (gBRCAm) advanced ovarian cancer who have been treated with three or more prior lines of chemotherapy. Select patients for therapy based on an FDA-approved companion diagnostic for Lynparza. (1.1, 2.2)

Talazoparib

Talazoparib is a poly(ADP-ribose) Polymerase 1, 2 (PARP 1;2 inhibitor). Talazoparib was approved by the FDA for use in germline BRCA mutated, HER2 negative, locally advanced or metastatic breast cancer on October 16, 2018 under the trade name Talzenna. Talazoparib prevents PARP-mediated repair of DNA damage in cancer cells, allowing accumulation of damage and PARP-DNA complexes. Repair related errors by error prone secondary repair pathways may also contribute to the cytotoxicity of Talazoparib. Talazoparib is indicated for the treatment of deleterious or suspected deleterious germline BRCA mutated, HER2 negative locally advanced or metastatic breast cancer in adults

Niraparib

Niraparib is an inhibitor of poly (ADP-ribose) polymerase (PARP) with potential antineoplastic activity. PARP Inhibitor MK4827 inhibits PARP activity, enhancing the accumulation of DNA strand breaks and promoting genomic instability and apoptosis. The PARP family of proteins detect and repair single strand DNA breaks by the base-excision repair (BER) pathway. The specific PARP family member target for PARP inhibitor MK4827 is unknown. (NCI Thesaurus)

ZEJULA is a poly(ADP-ribose) polymerase (PARP) inhibitor indicated for the maintenance treatment of adult patients with recurrent epithelial ovarian, fallopian tube, or primary peritoneal cancer who are in a complete or partial response to platinum-based chemotherapy.

Rucaparib

Rucaparib is a potent mammalian poly(ADP-ribose) polymerase 1, 2 and 3 inhibitor with anticancer properties (PARP 1;2;3 inhibitor).

Rucaparib is an inhibitor of PARP-1, PARP-2, and PARP-3. Via an inhibitory effect on the PARP enzymatic activity, rucaparib decreases the formation of PARP-DNA complexes resulting in DNA damage, apoptosis, and cell death. It is proposed that PARP inhibition specifically targets tumor cells with preexisting HRD, such as those cells possessing mutations in the BRCA1 or BRCA2 genes.

Daraxonasib

Daraxonasib (RMC-6236) is an orally administered, non-covalent RAS(ON) inhibitor designed to target the active, GTP-bound state of RAS proteins, including multiple oncogenic RAS variants. By inhibiting aberrant RAS signaling, daraxonasib is intended to suppress a key driver of tumor growth and survival in RAS-mutated cancers. The drug has demonstrated clinically meaningful antitumor activity in patients with advanced solid tumors, particularly pancreatic adenocarcinoma, and is being investigated across a broader range of RAS-mutated malignancies, including non-small cell lung cancer and colorectal cancer.

Potential Clinical Trials

Trial URL	Status	Title	Disease	Drug	Sites
https://clinicaltrials.gov/study/NCT04900818	Recruiting	A Phase 1 Study of TJ033721 in Subjects With Advanced or Metastatic Solid Tumors	Solid Tumor	TJ033721 (givastomig), TJ033721 (givastomig) , nivolumab, chemotherapy, TJ033721 (givastomig), chemotherapy, TJ033721 (givastomig), durvalumab, chemotherapy	Stern Center for Cancer Clinical Trials and Research, Orange, California 92868 UCHealth Cancer Care - Anschutz Medical Campus, Aurora, Colorado 80045 Mary Crowley Cancer Research, Dallas, Texas 75230

