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CLIA #: 05D2111917 CAP #: 9441574

Liquid Trace Solid Tumor

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

Collected Date:	06/01/2025	Time:	12:00 AM	Tumor Type:	Colon Adenocarcinoma
Received Date:	06/03/2025	Time:	10:18 AM	Stage:	Metastasis
Reported Date:	06/13/2025	Time:	02:59 PM		

Detected Genomic Alterations

Level 1 (FDA-Approved)	Level 2 (Standard of Care)	Level 3 (Clinical Evidence)	Level 4 (Biological Evidence)	Other
BRAF (V600E)	Tumor Mutation Burden Low: 4 Mut/Mb	TP53, APC (2 mutations), JAK3, DNMT3A	INHBA, KMT2C, SDHA, PTPRC (? Germline, VUS)	-t(X;22) (q28;q12.3) FLNA::MYH9 -Autosomal chromosomal structural analysis shows 1p-, -4, 5p+, 5q-, 8p-, 8q+, 13q+, 17p-, 17q+, 18p-, 19q+, and others

Results Summary

- **-Mutations in TP53, APC (2 mutations), BRAF, INHBA, JAK3, DNMT3A, KMT2C, and SDHA genes**
  -Possible germline mutation in PTPRC gene, heterozygous
  -t(X;22) (q28;q12.3) FLNA::MYH9 fusion mRNA
  -Autosomal chromosomal structural analysis shows 1p-, -4, 5p+, 5q-, 8p-, 8q+, 13q+, 17p-, 17q+, 18p-, 19q+, and others
  -No POLE or EGFR mutations
  -No evidence of ERBB2 (HER2) gene amplification
  -EBV viral RNA: Not detected
  -HPV viral RNA: Not detected
  -TTV viral RNA: Not detected
  -HLA Genotyping:
  -HLA-A: A*03:01-A*23:01
  -HLA-B: B*44:02-B*44:03
  -HLA-C: C*02:02-C*16:04

  -The findings suggest the presence of circulating solid tumor DNA/RNA. The DNMT3A mutation is most likely in myeloid cells, consistent with CHIP (clonal hematopoiesis of indeterminate potential).

  -BRAF mutation in colorectal tumors suggests response to treatment with triple therapy: BRAF inhibitor,

anti-EGFR, and MEK inhibitor (encorafenib, cetuximab, and binimetinib).

-JAK mutation suggests possible response to JAK inhibitors (tofacitinib (Xeljanz), peficitinib (Smyraf), …).

-APC mutations suggest possible response to WNT and COX-2 inhibitors.

-TP53 mutation suggests possible response to eprenetapopt (APR-246), Aurora kinase A and Wee1 inhibitors.

-The PTPRC mutation is detected at high level, raising the possibility of a germline mutation. This mutation leads to early termination (loss of function). However, there is no data on its clinical relevance and should be classified as of "uncertain significance" at this time.

See additional report information at the end of the report.

Tumor Heterogeneity

There are abnormal clones with TP53, APC (2 mutations), BRAF, INHBA, JAK3, DNMT3A, KMT2C, and SDHA mutations.
The PTPRC mutation is detected at a high level, possible germline abnormality.

Diagnostic Implications

TP53, APC (2 mutations), BRAF, INHBA, JAK3, DNMT3A, KMT2C, SDHA, PTPRC	-These findings suggest the presence of circulating solid tumor DNA/RNA (see results summary). -The PTPRC mutation is likely a germline variant.

FDA-Approved Therapeutics

BRAF (V600E)	Encorafenib + Cetuximab

Relevant Alteration Associated with Resistance

TP53 mutation is associated with resistance to therapy.
BRAF mutation may suggest resistance to targeted anti-EGFR therapy

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

TP53	Aurora kinase A inhibitors, Wee1 inhibitors, Chk1 inhibitors, kevetrin, APR-246, nutlins, gene therapy
APC	WNT, beta-catenin, and COX-2 inhibitors
BRAF (V600E)	ERK/MEK Inhibitors
JAK3	JAK inihibitor
DNMT3A	DNA methyltransferase inhibitors

Relevant Genes with NO Alteration		
No evidence of mutation in KRAS, NRAS, EGFR, or BRCA 1/2	No specific mutation in DPYD gene, associated with enzymatic deficiency	No evidence of METex14 skipping or EGFRvIII

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, and TTV), in cell-free (cf) DNA of 302 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

