GENOMIC TESTING COOPERATIVE
175 Technology Dr, Suite 100 Irvine, CA 92618
Tel: 1-949-450-9421 genomictestingcooperative.com

Hematology Profile Plus

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

Ethnicity:				Family History:	
MRN:				Indication for Testing:	
Collected Date:		Time :		Reason for Referral:	Malignant Neoplasm of Lung
Received Date:		Time :		Tumor Type:	Lung
Reported Date:		Time :		Stage:	T2B

Test Description:

This is a next generation sequencing (NGS) test to identify molecular abnormalities in DNA of 177 genes and RNA in 68 genes implicated in hematologic neoplasms, including leukemia, lymphoma melanoma and MDS. Whenever possible, clinical relevance and implications of detected abnormalities are described below.

Detected Genomic Alterations				
FLT3-ITD	IDH2	TET2	DNMT3A	NRAS
t(9;12)(p24;p13)				

Heterogeneity
IDH1 mutation is detected in very small subclone when compared with the rest of the mutations

Expression	
CD274	Low
MYC	High

Diagnostic Implications	
Acute Leukemia	Consistent with Acute Myeloid Leukemia (AML), but NRAS mutation suggests AMML, likely evolving from CMML background.
MDS	N/A
Lymphoma	N/A
Myeloma	N/A
Other	N/A

Therapeutic Implications	
FLT3-ITD	Rydapt (Midostaurin)

The professional and technical components of this assay were performed at Genomic Testing Cooperative, LCA, 27 Technology Drive, Suite 100, Irvine, CA 92618 (CLIA ID: 05D2111917).

IDH2 (Subclone)	Idhifa (Enasidenib)

Prognostic Implications	
FLT3-ITD	Poor
IDH2	Neutral
TET2	Neutral
DNMT3A	Poor
NRAS	Neutral
JAK2-ETV6 t(9;12)	Poor
Overall	Poor

Relevant Genes with No Alteration
NPM1

Results Summary

- There are mutations in FLT3-ITD, TET2, IDH2, DNMT3A, NRAS genes. In addition, a fusion JAK2-ETV6 RNA was detected.
- These findings are consistent with the diagnosis of AML. However, the presence of a mutation in NRAS gene is consistent with monocytic lineage involvement in the leukemic process and the diagnosis of acute monocytic leukemia or acute myelomonocytic leukemia.
- The presence of these abnormalities is consistent with aggressive disease and poor outcome.
- The presence of FLT3-ITD abnormality suggests response to FLT3 inhibitors and the presence of IDH2 mutation suggests possible response to IDH2 inhibitors, but since IDH2 mutation is detected in only subclone, therapy with IDH2 inhibitors may be only relevant for a subclone and may not affect the founding clone.

Biological Relevance of Detected Alterations

- FLT3 is an important cytokine receptor involved in normal hematopoiesis. Mutations in this gene are common in acute myeloid leukemia (AML) and screening for mutations in this gene has been recommended by the World Health Organization in patients with AML, particularly in cases of cytogenetically normal AML (CN-AML). FLT3 mutations commonly co-occur with mutations such as NPM1 that are associated with CN-AML and likely modulate prognostic impact. While FLT3-ITD mutations have been associated with poorer prognosis in AML, the prognostic impact of FLT3-TKD mutations are still up for debate.
- IDH2 mutations have been observed in a number of cancer types, including sarcomas, hematologic malignancies, colon cancer and brain cancer. Mutations in the two isocitrate dehydrogenase enzymes involved in cytoplasmic (IDH1) and mitochondrial (IDH2) conversion of alpha-ketoglutarate to D-2-hydroxyglutarate have been described as mutually exclusive in many of these cancer types. The most frequent mutations involve R132 (IDH1) and R172 (IDH2) involve the active site and result in neomorphic enzyme activity. Although IDH2 (R172) mutations are associated with poorer overall prognosis in AML patients, its utility as a prognostic marker in MDS is still under debate. Additionally, IDH2 (R140) has been associated with improved overall survival in AML. IDH2 mutations have been associated with improved prognosis in gliomas.
- TET2 (Tet Methylcytosine Dioxygenase 2) gene encodes a methylcytosine dioxygenase that catalyzes the conversion of methylcytosine to 5-hydroxymethylcytosine. The encoded protein is involved in myelopoiesis, and defects in this gene have been associated with several myeloproliferative disorders. In addition to its role in DNA demethylation, also involved in the recruitment of the O-GlcNAc transferase OGT to CpG-rich transcription start sites of active genes, thereby promoting histone H2B GlcNAcylation by OGT. No targeted therapy is available for this gene. However hypomethylation agents are considered to be relevant in treatment of diseases with abnormalities in this gene.