https://clinicaltrials.gov/study/NCT06874335	Recruiting	A Phase 1, Multicenter, Open-Label, Dose Escalation, Dose Expansion and Dose Optimization Study of BHV-1530 as Monotherapy and in Combination With Other Anti-Cancer Agents in Adult Participants With Advanced or Metastatic Solid Tumors	Solid Tumor	BHV-1530, Cemiplimab	Site-111, Newport Beach, California 92663 Site-106, West Valley City, Utah 84119 Site-107, Denver, Colorado 80218
https://clinicaltrials.gov/study/NCT05039801	Recruiting	A Phase 1 Open-Label, Dose-Escalation and Dose-Expansion Study to Investigate the Safety, Pharmacokinetics, and Anti-Tumor Activity of IACS-6274 as Monotherapy and in Combination in Patients With Advanced Solid Tumors	Solid Tumor	Bevacizumab, Glutaminase-1 Inhibitor IACS-6274, Paclitaxel, Capivasertib	M D Anderson Cancer Center, Houston, Texas 77030
https://clinicaltrials.gov/study/NCT07086768	https://clinicaltrials.gov/study/NCT07086768	A Phase Ia/Ib, 2-Part, Dose-Escalation and Dose-Expansion Study of BSI-082 Monotherapy and Combined Therapy in Patients With Advanced or Metastatic Solid Tumors	Solid Tumor	BSI-082, Trastuzumab deruxtecan	University of Texas Health Science Center at San Antonio, San Antonio, Texas 78229

Detailed Results

Single Nucleotide Variant (SNV) and Insertions-Deletions (INDELS)								
Gene name	Hgvsnp	Hgvsc	Amino acids	Codons	Consequence	Allele frequency	Read depth	Predicted effect on protein
KRAS	NP_203524.1:p.Gly12Asp	NM_033360.2:c.35G>A	G/D	gGt/gAt	missense_variant	29.17	761	deleterious
TP53	NP_000537.3:p.Ala159Pro	NM_000546.5:c.475G>C	A/P	Gcc/Ccc	missense_variant	21.72	1022	deleterious
KDM6A	NP_066963.2:p.Pro608AlafsTer13	NM_021140.2:c.1820dup	V/VX	gtg/gTtg	frameshift_variant	16.3	540	0
ELANE	NP_001963.1:p.Ala166Pro	NM_001972.2:c.496G>C	A/P	Gcc/Ccc	missense_variant	8.91	1032	tolerated
RAD51D	NP_002869.3:p.Arg291Cys	NM_002878.3:c.871C>T	R/C	Cgc/Tgc	missense_variant	7.26	882	deleterious
TERT	0	NM_198253.2:c.-124C>T	0	0	upstream_gene_variant	3.83	1018	0
MEN1	NP_570711.1:p.Pro545Leu	NM_130799.2:c.1634C>T	P/L	cCg/cTg	missense_variant	2.55	941	tolerated

Methodology and Test Background

This is a next generation sequencing (NGS) test that involves separate analysis of DNA and RNA panels for abnormalities that are reported to be altered in various types of solid tumors. The DNA panel is composed of 318 genes, and the RNA panel is composed of >1600 genes. The DNA and RNA components of this assay were developed, validated, and set up as separate workflows, with independent extraction, library preparation, sequencing, and analysis pipelines. For cases with detectable circulating solid tumor DNA, tumor mutation burden (TMB) is reported. The NGS assay also detects several viruses that are important in oncology, including EBV, HPV, HTLV 1, and TTV. TTV (torque teno virus) was first discovered in a patient with non-A-E hepatitis and is now regarded as a part of the human virome. In general, TTV does not cause pathology in immunocompetent individuals. TTV is considered as a marker of immune competence in patients with immunological impairment and inflammatory disorders. High TTV load is associated with increased risk of infection. In patients with organ transplant, low TTV load is associated with an increased risk of rejection.

