

## Liquid Trace Hematology

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

MRN:		Indication for Testing:	Other specified types of non-Hodgkin lymphoma, extranodal and solid organ sites (C85.89)
Collected Date:		Time:	12:00 AM
Received Date:		Time:	10:23 AM
Reported Date:		Time:	09:16 PM
		Tumor Type:	Lymphoma

Detected Genomic Alterations				
MYD88	MCL1 (2 mutations)	IKZF3	CD79B	PIM1 (3 mutations)
B2M	Autosomal chromosomes show +12.	B-cell clonality: Detectable (IgHV3-74 / IgKV2D-28)	T-cell clonality: Not detected	

### Results Summary

- **-Mutations in MYD88, MCL1 (2 mutations), IKZF3, CD79B, PIM1 (3 mutations), and B2M genes**
  - B-cell markers: Mildly increased with normal expression pattern.**
  - B-cell clonality: Detectable (IgHV3-74 / IgKV2D-28).**
  - T-cell clonality: Not detected.**
  - EBV viral RNA: Not detected.**
  - HPV viral RNA: Not detected.**
  - TTV viral RNA: Not detected.**
  - HLA Genotyping:**
    - HLA-A: A\*02:01-A\*11:01**
    - HLA-B: B\*55:01-B\*40:06**
    - HLA-C: C\*15:02-C\*01:02**
  - Autosomal chromosomes show +12.**
- These findings are most suggestive of diffuse large B-cell lymphoma.

**See additional report information at the end of the report.**

### Heterogeneity

There are abnormal clones with MYD88, MCL1 (2 mutations), IKZF3, CD79B, PIM1 (3 mutations), and B2M mutation.

### Expression

B-cell markers: Mildly increased with normal expression pattern

### Diagnostic Implications

MYD88, MCL1 (2 mutations), IKZF3, CD79B, PIM1 (3 mutations), B2M	These findings are consistent with a B-cell lymphoma
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### Therapeutic Implications

MYD88	BTK inhibitors
MCL1	Tubulins

### Prognostic Implications

MYD88	Poor
MCL1 (2 mutations)	Poor
IKZF3	Unknown
CD79B	Unknown
PIM1 (3 mutations)	Unknown
B2M	Unknown

### Relevant Genes with NO Alteration

No evidence of mutation in NOTCH, SF3B1, or TP53

## 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), 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 hematologic neoplasms, including leukemia, lymphoma, myeloma, myelodysplastic syndrome, and myeloproliferative neoplasms. Whenever possible, clinical relevance and implications of detected abnormalities are described below. If a gene is not reported, then no somatic mutations were detected. This assay facilitates myelodysplastic syndrome risk assessment as it includes evaluation for mutations and significant chromosomal gains and losses in all of the genes included in the IPSS-M risk calculator: ASXL1, BCOR, BCORL1, CBL, CEBPA, DNMT3A, ETNK1, ETV6, EZH2, FLT3, GATA2, GNB1, IDH1, IDH2, KMT2A (including KMT2A(MLL)-PTD), KRAS, NF1, NPM1, NRAS, PHF6, PPM1D, PRPF8, PTPN11, RUNX1, SETBP1, SF3B1, SRSF2, STAG2, TP53, U2AF1, and WT1.