- 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.
- Mutations in the RAS family of proteins have frequently been observed across cancer types. The amino acid positions G12, G13 and Q61 account for the overwhelming majority of these mutations. The isoforms, despite their raw similarity, also behave very differently when expressed in non-native tissue types, likely due to differences in the C-terminal hyper-variable regions. Mis-regulation of isoform expression has been shown to be a driving event in cancer, as well as missense mutations at the three hotspots previously mentioned. While highly recurrent in cancer, targeting these RAS mutants has also been very elusive, and has not yet become common practice in the clinic.

Drug Information

Midostaurin

FLT3 Inhibitor
1.1 Acute Myeloid Leukemia
RYDAPT is indicated, in combination with standard cytarabine and daunorubicin induction and cytarabine consolidation chemotherapy, for the treatment of adult patients with newly diagnosed acute myeloid leukemia (AML) who are FLT3 mutation-positive, as detected by a FDA approved test
Limitations of Use
RYDAPT is not indicated as a single-agent induction therapy for the treatment of patients with AML.
1.2 Systemic Mastocytosis
RYDAPT is indicated for the treatment of adult patients with aggressive systemic mastocytosis (ASM), systemic mastocytosis with associated hematological neoplasm (SM-AHN), or mast cell leukemia (MCL).

Enasidenib

IDH2 Inhibitor
Acute Myeloid Leukemia
IDHIFA is indicated for the treatment of adult patients with relapsed or refractory acute myeloid leukemia (AML) with an isocitrate dehydrogenase-2 (IDH2) mutation as detected by an FDA-approved test.

Potential Clinical Trials

Title	Conditions	Interventions	Locations	URL
Study of FF-10101-01 in Patients With Relapsed or Refractory Acute Myeloid Leukemia	AML, Adult	Drug: FF-10101-01	University Of California, San Francisco School of Medicine, San Francisco, California, United States|Northwestern University, Chicago, Illinois, United States|Johns Hopkins Hospital - Sidney Kimmel Cancer Center, Baltimore, Maryland, United States|University of Pennsylvania, Philadelphia, Pennsylvania, United States	https://ClinicalTrials.gov/show/NCT03194685
Combination Merestinib and LY2874455 for Patients With Relapsed or Refractory	Relapsed Adult Acute Myeloid Leukemia|Refractory Adult Acute	Drug: Merestinib|Drug: LY2874455	Brigham and Women's Hospital, Boston, Massachusetts, United States|Dana-Farber Cancer Institute, Boston, Massachusetts, United States	https://ClinicalTrials.gov/show/NCT03125239

