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CLIA #: 05D2111917 CAP #: 9441574

Liquid Trace Hematology

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

MRN:				Family History:	
Collected Date:		Time:		Indication for Testing:	Myelodysplastic syndrome, unspecified (D46.9), Diffuse large B-cell lymphoma (C83.3)
Received Date:		Time:			
Reported Date:	06/20/2025	Time:	02:52 PM	Tumor Type:	MDS, Lymphoma

Detected Genomic Alterations				
ATM	BCORL1	DNMT3A	FGFR4	ARID2
PPM1D	Low-level t(9;9)(q34;q34) ABL1::NUP214 fusion mRNA	CAR-T construct : CD247::CD28 fusion mRNA	Autosomal chromosomes show: 1p-, 3q+, 6q-, 7p+, 9p-(CDKN2A/B deletion), 11q-, 12p+, 13q-, and others.	T-cell clonality : Not detected
B-cell clonality : Detected (IgHV 4-4/ two IgKV clones 3-20 and 1-5).				

Results Summary

- **-Low-level somatic mutations in ATM, BCORL1, DNMT3A, FGFR4, ARID2, and PPM1D genes**
  -t(9;9)(q34;q34) ABL1::NUP214 fusion mRNA
  -CAR-T construct : CD247::CD28 fusion mRNA
  -EBV viral RNA: Not detected
  -HPV viral RNA: Not detected
  -TTV viral RNA: Not detected
  -HLA Genotyping:
  -HLA-A: A*01:01-A*02:01
  -HLA-B: B*50:01-B*51:01
  -HLA-C: C*06:02-C*02:02
  -Autosomal chromosomes show: 1p-, 3q+, 6q-, 7p+, 9p-(CDKN2A/B deletion), 11q-, 12p+, 13q-, and others.
  -T-cell clonality : Not detected.
  -B-cell clonality : Detected (IgHV 4-4/ two IgKV clones 3-20 and 1-5).
  -No increase inCD34 or TdT mRNA
  -Increased B-cell markers

  -These findings are consistent with high level aggressive B-cell lymphoma with residual CAR-T cells.

  -The ABL1::NUP214 is detected at low level. This fusion is typically seen in acute lymphoblastic

leukemia (ALL), but there is no increase in blast markers.

-The mutations in DNMT3A, PPM1D and BCORL1 are likely in myeloid cells, but detected at low level in the analyzed sample.

See additional report information at the end of the report.

Heterogeneity
There are abnormal low-level clones with BCORL1, DNMT3A, FGFR4, ARID2, and PPM1D mutations. The ATM mutation is detected at a high level.

Expression	
Increased B-cell markers	No increase inCD34 or TdT mRNA

Diagnostic Implications	
ATM, BCORL1, DNMT3A, FGFR4, ARID2, PPM1D	-These findings are consistent with aggressive B-cell lymphoma.

Therapeutic Implications	
DNMT3A	DNA methyltransferase inhibitors
ARID2	sensitivity to radiation therapy and PARP inhibitors
PPM1D	PPM1D inhibitors

Prognostic Implications	
BCORL1	Unknown
DNMT3A	Poor
FGFR4	Unknown
ARID2	Unknown
PPM1D	Poor

Relevant Genes with NO Alteration
No evidence of mutation in NOTCH, 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

- ATM. The protein encoded by this gene belongs to the PI3/PI4-kinase family. This protein is an important cell cycle checkpoint kinase that phosphorylates; thus, it functions as a regulator of a wide variety of downstream proteins, including tumor suppressor proteins p53 and BRCA1, checkpoint kinase CHK2, checkpoint proteins RAD17 and RAD9, and DNA repair protein NBS1. This protein and the closely related kinase ATR are thought to be master controllers of cell cycle checkpoint signaling pathways that are required for cell response to DNA damage and for genome stability. Mutations in this gene are associated with ataxia telangiectasia, an autosomal recessive disorder. [provided by RefSeq, Aug 2010]

- BCORL1. The protein encoded by this gene is a transcriptional corepressor that is found tethered to promoter regions by DNA-binding proteins. The encoded protein can interact with several different class II histone deacetylases to repress transcription. Two transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, May 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]

- FGFR4. The protein encoded by this gene is a tyrosine kinase and cell surface receptor for fibroblast growth factors. The encoded protein is involved in the regulation of several pathways, including cell proliferation, cell differentiation, cell migration, lipid metabolism, bile acid biosynthesis, vitamin D metabolism, glucose uptake, and phosphate homeostasis. This protein consists of an extracellular region, composed of three immunoglobulin-like domains, a single hydrophobic membrane-spanning segment, and a cytoplasmic tyrosine kinase domain. The extracellular portion interacts with fibroblast growth factors, setting in motion a cascade of downstream signals, ultimately influencing mitogenesis and differentiation. [provided by RefSeq, Aug 2017]