## Biological relevance of detected Alterations

- **MYD88.** This gene encodes a cytosolic adapter protein that plays a central role in the innate and adaptive immune response. This protein functions as an essential signal transducer in the interleukin-1 and Toll-like receptor signaling pathways. These pathways regulate that activation of numerous proinflammatory genes. The encoded protein consists of an N-terminal death domain and a C-terminal Toll-interleukin1 receptor domain. Patients with defects in this gene have an increased susceptibility to pyogenic bacterial infections. Alternate splicing results in multiple transcript variants. [provided by RefSeq, Feb 2010]
- **MCL1.** This gene encodes an anti-apoptotic protein, which is a member of the Bcl-2 family. Alternative splicing results in multiple transcript variants. The longest gene product (isoform 1) enhances cell survival by inhibiting apoptosis while the alternatively spliced shorter gene products (isoform 2 and isoform 3) promote apoptosis and are death-inducing. [provided by RefSeq, Oct 2010]
- **IKZF3.** This gene encodes a member of the Ikaros family of zinc-finger proteins. Three members of this protein family (Ikaros, Aiolos and Helios) are hematopoietic-specific transcription factors involved in the regulation of lymphocyte development. This gene product is a transcription factor that is important in the regulation of B lymphocyte proliferation and differentiation. Both Ikaros and Aiolos can participate in chromatin remodeling. Regulation of gene expression in B lymphocytes by Aiolos is complex as it appears to require the sequential formation of Ikaros homodimers, Ikaros/Aiolos heterodimers, and Aiolos homodimers. Several alternative transcripts encoding different isoforms have been described, as well as some non-protein coding variants. [provided by RefSeq, Apr 2012]
- **CD79B.** The B lymphocyte antigen receptor is a multimeric complex that includes the antigen-specific component, surface immunoglobulin (Ig). Surface Ig non-covalently associates with two other proteins, Ig-alpha and Ig-beta, which are necessary for expression and function of the B-cell antigen receptor. This gene encodes the Ig-beta protein of the B-cell antigen component. Alternatively spliced transcript variants encoding different isoforms have been described. [provided by RefSeq, Jul 2008]
- **PIM1.** The protein encoded by this gene belongs to the Ser/Thr protein kinase family, and PIM subfamily. This gene is expressed primarily in B-lymphoid and myeloid cell lines, and is overexpressed in hematopoietic malignancies and in prostate cancer. It plays a role in signal transduction in blood cells, contributing to both cell proliferation and survival, and thus provides a selective advantage in tumorigenesis. Both the human and orthologous mouse genes have been reported to encode two isoforms (with preferential cellular localization) resulting from the use of alternative in-frame translation initiation codons, the upstream non-AUG (CUG) and downstream AUG codons (PMIDs:16186805, 1825810).[provided by RefSeq, Aug 2011]
- **B2M.** This gene encodes a serum protein found in association with the major histocompatibility complex (MHC) class I heavy chain on the surface of nearly all nucleated cells. The protein has a predominantly beta-pleated sheet structure that can form amyloid fibrils in some pathological conditions. The encoded antimicrobial protein displays antibacterial activity in amniotic fluid. A mutation in this gene has been shown to result in hypercatabolic hypoproteinemia.[provided by RefSeq, Aug 2014]

## Drug Information

### Rituximab (Rituxan)

Rituximab is a monoclonal antibody that targets the CD20 antigen, which is expressed on the surface of pre-B and mature B-lymphocytes. After binding to CD20, rituximab mediates B-cell lysis (or breakdown). The possible mechanisms of cell lysis include complement dependent cytotoxicity (CDC) and antibody dependent cell-mediated cytotoxicity (ADCC).

Rituximab is indicated in the following conditions:

- Non-Hodgkin Lymphoma (NHL)
- Chronic Lymphocytic Leukemia (CLL)
- Rheumatoid Arthritis (RA) in combination with methotrexate in adult patients with moderately-to severely-active RA
- Granulomatosis with Polyangiitis (GPA) (Wegener Granulomatosis) and Microscopic Polyangiitis (MPA)
- Moderate to severe Pemphigus Vulgaris (PV) in adult patients

### Ibrutinib (Imbruvica)

Ibrutinib is a small molecule that acts as an irreversible potent inhibitor of Bruton tyrosine kinase (BTK). It is designated as a targeted covalent drug and it presents a very promising activity in B cell malignancies. Ibrutinib forms a covalent bond with a cysteine residue in the active site of BTK (Cys481), leading to its inhibition. The inhibition of BTK plays a role in the B-cell receptor signaling and thus, the presence of ibrutinib prevents the phosphorylation of downstream substrates such as PLC-gamma.

Ibrutinib was developed by Pharmacyclics Inc and in November 2013 was FDA-approved for the treatment of mantle cell lymphoma. Later, in February 2014, ibrutinib was approved for the treatment of chronic lymphocytic leukemia and it is also indicated for the treatment of patients with Waldenstrom Macroglobulinemia. Ibrutinib has also been approved by the EMA for the treatment of chronic lymphocytic leukemia and mantle cell lymphoma. Ibrutinib was approved for use in chronic graft versus host disease in August 2017.

