Genomic Testing Cooperative, LCA, 175 Technology Drive, Suite 100, Irvine, CA 92618
Tel: 1-866-484-8870 www.genomictestingcooperative.com
CLIA #: 05D2111917 CAP #: 9441547

Solid Tumor Profile Plus

Patient Name:	[redacted]	Ordering Physician:	[redacted]
Date of Birth:	[redacted]	Accession #:	[redacted]
Gender (M/F):	[redacted]	Specimen Type:	[redacted]
Client:	[redacted]	Specimen ID:	[redacted]
Case #:	[redacted]		
Body Site:	[redacted]		

MRN:	[redacted]			Indication for Testing:	C16.6 Malignant neoplasm of greater curvature of stomach, unspecified
Collected Date:	[redacted]	Time:	11:18 AM		
Received Date:	[redacted]	Time:	01:00 PM		
Reported Date:	[redacted]	Time:	05:56 PM		

Test Description:

This is a comprehensive molecular profile which uses next generation sequencing (NGS), fragment length analysis and Sanger Sequencing testing to identify molecular abnormalities (including SNVs, INDELS, CNVs, TMB, MSI and HRD) in DNA of 434 genes and RNA in 1408 genes implicated in solid tumors. Whenever possible, clinical relevance and implications of detected abnormalities are described below.

Detected Genomic Alterations

Level 1 (FDA-Approved)	Level 2 (Standard of Care)	Level 3 (Clinical Evidence)	Level 4 (Biological Relevance)	Other
ERBB2 amplification	-Homologous recombination deficiency (HRD): Intermediate -Tumor Mutation Burden Low: 9 Mut/Mb -No evidence of microsatellite instability	SYK, TP53, DNMT3A	FANCI, SETBP1, RANBP2	Chromosomal structural analysis shows 11q+, 17q+ (CDK12 and ERBB2 amplification) and 19q+ (CCNE1 amplification)

PD-L1 testing by immunohistochemistry (IHC) as performed and reported by CSI Laboratories: Clone 22C3 : Tumor cells: <1%; Immune cells: 10% Combined Positive Score (CPS): 10. PD-L1 expression level: Positive

Tumor Heterogeneity

There is a dominant abnormal clone with FANCI mutation. The SYK mutation is detected in a subclone. There are abnormal low-level clones with TP53, SETBP1, DNMT3A, and RANBP2 mutations.

Expression

Markedly high ERBB2 mRNA

Diagnostic Implications	
FANCI, SYK, TP53, SETBP1, DNMT3A, RANBP2	These abnormalities are consistent with aggressive neoplasm.

Prognostic Implications	
FANCI	Unknown
SYK	Poor
TP53	Poor
SETBP1	Unknown
DNMT3A	Poor
RANBP2	Unknown

FDA-Approved Therapeutics	
ERBB2 amplification	Trastuzumab, Ado-Trastuzumab Emtansine, Lapatinib, Pertuzumab+Trastuzumab, Lapatinib+Trastuzumab, Neratinib, Neratinib + Capecitabine, Trastuzumab + Pertuzumab + Chemotherapy, Trastuzumab + Tucatinib + Capecitabine, Trastuzumab Deruxtecan, Margetuximab + Chemotherapy, Tucatinib, Trastuzumab + Chemotherapy...Trastuzumab, Pembrolizumab + Trastuzumab + Chemotherapy, Trastuzumab + Chemotherapy, Trastuzumab Deruxtecan

FDA-Approved Therapeutics in Other Tumor Types	
HRD Intermediate	Niraparib + platinum-based chemotherapy

Relevant Alteration Associated with Resistance
TP53 mutation is associated with resistance to therapy.