- 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]

- APC. This gene encodes a tumor suppressor protein that acts as an antagonist of the Wnt signaling pathway. It is also involved in other processes including cell migration and adhesion, transcriptional activation, and apoptosis. Defects in this gene cause familial adenomatous polyposis (FAP), an autosomal dominant pre-malignant disease that usually progresses to malignancy. Mutations in the APC gene have been found to occur in most colorectal cancers. Disease-associated mutations tend to be clustered in a small region designated the mutation cluster region (MCR) and result in a truncated protein product. [provided by RefSeq, Dec 2019]

- BRAF. This gene encodes a protein belonging to the RAF family of serine/threonine protein kinases. This protein plays a role in regulating the MAP kinase/ERK signaling pathway, which affects cell division, differentiation, and secretion. Mutations in this gene, most commonly the V600E mutation, are the most frequently identified cancer-causing mutations in melanoma, and have been identified in various other cancers as well, including non-Hodgkin lymphoma, colorectal cancer, thyroid carcinoma, non-small cell lung carcinoma, hairy cell leukemia and adenocarcinoma of lung. Mutations in this gene are also associated with cardiofaciocutaneous, Noonan, and Costello syndromes, which exhibit overlapping phenotypes. A pseudogene of this gene has been identified on the X chromosome. [provided by RefSeq, Aug 2017]

- INHBA. This gene encodes a member of the TGF-beta (transforming growth factor-beta) superfamily of proteins. The encoded preproprotein is proteolytically processed to generate a subunit of the dimeric activin and inhibin protein complexes. These complexes activate and inhibit, respectively, follicle stimulating hormone secretion from the pituitary gland. The encoded protein also plays a role in eye, tooth and testis development. Elevated expression of this gene may be associated with cancer cachexia in human patients. [provided by RefSeq, Aug 2016]

- JAK3. The protein encoded by this gene is a member of the Janus kinase (JAK) family of tyrosine kinases involved in cytokine receptor-mediated intracellular signal transduction. It is predominantly expressed in immune cells and transduces a signal in response to its activation via tyrosine phosphorylation by interleukin receptors. Mutations in this gene are associated with autosomal SCID (severe combined immunodeficiency disease). [provided by RefSeq, Jul 2008]

- DNMT3A. CpG methylation is an epigenetic modification that is important for embryonic development, imprinting, and X-chromosome inactivation. Studies in mice have demonstrated that DNA methylation is required for mammalian development. This gene encodes a DNA methyltransferase that is thought to function in de novo methylation, rather than maintenance methylation. The protein localizes to the cytoplasm and nucleus and its expression is developmentally regulated. [provided by RefSeq, Mar 2016]

- KMT2C. This gene is a member of the myeloid/lymphoid or mixed-lineage leukemia (MLL) family and encodes a nuclear protein with an AT hook DNA-binding domain, a DHHC-type zinc finger, six PHD-type zinc fingers, a SET domain, a post-SET domain and a RING-type zinc finger. This protein is a member of the ASC-2/NCOA6 complex (ASCOM), which possesses histone methylation activity and is involved in transcriptional coactivation. [provided by RefSeq, Jul 2008]

- SDHA. This gene encodes a major catalytic subunit of succinate-ubiquinone oxidoreductase, a complex of the mitochondrial respiratory chain. The complex is composed of four nuclear-encoded subunits and is localized in the mitochondrial inner membrane. Mutations in this gene have been associated with a form of mitochondrial respiratory chain deficiency known as Leigh Syndrome. A pseudogene has been identified on chromosome 3q29. Alternatively spliced transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq,

Jun 2014]

- PTPRC. The protein encoded by this gene is a member of the protein tyrosine phosphatase (PTP) family. PTPs are known to be signaling molecules that regulate a variety of cellular processes including cell growth, differentiation, mitosis, and oncogenic transformation. This PTP contains an extracellular domain, a single transmembrane segment and two tandem intracytoplasmic catalytic domains, and thus is classified as a receptor type PTP. This PTP has been shown to be an essential regulator of T- and B-cell antigen receptor signaling. It functions through either direct interaction with components of the antigen receptor complexes, or by activating various Src family kinases required for the antigen receptor signaling. This PTP also suppresses JAK kinases, and thus functions as a regulator of cytokine receptor signaling. Alternatively spliced transcripts variants of this gene, which encode distinct isoforms, have been reported. [provided by RefSeq, Jun 2012]

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 potently 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).