Ibrutinib is indicated for:

- treatment of mantle cell lymphoma who have received at least one prior therapy.
- treatment of chronic lymphocytic leukemia (CLL) who have at least one prior therapy.
- treatment of chronic lymphocytic leukemia (CLL) with 17p deletion.
- treatment of patients with Waldenstrom Macroglobulinemia (WM).

### Venetoclax (Venclexta)

Venetoclax is a selective inhibitor of both BCL-2 and BCL-2-like 1 (BCL-X(L)), which has demonstrated clinical efficacy in some BCL-2-dependent hematological cancers. Selective inhibition of BCL-2 by venetoclax, sparing BCL-xL enables therapeutic induction of apoptosis without the negative effect of thrombocytopenia. Venetoclax helps restore the process of apoptosis by binding directly to the BCL-2 protein, displacing pro-apoptotic proteins, leading to mitochondrial outer membrane permeabilization and the activation of caspase enzymes. In nonclinical studies, venetoclax has shown cytotoxic activity in tumor cells that overexpress BCL-2

### Zanubrutinib

Zanubrutinib, sold under the brand name Brukinsa, is a medication for the treatment of mantle cell lymphoma and Waldenström macroglobulinemia. It was approved for medical use in the United States in November 2019. Zanubrutinib is classified as a Bruton tyrosine kinase inhibitor. Zanubrutinib is an immunomodulating agent that decreases the survival of malignant B cells. Zanubrutinib inhibits BTK by forming a covalent bond with cysteine 481 residue in the adenosine triphosphate (ATP)-binding pocket of BTK, which is the enzyme's active site.

### Potential Clinical Trials

Trial URL	Status	Title	Disease	Drug	Sites
<a href="https://clinicaltrials.gov/study/NCT05828589">https://clinicaltrials.gov/study/NCT05828589</a>	Recruiting	A Phase 1/1b Open-Label Dose-Escalation and Dose-Optimization Study of Bcl-2 Inhibitor BGB-21447 in Patients With Mature B-Cell Malignancies	B-cell Lymphoma	BGB-21447	University of Iowa Hospitals and Clinics, Iowa City, Iowa 52242-1009 Mission Cancer and Blood, Waukee, Iowa 50263 Sidney Kimmel Comprehensive Cancer At Johns Hopkins, Baltimore, Maryland 21287
<a href="https://clinicaltrials.gov/study/NCT05006716">https://clinicaltrials.gov/study/NCT05006716</a>	Recruiting	A Phase 1/2, Open-Label, Dose-Escalation and -Expansion Study of the Bruton Tyrosine Kinase Targeted Protein Degradator BGB-16673 in Patients With B-Cell Malignancies	B-cell Lymphoma	BGB-16673	University of Alabama At Birmingham Hospital, Birmingham, Alabama 35294-0004 Western Regional Medical Center, Llc, Goodyear, Arizona 85338-3007 Mayo Clinic Phoenix, Phoenix, Arizona 85054-4502
<a href="https://clinicaltrials.gov/study/NCT05544019">https://clinicaltrials.gov/study/NCT05544019</a>	Recruiting	A Phase 1, Open-Label, Multicenter, Dose Escalation Study of SGR-1505 as Monotherapy in Subjects With Mature B-Cell Malignancies	B-cell Lymphoma	SGR-1505	Banner Health - MD Anderson Cancer Center, Gilbert, Arizona 85234 Christiana Care Hospital - Helen F Graham Cancer Center, Newark, Delaware 19713 Napa Research, Pompano Beach, Florida 99064