Levels 2, 3 & 4 (Standard of Care and Clinical/Biological Evidence)	
SYK	SYK Inhibitor (FOSTAMATINIB)
TP53	Aurora kinase A inhibitors, Wee1 inhibitors, Chk1 inhibitors, kevetrin, APR-246, nutlins, gene therapy
DNMT3A	DNA methyltransferase inhibitors
HRD Intermediate	PARP Inhibitors and Platinum based chemotherapy
ERBB2 amplification	anti-HER2 targeted therapy

Relevant Genes with NO Alteration		
No evidence of mutation in: KRAS, NRAS, EGFR, BRAF,	No evidence of fusion mRNA involving ALK, RET, ROS1, or NTRK	No evidence of MET14 deletion, EGFR Viii

Results Summary

- **-Mutations in FANCI, SYK, TP53, SETBP1, DNMT3A, and RANBP2 genes**
  -Homologous recombination deficiency (HRD): Intermediate
  -Chromosomal structural anlysis shows 11q+, 17q+ (CDK12 and ERBB2 amplification) and 19q+ (CCNE1 amplification)
  -Tumor Mutation Burden Low: 9 Mut/Mb
  -No evidence of microsatellite instability
  -PD-L1 testing by immunohistochemistry (IHC) as performed and reported by CSI Laboratories: Clone 22C3 : Tumor cells: <1%; Immune cells: 10% Combined Positive Score (CPS): 10. PD-L1 expression level: Positive

  -ERBB2 gene amplification and overexpression suggest response to anti-HER2 therapy.

  -Borderline positive homologous recombination deficiency (HRD) suggests response to platinum-based chemotherapy and PARP inhibitors.

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

  -CCNE1 amplification suggests possible response to CDK inhibitors (Palbociclib).

Biological relevance of detected Alterations

- FANCI: The Fanconi anemia complementation group (FANC) currently includes FANCA, FANCB, FANCC, FANCD1 (also called BRCA2), FANCD2 FANCE, FANCF, FANCG, FANCI, FANCJ (also called BRIP1), FANCL, FANCM and FANCN (also called PALB2). The previously defined group FANCH is the same as FANCA. Fanconi anemia is a genetically heterogeneous recessive disorder characterized by cytogenetic instability, hypersensitivity to DNA crosslinking agents, increased chromosomal breakage, and defective DNA repair. The members of the Fanconi anemia complementation group do not share sequence similarity; they are related by their assembly into a common nuclear protein complex. This gene encodes the protein for complementation group I. Alternative splicing results in two transcript variants encoding different isoforms. [provided by RefSeq, Jul 2008]

- SYK encodes a member of the family of non-receptor type Tyr protein kinases. This protein is widely expressed in hematopoietic cells and is involved in coupling activated immunoreceptors to downstream signaling events that mediate diverse cellular responses, including proliferation, differentiation, and phagocytosis. It is thought to be a modulator of epithelial cell growth and a potential tumour suppressor in human breast carcinomas. Alternatively spliced transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, Mar 2010]

- TP53 mutations are universal across cancer types. The loss of a tumor suppressor is most often through large deleterious events, such as frameshift mutations, or premature stop codons. In TP53 however, many of the observed mutations in cancer are found to be single nucleotide missense variants. These variants are broadly distributed throughout the gene, but with the majority localizing in the DNA binding domain. There is no single hotspot in the DNA binding domain, but a majority of mutations occur in amino acid positions 175, 245, 248, 273, and 282 (NM_000546) (Olivier et al., 2010). To fulfill its proper biological function four TP53 polypeptides must form a tetramer which functions as a transcription factor, therefore even if one out of four polypeptides has inactivating mutation it may lead to dominant negative phenotype of variable degree. While a large proportion of cancer genomics research is focused on somatic variants, TP53 is also of note in the germline. Germline TP53 mutations are the hallmark of Li-Fraumeni syndrome, and many (both germline and somatic) variants have been found to have a prognostic impact on patient outcomes. The significance of many polymorphisms for susceptibility and prognosis of disease is still very much up for debate.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]

- SETBP1 encodes a protein which contains a several motifs including a ski homology region and a SET-binding region in addition to three nuclear localization signals. The encoded protein has been shown to bind the SET nuclear oncogene which is involved in DNA replication.