Nucleic acid is isolated from peripheral blood plasma or CSF. When CSF sample is submitted, RNA sequencing is performed on the CSF cell pellet instead of cfRNA due to degradation. Performance of the assays may vary depending on the quantity and quality of nucleic acid, sample preparation and sample age. Testing is performed using massive parallel sequencing of the coding DNA of the listed genes. This includes sequencing of all the exons as well as approximately 50 nucleotides at the 5' and 3' ends of each coding exon to detect splice site abnormalities. The TERT promoter region, including the hotspots at -124 and -146 bp, is also covered. Our cfDNA sequencing method has a sensitivity of 0.1% for detecting hot spot mutations, 0.5% for detecting single nucleotide variants (SNVs) and 1% for small (<60 bp) insertions/ deletions (indels). Known hot spots in specific genes such as IDH1/2, NRAS, and KRAS are reported at levels of 0.01% and higher when both cfRNA and cfDNA results are combined. Significant gene amplification and deletion (copy number variants) are also reported. TMB is calculated and cut-off points were determined based on comparison with tissue samples obtained from the same patient. Using cut-off of 6 mut/Mb, 17% of cases called as negative by cfDNA are false negative (FN). However, cases with ≤ 3 show only 6% FN. Intermediate cases (TMB between 6 and 9 mut/Mb) show 51% false positivity. Positive cases (TMB ≥ 9 Mut/Mb) show only 7% false positive. Cases without circulating solid tumor DNA are reported as "unable to evaluate" for TMB. Targeted RNA NGS is performed by hybrid capture and duplicates are excluded for levels measurements. The Universal Human Reference (UHR) RNA is used as control. All detected fusion transcripts are reported. While the major focus of the RNA analysis is the detection of fusion mRNA, mutations in the expressed RNA of the analyzed genes, HLA class I genotyping, and Epstein-Barr virus (EBV), human papillomavirus (HPV), human T-lymphotropic virus type-1 (HTLV1), and torque teno virus (TTV) viral RNA are also analyzed and reported. B- and T-cell clonality will be reported, if clonal or clinically relevant. The sensitivity of this assay in detecting fusion mRNA is between 5% and 10%. This test specifically covers translocations that lead to the expression of fusion RNA. Translocations that lead to deregulation of expression can be addressed by this test if compared to the expression proper normal control. Since the clinical relevance of the RNA expression level of most of the genes is not characterized at this time, only a few specific genes will be commented on when abnormalities are detected. CD274 (PD-L1) mRNA levels are reported when they are significantly elevated. The testing is not validated to detect germline abnormalities. Potential germline mutations detected incidentally should be confirmed by tests specifically designed for detecting germline abnormalities.

Based on our validation study, the following exonic regions of the genes listed below are not covered appropriately <100 X coverage and sequencing by NGS may not be reliable in these regions. The poor coverage is primarily due to the inherent difficulty in obtaining adequate sequencing coverage in regions with high GC content. No well-characterized hotspots are present in these regions. RAD51 NM_133487 chr15:40994004-40994124, BRCA1 NM_007300 chr17:41231351-41231416, FUBP1 NM_003902 chr1:78435609-78435699, CBLB NM_170662 chr3:105420938-105421303, TERT NM_198253 chr5:1295183-1295250, ARID1B NM_017519 chr6:157098715-157100605, CUX1 NM_001202543 chr7:101740644-101740781, KMT2C NM_170606 chr7:151891314-151891346 and 151935792-151935911, GALNT12 NM_024642 chr9:101569952-101570351, ATM NM_000051 chr11:108164040-108164204, CDK17 NM_001170464 chr12:96679880-96679926, RB1 NM_000321 chr13:48954189-48954220, SETBP1 NM_015559 chr18:42643044-42643692, KMT2B NM_014727 chr19:36208921-36209283, AR NM_000044 chrX:66764889-66766604, STAG2 NM_001042749 chrX:123200025-123200112. Data was annotated against GRCh37

(hg19) reference human genome using Nirvana v3.17.0. This human reference is based on the GRCh37.p6 version of the human genome assembly.

DEX Z-Code Z00WY is approved by MolDx for use in assessing DNA SNVs/Indels only.