- ARID2. This gene encodes a member of the AT-rich interactive domain (ARID)-containing family of DNA-binding proteins. Members of the ARID family have roles in embryonic patterning, cell lineage gene regulation, cell cycle control, transcriptional regulation and chromatin structure modification. This protein functions as a subunit of the polybromo- and BRG1-associated factor or PBAF (SWI/SNF-B) chromatin remodeling complex which facilitates ligand-dependent transcriptional activation by nuclear receptors. Mutations in this gene are associated with hepatocellular carcinomas. A pseudogene of this gene is found on chromosome1. [provided by RefSeq, Dec 2016]

- PPM1D. The protein encoded by this gene is a member of the PP2C family of Ser/Thr protein phosphatases. PP2C family members are known to be negative regulators of cell stress response pathways. The expression of this gene is induced in a p53-dependent manner in response to various environmental stresses. While being induced by tumor suppressor protein TP53/p53, this phosphatase negatively regulates the activity of p38 MAP kinase, MAPK/p38, through which it reduces the phosphorylation of p53, and in turn suppresses p53-mediated transcription and apoptosis. This phosphatase thus mediates a feedback regulation of p38-p53 signaling that contributes to growth inhibition and the suppression of stress induced apoptosis. This gene is located in a chromosomal region known to be amplified in breast cancer. The amplification of this gene has been detected in both breast cancer cell line and primary breast tumors, which suggests a role of this gene in cancer development. [provided by RefSeq, Jul 2008]

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

Potential Clinical Trials

Trial URL	Status	Title	Disease	Drug	Sites
https://clinicaltrials.gov/study/NCT05828589	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	Sidney Kimmel Comprehensive Cancer At Johns Hopkins, Baltimore, Maryland 21287 Thomas Jefferson University, Philadelphia, Pennsylvania 19107-4216 University of Iowa Hospitals and Clinics, Iowa City, Iowa 52242-1009
https://clinicaltrials.gov/study/NCT05934838	Recruiting	A Feasibility Trial of Tazemetostat Plus CAR T Cell Therapy in B-cell Lymphomas	B-cell Lymphoma	Tazemetostat Pill	Weill Cornell Medicine/NewYork-Presbyterian Hospital, New York, New York 10065

https://clinicaltrials.gov/study/NCT05512390	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 Health Network_Princess Margaret Cancer Centre /ID# 243936, Toronto, Ontario M5G 2M9 Novant Health Presbyterian Medical Center /ID# 246719, Charlotte, North Carolina 28204 Memorial Sloan Kettering Cancer Center-Koch Center /ID# 249246, New York, New York 10065-6007
https://clinicaltrials.gov/study/NCT05006716	Recruiting	A Phase 1/2, Open-Label, Dose-Escalation and -Expansion Study of the Bruton Tyrosine Kinase Targeted Protein Degrader BGB-16673 in Patients With B-Cell Malignancies	B-cell Lymphoma	BGB-16673	Karmanos Cancer Institute, Detroit, Michigan 48201-2013 Norton Cancer Institute Pavilion, Louisville, Kentucky 40207-4700 Roswell Park Comprehensive Cancer Center, Buffalo, New York 14203

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
ATM	NP_000042.3:p.Lys1192Ter	NM_000051.3:c.3574A>T	K/*	Aag/Tag	"stop_gained,splice_region_variant"	73.4	282	0
BCORL1	NP_068765.3:p.Ala839Val	NM_021946.4:c.2516C>T	A/V	gCg/gTg	missense_variant	3.61	415	0
DNMT3A	NP_783328.1:p.Arg882His	NM_175629.2:c.2645G>A	R/H	cGc/cAc	missense_variant	2.33	727	deleterious (0)
FGFR4	NP_002002.3:p.Glu39Gln	NM_002011.3:c.115G>C	E/Q	Gag/Cag	missense_variant	2.29	874	tolerated (0.25)
ARID2	NP_689854.2:p.Lys275Arg	NM_152641.2:c.824A>G	K/R	aAg/aGg	missense_variant	1.66	901	tolerated (0.19)
PPM1D	NP_003611.1:p.Cys414SerfsTer15	NM_003620.3:c.1239_1252delATGTTCTACACCAC	PCSTP/X	cCATGTTCTACACCA/c	frameshift_variant	0.24	841	0

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. 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. B-cell lymphoma: Advances in pathogenesis, diagnosis, and targeted therapies. Patil S, Rajput S, Patil S, Mhaiskar A. Pathol Res Pract. 2025 Jul;271:156036. doi: 10.1016/j.prp.2025.156036. Epub 2025 May 26. PMID: 40435909.