Mutations in this gene are associated with Schinzel-Giedion midface retraction syndrome. Multiple transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, Aug 2011]

- DNMT3A is one of several epigenetic modifiers identified as recurrently mutated in acute myeloid leukemia (AML). DNMT3A mutations are associated with cytogenetically normal AML. In vitro experiments indicate that the R882H mutation acts in a dominant negative manner to disrupt the de novo methyltransferase activity of wildtype homotetramers. AML patient bone marrow harboring R882 mutations were similarly demonstrated to be hypomethylated compared to patients with wildtype DNMT3A. These studies also indicated that non-R882 DNMT3A mutations may act in a functionally distinct manner from R882 mutations. Alternative mechanisms indicate independent prognostic outcomes and treatment protocols may need to be considered for these two classes of DNMT3A mutations.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]

- RANBP2: RAN is a small GTP-binding protein of the RAS superfamily that is associated with the nuclear membrane and is thought to control a variety of cellular functions through its interactions with other proteins. This gene encodes a very large RAN-binding protein that immunolocalizes to the nuclear pore complex. The protein is a giant scaffold and mosaic cyclophilin-related nucleoporin implicated in the Ran-GTPase cycle. The encoded protein directly interacts with the E2 enzyme UBC9 and strongly enhances SUMO1 transfer from UBC9 to the SUMO1 target SP100. These findings place sumoylation at the cytoplasmic filaments of the nuclear pore complex and suggest that, for some substrates, modification and nuclear import are linked events. This gene is partially duplicated in a gene cluster that lies in a hot spot for recombination on chromosome 2q. [provided by RefSeq, Jul 2008]

Drug Information

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 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.

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.

Olaparib

Olaparib 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 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)

Lynparza is 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.

Trastuzumab

Trastuzumab (Herceptin) is a monoclonal antibody, a man-made version of a very specific immune system protein, which targets the HER2 protein. Giving trastuzumab with chemo can help some patients with advanced, HER2-positive stomach cancer live longer than giving chemo alone.

-It is suggested that the overexpression or gene amplification of HER2 has been found in about 20–30% of breast cancers and elevated activation of HER2 triggers multiple downstream pathways leading to abnormal proliferation of cancer cells. Trastuzumab binds to HER2 and suppresses cancer cells growth, proliferation, and survival directly and indirectly.

-In December 2017, FDA approved Ogivri (trastuzumab-dkst) as a biosimilar to Herceptin (trastuzumab) for the treatment of patients with breast or metastatic stomach cancer (gastric or gastroesophageal junction adenocarcinoma) whose tumors overexpress the HER2 gene (HER2+).

Pertuzumab

Pertuzumab is a humanized monoclonal antibody designed to bind to the HER2 receptor and inhibit the ability of HER2 to interact with other HER family members (HER1, HER2, HER3, and HER4) on the surface of cancer cells. The HER signaling pathway plays a role in the formation and growth of numerous cancers, and previous clinical trials of pertuzumab in a single agent setting had suggested clinical activity - including stable disease -

in heavily pretreated patients with advanced ovarian and breast cancers.

Pertuzumab is indicated for use in combination with trastuzumab and docetaxel for the treatment of patients with HER2-positive metastatic breast cancer who have not received prior anti-HER2 therapy or chemotherapy for metastatic disease.

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).

Azacitidine

Azacitidine is a pyrimidine analogue that inhibits DNA methyltransferase, impairing DNA methylation. It is also an antimetabolite of cytidine, incorporated primarily into RNA. Azacytidine has been used as an antineoplastic agent.

