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

Hematology Profile Plus

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

MRN:				Indication for Testing:	D47.Z9 Other specified neoplasms of uncertain behavior of lymphoid, hematopoietic and related tissue
Collected Date:		Time:	12:00 AM		
Received Date:		Time:	10:37 AM		
Reported Date:		Time:	05:21 PM	Tumor Type:	lymphoma

Detected Genomic Alterations

MPL (?Germline)	NOTCH2	PLCG1	SOX2	NPM1
DOT1L	LMO7 (?Germline)	Autosomal chromosomal structural analysis shows: +7, distal 8q+, 11q+, 17p- (TP53 deletion), 17q+, 19p-	T-cell clonality: Detected [TRAV1-1 (79%), TRBV7-9 (93%), TRGV5 (78%)]	B-cell clonality: Not detected

Results Summary

- **-Low-level somatic mutations in NOTCH2, PLCG1, SOX2, NPM1, and DOT1L genes**
  -Possible germline mutations in MPL and LMO7 genes, heterozygous
  -Autosomal chromosomal structural analysis shows: +7, distal 8q+, 11q+, 17p- (TP53 deletion), 17q+, 19p-
  -T-cell clonality: Detected [TRAV1-1 (79%), TRBV7-9 (93%), TRGV5 (78%)]
  -B-cell clonality: Not detected
  -T cell markers: Increased with abnormal pattern
  -Increased KI67, CD4, CD30, GATA3 mRNA
  -No significant increase in TBX21 mRNA
  -EBV, HPV, TTV, and HTLV1 viral mRNA: Not detected
  -HLA Genotyping:
  -HLA-A: A*01:01-A*02:01
  -HLA-B: B*07:02-B*15:01
  -HLA-C: C*07:02-C*01:02
  -T-cell markers RNA ratios:
  CD3D:CD3E Ratio : 2.40
  CD4:CD8A Ratio : 9.73
  CD4:CD8B Ratio : 11.96
  CD4:CD26 Ratio : 1.14
  CD4:CD7 Ratio : 5.57

-The findings are consistent with advanced T-cell lymphoma with monoallelic TP53 deletion.

-The MPL and LMO7 mutations are detected at high level, raising the possibility of germline mutations. These mutations lead to early termination (loss of function). However, there is no data on their clinical relevance and should be classified as of "uncertain significance" at this time.

-See additional report information at the end of the report

Heterogeneity

There are abnormal low-level clones with NOTCH2, PLCG1, SOX2, NPM1, and DOT1L mutations.
The MPL and LMO7 mutations are detected at high levels, possible germline abnormalities.

Expression

T cell markers: Increased with abnormal pattern	Increased KI67, CD4, CD30, GATA3 mRNA
No significant increase in TBX21 mRNA	

Diagnostic Implications

MPL, NOTCH2, PLCG1, SOX2, NPM1, DOT1L, LMO7	The findings are consistent with T-cell lymphoma The MPL and LMO7 mutations are likely germline variants.

Therapeutic Implications

NOTCH2	NOTCH inhibitors
DOT1L	DOT1L inhibitors

Prognostic Implications

NOTCH2	Poor
PLCG1	Unknown
SOX2	Unknown
NPM1	Favorable
DOT1L	Unknown

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, IgVH mutation analysis, and viruses (HPV, EBV, HTLV1, and TTV), in DNA of 302 genes and RNA 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