Acute Myeloid Leukemia	Myeloid Leukemia			
Efficacy of Intermediate-Dose Cytarabine Induction Regimen in Adult AML	AML	Drug: Daunomycin and Cytarabine (DA Regimen)|Drug: Daunomycin and Cytarabine (Intermediate Dose of DA Regimen)	Institute of Hematology & Blood Diseases Hospital, Tianjin, Tianjin, China	https://ClinicalTrials.gov/show/NCT03021330
Safety and Activity of Digoxin With Decitabine in Adult AML and MDS	Acute Myeloid Leukemia|Myelodysplastic Syndromes	Drug: Decitabine|Drug: Digoxin	Fox Chase Cancer Center, Philadelphia, Pennsylvania, United States|Jeans Hospital, Philadelphia, Pennsylvania, United States	https://ClinicalTrials.gov/show/NCT03113071
Treatment of Older Adult Acute Myeloid Leukemia Patients Aged 55 to 65 Years	Acute Myeloid Leukemia	Drug: Daunorubicin|Drug: Cytarabine	Treatment and Diagnosis Center of Leukemia, Tianjin, Tianjin, China	https://ClinicalTrials.gov/show/NCT02432872
Metabolic Changes in Blood Samples From Patients With Acute Myeloid Leukemia	Recurrent Adult Acute Myeloid Leukemia|Untreated Adult Acute Myeloid Leukemia	Other: Cytology Specimen Collection Procedure|Other: Laboratory Biomarker Analysis	Comprehensive Cancer Center of Wake Forest University, Winston-Salem, North Carolina, United States	https://ClinicalTrials.gov/show/NCT02581917
Selinexor With Induction, Consolidation, and Maintenance Therapy in Treating Older Patients With Acute Myeloid Leukemia	Untreated Adult Acute Myeloid Leukemia	Drug: Cytarabine|Drug: Daunorubicin Hydrochloride|Drug: Selinexor	Comprehensive Cancer Center of Wake Forest University, Winston-Salem, North Carolina, United States	https://ClinicalTrials.gov/show/NCT02835222
Outpatient Induction Chemotherapy in Treating Patients With Acute Myeloid Leukemia or Advanced Myelodysplastic Syndrome	Adult Acute Myeloid Leukemia|Adult Myelodysplastic Syndrome	Drug: Chemotherapy	Bozeman Deaconess Hospital, Bozeman, Montana, United States|Kadlec Clinic Hematology and Oncology, Kennewick, Washington, United States|EvergreenHealth Medical Center, Kirkland, Washington, United States|Skagit Valley Hospital, Mount Vernon, Washington, United States|Olympic Medical Center, Port Angeles, Washington, United States|Group Health Cooperative, Redmond, Washington, United States|Fred Hutch/University of	https://ClinicalTrials.gov/show/NCT01807091

			Washington Cancer Consortium, Seattle, Washington, United States|Multicare Health System, Tacoma, Washington, United States|Wenatchee Valley Hospital and Clinics, Wenatchee, Washington, United States	
FLT PET/CT in Measuring Response in Patients With Previously Untreated Acute Myeloid Leukemia	Acute Myeloid Leukemia|Untreated Adult Acute Myeloid Leukemia	Drug: Chemotherapy|Procedure: Computed Tomography|Drug: Cytarabine|Other: Fluorothymidine F-18|Other: Laboratory Biomarker Analysis|Procedure: Positron Emission Tomography	University of Alabama at Birmingham Cancer Center, Birmingham, Alabama, United States|Mayo Clinic, Rochester, Minnesota, United States|Washington University School of Medicine, Saint Louis, Missouri, United States|Mount Sinai Hospital, New York, New York, United States|UNC Lineberger Comprehensive Cancer Center, Chapel Hill, North Carolina, United States|University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma, United States|University of Pennsylvania/Abramson Cancer Center, Philadelphia, Pennsylvania, United States|Fox Chase Cancer Center, Philadelphia, Pennsylvania, United States|Vanderbilt University/Ingram Cancer Center, Nashville, Tennessee, United States|UT Southwestern/Simmons Cancer Center-Dallas, Dallas, Texas, United States|Huntsman Cancer Institute/University of Utah, Salt Lake City, Utah, United States|University of Wisconsin Hospital and Clinics, Madison, Wisconsin, United States	https://ClinicalTrials.gov/show/NCT02392429
CIP-613, Cytarabine, and Mitoxantrone Hydrochloride in Treating Patients With Relapsed or Refractory Acute Myeloid Leukemia or Granulocytic Sarcoma	Granulocytic Sarcoma|Recurrent Adult Acute Myeloid Leukemia	Drug: 6,8-Bis(benzylthio)octanoic Acid|Drug: Cytarabine|Procedure: Hematopoietic Cell Transplantation|Drug: Mitoxantrone Hydrochloride	Comprehensive Cancer Center of Wake Forest University, Winston-Salem, North Carolina, United States	https://ClinicalTrials.gov/show/NCT02484391