Azacitidine for injection is a nucleoside metabolic inhibitor indicated for the treatment of patients with the following FAB myelodysplastic syndrome (MDS) subtypes: Refractory anemia (RA) or refractory anemia with ringed sideroblasts (RARS) (if accompanied by neutropenia or thrombocytopenia or requiring transfusions), refractory anemia with excess blasts (RAEB), refractory anemia with excess blasts in transformation (RAEB-T), and chronic myelomonocytic leukemia (CMMoL). (1)

Azacitidine for injection is indicated for treatment of patients with the following French-American-British (FAB) myelodysplastic syndrome subtypes: refractory anemia (RA) or refractory anemia with ringed sideroblasts (if accompanied by neutropenia or thrombocytopenia or requiring transfusions), refractory anemia with excess blasts (RAEB), refractory anemia with excess blasts in transformation (RAEB-T), and chronic myelomonocytic leukemia (CMMoL).

Decitabine

Decitabine is a cytidine antimetabolite analogue with potential antineoplastic activity. Decitabine incorporates into DNA and inhibits DNA methyltransferase, resulting in hypomethylation of DNA and intra-S-phase arrest of DNA replication.

Decitabine for injection is indicated for treatment of adult patients with myelodysplastic syndromes (MDS) including previously treated and untreated, de novo and secondary MDS of all French-American-British subtypes (refractory anemia, refractory anemia with ringed sideroblasts, refractory anemia with excess blasts, refractory anemia with excess blasts in transformation, and chronic myelomonocytic leukemia) and intermediate-1, intermediate-2, and high-risk International Prognostic Scoring System groups.

Decitabine also has EU approval for acute myeloid leukemia (AML).

Palbociclib

Palbociclib is an investigational selective, small-molecule inhibitor of CDK4 and CDK6. CDK4 and CDK6 along with their regulatory partner cyclin D1 play a key role in regulating the G1- to S-phase cell-cycle transition via regulation of phosphorylation of the retinoblastoma (Rb) protein. Inhibition of these proteins leads to reduced phosphorylation of Rb, inhibition of downstream signalling, and increased tumor growth arrest.

Palbociclib is indicated in combination with letrozole for the treatment of postmenopausal women with estrogen receptor (ER)-positive, human epidermal growth factor receptor 2 (HER2)-negative advanced breast cancer as initial endocrine-based therapy for their metastatic disease.

Potential Clinical Trials

Trial URL	Status	Title	Disease	Drug	Sites
https://ClinicalTrials.gov/show/NCT04379596	Recruiting	Ph1b/2 Study of the Safety and Efficacy of T-DXd Combinations in Advanced HER2+ Gastric Cancer (DESTINY-Gastric03)	Gastric Cancer	Fluorouracil (5-FU) Capecitabine Durvalumab Oxaliplatin Trastuzumab Trastuzumab	Research Site, La Jolla, California, United States Research Site, Santa Monica, California, United States

				deruxtecan Cisplatin Pembrolizumab	Research Site, Stanford, California, United States
https://ClinicalTrials.gov/show/NCT04725994	Recruiting	Study to Assess the Safety, Tolerability, and Efficacy of IDX-1197 in Combination With XELOX or Irinotecan in Patients With Advanced Gastric Cancer	Gastric Cancer	IDX-1197+XELOX IDX-1197+Irinotecan	USC Norris Comp. Cancer Ctr Hospital, Los Angeles, California, United States Emory University Winship Cancer Institute, Atlanta, Georgia, United States Cleveland Clinic, Cleveland, Ohio, United States
https://ClinicalTrials.gov/show/NCT04082364	Recruiting	Combination Margetuximab, Retifanlimab, Tebotelimab, and Chemotherapy Phase 2/3 Trial in HER2+ Gastric/GEJ Cancer	Gastric Cancer	margetuximab Retifanlimab Tebotelimab Trastuzumab Chemotherapy	Mayo Clinic - Scottsdale, Scottsdale, Arizona, United States City of Hope Comprehensive Cancer Center - Duarte, Duarte, California, United States Norris Comprehensive Cancer Center (USC), Los Angeles, California, United States

Detailed Results

Single Nucleotide Variant (SNV)

