GENOMIC TESTING COOPERATIVE

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

Solid Tumor Profile Plus

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

MRN:				Indication for Testing:	D32.9 Benign neoplasm of meninges unspecified
Collected Date:		Time:	09:17 AM		
Received Date:		Time:	09:49 AM		
Reported Date:		Time:	11:13 AM		

Detected Genomic Alterations

Level 1 (FDA-Approved)	Level 2 (Standard of Care)	Level 3 (Clinical Evidence)	Level 4 (Biological Evidence)	Other
-inv(12)(q13q13) NAB2::STAT6 fusion mRNA -No evidence of microsatellite instability -Detected TERTp mutation --No evidence of NF2, SMO, SUFU, PTCH1, BAP1, SMARCE1, AKT1, KLF4 and TP53 mutations -No evidence of CDKN2A/B gene deletion	-Tumor Mutation Burden Low: 4 Mut/Mb -Homologous recombination deficiency (HRD): Negative	PIK3CG	SLX4, PDE4DIP	Autosomal chromosomes show low level -7, 8p+ and +18.

Results Summary

- **-Mutations in TERT, SLX4, PIK3CG, and PDE4DIP genes**
  -inv(12)(q13q13) NAB2::STAT6 fusion mRNA
  -No evidence of microsatellite instability
  -Tumor Mutation Burden Low: 4 Mut/Mb
  -Homologous recombination deficiency (HRD): Negative
  -No evidence of fusion mRNA involving ALK, RET, ROS1, or NTRK
  -No evidence of NF2, TRAF7, AKT1, KLF4, SMO, PIK3CA, SMARCE1 or BAP1 mutations
  -No evidence of CDKN2A/B gene deletion
  -EBV viral RNA: Not detected
  -HPV viral RNA: Not detected

-TTV viral RNA: Not detected
-HLA Genotyping:
-HLA-A: A*29:02-A*68:02
-HLA-B: B*07:02-B*49:01
-HLA-C: C*07:01-C*07:02
-Autosomal chromosomes show low level -7, 8p+ and +18.
-Increased EGFR mRNA
-Marked increase in PDGFRA mRNA

-The NAB2::STAT6 fusion is consistent with solitary fibrous tumor (SFT)

-The presence of NAB2::STAT6 fusion suggests possible respoonse to anti-angiogenic agents (e.g., pazopanib, sunitinib, sorafenib), and Temozolomide + bevacizumab

-PIK3CG abnormality suggests response to PI3K inhibitors duvelisib (Copiktra).

See additional report information at the end of the report.

Tumor Heterogeneity
There are abnormal clones with TERT, SLX4, PIK3CG, and PDE4DIP mutations.

Expression	
Marked increase in PDGFRA mRNA	Increased EGFR mRNA

Diagnostic Implications	
TERT, SLX4, PIK3CG, PDE4DIP	These findings are consistent with aggressive neoplasm.

Levels 2, 3 & 4 (Standard of Care and Clinical/Biological Evidence)	
PIK3CG	PI3K, AKT, MTOR inhibitors

Relevant Genes with NO Alteration		
-No evidence of mutation in KRAS, NRAS, EGFR, BRAF, TP53, or BRCA 1/2 -No specific mutation in DPYD gene, associated with enzymatic deficiency	No evidence of fusion mRNA involving ALK, RET, ROS1, or NTRK	-No evidence of METex14 skipping or EGFRvIII -No evidence of ERBB2 (HER2) amplification

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, tumor mutational burden (TMB), microsatellite instability (MSI), homologous recombination deficiency (HRD), B- and T-cell clonality, and viruses (HPV, EBV, and TTV), in DNA of 434 genes and RNA in greater than 1600 genes implicated in solid tumors. Whenever possible, clinical relevance and implications of detected abnormalities are described below.

