GENOMIC TESTING COOPERATIVE®
25371 Commercentre Drive, Lake Forest, CA 92630
Tel: 1-866-484-8870 | genomictestingcooperative.com
CLIA # 05D2111917 CAP # 9441574

Liquid Trace Hematology

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

MRN:				Indication for Testing:	Small cell B-cell lymphoma, unspecified site (C83.00)
Collected Date:		Time:	12:00 AM		
Received Date:		Time:	10:28 AM		
Reported Date:		Time:	05:09 PM		

Detected Genomic Alterations				
FLCN (?Germline)	SRSF2	DNMT3A (2 mutations)	BRAF (G466R)	NOTCH1
PRKDC	TTV viral RNA: Detected (23 copies)	Autosomal chromosomes show low level 13q-.	B cell clonality: Detected, triclonal (one heavy chain: IGHV4-4 and three light chains: IGKV2D-28, IGKV1D-39, IGLV10-54)	T cell clonality: Not detected

Results Summary

- **-Low level somatic mutations in SRSF2, DNMT3A (2 mutations), BRAF, NOTCH1, and PRKDC genes**
  -Possible germline mutation in FLCN gene, heterozygous
  -Autosomal chromosomes show low level 13q-.
  -B cell clonality: Detected, triclonal (one heavy chain: IGHV4-4 and three light chains: IGKV2D-28, IGKV1D-39, IGLV10-54)
  -T cell clonality: Not detected
  -B cell markers: Increased with normal pattern
  -BCL2 and CD5 mRNA: Increased
  -EBV viral RNA: Not detected
  -HPV viral RNA: Not detected
  -TTV viral RNA: Detected (23 copies)
  -HLA Genotyping:
  -HLA-A: A*24:02-A*32:01
  -HLA-B: B*35:02-B*27:02
  -HLA-C: C*04:01-C*02:02

  -These findings are consistent with chronic lymphocytic leukemia / small lymphocytic lymphoma (CLL/SLL). However, the SRSF2 and DNMT3A mutations are likely in myeloid cells, consistent with CHIP (clonal hematopoiesis of indeterminate potential).

-The FLCN mutation is detected at high level raising the possibility of a germline abnormality. This mutation has been reported as a germline pathogenic abnormality associated with predisposition to cancer.

See additional report information at the end of the report.

Heterogeneity

There is an abnormal low-level clone with SRSF2, DNMT3A (2 mutations), BRAF, NOTCH1, and PRKDC mutations.
The FLCN mutation is detected at a high level possible germline abnormality.

Expression

B cell markers: Increased with normal pattern	BCL2 and CD5 mRNA: Increased

Diagnostic Implications

FLCN, SRSF2, DNMT3A (2 mutations), BRAF, NOTCH1, PRKDC	-These findings are consistent with chronic lymphocytic leukemia / small lymphocytic lymphoma (CLL/SLL). -The FLCN mutation is likely a germline variant.

Therapeutic Implications

SRSF2	Spliceosome modifiers
DNMT3A	DNA methyltransferase inhibitors
BRAF	ERK/MEK Inhibitors
NOTCH1	NOTCH inhibitors
PRKDC	PI3K/AKT, PARP inhibitors

Prognostic Implications

SRSF2	Poor
DNMT3A (2 mutations)	Poor
BRAF	Poor
NOTCH1	Poor
PRKDC	Poor

Relevant Genes with NO Alteration

No evidence of mutation in: SF3B1, TP53, or MYD88

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

- FLCN. This gene is located within the Smith-Magenis syndrome region on chromosome 17. Mutations in this gene are associated with Birt-Hogg-Dube syndrome, which is characterized by fibrofolliculomas, renal tumors, lung cysts, and pneumothorax. Alternative splicing of this gene results in two transcript variants encoding different isoforms. [provided by RefSeq, Jul 2008]