Gene Name	Hgvsp	Hgvsc	Amino Acids	Codons	Consequence	Allele frequency	Read depth	Predicted effect on protein
FANCI	NP_001106849.1:p.His99IlefsTer10	NM_001113378.1:c.295delC	H/X	caC/ca	frameshift_variant	49.35	154	0
SYK	NP_003168.2:p.Glu440Lys	NM_003177.5:c.1318G>A	E/K	Gag/Aag	missense_variant	23.53	238	tolerated (0.28)
TP53	NP_000537.3:p.Glu204Ter	NM_000546.5:c.610G>T	E/*	Gag/Tag	stop_gained	6.7	194	0
SETBP1	NP_056374.2:p.Val448Leu	NM_015559.2:c.1342G>C	V/L	Gtt/Ctt	missense_variant	5.91	220	tolerated (0.19)
DNMT3A	NP_783328.1:p.Phe384Ser	NM_175629.2:c.1151T>C	F/S	tTc/tCc	missense_variant	5.0	80	deleterious (0.01)
RANBP2	NP_006258.3:p.Ser832Arg	NM_006267.4:c.2496C>G	S/R	agC/agG	missense_variant	3.95	152	tolerated (0.06)

Methodology and Test Background

This is a next generation sequencing (NGS) test that analyzes DNA for abnormalities in 434 genes that are reported to be altered in various types of tumors. Nucleic acid is isolated from paraffin-embedded tissue. 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 50 nucleotides at the 5' and 3' ends of each coding exon. Our sequencing method has a typical sensitivity of 3% for detecting common specific mutations and 5% for other mutations. MSI status is inferred by interrogating all available genomic microsatellites covered. Tumor mutational burden (TMB) is measured by counting all non-

synonymous variants and filter settings as follows: (A) Pass all filters; (B) inside genes; (C) had a mutant allele frequency >5%; (D) not found in the dbSNP (to exclude germline variations). The median for TMB is 10 based on lung carcinoma analysis. The cut off for other types of tumors is not well established at this time. Performance of the assay may vary dependent on the quantity and quality of nucleic acid, sample preparation and sample age. The assay is designed to detect significant gene amplification and deletion in addition to various single nucleotide variations (SNV) and indels.

In addition to DNA analysis, targeted RNA NGS analysis is performed. This is a next generation sequencing (NGS) test that analyzes targeted RNA on 1,408 genes implicated in solid tumors. It is based on hybrid capture of targeted RNA. Duplicates are excluded for levels measurements. While the major focus of the analysis is the detection of fusion mRNA, mutations in the expressed RNA of the analyzed genes are also analyzed and reported. mRNA expression levels are evaluated, and only significant high expression of specific genes are relatively reported. CD274 (PD-L1) mRNA levels are reported when they are significantly elevated. All detect fusion transcripts are reported. 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. The sensitivity of this assay in detecting fusion mRNA is between 1% and 5%. This assay is not designed to detect minimal residual disease and should be used for diagnosis. For optimal results neoplastic cells should be >30% of the analyzed cells. The Universal Human Reference (UHR) RNA is used as control.

Based on our validation study, the following regions of the genes listed below are not covered appropriately (<100 X coverage) and sequencing by NGS may not be reliable in these regions. This poor coverage is due to high GC content with inherited problem in obtaining adequate coverage. CSF3R, 36937661, 36937745, R343-K357, 1. RANBP2, 109378551, 109378656, V868-k899, 2. PBRM1, 52677258, 52677364, R300-R332, 3. BAP1, 52443852, 52443899, M1-G13, 3. FLT4, 180035275, 180035289, R1298 (Last AA), 5. RHEB, 151216540, 151216602, M1-G18, 7. RHEB, 151195169, 151195198, Intronic region, 7. ANKRD26, 27368955, 27369112, Intonic region, 10. ERBB3, 56492278, 56492364, T873-G898, 12. DDX11, 31240866, 31240922, R186-K202, 12. IRS2, 110437064, 110437332, S357-A441, 13. FLT3, 28674599, 28674652, M1-V15, 13. CCNE1, 30303457, 30303490, M1-R8, 19. MED12, 70361074, 70361225, Q2090-2136, X