Biological relevance of detected Alterations

- TERT. Telomerase is a ribonucleoprotein polymerase that maintains telomere ends by addition of the telomere repeat TTAGGG. The enzyme consists of a protein component with reverse transcriptase activity, encoded by this gene, and an RNA component which serves as a template for the telomere repeat. Telomerase expression plays a role in cellular senescence, as it is normally repressed in postnatal somatic cells resulting in progressive shortening of telomeres. Deregulation of telomerase expression in somatic cells may be involved in oncogenesis. Studies in mouse suggest that telomerase also participates in chromosomal repair, since de novo synthesis of telomere repeats may occur at double-stranded breaks. Alternatively spliced variants encoding different isoforms of telomerase reverse transcriptase have been identified; the full-length sequence of some variants has not been determined. Alternative splicing at this locus is thought to be one mechanism of regulation of telomerase activity. [provided by RefSeq, Jul 2008] In addition, recurring somatic mutations at multiple spots in the proximal promoter (particularly at 124bp and 146bp upstream of the translation start site) are found in tumors of many tissue origins. These mutations are thought to affect binding of Ets family proteins and nuclear factor kappa B and alter secondary structure and long-range interactions, leading to increased promoter activity. [provided by RefSeq, May 2023]

- SLX4. This gene encodes a protein that functions as an assembly component of multiple structure-specific endonucleases. These endonuclease complexes are required for repair of specific types of DNA lesions and critical for cellular responses to replication fork failure. Mutations in this gene were found in patients with Fanconi anemia. [provided by RefSeq, Sep 2016]

- PIK3CG. Phosphoinositide 3-kinases (PI3Ks) phosphorylate inositol lipids and are involved in the immune response. The protein encoded by this gene is a class I catalytic subunit of PI3K. Like other class I catalytic subunits (p110-alpha p110-beta, and p110-delta), the encoded protein binds a p85 regulatory subunit to form PI3K. This gene is located in a commonly deleted segment of chromosome 7 previously identified in myeloid leukemias. Several transcript variants encoding the same protein have been found for this gene. [provided by RefSeq, Jun 2015]

- PDE4DIP. The protein encoded by this gene serves to anchor phosphodiesterase 4D to the Golgi/centrosome region of the cell. Defects in this gene may be a cause of myeloproliferative disorder (MBD) associated with eosinophilia. Several transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, Aug 2010]

Drug Information

Duvelisib

Duvelisib acts as a strong reversible inhibitor of the isoform gamma and delta of the phosphoinositide3-kinase (PI3K) [PI3K/AKT/mTOR].

Alpelisib

Alpelisib is an orally bioavailable phosphatidylinositol 3-kinase (PI3K) inhibitor with potential antineoplastic activity. Alpelisib specifically inhibits PIK3 in the PI3K/AKT kinase (or protein kinase B) signaling pathway, thereby inhibiting the activation of the PI3K signaling pathway. This may result in inhibition of tumor cell growth and survival in susceptible tumor cell populations. Activation of the PI3K signaling pathway is frequently associated with tumorigenesis. Dysregulated PI3K signaling may contribute to tumor resistance to a variety of antineoplastic agents.

Potential Clinical Trials

Trial URL	Status	Title	Disease	Drug	Sites

https://clinicaltrials.gov/study/NCT05425004	Recruiting	A Phase II Study of Cabozantinib for Patients With Recurrent or Progressive Meningioma	Meningioma	Cabozantinib	Memorial Sloan Kettering Cancer Center, New York, New York 100653 Vanderbilt University Medical Center, Nashville, Tennessee 37232 Miami Cancer Institute at Baptist Health, Inc., Miami, Florida 33176
https://clinicaltrials.gov/study/NCT06014905	Recruiting	Pilot/Phase I Study of Feasibility of Acquiring Hyperpolarized Imaging in Patients With Meningioma	Meningioma	Hyperpolarized carbon C 13 pyruvate, Magnetic Resonance Image (MRI), Saline	University of California, San Francisco, San Francisco, California 94143
https://clinicaltrials.gov/study/NCT04081701	Recruiting	68Ga(Gallium)-DOTATATE Positron Emission Tomography (PET)/MRI in the Diagnosis and Management of Somatostatin Receptor Positive Central Nervous System CNS Tumors.	Meningioma	Ga68-DOTATATE-PET/MRI	Weill Cornell Medicine, New York, New York 10065
https://clinicaltrials.gov/study/NCT06322342	Recruiting	A Multi-Center, Phase 2, Open Label, Ascending Dose Study to Evaluate the Safety and Efficacy of RVP-001 and to Identify an Appropriate Dose to Detect CNS Lesions in Adult Patients	Meningioma	RVP-001	Yale New Haven Hospital, New Haven, Connecticut 06510 Massachusetts General Hospital, Boston, Massachusetts 02114 Brigham & Women's Hospital, Boston, Massachusetts 02115