2. Evolving molecular classification of aggressive B-cell lymphoma. Alig SK, Chapuy B, Ennishi D, Dunleavy K, Hodson DJ. Histopathology. 2025 Jan;86(1):94-105. doi: 10.1111/his.15350. Epub 2024 Nov 15. PMID: 39545339.

3. 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.

4. 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.

Electronic Signature

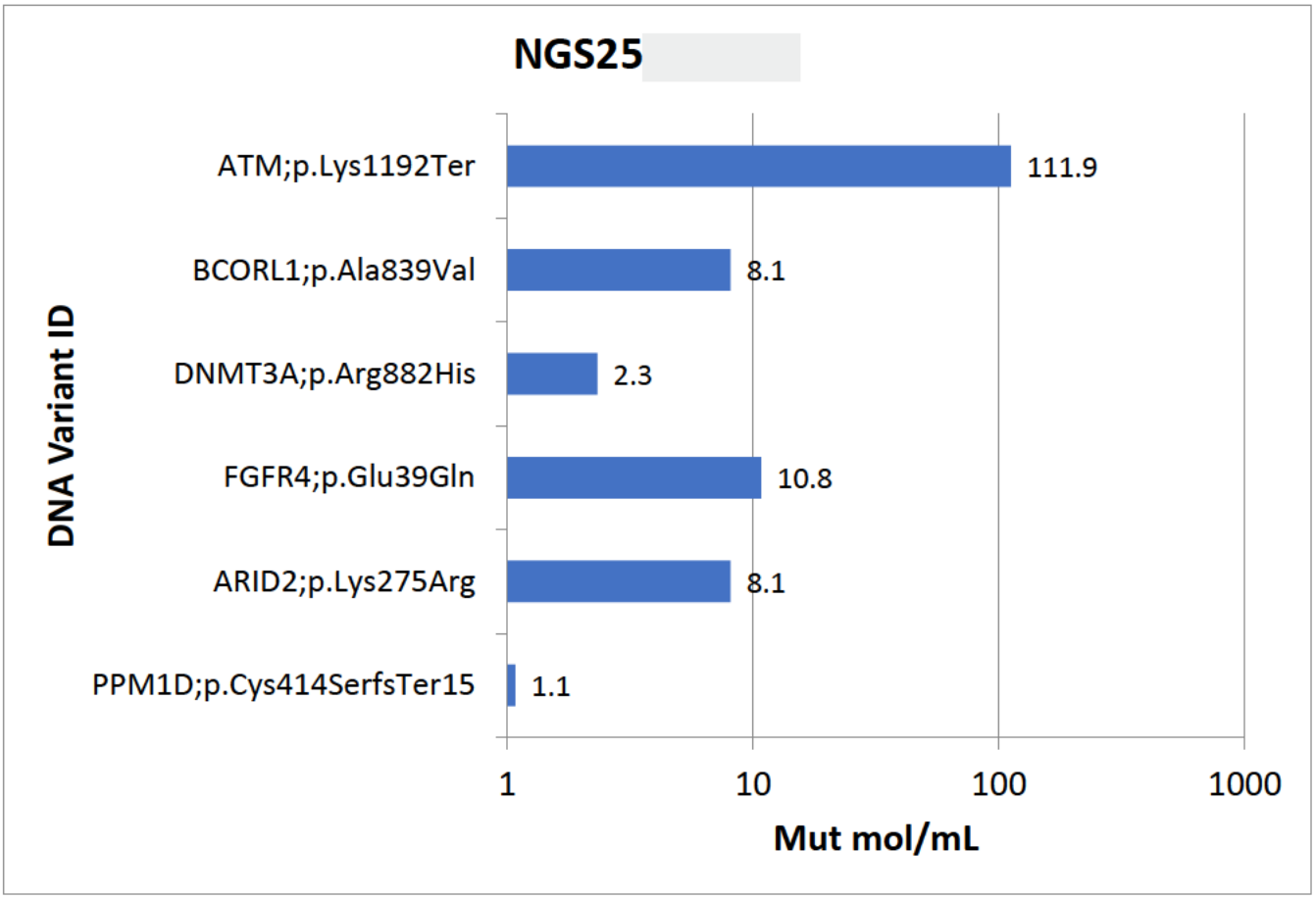
Maher Albitar, M.D.

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)



Additional Report Information

Chromosomal Abnormality Graph