- MPL. In 1990 an oncogene, v-mpl, was identified from the murine myeloproliferative leukemia virus that was capable of immortalizing bone marrow hematopoietic cells from different lineages. In 1992 the human homologue, named, c-mpl, was cloned. Sequence data revealed that c-mpl encoded a protein that was homologous with members of the hematopoietic receptor superfamily. Presence of anti-sense oligodeoxynucleotides of c-mpl inhibited megakaryocyte colony formation. The ligand for c-mpl, thrombopoietin, was cloned in 1994. Thrombopoietin was shown to be the major regulator of megakaryocytopoiesis and platelet formation. The protein encoded by the c-mpl gene, CD110, is a 635 amino acid transmembrane domain, with two extracellular cytokine receptor domains and two intracellular cytokine receptor box motifs . TPO-R deficient mice were severely thrombocytopenic, emphasizing the important role of CD110 and thrombopoietin in megakaryocyte and platelet formation. Upon binding of thrombopoietin CD110 is dimerized and the JAK family of non-receptor tyrosine kinases, as well as the STAT family, the MAPK family, the adaptor protein Shc and the receptors themselves become tyrosine phosphorylated. [provided by RefSeq, Jul 2008]

- NOTCH2. This gene encodes a member of the Notch family. Members of this Type 1 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 family members play a role in a variety of developmental processes by controlling cell fate decisions. The Notch signaling network is an evolutionarily conserved intercellular signaling pathway which regulates interactions between physically adjacent cells. In Drosophilia, notch interaction with its cell-bound ligands (delta, serrate) establishes an intercellular signaling pathway that plays a key role in development. Homologues of the notch-ligands have also been identified in human, but precise interactions between these ligands and the human notch homologues remain to be determined. This protein is cleaved in the trans-Golgi network, and presented on the cell surface as a heterodimer. This protein functions as a receptor for membrane bound ligands, and may play a role in vascular, renal and hepatic development. Two transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, Jan 2011]

- PLCG1. The protein encoded by this gene catalyzes the formation of inositol 1,4,5-trisphosphate and diacylglycerol from phosphatidylinositol 4,5-bisphosphate. This reaction uses calcium as a cofactor and plays an important role in the intracellular transduction of receptor-mediated tyrosine kinase activators. For example, when activated by SRC, the encoded protein causes the Ras guanine nucleotide exchange factor RasGRP1 to translocate to the Golgi, where it activates Ras. Also, this protein has been shown to be a major substrate for heparin-binding growth factor 1 (acidic fibroblast growth factor)-activated tyrosine kinase. Two transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, Jul 2008]

- SOX2. This intronless gene encodes a member of the SRY-related HMG-box (SOX) family of transcription factors involved in the regulation of embryonic development and in the determination of cell fate. The product of this gene is required for stem-cell maintenance in the central nervous system, and also regulates gene expression in the stomach. Mutations in this gene have been associated with optic nerve hypoplasia and with syndromic microphthalmia, a severe form of structural eye malformation. This gene lies within an intron of another gene called SOX2 overlapping transcript (SOX2OT). [provided by RefSeq, Jul 2008]

- NPM1. The protein encoded by this gene is involved in several cellular processes, including centrosome duplication, protein chaperoning, and cell proliferation. The encoded phosphoprotein shuttles between the nucleolus, nucleus, and cytoplasm, chaperoning ribosomal proteins and core histones from the nucleus to the cytoplasm. This protein is also known to sequester the tumor suppressor ARF in the nucleolus, protecting it from degradation until it is needed. Mutations in this gene are associated with acute myeloid leukemia. Dozens of pseudogenes of this gene have been identified. [provided by RefSeq, Aug 2017]

- DOT1L. The protein encoded by this gene is a histone methyltransferase that methylates lysine-79 of histone H3. It is inactive against free core histones, but shows significant histone methyltransferase activity against nucleosomes. [provided by RefSeq, Aug 2011]

- LMO7. This gene encodes a protein containing a calponin homology (CH) domain, a PDZ domain, and a LIM domain, and may be involved in protein-protein interactions. Several alternatively spliced transcript variants encoding different isoforms have been found for this gene, however, the full-length nature of some variants is not known. [provided by RefSeq, Jan 2009]

Drug Information

Brontictuzumab

Brontictuzumab is a humanized monoclonal antibody directed against the Notch-1 receptor with potential antineoplastic activity. Upon administration, brontictuzumab binds to Notch-1 on the cell surface, thereby inhibiting Notch-mediated signaling and tumor cell proliferation. Notch 1, a type 1 transmembrane protein belonging to the Notch family, functions as a receptor for membrane bound ligands and has various roles during development; dysregulated Notch signaling is associated with increased cell growth and chemoresistance in cancers.