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
TERT	0	NM_198253.2:c.-146C>T	0	0	upstream_gene_variant	50.0	30	0
SLX4	NP_115820.2:p.Asn847Ser	NM_032444.2:c.2540A>G	N/S	aAt/aGt	missense_variant	38.59	298	deleterious
PIK3CG	NP_002640.2:p.Asp422Ala	NM_002649.2:c.1265A>C	D/A	gAc/gCc	missense_variant	30.88	285	deleterious
PDE4DIP (RNA)	NP_071754.3:p.Cys18Ter	NM_022359.5:c.54C>A	C/*	tgC/tgA	stop_gained	12.42	475	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 to be altered in various types of solid tumors. The DNA panel is composed of 434 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 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 paraffin-embedded tissue. For optimal results neoplastic cells should be greater than 30% of the analyzed cells. H&E-sections are reviewed by a pathologist and tumor-enrichment is performed by macrodissection when possible. 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 3% for detecting hotspot mutations and 5% for detecting single nucleotide variants (SNVs) and small (<60 bp) insertions/ deletions (indels). MSI status is inferred by interrogating all available genomic microsatellites covered. Borderline MSI results by NGS are confirmed by fragment analysis. Tumor mutational burden (TMB) is measured by counting all nonsynonymous variants and filter settings as follows: (A) Pass all filters; (B) inside genes; (C) had a mutant allele frequency >5%; (D) not found in the dbSNP (to exclude germline variations). The median for TMB is 10 mutations/Mb based on lung carcinoma analysis. The cut off for other types of tumors is not well-established at this time. 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, HLA class I genotyping, and Epstein-Barr virus (EBV), human papillomavirus (HPV) and torque teno virus (TTV) viral RNA are also analyzed and reported. B- and T-cell clonality will be reported, if clonal or clinically relevant. 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. CD274 (PD-L1) mRNA levels are reported when they are significantly elevated. This assay is not designed to detect minimal residual disease and should be used for diagnosis. Performance of the assays 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 <100X 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. ASXL1 NM_001164603 20:30946620- 30946635, ATM NM_000051 11:108186550-108186638, BAP1 NM_004656 3:52443858-52443894, BCR NM_004327 22:23652510-23652620, BRD4 NM_058243 19:15353808-15354193,5355041-15355411, CCNE1 NM_001238 19:30303463-30303485, CD274 NM_001267706 9:5456109-5456165, CD79A NM_001783 19:42384736-42384805, CSF3R NM_000760 1:36937667-36937740, DDX11 NM_001257144 12:31240872-31240917, ERBB3 NM_001982 12:56492284-56492359, FANCI NM_001113378 15:89835919-89836052, FLT3 NM_004119 13:28674605-28674652, FLT4 NM_002020 5:180035281-180035284, GEN1 NM_001130009 2:17954486-17954525, H3-3A NM_002107 1:226259140-226259180, IRS2 NM_003749 13:110437126-110437363, 110437805-110437899, 110438359- 110438400, JAK1 NM_002227 1:65309747-65309771, MAGI2 NM_012301 7:77648719-77649044, MITF NM_000248 3:70005606-70005681, MYCL NM_001033081 1:40367518-40367565, NF1 NM_000267 17:29664837-29664898, NOTCH2 NM_001200001 1:120572528-120572610, PBRM1 NM_018313 3:52677264-52677322, PIK3R2 NM_005027 19:18272089-18272305, PMS2 NM_000535 7:6013024-6013173, RANBP2 NM_006267 2:109363166-109363254, 109367779-109367838, 109367984-109368069, 109369453-109369497, 109378578-109378651, .RHEB NM_005614 7:151216546-151216597, SUFU NM_001178133 10:104263911-104264039, TNFRSF14 NM_003820 1:2494304- 2494335.