Sulindac

Sulindac is a COX-1/COX-2 inhibitor. It is being evaluated as a potential cancer chemo-preventive and therapeutic drug in clinical trials for a variety of malignancies.

Celecoxib

Celecoxib is a selective cyclooxygenase-2 (COX-2) inhibitor, is a nonsteroidal anti-inflammatory drug (NSAID). It is used to manage symptoms of various types of arthritis pain and in familial adenomatous polyposis (FAP) to reduce precancerous polyps in the colon. It is marketed by Pfizer under the brand name Celebrex, and was initially granted FDA approval in 1998. Interestingly, selective COX-2 inhibitors (especially celecoxib), have been evaluated as potential cancer chemopreventive and therapeutic drugs in clinical trials for a variety of malignancies.

Trametinib

Trametinib is an orally bioavailable inhibitor of mitogen-activated protein kinase kinase (MEK MAPK/ERK kinase) with potential antineoplastic activity. Trametinib specifically binds to and inhibits MEK 1 and 2, resulting in an inhibition of growth factor-mediated cell signaling and cellular proliferation in various cancers. MEK 1 and 2, dual specificity threonine/tyrosine kinases often upregulated in various cancer cell types, play a key role in the activation of the RAS/RAF/MEK/ERK signaling pathway that regulates cell growth.

Tofacitinib

Tofacitinib is a partial and reversible janus kinase (JAK) inihibitor that will prevent the body from responding to cytokine signals. By inhibiting JAKs, tofacitinib prevents the phosphorylation and activation of STATs. The JAK-STAT signalling pathway is involved in the transcription of cells involved in hematopoiesis, and immune cell function. Tofacitinib works therapeutically by inhibiting the JAK-STAT pathway to decrease the inflammatory response. However, there is evidence to suggest that it may also achieve efficacy via other pathways as well.

Dabrafenib

Dabrafenib mesylate (Tafinlar) is a reversible ATP-competitive kinase inhibitor and targets the MAPK pathway.

Dabrafenib is an orally bioavailable inhibitor of B-raf (BRAF) protein with antineoplastic activity. Dabrafenib selectively binds to and inhibits the activity of B-raf, which may inhibit the proliferation of tumor cells which contain a mutated BRAF gene.

Dabrafenib causes an inhibition of phosphorylated extracellular signal-regulated kinase (ERK). This indicates a decrease in cell proliferation. Furthermore, within 24 hours of administration, downstream mediators of the MAPK pathway are inhibited. BRAF belongs to the raf/mil family of serine/threonine protein kinases and plays a role in regulating the MAP kinase/Extracellular Signal-regulated Kinases signaling pathway, which may be constitutively activated due to BRAF gene mutations.

Vemurafenib

Vemurafenib is a competitive kinase inhibitor with activity against BRAF kinase with mutations like V600E. Vemurafenib blocks downstream processes to inhibit tumour growth and eventually trigger apoptosis. Vemurafenib does not have antitumour effects against melanoma cell lines with the wild-type BRAF mutation. It exerts its function by binding to the ATP-binding domain of the mutant BRAF. Vemurafenib was co-developed by Roche and Plexxikon and it obtained its FDA approval on August 17, 2011, under the company Hoffmann La Roche. BRAF activation results in cell growth, proliferation, and metastasis. BRAF is an intermediary molecule in MAPK whose activation depends on ERK activation, elevation of cyclin D1 and cellular proliferation. The mutation V600E produces a constitutively form of BRAF. Vemurafenib has been shown to reduce all activation markers related to BRAF; in clinical trials, vemurafenib treatment showed a reduction of cytoplasmic phosphorylated ERK and a cell proliferation driven by Ki-67. Studies also reported decrease in MAPK-related metabolic activity. All the different reports indicate thet Vemurafenib generates an almost complete inhibition of the MAPK pathway.