- SRSF2. The protein encoded by this gene is a member of the serine/arginine (SR)-rich family of pre-mRNA splicing factors, which constitute part of the spliceosome. Each of these factors contains an RNA recognition motif (RRM) for binding RNA and an RS domain for binding other proteins. The RS domain is rich in serine and arginine residues and facilitates interaction between different SR splicing factors. In addition to being critical for mRNA splicing, the SR proteins have also been shown to be involved in mRNA export from the nucleus and in translation. Two transcript variants encoding the same protein and one non-coding transcript variant have been found for this gene. In addition, a pseudogene of this gene has been found on chromosome 11. [provided by RefSeq, Sep 2010]

- 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]

- 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]

- NOTCH1. This gene encodes a member of the NOTCH family of proteins. Members of this Type I transmembrane protein family share structural characteristics including an extracellular domain consisting of multiple epidermal growth factor-like (EGF) repeats, and an intracellular domain consisting of multiple different domain types. Notch signaling is an evolutionarily conserved intercellular signaling pathway that regulates interactions between physically adjacent cells through binding of Notch family receptors to their cognate ligands. The encoded preproprotein is proteolytically processed in the trans-Golgi network to generate two polypeptide chains that heterodimerize to form the mature cell-surface receptor. This receptor plays a role in the development of numerous cell and tissue types. Mutations in this gene are associated with aortic valve disease, Adams-Oliver syndrome, T-cell acute lymphoblastic leukemia, chronic lymphocytic leukemia, and head and neck squamous cell carcinoma. [provided by RefSeq, Jan 2016]

- PRKDC. This gene encodes the catalytic subunit of the DNA-dependent protein kinase (DNA-PK). It functions with the Ku70/Ku80 heterodimer protein in DNA double strand break repair and recombination. The protein encoded is a member of the PI3/PI4-kinase family.[provided by RefSeq, Jul 2010]

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)

A BCL-2 inhibitor indicated for the treatment of patients with chronic lymphocytic leukemia (CLL) or small lymphocytic lymphoma (SLL), with or without 17p deletion, who have received at least one prior therapy. Venetoclax induces rapid and potent onset apoptosis of CLL cells, powerful enough to act within 24h and to lead to tumor lysis syndrome. Selective targeting of BCL2 with venetoclax has demonstrated a manageable safety profile and has been shown to induce significant response in patients with relapsed CLL (chronic lymphocytic leukemia) or SLL (small lymphocytic leukemia), including patients with poor prognostic features.

Potential Clinical Trials

Trial URL	Status	Title	Disease	Drug	Sites
https://clinicaltrials.gov/study/NCT06073821	Recruiting	A Phase 3, Open-Label, Randomized Study of Sonrotoclax (BGB-11417) Plus Zanubrutinib (BGB-3111) Compared With Venetoclax Plus Obinutuzumab in Patients With Previously Untreated Chronic Lymphocytic Leukemia	Chronic Lymphocytic Leukemia	Sonrotoclax, Zanubrutinib, Venetoclax, Obinutuzumab	John Theurer Cancer Center Hackensack University Medical Center, Hackensack, New Jersey 07601-2191 Laura and Isaac Perlmutter Cancer Center At Nyu Langone Health, New York, New York 10016-2708 Columbia University Medical Center, New York, New York 10032
https://clinicaltrials.gov/study/NCT05963074	Recruiting	Multicohort Study to Customize Ibrutinib Treatment Regimens for Patients With Previously Untreated Chronic Lymphocytic Leukemia	Chronic Lymphocytic Leukemia	Ibrutinib, Venetoclax	Summit Medical Group, Florham Park, New Jersey 07932 Hematology Oncology Associates of Rockland, Nyack, New York 10960 Hunterdon Hematology Oncology, Flemington, New Jersey 08822