Detailed Results

The professional and technical components of this assay were performed at Genomic Testing Cooperative, LCA, 27 Technology Drive, Suite 100, Irvine, CA 92618 (CLIA ID: 05D2111917).
The assay is FDA cleared and the performance characteristics were established at this location and was

27 Technology Dr, Suite 100 Irvine, CA 92618
Tel: 1-949-450-9421 genomictestingcooperative.com

Single Nucleotide Variant (SNV)								
Gene name	Hgvsp	Hgvsc	Aminoacids	Codons	Consequence	Allele frequency	Read depth	Predicted effect on protein
TET2	NP_001120680.1:p.Gln138Ter	NM_001127208.2:c.412C>T	Q/*	Caa/Taa	stop_gained	35.3	645	-
DNMT3A	NP_783328.1:p.Met224IlefsTer92	NM_175629.2:c.672delG	M/X	atG/at	frameshift_variant	30.6	395	-
NRAS	NP_002515.1:p.Gly60Glu	NM_002524.4:c.179G>A	G/E	gGa/gAa	missense_variant	39.2	213	deleterious (0)
FLT3 (ITD)	NP_004110.2:p.Tyr597_Glu598insAspTyrValAspPheArgGluTyr	NM_004119.2:c.1770_1793dupCTACGTTGATTTCAGAGAATATGA	-	-	inframe_insertion	30.8	120	-
IDH2	NP_002159.2:p.Arg140Gln	NM_002168.2:c.419G>A	R/Q	cGg/cAg	missense_variant	12.2	546	deleterious

Fusion	
JAK2-ETV6 / t(9;12)(p24;p13)	51%

Methodology and Test Background

This is a next generation sequencing (NGS) test that analyzes DNA for abnormalities in 177 genes that are reported to be altered in various types of hematologic neoplasms. Nucleic acid is isolated from plasma, fresh cells peripheral blood cells or bone marrow or body fluid), or 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. Fragment length analysis is also performed on CALR, FLT3, and NPM1 to enhance the detection of insertion/deletion mutations in these genes. Our sequencing method has a typical sensitivity of 3% for detecting common specific mutations and 5% for other mutations. Known hot spots in specific genes such as IDH1/2, NRAS, and KRAS are reported at levels of 1% and higher. The FLT3-ITD fragment analysis assay has a sensitivity of 2%-5% for detecting FLT3-ITD in wildtype background. The CALR fragment analysis test has a sensitivity of 2%-5% for detecting heterozygous insertion/deletions in the wild-type background. 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 was performed. This analyzes targeted RNA with a focus on 68 genes. 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, mainly to distinguish B-cell neoplasms from myeloid. CRLF2 mRNA levels are reported in acute lymphoblastic leukemia. CD274 (PD-L1) mRNA levels are reported when they are significantly elevated. If requested, detailed expression levels will be provided as a research data and not for clinical use. 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. Since the clinical relevance of the expression level of most of these genes is not characterized at this time, only few specific genes (MYC, BCL2, CD274, CD19, CD22, CD79A, CD79B) will be commented on. 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 when neoplastic cells are >10% of the analyzed cells. The Universal Human Reference (UHR) RNA is used as control.

GENOMIC TESTING COOPERATIVE
27 Technology Dr, Suite 100 Irvine, CA 92618
Tel: 1-949-450-9421 genomictestingcooperative.com