Potential Clinical Trials

Trial URL	Status	Title	Disease	Drug	Sites
https://clinicaltrials.gov/study/NCT06728072	Recruiting	Pilot Study Evaluating Microbiome Modulation Therapy (MBMT) With Ciprofloxacin, Metronidazole, and Aspirin in Addition to Standard of Care Chemotherapy in Patients Undergoing First-Line Therapy for Metastatic Colorectal Cancer	Colorectal Cancer	Standard of Care Chemotherapy + Metronidazole, ciprofloxacin, aspirin	Virginia Commonwealth University, Richmond, Virginia 23298
https://clinicaltrials.gov/study/NCT05948826	Recruiting	A Phase 1, First in Human, Dose-Escalation Study of TORL-3-600 in Participants With Advanced Cancer	Colorectal Cancer	TORL-3-600	Providence Medical Foundation, Fullerton, California 92835 UCLA - JCCC Clinical Research Unit, Los Angeles, California 90095 Sarah Cannon Research Institute, Denver, Colorado 80218
https://clinicaltrials.gov/study/NCT05379595	Recruiting	A Phase 1b/2, Open-Label Study of Amivantamab Monotherapy and in Addition to Standard-of-Care Chemotherapy in Participants With Advanced or Metastatic Colorectal Cancer	Colorectal Cancer	Amivantamab, Fluorouracil, Leucovorin, Oxaliplatin, Irinotecan	O Neal Comprehensive Cancer Center at UAB, Birmingham, Alabama 35233 University of Southern California, Los Angeles, California 90033 University of California, Los Angeles UCLA, Los Angeles, California 90404
https://clinicaltrials.gov/study/NCT05783622	Recruiting	An Open-Label, Multicenter, Phase 1/1b Study of JANX008 in Subjects with Advanced or Metastatic Solid Tumor Malignancies	Colorectal Cancer	JANX008	City of Hope Medical Center, Duarte, California 91010 University of California San Diego Moores Cancer Center, San Diego, California 92093 University of Chicago Medical Center, Chicago, Illinois 60637

Detailed Results

Single Nucleotide Variant (SNV) and Insertions-Deletions (INDELS)								
Gene name	Hgvsp	Hgvsc	Amino acids	Codons	Consequence	Allele frequency	Read depth	Predicted effect on protein
TP53	NP_000537.3:p.Pro278Ser	NM_000546.5:c.832C>T	P/S	Cct/Tct	missense_variant	40.08	3034	deleterious (0.03)
APC	NP_000029.2:p.Arg876Ter	NM_000038.5:c.2626C>T	R/*	Cga/Tga	stop_gained	39.22	3095	0
BRAF	NP_004324.2:p.Val600Glu	NM_004333.4:c.1799T>A	V/E	gTg/gAg	missense_variant	30.55	3935	deleterious (0)
INHBA	NP_002183.1:p.Ala82Ser	NM_002192.2:c.244G>T	A/S	Gcg/Tcg	missense_variant	29.91	4768	deleterious (0.01)
JAK3	NP_000206.2:p.Arg103His	NM_000215.3:c.308G>A	R/H	cGc/cAc	"missense_variant,splice_region_variant"	19.09	3169	deleterious (0)
DNMT3A	NP_783328.1:p.Asp658Tyr	NM_175629.2:c.1972G>T	D/Y	Gac/Tac	missense_variant	4.02	3085	deleterious (0)
KMT2C	NP_733751.2:p.Asp372Val	NM_170606.2:c.1115A>T	D/V	gAt/gTt	missense_variant	2.17	5333	0
SDHA	NP_004159.2:p.Ter665CysextTer1	NM_004168.2:c.1995A>T	*/C	tgA/tgT	stop_lost	1.03	3398	0
APC	NP_000029.2:p.Thr1556AsnfsTer3	NM_000038.5:c.4666dupA	E/EX	gaa/gAaa	frameshift_variant	0.63	3666	0
PTPRC (RNA)	NP_002829.3:p.Ter1307GlnextTer4	NM_002838.4:c.3919T>C	*/Q	Tag/Cag	stop_lost	53.5	585	0

Methodology and Test Background

This is a next generation sequencing (NGS) test that analyzes cfDNA in 302 genes and cfRNA in >1600 genes for abnormalities that are reported to be altered in various types of solid tumors. For cases with detectable circulating solid tumor DNA, tumor mutation burden (TMB) is reported. The assay also detects several viruses that are important in oncology, including EBV, HPV 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. 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) 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.

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.