https://clinicaltrials.gov/study/NCT04904588	Recruiting	A Multi-Center, Phase II Trial of HLA-Mismatched Unrelated Donor Hematopoietic Cell Transplantation With Post-Transplantation Cyclophosphamide for Patients With Hematologic Malignancies	Chronic Lymphocytic Leukemia	Busulfan, Busulfan, Fludarabine, Total-body irradiation, Cyclophosphamide, Melphalan, PBSC Hematopoietic Stem Cell Transplantation (HSCT), Bone Marrow Hematopoietic Stem Cell Transplantation, Post-transplant Cyclophosphamide, Mesna, Tacrolimus, Mycophenolate Mofetil, Patient-Reported Outcomes	Columbia University Medical Center, New York, New York 10032 Memorial Sloan Kettering Cancer Center, New York, New York 10065 University of Pennsylvania, Philadelphia, Pennsylvania 19104
https://clinicaltrials.gov/study/NCT04728893	Recruiting	A Phase 2 Study to Evaluate the Efficacy and Safety of MK-1026 in Participants With Hematologic Malignancies	Chronic Lymphocytic Leukemia	Nemtabrutinib	John Theurer Cancer Center at Hackensack University Medical Center (Site 2704), Hackensack, New Jersey 07601 Astera Cancer Care (Site 2732), East Brunswick, New Jersey 08816 The Ottawa Hospital (Site 0404), Ottawa, Ontario K1H 8L6
https://clinicaltrials.gov/study/NCT05602363	Recruiting	A Phase 1b Study of Oral AS-1763 in Patients With Previously Treated Chronic Lymphocytic Leukemia/Small Lymphocytic Lymphoma or Non-Hodgkin Lymphoma	Chronic Lymphocytic Leukemia	Docirbrutinib	Clinical Research Alliance, Inc., Westbury, New York 11590 University of Maryland Medical Center - Greenebaum Comprehensive Cancer Center, Baltimore, Maryland 21201 University of Massachusetts Memorial Medical Center, Worcester, Massachusetts 01655

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
FLCN	0	NM_144997.5:c.1063-2A>G	0	0	splice_acceptor_variant	48.08	1383	0
SRSF2	NP_001182356.1:p.Pro95Arg	NM_001195427.1:c.284C>G	P/R	cCc/cGc	missense_variant	2.81	2025	tolerated
DNMT3A	NP_072046.2:p.Gln692GlufsTer20	NM_022552.4:c.2074_2075del	Q/X	CAg/g	frameshift_variant	0.63	2393	0
BRAF	NP_004324.2:p.Gly466Arg	NM_004333.4:c.1396G>A	G/R	Gga/Aga	missense_variant	0.59	2353	deleterious
DNMT3A	NP_072046.2:p.Arg736His	NM_022552.4:c.2207G>A	R/H	cGc/cAc	missense_variant	0.42	2381	tolerated

NOTCH1	NP_060087.3:p.Pro2514ArgfsTer4	NM_017617.3:c.7541_7542del	P/X	cCT/c	frameshift_variant	0.41	2932	0
PRKDC	NP_008835.5:p.Lys3681Asn	NM_006904.6:c.11043A>C	K/N	aaA/aaC	missense_variant	0.27	2926	deleterious

Methodology and Test Background

This is a next generation sequencing (NGS) test that analyzes cfDNA for abnormalities in 302 genes and cfRNA 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 cfRNA due to degradation. Performance of the assays may vary depending on the quantity and quality of nucleic acid, sample preparation and sample age. Testing is performed using massive parallel sequencing of the coding DNA of the listed genes. This includes sequencing of all the exons as well as approximately 50 nucleotides at the 5' and 3' ends of each coding exon to detect splice site abnormalities. The TERT promoter region, including the hotspots at -124 and -146 bp, is also covered. Our cfDNA sequencing method has a sensitivity of 0.1% for detecting hot spot mutations, 0.5% for detecting single nucleotide variants (SNVs) and 1% for small (<60 bp) insertions/ deletions (indels). Known hot spots in specific genes such as IDH1/2, NRAS, and KRAS are reported at levels of 0.01% and higher when both cfRNA and cfDNA results are combined. Significant gene amplification and deletion (copy number variants) are also reported. 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	XPO1
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	HNF1A	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. Chronic Lymphocytic Leukemia: 2025 Update on the Epidemiology, Pathogenesis, Diagnosis, and Therapy. Hallek M. Am J Hematol. 2025 Mar;100(3):450-480. doi: 10.1002/ajh.27546. Epub 2025 Jan 28. PMID: 39871707.