Tested genes

Genes Tested for Abnormalities in Coding Sequen												
ABL1	BCL2	CBL	CDKN2C	DICER1	FAS	IDH2	KMT2A	MPL	PAX5	PTCH1	SMAD2	TGFBR2
AKT1	BCL2L1	CBLB	CEBPA	DNMT3A	FBXW7	IGF1R	KMT2B	MRE11A	PBRM1	PTEN	SMAD4	TP53
AKT2	BCL6	CBLC	CHEK1	EP300	FLT3	IKZF1	KMT2C	MTOR	PDGFRA	PTPN11	SMARCA4	TSC1
AKT3	BCOR	CCND1	CHEK2	ERG	GATA1	IKZF3	KMT2D	MUTYH	PDGFRB	RAD21	SMARCB1	TSC2
ALK	BCORL1	CCND3	CIC	ETV6	GATA2	IRF4	KRAS	MYC	PHF6	RAD50	SMC1A	TSHR
AMER1	BCR	CD274	CREBBP	EZH2	GATA3	JAK1	MAP2K1	MYD88	PIK3CA	RAD51	SMO	WT1
APC	BIRC3	CD79A	CRLF2	FAM175A	GEN1	JAK2	MAP2K2	NFKBIA	PIK3R1	RB1	SOCS1	ZNF217
ARID1A	BLM	CD79B	CSF1R	FAM46C	GNAQ	JAK3	MAP2K4	NOTCH1	PIK3R2	RHOA	SRC	ZRSR2
ARID1B	BRAF	CDH1	CSF3R	FANCA	GNAS	KAT6A	MAP3K1	NOTCH2	PIM1	RNF43	SRSF2	MEF2B
ARID2	BRCA1	CDK12	CTNNA1	FANCC	H3F3A	KDM5C	MAP3K14	NOTCH3	PLCG1	RUNX1	STAG2	
ASXL1	BRCA2	CDK4	CTNNB1	FANCD2	HNF1A	KDM6A	MAPK1	NPM1	POLD1	SDHB	STAT3	
ATM	BTK	CDK6	CUX1	FANCE	HOXB13	KDR	MCL1	NRAS	POLE	SETBP1	STK11	
ATRX	CALR	CDKN2A	CXCR4	FANCF	HSP90AA1	KEAP1	MDM2	NSD1	PPM1D	SETD2	TERT	
B2M	CARD11	CDKN2B	DDR2	FANCG	IDH1	KIT	MDM4	PALB2	PPP2R1A	SF3B1	TET2	

Add-on RNA Fusions/Expression

Fusion/Expression																
ABL1	ALK	BRAF	CREBBP	EPOR	ETV5	FGFR2	FOXO1	*JAK2*	MAP3K1	NOTCH1	NUP214	PCM1	PICALM	RET	RUNX1T1	TCF3
ABL2	BCL1	CBFB	*CRLF2*	ERG	ETV6	FGFR3	FUS	KMT2A	MECOM	NTRK1	NUP98	PDGFRA	PML	RHOA	SS18	TCF3
AKT3	BCL2	*CBL*	*CSF1R*	ETV1	EWSR1	FIP1L1	GLI1	KRT18P6	MYC	NTRK2	P2RY8	PDGFRB	*PTK2B*	ROS2	STAT6	TFG
ALK	BCL6	CIC	EGFR	ETV4	*FGFR1*	*FLT3*	IKZF3	*LYN*	*MYH9*	*NTRK3*	PBX1	PD-L1	RARA	RUNX1	TAL1	*TYK2*