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	BRAF	CDK12	CUX1	EPHA5	FGF4	GNAQ	IL7R	MAP2K1	MSH3	NPM1	PIM1	RAD21	SDHB	SRSF2	TRAF3
ABRAXAS1	AURKA	BRCA1	CDK4	CXCR4	ERBB2	FGF6	GNAS	INHBA	MAP2K2	MSH6	NRAS	PLCG1	RAD50	SDHC	STAG2	TSC1
ACVR1B	AURKB	BRCA2	CDK6	CYLD	ERBB3	FGFR1	GNB1	IRF4	MAP2K4	MTOR	NSD1	PMS1	RAD51	SDHD	STAT3	TSC2
AKT1	AURKC	BRIP1	CDKN1B	DAXX	ERBB4	FGFR2	GREM1	JAK1	MAP3K1	MUTYH	NSD2 (WHSC1)	PMS2	RAD51C	SETBP1	STAT5B	TSHR
AKT2	AXIN1	BTK	CDKN2A	DDR2	ERG	FGFR3	GRIN2A	JAK2	MAP3K14	MYC	NTHL1	POLD1	RAD51D	SETD2	STK11	U2AF1
AKT3	AXIN2	CALR	CDKN2B	DDX41	ESR1	FGFR4	H3-3A (H3F3A)	JAK3	MAPK1	MYCL	NTRK1	POLE	RAF1	SF3B1	SUFU	U2AF2
ALK	B2M	CARD11	CDKN2C	DICER1	ETNK1	FH	H3C2	KAT6A	MCL1	MYCN	NTRK2	POT1	RB1	SMAD2	SUZ12	UBA1
AMER1	BAP1	CBL	CEBPA	DNM2	ETV6	FLCN	HGF	KDM5C	MDM2	MYD88	NTRK3	PPM1D	RET	SMAD4	TAL1	VHL
ANKRD26	BARD1	CBLB	CHEK1	DNMT3A	EXO1	FLT3	HNF1A	KDM6A	MDM4	NBN	PAK3	PPP2R1A	RHEB	SMARCA4	TCF3	WT1
APC	BCL2	CBLC	CHEK2	DOT1L	EZH2	FLT4	HOXB13	KDR	MED12	NF1	PALB2	PRDM1	RHOA	SMARCB1	TENT5C (FAM46C)	XPO1
AR	BCL2L1	CCND1	CIC	EED	FANCA	FOXL2	HRAS	KEAP1	MEF2B	NF2	PAX5	PRKAR1A	RIT1	SMC1A	TERC	XRCC2
ARAF	BCL6	CCND3	CREBBP	EGFR	FANCC	FUBP1	HSP90AA1	KIT	MEN1	NFE2	PBRM1	PRKDC	RNF43	SMC3	TERT	XRCC3
ARID1A	BCOR	CCNE1	CRLF2	EGLN1	FANCD2	GALNT12	ID3	KMT2A	MET	NFE2L2	PDGFRA	PRPF8	ROS1	SMO	TET2	ZNF217
ARID1B	BCORL1	CD274	CSF1R	ELANE	FANCE	GATA1	IDH1	KMT2B	MITF	NFKBIA	PDGFRB	PRSS1	RUNX1	SOCS1	TGFBR2	ZRSR2
ARID2	BCR	CD79A	CSF3R	EP300	FANCF	GATA2	IDH2	KMT2C	MLH1	NKX2-1	PHF6	PTCH1	SAMD9	SOX2	TMEM127	-
ASXL1	BIRC3	CD79B	CTCF	EPAS1	FANCG	GATA3	IGF1R	KMT2D	MPL	NOTCH1	PIK3CA	PTEN	SAMD9L	SOX9	TNFAIP3	-
ATM	BLM	CDC73	CTNNA1	EPCAM	FAS	GEN1	IKZF1	KRAS	MRE11	NOTCH2	PIK3R1	PTPN11	SDHA	SPOP	TNFRSF14	-
ATR	BMPR1A	CDH1	CTNNB1	EPHA3	FBXW7	GNA11	IKZF3	LRP1B	MSH2	NOTCH3	PIK3R2	RAC1	SDHAF2	SRC	TP53	-

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	YWHAE
BCL2	CCND1	ERBB2	ETV6	FIP1L1	HMGA2	MAML2	NOTCH1	NUP214	PICALM	RARA	RUNX1T1	TFG	ZFTA

Reference

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3. Advancements in combining targeted therapy and immunotherapy for colorectal cancer. Singh M, Morris VK, Bandey IN, Hong DS, Kopetz S. Trends Cancer. 2024 Jul;10(7):598-609. doi: 10.1016/j.trecan.2024.05.001. Epub 2024 May 31. PMID: 38821852.

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Electronic Signature

Ahmad Charifa, M.D.

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.

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)