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/solid-tumor-profile-plus/*(click the DNA tab)*

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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/solid-tumor-profile-plus/ *(click the RNA tab)*

Tested genes

Genes Tested for Abnormalities in Coding Sequence																
ABCB7	ATRX	BTK	CDKN2B	DKC1	FANCA	FLI1	GREM1	INPP4B	LIG4	MSH2	NSD2 (WHSC1)	POLE	RAF1	SDHD	STAG2	TP53
ABL1	AURKA	CALR	CDKN2C	DNM2	FANCB	FLT1	GRIN2A	IRF2	LMO1	MSH6	NTRK1	POT1	RANBP2	SEC23B	STAT3	TRAF3
ABL2	AURKB	CARD11	CEBPA	DNMT3A	FANCC	FLT3	GRM3	IRF4	LPIN2	MTOR	NTRK2	PPM1D	RARA	SETBP1	STAT4	TSC1
ABRAXAS1	AURKC	CBFB	CHD2	DOT1L	FANCD2	FLT4	GSK3B	IRS2	LRP1B	MUTYH	NTRK3	PPP2R1A	RB1	SETD2	STAT6	TSC2
ACD	AXIN1	CBL	CHD4	EED	FANCE	FOXL2	GSKIP	JAGN1	LYN	MVK	NUP93	PRDM1	RBBP6	SF3B1	STK11	TSHR
ACVR1B	AXIN2	CBLB	CHEK1	EGFR	FANCF	FOXP1	H3-3A (H3F3A)	JAK1	LYST	MYC	PAK3	PREX2	RBM10	SLIT2	SUFU	U2AF1
ADA	AXL	CBLC	CHEK2	EGLN1	FANCG	FRS2	H3C2	JAK2	LZTR1	MYCL	PALB2	PRKAR1A	RBM8A	SLX4	SUZ12	U2AF2
ADGRA2	B2M	CCN6 (WISP3)	CIC	ELANE	FANCI	FUBP1	HAX1	JAK3	MAGI2	MYCN	PAX5	PRKCI	REEP5	SMAD2	SYK	VEGFA
AK2	BAP1	CCND1	CREBBP	EMSY	FANCL	G6PC3	HGF	JUN	MAP2K1	MYD88	PBRM1	PRKDC	RET	SMAD3	TAF1	VHL
AKT1	BARD1	CCND2	CRKL	EP300	FANCM	GABRA6	HNF1A	KAT6A	MAP2K2	NBN	PDCD1LG2	PRKN (PARK2)	RHEB	SMAD4	TAL1	WAS
AKT2	BCL2	CCND3	CRLF2	EPAS1	FAS	GALNT12	HOXA11	KDM5A	MAP2K4	NF1	PDGFRA	PRSS1	RHOA	SMAD9	TBX3	WT1
AKT3	BCL2L1	CCNE1	CSF1R	EPCAM	FAT1	GATA1	HOXB13	KDM5C	MAP3K1	NF2	PDGFRB	PRSS8	RICTOR	SMARCA4	TCF3	XPO1
ALK	BCL2L2	CD274	CSF3R	EPHA3	FBXW7	GATA2	HRAS	KDM6A	MAP3K14	NFE2L2	PDK1	PSTPIP1	RIT1	SMARCB1	TCIRG1	XRCC2
AMER1	BCL6	CD79A	CTC1	EPHA5	FGF10	GATA3	HSD3B1	KDR	MAPK1	NFKBIA	PHF6	PTCH1	RNF168	SMC1A	TENT5C (FAM46C)	XRCC3
ANKRD26	BCOR	CD79B	CTCF	EPHA7	FGF14	GATA4	HSP90AA1	KEAP1	MCL1	NHP2	PIK3C2B	PTEN	RNF43	SMC3	TERC	ZBTB2
APC	BCORL1	CDAN1	CTNNA1	EPHB1	FGF19	GATA6	ID3	KEL	MDM2	NKX2-1	PIK3CA	PTPN11	ROS1	SMO	TERF1	ZNF217
AR	BCR	CDC73	CTNNB1	ERBB2	FGF23	GEN1	IDH1	KIF23	MDM4	NLRP3	PIK3CB	QKI	RPTOR	SNCAIP	TERF2	ZNF703
ARAF	BIRC3	CDH1	CUL3	ERBB3	FGF3	GFI1	IDH2	KIT	MED12	NME1	PIK3CG	RAB27A	RTEL1	SOCS1	TERF2IP	ZRSR2
ARFRP1	BLM	CDIN1 (C15orf41)	CUX1	ERBB4	FGF4	GFI1B	IGF1R	KLF1	MEF2B	NOP10	PIK3R1	RAC1	RUNX1	SOX10	TERT	-
ARID1A	BMPR1A	CDK12	CXCR4	ERCC4	FGF6	GID4	IGF2	KLHL6	MEFV	NOTCH1	PIK3R2	RAD21	RUNX1T1	SOX2	TET2	-
ARID1B	BRAF	CDK4	CYLD	ERG	FGFR1	GLI1	IKBKE	KLLN	MEN1	NOTCH2	PIM1	RAD50	SAMD9L	SOX9	TGFBR2	-
ARID2	BRCA1	CDK6	DAXX	ERRFI1	FGFR2	GLI2	IKZF1	KMT2A	MET	NOTCH3	PLCG1	RAD51	SBDS	SPEN	TNFAIP3	-
ASXL1	BRCA2	CDK8	DDR2	ESR1	FGFR3	GNA11	IKZF3	KMT2B	MITF	NPM1	PLCG2	RAD51B	SBF2	SPOP	TNFRSF14	-
ATG2B	BRD4	CDKN1A	DDX11	ETV6	FGFR4	GNA13	IL2RG	KMT2C	MLH1	NR0B1	PMS1	RAD51C	SDHA	SPTA1	TNFRSF1A	-
ATM	BRIP1	CDKN1B	DDX41	EXO1	FH	GNAQ	IL7R	KMT2D	MPL	NRAS	PMS2	RAD51D	SDHB	SRC	TOP1	-
ATR	BTG1	CDKN2A	DICER1	EZH2	FLCN	GNAS	INHBA	KRAS	MRE11	NSD1	POLD1	RAD54L	SDHC	SRSF2	TOP2A	-