Potential Clinical Trials

Trial URL	Status	Title	Disease	Drug	Sites
https://clinicaltrials.gov/study/NCT05996185	Recruiting	Phase II Study of Mogamulizumab With DA-EPOCH or CHOEP in Patients With Aggressive T-cell Lymphoma	T-cell Lymphoma	Mogamulizumab, DA-EPOCH Protocol, CHOEP protocol	Icahn School of Medicine at Mount Sinai, New York, New York 10029
https://clinicaltrials.gov/study/NCT04068597	Recruiting	An Open-label Phase I/IIa Study to Evaluate the Safety and Efficacy of CCS1477 as Monotherapy and in Combination in Patients With Advanced Haematological Malignancies.	T-cell Lymphoma	CCS1477, Pomalidomide, Dexamethasone, Azacitidine, Venetoclax, Bortezomib, Ixazomib, Elranatamab, Teclistamab, Lenalidomide, Daratumumab	Penn Medicine - Abramson Cancer Center Perelman, Philadelphia, Pennsylvania 19104
https://clinicaltrials.gov/study/NCT04104776	Recruiting	A Phase 1/2 Study of DZR123 (CPI-0209) in Patients With Advanced Solid Tumors and Lymphomas	T-cell Lymphoma	Tulmimetostat, Enzalutamide	Montefiore Einstein Center for Cancer Care, The Bronx, New York 10467-2490

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
MPL	NP_005364.1:p.Glu259Ter	NM_005373.2:c.775G>T	E/*	Gaa/Taa	stop_gained	49.64	413	0
NOTCH2	NP_077719.2:p.Gly1336Val	NM_024408.3:c.4007G>T	G/V	gGa/gTa	"missense_variant,splice_region_variant"	19.57	281	deleterious
PLCG1	NP_002651.2:p.Ser345Phe	NM_002660.2:c.1034C>T	S/F	tCc/tTc	missense_variant	17.87	414	tolerated
SOX2	NP_003097.1:p.Ser52Phe	NM_003106.3:c.155_156delCCinsTT	S/F	tCC/Ttt	missense_variant	17.28	434	deleterious
NPM1	NP_002511.1:p.Arg142Pro	NM_002520.6:c.425G>C	R/P	cGg/cCg	missense_variant	17.24	464	deleterious

DOT1L	NP_115871.1:p.Asp64Tyr	NM_032482.2:c.190G>T	D/Y	Gac/Tac	missense_variant	3.92	153	deleterious
LMO7 (RNA)	NP_005349.3:p.Asp147TyrfsTer16	NM_005358.5:c.439_451del	DLQDL/X	GATCTACAGGATTta/ta	frameshift_variant	43.38	302	0