<a href="https://clinicaltrials.gov/study/NCT05512390">https://clinicaltrials.gov/study/NCT05512390</a>	Recruiting	A First In Human Multicenter, Open-Label Study to Determine the Safety, Tolerability, Pharmacokinetics, and Preliminary Efficacy of ABBV-319 in B-cell Malignancies	B-cell Lymphoma	ABBV-319	University of Arizona Cancer Center - Tucson /ID# 247752, Tucson, Arizona 85724 Sylvester Comprehensive Cancer Center - University of Miami /ID# 247232, Miami, Florida 33136 Allina Health System /ID# 251782, Minneapolis, Minnesota 55407-1321
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## 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
MYD88	NP_002459.2:p.Leu265Pro	NM_002468.4:c.794T>C	L/P	cTg/cCg	missense_variant	35.71	14	deleterious
MCL1	NP_068779.1:p.Ser206Asn	NM_021960.4:c.617G>A	S/N	aGc/aAc	missense_variant	33.33	9	tolerated
IKZF3	NP_001244337.1:p.Asn97Ser	NM_001257408.1:c.290A>G	N/S	aAt/aGt	missense_variant	31.58	19	deleterious
CD79B	NP_000617.1:p.Tyr196His	NM_000626.2:c.586T>C	Y/H	Tac/Cac	missense_variant	31.58	19	deleterious
PIM1	NP_001230115.1:p.Pro172Ser	NM_001243186.1:c.514C>T	P/S	Cct/Tct	"missense_variant,splice_region_variant"	30.77	13	0
PIM1	NP_001230115.1:p.Glu121Lys	NM_001243186.1:c.361G>A	E/K	Gag/Aag	missense_variant	30.0	10	0
PIM1	NP_001230115.1:p.Asn98Lys	NM_001243186.1:c.294C>G	N/K	aaC/aaG	missense_variant	20.0	10	0
MCL1	NP_068779.1:p.Asn260Thr	NM_021960.4:c.779A>C	N/T	aAc/aCc	missense_variant	20.0	20	deleterious
B2M	NP_004039.1:p.?	NM_004048.2:c.1A>G	M/V	Atg/Gtg	start_lost	17.65	17	deleterious

## Methodology and Test Background

This is a next generation sequencing (NGS) test that analyzes cfDNA for abnormalities in 302 genes and cfrRNA of >1600 genes for abnormalities that are reported in various types of hematologic neoplasms. 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 or CSF. When CSF sample is submitted, RNA sequencing is performed on the CSF cell pellet instead of cfrRNA 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. 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, B- and T-cell clonality, HLA class I genotyping, and Epstein-Barr virus (EBV), human papillomavirus (HPV) and torque teno virus (TTV) viral RNA are also analyzed and reported. 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.

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.

**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-hematologic-malignancies/> (click the DNA tab)

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

## Tested genes

Genes Tested for Abnormalities in Coding Sequence												
ABL1	B2M	CCNE1	CUX1	ETNK1	GALNT12	IL7R	MCL1	NFE2L2	PIM1	RB1	SMO	TRAF3
ABRAXAS1	BAP1	CD274	CXCR4	ETV6	GATA1	INHBA	MDM2	NFKBIA	PLCG1	RET	SOCS1	TSC1
ACVR1B	BARD1	CD79A	CYLD	EXO1	GATA2	IRF4	MDM4	NKX2-1	PMS1	RHEB	SOX2	TSC2
AKT1	BCL2	CD79B	DAXX	EZH2	GATA3	JAK1	MED12	NOTCH1	PMS2	RHOA	SOX9	TSHR
AKT2	BCL2L1	CDC73	DDR2	FANCA	GEN1	JAK2	MEF2B	NOTCH2	POLD1	RIT1	SPOP	U2AF1
AKT3	BCL6	CDH1	DDX41	FANCC	GNA11	JAK3	MEN1	NOTCH3	POLE	RNF43	SRC	U2AF2
ALK	BCOR	CDK12	DICER1	FANCD2	GNAQ	KAT6A	MET	NPM1	POT1	ROS1	SRSF2	UBA1
AMER1	BCORL1	CDK4	DNM2	FANCE	GNAS	KDM5C	MITF	NRAS	PPM1D	RUNX1	STAG2	VHL
ANKRD26	BCR	CDK6	DNMT3A	FANCF	GNB1	KDM6A	MLH1	NSD1	PPP2R1A	SAMD9	STAT3	WT1
APC	BIRC3	CDKN1B	DOT1L	FANCG	GREM1	KDR	MPL	NSD2 (WHSC1)	PRDM1	SAMD9L	STAT5B	XP01
AR	BLM	CDKN2A	EED	FAS	GRIN2A	KEAP1	MRE11	NTHL1	PRKAR1A	SDHA	STK11	XRCC2
ARAF	BMPR1A	CDKN2B	EGFR	FBXW7	H3-3A (H3F3A)	KIT	MSH2	NTRK1	PRKDC	SDHAF2	SUFU	XRCC3
ARID1A	BRAF	CDKN2C	EGLN1	FGF4	H3C2 (HIST1H3B)	KMT2A	MSH3	NTRK2	PRPF8	SDHB	SUZ12	ZNF217
ARID1B	BRCA1	CEBPA	ELANE	FGF6	HGF	KMT2B	MSH6	NTRK3	PRSS1	SDHC	TAL1	ZRSR2