RNA Fusions/Expression

Fusion/Expression													
ABL1	BCL6	CD274 (PD-L1)	EGFR	EWSR1	FLI1	IKZF3	MAP3K1	NRG1	NUP98	PML	RET	SS18	THADA
AKT3	BRAF	CIC	ERG	FGFR1	FOXO1	JAK2	MECOM	NTRK1	PAX8	PPARG	RHOA	STAT6	TMPRSS2
ALK	CAMTA1	CREB1	ETS1	FGFR2	FUS	KIAA1549	MYB	NTRK2	PDGFRA	PRKACA	ROS1	TAL1	YAP1
AR	CBFB	CREBBP	ETV1	FGFR3	GLI1	KMT2A	MYC	NTRK3	PDGFRB	RAF1	RUNX1	TCF3	YWHAE
BCL2	CCND1	ERBB2	ETV6	FIP1L1	HMGA2	MAML2	NOTCH1	NUP214	PICALM	RARA	RUNX1T1	TFG	ZFTA

Reference

1. Current immunotherapy techniques in meningioma. White AJ, Harary M, Casaos J, Everson RG. Expert Rev Anticancer Ther. 2024 Oct;24 (10):931-941. doi: 10.1080/14737140.2024.2399252. Epub 2024 Sep 4. PMID: 39233324.

2. Meningioma: Molecular Updates from the 2021 World Health Organization Classification of CNS Tumors and Imaging Correlates. Soni N, Ora M, Bathla G, Szekeres D, Desai A, Pillai JJ, Agarwal A. AJNR Am J Neuroradiol. 2025 Feb 3;46(2):240-250. doi: 10.3174/ajnr.A8368. PMID: 38844366.

3. Evolving concepts in meningioma management in the era of genomics. Hsieh AL, Bi WL, Ramesh V, Brastianos PK, Plotkin SR. Cancer. 2024 Aug 1;130(15):2586-2600. doi: 10.1002/cncr.35279. Epub 2024 May 16. PMID: 38753473.

4. Radiotherapy for Meningioma. Susko MS, Raleigh DR. Adv Exp Med Biol. 2023;1416:95-106. doi: 10.1007/978-3-031-29750-2_8. PMID: 37432622.

Electronic Signature

Maher Albitar, M.D.