Methodology and Test Background

This is a next generation sequencing (NGS) test that involves separate analysis of DNA and RNA panels for abnormalities that are reported in various types of hematologic neoplasms. The DNA panel is composed of 302 genes and the RNA panel is composed of >1600 genes. The DNA and RNA components of this assay were developed, validated, and set up as separate workflows, with independent extraction, library preparation, sequencing, and analysis pipelines. The NGS assay also detects several viruses that are important in oncology, including EBV, HPV, HTLV 1, 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 fresh cells, peripheral blood cells, bone marrow, body fluid, or paraffin-embedded tissue. For optimal results, neoplastic cells should be >30% of the analyzed cells. For fresh bone marrow specimens with the clinical indication of myeloma, enrichment for CD138-positive cells may be performed using immunomagnetic positive selection and both the CD138-positive and CD138-negative cell fractions extracted for NGS testing and the findings integrated within the final report. 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 DNA sequencing method has a sensitivity of 1% for detecting single nucleotide variants (SNVs) and small (<60 bp) insertions/ deletions (indels). Significant gene amplification and deletion (copy number variants) are also reported. In addition, fragment length analysis is performed for CALR, FLT3, and NPM1 to enhance the detection of large indels and has a sensitivity of 2%-5% for detecting CALR, FLT3-ITD, and NPM1 indels in wildtype background. For cases with indication of acute myeloid leukemia, preliminary FLT3-ITD results based on fragment analysis will be 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), human T-lymphotropic virus type-1 (HTLV1), and torque teno virus (TTV) viral RNA are also analyzed and reported. In cases of suspected chronic lymphocytic leukemia (CLL), IgVH mutation rate will also be reported. The sensitivity of this assay for detecting fusion mRNA is between 5% and 10%. This test specifically detects 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 proper normal expression control. Since the clinical relevance of the RNA expression level of most of the genes is not well-characterized at this time, only a small subset of the genes may be described based on the suspected disease, including but not limited to MYC, BCL2, CD274, CD19, CD22, CD34, and CD138. CRLF2 mRNA levels are reported in acute lymphoblastic leukemia. CD274 (PD-L1) mRNA levels are reported when they are significantly elevated. Performance of the assay may vary dependent on the quantity and quality of nucleic acid, sample preparation, and sample age. Decalcified specimens have not been validated. Decalcification with strong acids is not recommended and may lead to poor nucleic acid quality and suboptimal results.

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/gtc-hematology-profile-plus/ *(click the DNA tab)*

The table below contains a partial list of the tested RNA genes (Fusions/Expression). For a complete list, please go to: https://genomictestingcooperative.com/genomic-tests/gtc-hematology-profile-plus/ *(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. Advances in Novel Systemic Therapies for the Management of Cutaneous T Cell Lymphoma (CTCL). Case KB, Allen PB. Curr Hematol Malig Rep. 2025 Jan 13;20(1):5. doi: 10.1007/s11899-024-00746-7. PMID: 39800801.

2. New insights into the biology of T-cell lymphomas. Iqbal J, Inghirami G, Chan WC. Blood. 2024 Oct 31;144(18):1873-1886. doi: 10.1182/blood.2023021787. PMID: 39213420.

3. Aggressive T-cell lymphomas: 2024: Updates on diagnosis, risk stratification, and management. Ong SY, Zain JM. Am J Hematol. 2024 Mar;99 (3):439-456. doi: 10.1002/ajh.27165. PMID: 38304959.

4. T-cell lymphoma: the CAR-T revolution is coming. Grover NS, Beaven AW. Blood. 2024 Mar 28;143(13):1201-1202. doi: 10.1182/blood.2023023443. PMID: 38546636.