Tested genes

Genes Tested for Abnormalities in Coding Sequence															
ABCS3:AE27B7	AURKB	C15ORF41	CEBPA	DICER1	FANCC	FLT3	GRIN2A	IRF2	LMO1	MSH2	NTRK1	POLE	RANBP2	SETD2	STAT4	TSC2
ABL1	AURKC	CALR	CHD2	DOT1L	FANCD2	FLT4	GRM3	IRF4	LPIN2	MSH6	NTRK2	POT1	RARA	SF3B1	STAT6	TSHR
ABL2	AXIN1	CARD11	CHD4	EED	FANCE	FOXL2	GSK3B	IRS2	LRP1B	MTOR	NTRK3	PPM1D	RB1	SLIT2	STK11	U2AF1
ACD	AXIN2	CBFB	CHEK1	EGFR	FANCF	FOXP1	GSKIP	JAGN1	LYN	MUTYH	NUP93	PPP2R1A	RBBP6	SLX4	SUFU	U2AF2
ACVR1B	AXL	CBL	CHEK2	EGLN1	FANCG	FRS2	H3F3A	JAK1	LYST	MVK	PAK3	PRDM1	RBM10	SMAD2	SUZ12	VEGFA
ADA	B2M	CBLB	CIC	ELANE	FANCI	FUBP1	HAX1	JAK2	LZTR1	MYC	PALB2	PREX2	RBM8A	SMAD3	SYK	VHL
AK2	BAP1	CBLC	CREBBP	EP300	FANCL	G6PC3	HGF	JAK3	MAGI2	MYCL	PARK2	PRKAR1A	RET	SMAD4	TAF1	WAS
AKT1	BARD1	CCND1	CRKL	EPAS1	FANCM	GABRA6	HIST1H3B	JUN	MAP2K1	MYCN	PAX5	PRKCI	RHEB	SMAD9	TAL1	WHSC1
AKT2	BCL2	CCND2	CRLF2	EPCAM	FAS	GALNT12	HNF1A	KAT6A	MAP2K2	MYD88	PBRM1	PRKDC	RHOA	SMAD9L	TBX3	WISP3
AKT3	BCL2L1	CCND3	CSF1R	EPHA3	FAT1	GATA1	HOXA11	KDM5A	MAP2K4	NBN	PDCD1LG2	PRSS1	RICTOR	SMARCA4	TCF3	WT1
ALK	BCL2L2	CCNE1	CSF3R	EPHA5	FBXW7	GATA2	HOXB13	KDM5C	MAP3K1	NF1	PDGFRA	PRSS8	RIT1	SMARCB1	TCIRG1	XPO1
AMER1	BCL6	CD274	CTC1	EPHA7	FGF10	GATA3	HRAS	KDM6A	MAP3K14	NF2	PDGFRB	PSTPIP1	RNF168	SMC1A	TERC	XRCC2
ANKRD26	BCOR	CD79A	CTCF	EPHB1	FGF14	GATA4	HSD3B1	KDR	MAPK1	NFE2L2	PDK1	PTCH1	RNF43	SMC3	TERF1	XRCC3
APC	BCORL1	CD79B	CTNNA1	ERBB2	FGF19	GATA6	HSP90AA1	KEAP1	MCL1	NFKBIA	PHF6	PTEN	ROS1	SMO	TERF2	ZBTB2
AR	BCR	CDAN1	CTNNB1	ERBB3	FGF23	GEN1	ID3	KEL	MDM2	NHP2	PIK3C2B	PTPN11	RPTOR	SNCAIP	TERF2IP	ZNF217
ARAF	BIRC3	CDC73	CUL3	ERBB4	FGF3	GFI1	IDH1	KIF23	MDM4	NKX2-1	PIK3CA	QKI	RTEL1	SOCS1	TERT	ZNF703
ARFRP1	BLM	CDH1	CUX1	ERCC4	FGF4	GFI1B	IDH2	KIT	MED12	NLRP3	PIK3CB	RAB27A	RUNX1	SOX10	TET2	ZRSR2
ARID1A	BMPR1A	CDK12	CXCR4	ERG	FGF6	GID4	IGF1R	KLF1	MEF2B	NME1	PIK3CG	RAC1	RUNX1T1	SOX2	TGFBR2	
ARID1B	BRAF	CDK4	CYLD	ERRFI1	FGFR1	GLI1	IGF2	KLHL6	MEFV	NOP10	PIK3R1	RAD21	SBDS	SOX9	TNFAIP3	
ARID2	BRCA1	CDK6	DAXX	ESR1	FGFR2	GLI2	IKBKE	KLLN	MEN1	NOTCH1	PIK3R2	RAD50	SBF2	SPEN	TNFRSF14	
ASXL1	BRCA2	CDK8	DDR2	ETV6	FGFR3	GNA11	IKZF1	KMT2A	Merged	NOTCH2	PIM1	RAD51	SDHA	SPOP	TNFRSF1A	
ATG2B	BRD4	CDKN1A	DDX11	EXO1	FGFR4	GNA13	IKZF3	KMT2B	MET	NOTCH3	PLCG1	RAD51B	SDHB	SPTA1	TOP1	