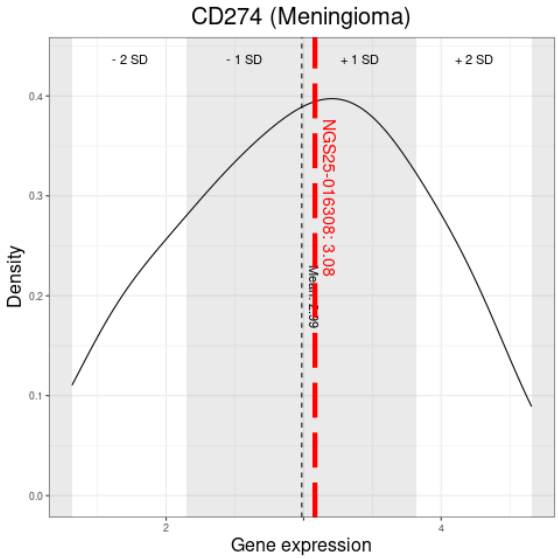
The test (sample processing, sequencing and data generation) was performed at Anthology Diagnostics-JFK Medical Center Lab, 80 James Street Edison, NJ 08820. Medical Director Clinton Ewing, M.D. Analysis of the data was performed by Genomic Testing Cooperative, LCA, 25371 Commercentre Drive, Lake Forest, 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.

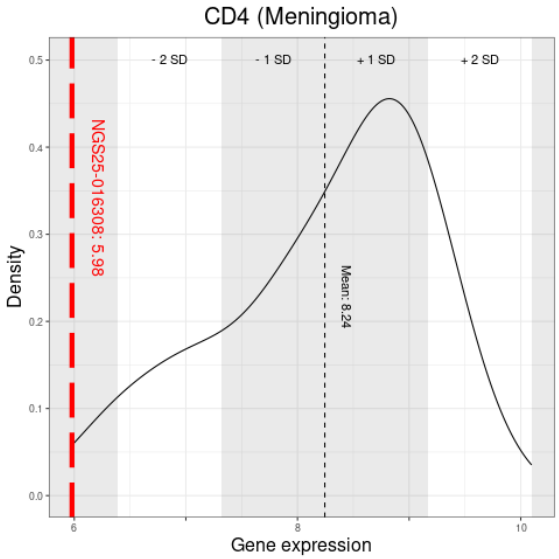
The test was developed and its performance characteristics have been determined by Anthology Diagnostics-JFK Medical Center Lab. This test has not been approved by the FDA. The FDA has determined such clearance or approval is not necessary. This laboratory is CLIA certified to perform high complexity clinical testing.

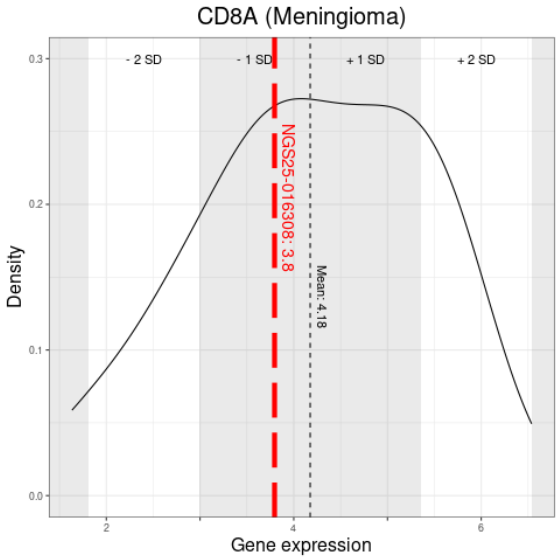
Additional Report Information

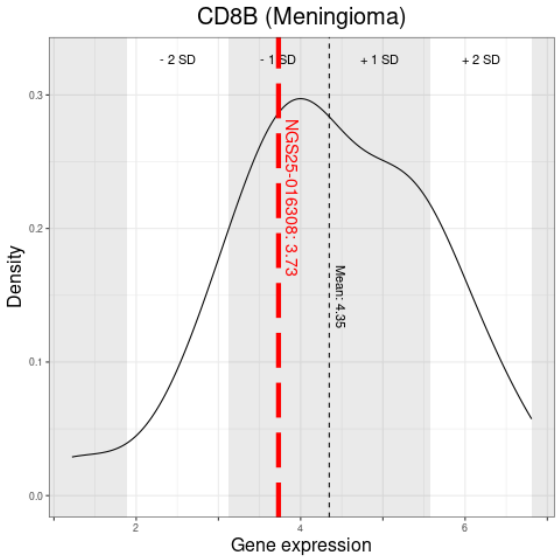
RNA Expression Plots

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