Electronic Signature

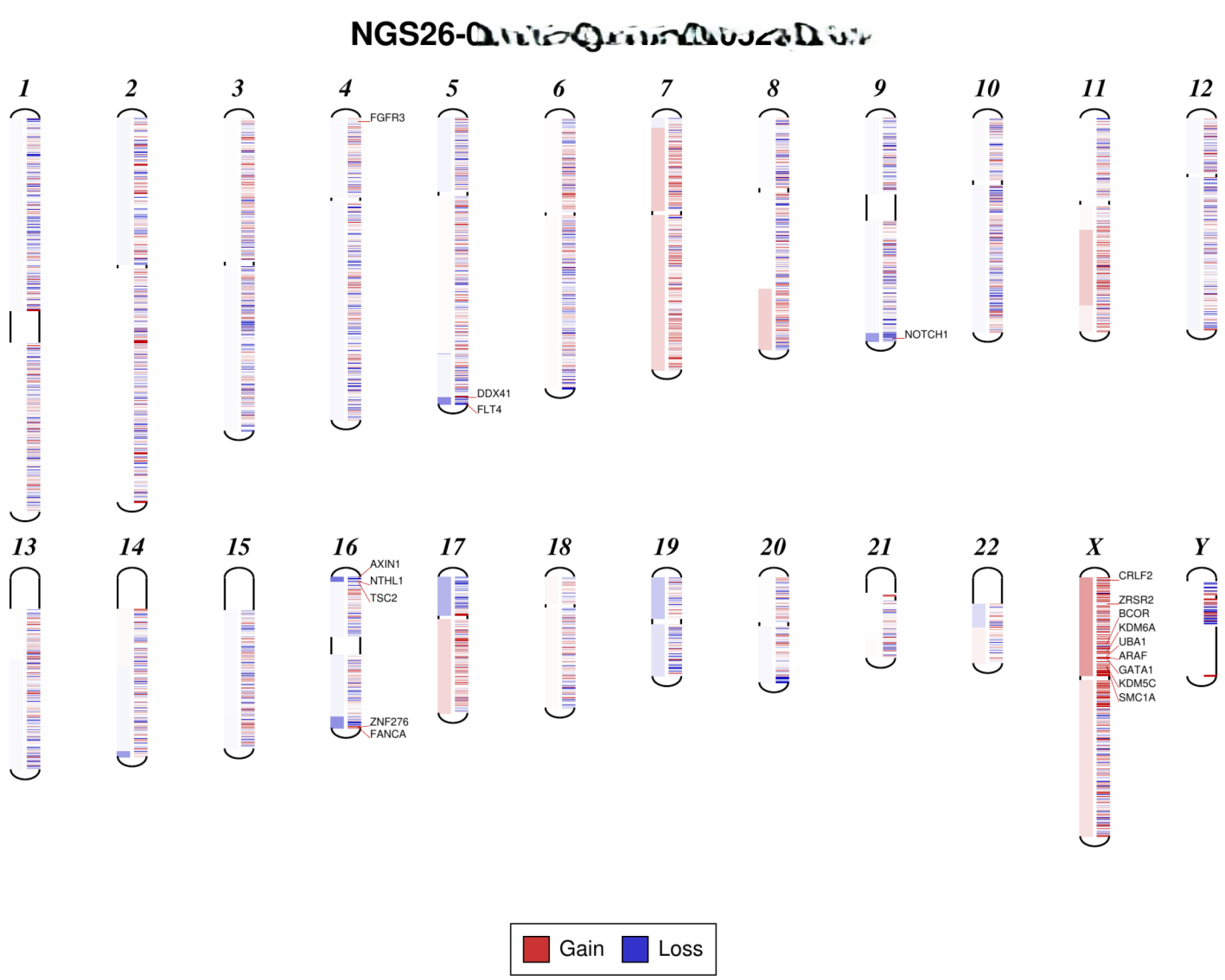
Maher Albitar, M.D.

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. (CLIA #: 05D2111917 CAP #: 9441574). The signing pathologist is fully responsible for the accuracy and interpretation of results and the release of this report.