ATM	BRIP1	CDKN1B	DDX41	EZH2	FH	GNAQ	IL2RG	KMT2C	MITF	NPM1	PLCG2	RAD51C	SDHC	SRC	TOP2A	
ATR	BTG1	CDKN2A	DKC1	FAM175A	FLCN	GNAS	IL7R	KMT2D	MLH1	NRAS	PMS1	RAD51D	SDHD	SRSF2	TP53	
ATRX	BTK	CDKN2B	DNM2	FAM46C	FLI1	GPR124	INHBA	KRAS	MPL	NROB1	PMS2	RAD54L	SEC23B	STAG2	TRAF3	
AURKA	C11orf30	CDKN2C	DNMT3A	FANCA	FLT1	GREM1	INPP4B	LIG4	MRE11A	NSD1	POLD1	RAF1	SETBP1	STAT3	TSC1	
* Microsatellite markers BAT25, BAT26, D2S123, D5S346, and D17S250 are included.																

RNA Fusions/Expression

Fusion/Expression													
ABL1	BCL2	CBFB	ERG	FGFR2	FOXO1	IKZF3	MAP3K1	NTRK1	NUP98	PICALM	RHOA	SS18	TCF3
AKT3	BCL6	CIC	ETV6	FGFR3	FUS	JAK2	MECOM	NTRK2	PDGFRA	PML	ROS2	STAT6	TFG
ALK	BRAF	CREBBP	EWSR1	FIP1L1	GLI1	KIAA1549	MYC	NTRK3	PDGFRB	RARA	RUNX1	TAFG	YWHAE
BCL1	CAMTA1	EGFR	FGFR1	FLAG1	HMGA2	KMT2A	NOTCH1	NUP214	PD-L1	RET	RUNX1T1	TAL1	

Reference

1. Integration of Trastuzumab, with or without Pertuzumab, into Perioperative Chemotherapy of HER2- Positive Stomach Cancer: The INNOVATION Trial (EORTC-1203-GITCG). [No authors listed] [No authors listed] Oncol Res Treat. 2016;39(3):153-4; discussion 155. doi: 10.1159/000444702. Oncol Res Treat. 2016. PMID: 27486629

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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, Genomic Testing Cooperative, LCA, 175 Technology Drive, Suite 100, Irvine, CA 92618. Medical Director Maher Albitar, M.D. Analysis of the data was performed in part at Genomic Testing Cooperative, LCA, 175 Technology Drive, Suite 100, Irvine, CA 92618. 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.