2. Frontline Therapy of CLL-Changing Treatment Paradigms. Coombs CC. Curr Hematol Malig Rep. 2024 Apr;19(2):65-74. doi: 10.1007/s11899-024-00726-x. Epub 2024 Feb 10. PMID: 38337108.

3. First line therapy of CLL. Hallek M. Hematol Oncol. 2023 Jun;41 Suppl 1:129-135. doi: 10.1002/hon.3145. PMID: 37294974.

4. Diagnosis and Treatment of Chronic Lymphocytic Leukemia: A Review. Shadman M. JAMA. 2023 Mar 21;329(11):918-932. doi: 10.1001/jama.2023.1946. PMID: 36943212.

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Electronic Signature

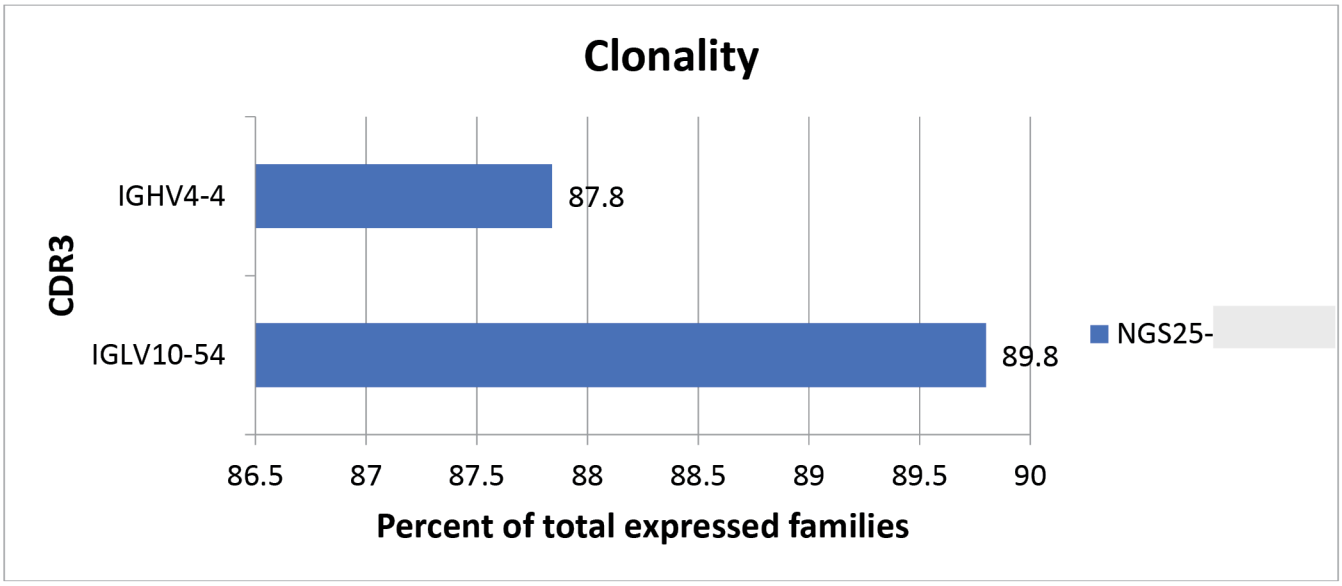
Maher Albitar, M.D.

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

B-cell/T-cell Clonality



Additional Report Information

Mutations Load (mol/mL)