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

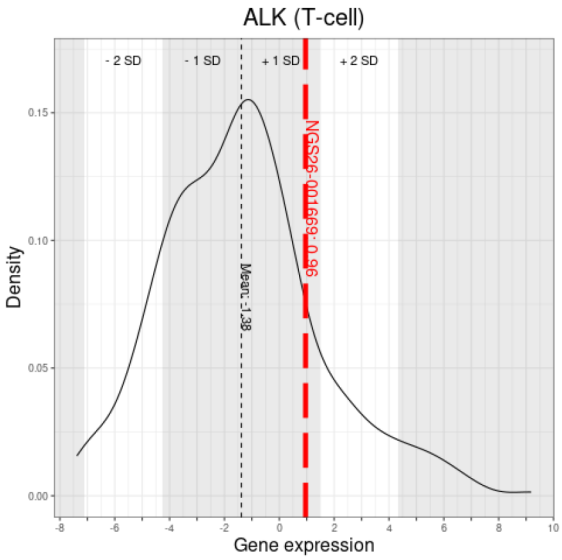
Chromosomal Abnormality Graph

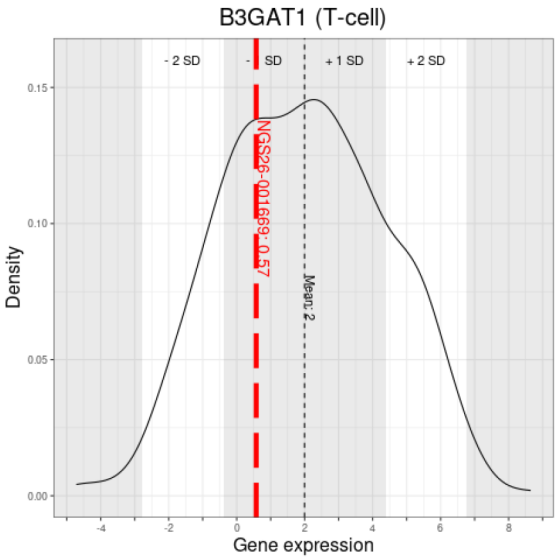


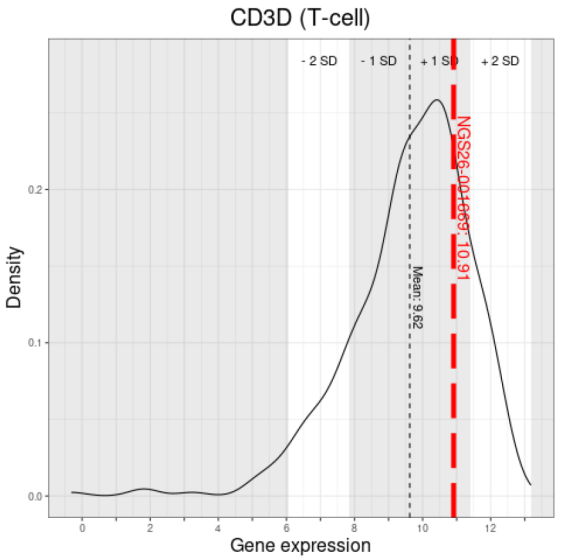
Additional Report Information

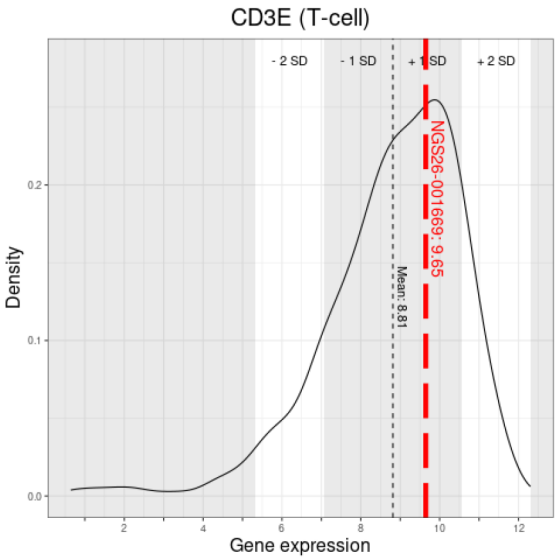
RNA Expression Plots

These plots represent the distribution of the expression in log2 transformed TPM (transcript per million) for each gene across GTC's history for the specified disease. The mean for each distribution is denoted by the black dotted line, while the alternating shaded areas depict the standard deviation. The expression for the current patient is marked by the red dotted line.