The table below may contain a partial list of the tested DNA genes. For a complete list, please go to:
<https://genomictestingcooperative.com/genomic-tests/liquid-trace-solid-tumor/> (click the DNA tab)

For a complete list of tested RNA genes (Fusions/Expression), please go to:
<https://genomictestingcooperative.com/genomic-tests/liquid-trace-solid-tumor/> (click the RNA tab)

Tested genes

Genes Tested for Abnormalities in Coding Sequence																	
ABL1	ATRX	BRCA1	CDK4	CYLD	ERBB4	FGFR3	H3-3A (H3F3A)	KAT6A	MAP3K14	MTOR	NSD2 (WHSC1)	PMS2	RAD51D	SF3B1	STK11	TSHR	
ABRAXAS1	AURKA	BRCA2	CDK6	DAXX	ERG	FGFR4	H3C2	KAT6B	MAPK1	MUTYH	NTHL1	POLD1	RAF1	SH2B3	SUFU	TTYH1	
ACSL4	AURKB	BRIP1	CDKN1B	DDR2	ESR1	FH	HGF	KDM5C	MCL1	MYC	NTRK1	POLE	RB1	SLC6A16	SUZ12	U2AF1	
ACVR1B	AURKC	BTX	CDKN2A	DDX41	ETNK1	FLCN	HNF1A	KDM6A	MDM2	MYCL	NTRK2	POT1	RET	SMAD2	TAL1	U2AF2	
AKT1	AXIN1	CALR	CDKN2B	DICER1	ETV6	FLT3	HOXB13	KDR	MDM4	MYCN	NTRK3	PPM1D	RHEB	SMAD4	TCF3	UBA1	
AKT2	AXIN2	CARD11	CDKN2C	DNM2	EXO1	FLT4	HRAS	KEAP1	MED12	MYD88	NUDT15	PPP2R1A	RHOA	SMARCA4	TENT5C (FAM46C)	UBTF	
AKT3	B2M	CBL	CEBPA	DNMT3A	EZH2	FOXL2	HSP90AA1	KIT	MEF2B	NBN	PAK3	PRDM1	RIT1	SMARCB1	TERC	VHL	
ALK	BAP1	CBLB	CHEK1	DOT1L	FANCA	FUBP1	ID3	KMT2A	MEN1	NF1	PALB2	PRKAR1A	RNF43	SMC1A	TERT	WT1	
AMER1	BARD1	CBLC	CHEK2	EED	FANCC	GALNT12	IDH1	KMT2B	MET	NF2	PAX5	PRKDC	ROS1	SMC3	TET2	XP01	
ANKRD26	BCL2	CCND1	CIC	EGFR	FANCD2	GATA1	IDH2	KMT2C	MGA	NFE2	PBRM1	PRPF8	RUNX1	SMO	TGFB2	XRCC2	
APC	BCL2L1	CCND3	CREBBP	EGLN1	FANCE	GATA2	IGF1R	KMT2D	MIR15A	NFE2L2	PDGFRA	PRSS1	SAMD9	SOC1	TMEM127	XRCC3	
AR	BCL6	CCNE1	CRLF2	ELANE	FANCF	GATA3	IKZF1	KRAS	MIR16-1	NFKBIA	PDGFRB	PTCH1	SAMD9L	SOX2	TNFAIP3	ZIC2	
ARAF	BCOR	CD274	CSF1R	EP300	FANCG	GEN1	IKZF3	LIG4	MITF	NKX2-1	PHF6	PTEN	SDHA	SOX9	TNFRSF14	ZNF217	
ARID1A	BCORL1	CD79A	CSF3R	EPAS1	FAS	GNA11	IL7R	LIN28A	MLH1	NOTCH1	PIK3CA	PTPN11	SDHB	SPOP	TP53	ZRSR2	
ARID1B	BCR	CD79B	CTCF	EPCAM	FBXW7	GNAQ	INHBA	LRP1B	MPL	NOTCH2	PIK3R1	RAC1	SDHAF2	SRC	TPMT	-	
ARID2	BIRC3	CDA	CTNNA1	EPHA3	FGF4	GNAS	IRF4	MAP2K1	MRE11	NOTCH3	PIK3R2	RAD21	SDHC	SRSF2	TRAF3	-	
ASXL1	BLM	CDC73	CTNNA1	EPHA5	FGF6	GNB1	JAK1	MAP2K2	MSH2	NPM1	PIM1	RAD50	SDHD	STAG2	TRPM4	-	
ATM	BMPRTA	CDH1	CUX1	ERBB2	FGFR1	GREM1	JAK2	MAP2K4	MSH3	NRAS	PLCG1	RAD51	SETBP1	STAT3	TSC1	-	
ATR	BRAF	CDK12	CXCR4	ERBB3	FGFR2	GRIN2A	JAK3	MAP3K1	MSH6	NSD1	PMS1	RAD51C	SETD2	STAT5B	TSC2	-	