References

1. Midostaurin does not prolong cardiac repolarization defined in a thorough electrocardiogram trial in healthy volunteers. del Corral A, Dutreix C, Huntsman-Labed A, Lorenzo S, Morganroth J, Harrell R, Wang Y. Cancer Chemother Pharmacol. 2012 May;69(5):1255-63. doi: 10.1007/s00280-012-1825-y.
2. Comparison of two endogenous biomarkers of CYP3A4 activity in a drug-drug interaction study between midostaurin and rifampicin. Dutreix C, Lorenzo S, Wang Y. Eur J Clin Pharmacol. 2014 Aug;70(8):915-20. doi: 10.1007/s00228-014-1675-0. Epub 2014 May 21.
3. Mast cell leukemia with prolonged survival on PKC412/midostaurin. Xu X, Kreisel FH, Frater JL, Hassan A. Int J Clin Exp Pathol. 2014 May 15;7(6):3439-43. eCollection 2014.
4. The DNA Methyltransferase DNMT1 and Tyrosine-Protein Kinase KIT Cooperatively Promote Resistance to 5-Aza-2'-deoxycytidine (Decitabine) and Midostaurin (PKC412) in Lung Cancer Cells. Yan F, Shen N, Pang J, Molina JR, Yang P, Liu S. J Biol Chem. 2015 Jul 24;290(30):18480-94. doi: 10.1074/jbc.M114.633693. Epub 2015 Jun 17.
5. IDH2 inhibition in AML: Finally progress? Stein EM. Best Pract Res Clin Haematol. 2015 Jun-Sep;28(2-3):112-5. doi: 10.1016/j.beha.2015.10.016. Epub 2015 Oct 19. Review.
6. Overview: A New Era of Cancer Genome in Myeloid Malignancies. Kiyoi H. Oncology. 2015;89 Suppl 1:1-3. doi: 10.1159/000431054. Epub 2015 Nov 10. Review.
7. Cancer metabolism pipeline breaks new ground. Mullard A. Nat Rev Drug Discov. 2016 Nov 3;15(11):735-737. doi: 10.1038/nrd.2016.223.
8. Enasidenib in mutant IDH2 relapsed or refractory acute myeloid leukemia. Stein EM, DiNardo CD, Pollyea DA, Fathi AT, Roboz GJ, Altman JK, Stone RM, DeAngelo DJ, Levine RL, Flinn IW, Kantarjian HM, Collins R, Patel MR, Frankel AE, Stein A, Sekeres MA, Swords RT, Medeiros BC, Willekens C, Vyas P, Tosolini A, Xu Q, Knight RD, Yen KE, Agresta S, de Botton S, Tallman MS. Blood. 2017 Aug 10;130(6):722-731. doi: 10.1182/blood-2017-04-779405. Epub 2017 Jun 6.
9. AG-221, a First-in-Class Therapy Targeting Acute Myeloid Leukemia Harboring Oncogenic IDH2 Mutations. Yen K, Travins J, Wang F, David MD, Artin E, Straley K, Padyana A, Gross S, DeLaBarre B, Tobin E, Chen Y, Nagaraja R, Choe S, Jin L, Konteatis Z, Cianchetta G, Saunders JO, Salituro FG, Quivoron C, Opolon P, Bawa O, Saada V, Paci A, Broutin S, Bernard OA, de Botton S, Marteyn BS, Pilichowska M, Xu Y, Fang C, Jiang F, Wei W, Jin S, Silverman L, Liu W, Yang H, Dang L, Dorsch M, Penard-Lacronique V, Biller SA, Su SM. Cancer Discov. 2017 May;7(5):478-493. doi: 10.1158/2159-8290.CD-16-1034. Epub 2017 Feb 13.
10. Reasons for optimism in the therapy of acute leukemia. Rowe JM. Best Pract Res Clin Haematol. 2015 Jun-Sep;28(2-3):69-72. doi: 10.1016/j.beha.2015.10.002. Epub 2015 Oct 22. Review.
11. Enasidenib. Drugs and Lactation Database (LactMed) [Internet]. Bethesda (MD): National Library of Medicine (US); 2006.
12. Optimizing Next-Generation AML Therapy: Activity of Mutant IDH2 Inhibitor AG-221 in Preclinical Models. Thomas D, Majeti R. Cancer Discov. 2017 May;7(5):459-461. doi: 10.1158/2159-8290.CD-17-0270.
13. A pharmacogenomic approach validates AG-221 as an effective and on-target therapy in IDH2 mutant AML. Kats LM, Vervoort SJ, Cole R, Rogers AJ, Gregory GP, Vidacs E, Li J, Nagaraja R, Yen KE, Johnstone RW. Leukemia. 2017 Jun;31(6):1466-1470. doi: 10.1038/leu.2017.84. Epub 2017 Mar 10. No abstract available.
14. The role of mutant IDH1 and IDH2 inhibitors in the treatment of acute myeloid leukemia. Nassereddine S, Lap CJ, Haroun F, Tabbara I. Ann Hematol. 2017 Dec;96(12):1983-1991. doi: 10.1007/s00277-017-3161-0. Epub 2017 Oct 31. Review.

Electronic Signature

Maher Albitar, M.D., Pathologist - GTC Laboratories

The Technical Component Processing, Analysis and Professional Component of this test was completed at GTC Laboratories, 21 Technology Dr. #100, Irvine, CA / 92618/ Medical Director: Maher Albitar, M.D. .

The performance characteristics of this test have been determined by GTC Laboratories. 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.