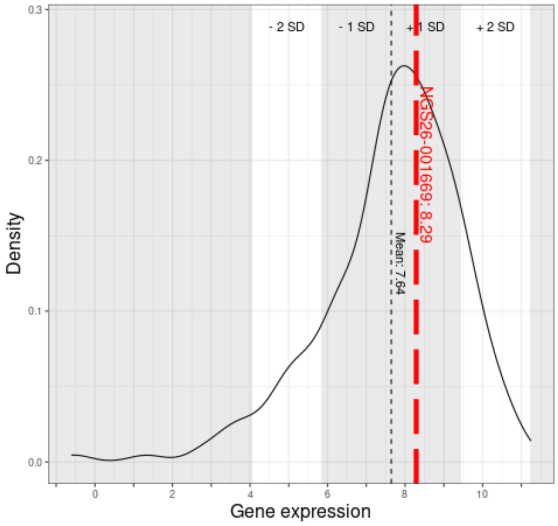




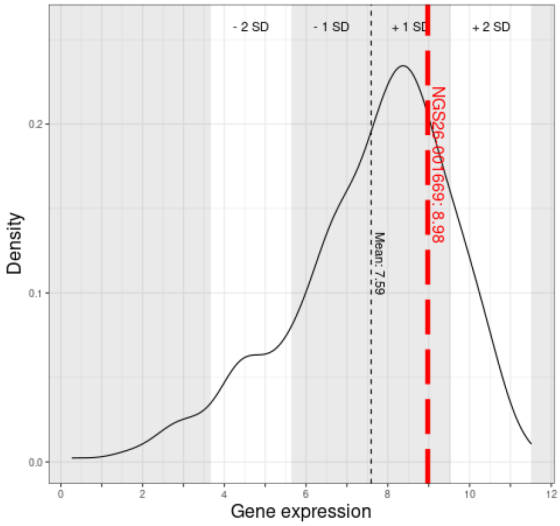
Additional Report Information

RNA Expression Plots

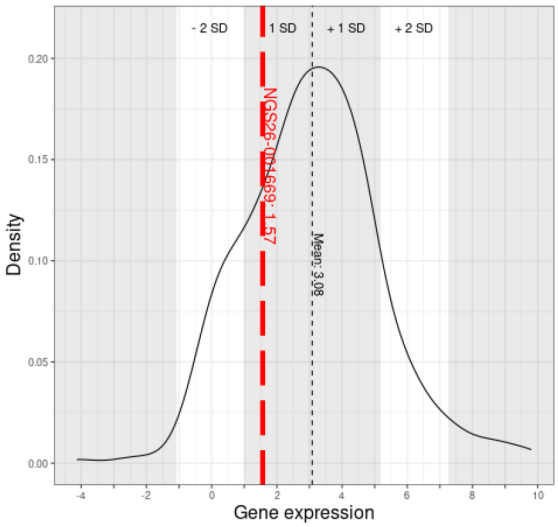
CD3G (T-cell)



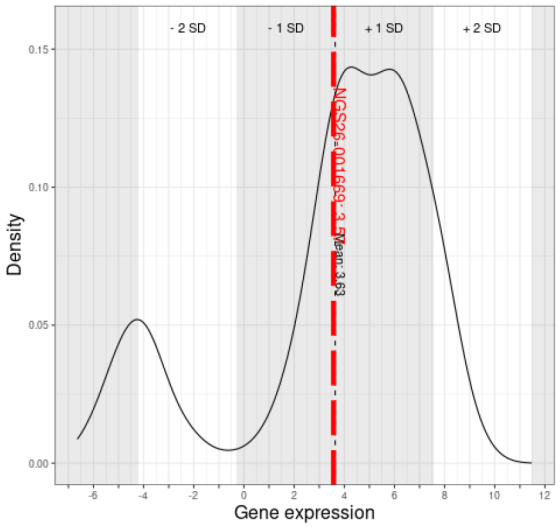
GATA3 (T-cell)



NCAM1 (T-cell)



TBX21 (T-cell)



TNFRSF8 (CD30) (T-cell)