ARID2	BRCA2	CHEK1	EP300	FGFR1	HNFA1	KMT2C	MTOR	PAK3	PTCH1	SDHD	TCF3	-
ASXL1	BRIP1	CHEK2	EPAS1	FGFR2	HOXB13	KMT2D	MUTYH	PALB2	PTEN	SETBP1	TENT5C (FAM46C)	-
ATM	BTK	CIC	EPCAM	FGFR3	HRAS	KRAS	MYC	PAX5	PTPN11	SETD2	TERC	-
ATR	CALR	CREBBP	EPHA3	FGFR4	HSP90AA1	LRP1B	MYCL	PBRM1	RAC1	SF3B1	TERT	-
ATRX	CARD11	CRLF2	EPHA5	FH	ID3	MAP2K1	MYCN	PDGFRA	RAD21	SMAD2	TET2	-
AURKA	CBL	CSF1R	ERBB2	FLCN	IDH1	MAP2K2	MYD88	PDGFRB	RAD50	SMAD4	TGFBR2	-
AURKB	CBLB	CSF3R	ERBB3	FLT3	IDH2	MAP2K4	NBN	PHF6	RAD51	SMARCA4	TMEM127	-
AURKC	CBLC	CTCF	ERBB4	FLT4	IGF1R	MAP3K1	NF1	PIK3CA	RAD51C	SMARCB1	TNFAIP3	-
AXIN1	CCND1	CTNNA1	ERG	FOXL2	IKZF1	MAP3K14	NF2	PIK3R1	RAD51D	SMC1A	TNFRSF14	-
AXIN2	CCND3	CTNNB1	ESR1	FUBP1	IKZF3	MAPK1	NFE2	PIK3R2	RAF1	SMC3	TP53	-

## RNA Fusions/Expression

Fusion/Expression																
ABL1	BCL2	CCND1	CREBBP	EGFR	ETV4	FGFR2	FOXO1	IKZF3	MAP3K1	MYH9	NTRK3	PAX5	PDGFRB	PTK2B	ROS1	TAL1
ABL2	BCL6	CD274 (PD-L1)	CRLF2	EPOR	ETV5	FGFR3	FUS	JAK2	MECOM	NOTCH1	NUP214	PBX1	PICALM	RARA	RUNX1	TCF3
AKT3	BRAF	CBL	CSF1R	ERG	ETV6	FIP1L1	GLI1	KMT2A	MRTFA	NTRK1	NUP98	PCM1	PIGA	RET	RUNX1T1	TFG
ALK	CBFB	CIC	DUSP22	ETV1	FGFR1	FLT3	HLF	LYN	MYC	NTRK2	P2RY8	PDGFRA	PML	RHOA	STAT6	TYK2

## Reference

1. B-cell lymphoma: Advances in pathogenesis, diagnosis, and targeted therapies. Patil S, Rajput S, Patil S, Mhaikar A. Pathol Res Pract. 2025 Jul;271:156036. doi: 10.1016/j.prp.2025.156036. Epub 2025 May 26. PMID: 40435909.
2. Bispecific antibodies for the treatment of B-cell lymphoma: promises, unknowns, and opportunities. Falchi L, Vardhana SA, Salles GA. Blood. 2023 Feb 2;141(5):467-480. doi: 10.1182/blood.2021011994. PMID: 36322929.
3. Effects of B-Cell Lymphoma on the Immune System and Immune Recovery after Treatment: The Paradigm of Targeted Therapy. Mancuso S, Mattana M, Carlisi M, Santoro M, Siragusa S. Int J Mol Sci. 2022 Mar 21;23(6):3368. doi: 10.3390/ijms23063368. PMID: 35328789.
4. Targeting the tumor microenvironment in B-cell lymphoma: challenges and opportunities. Liu Y, Zhou X, Wang X. J Hematol Oncol. 2021 Aug 17;14(1):125. doi: 10.1186/s13045-021-01134-x. PMID: 34404434.

## Electronic Signature

Maher Albitar, 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)