RNA Fusions/Expression

Fusion/Expression													
ABL1	BCL6	CD274 (PD-L1)	EGFR	EWSR1	FLI1	IKZF3	MAP3K1	NRG1	NUP98	PML	RET	SS18	THADA
AKT3	BRAF	CIC	ERG	FGFR1	FOXO1	JAK2	MECOM	NTRK1	PAX8	PPARG	RHOA	STAT6	TMPRSS2
ALK	CAMTA1	CREB1	ETS1	FGFR2	FUS	KIAA1549	MYB	NTRK2	PDGFRA	PRKACA	ROS1	TAL1	YAP1
AR	CBFB	CREBBP	ETV1	FGFR3	GLI1	KMT2A	MYC	NTRK3	PDGFRB	RAF1	RUNX1	TCF3	YWHA
BCL2	CCND1	ERBB2	ETV6	FIP1L1	HMG2	MAML2	NOTCH1	NUP214	PICALM	RARA	RUNX1T1	TFG	ZFTA

Reference

- Recent Advances in CAR-Based Solid Tumor Immunotherapy. Shin MH, Oh E, Kim Y, Nam DH, Jeon SY, Yu JH, Minn D. Cells. 2023 Jun 11;12(12):1606. doi: 10.3390/cells12121606. PMID: 37371075.
- Tumor-Associated Fibroblast-Targeting Nanoparticles for Enhancing Solid Tumor Therapy: Progress and Challenges. Li W, Little N, Park J, Foster CA, Chen J, Lu J. Mol Pharm. 2021 Aug 2;18(8):2889-2905. doi: 10.1021/acs.molpharmaceut.1c00455. Epub 2021 Jul 14. PMID: 34260250.

3. Mechanisms of Therapeutic Antitumor Monoclonal Antibodies. Tsao LC, Force J, Hartman ZC. Cancer Res. 2021 Sep 15;81(18):4641-4651. doi: 10.1158/0008-5472.CAN-21-1109. Epub 2021 Jun 18. PMID: 34145037.
4. Overcoming biological barriers to improve solid tumor immunotherapy. Ukidve A, Cu K, Kumbhojkar N, Lahann J, Mitragotri S. Drug Deliv Transl Res. 2021 Dec;11(6):2276-2301. doi: 10.1007/s13346-021-00923-8. Epub 2021 Feb 20. PMID: 33611770.

Electronic Signature

Ahmad Charifa, M.D.

Genomic Testing Cooperative, LCA serves as the coordinating laboratory for this assay and is responsible for overall test performance and reporting. The test (sample processing, sequencing and data generation) was performed at Genomic Testing Cooperative, LCA, 25371 Commercentre Drive Lake Forest, CA 92630. Medical Director Maher Albitar, M.D. Analysis of the data was performed by Genomic Testing Cooperative, LCA, 25371 Commercentre Drive, Lake Forest, CA 92630. Medical Director: Maher Albitar, M.D. (CLIA #: 05D2111917 CAP #: 9441574). The signing pathologist / Medical Director is responsible for the accuracy and interpretation of results and the release of this report.

The test was developed and its performance characteristics have been determined by Genomic Testing Cooperative, LCA. This test has not been approved by the FDA. The FDA has determined such clearance or approval is not necessary. This laboratory is CLIA certified to perform high complexity clinical testing.

Additional Report Information

Mutations Load (mol/mL)

