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CLIA #: 05D2111917 CAP #: 9441547

Solid Tumor Profile Plus

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

MRN:		Reason for Referral:	
Collected Date:			
Received Date:			
Reported Date:			

Detected Genomic Alterations

Level 1 (FDA-Approved)	Level 2 (Standard of Care)	Level 3 (Clinical Evidence)	Level 4 (Biological Evidence)	Other
-PIK3R1 mutation -Combined +7/-10 chromosomal changes -CDK4 and MDM2 amplification	-Tumor Mutation Burden Low: 5 Mut/Mb -No evidence of microsatellite instability -Homologous recombination deficiency (HRD): Negative -MGMT promoter methylation: Not detected	FANCA, IGF1R	FANCM, TERTp, DST, CNTRL	Chromosomal structural analysis shows +7, -10, -11, +12 (CDK4 and MDM2 amplification), 14q-, and 20p+

Results Summary

- **-Mutations in FANCM, PIK3R1, FANCA, TERTp, IGF1R, DST, and CNTRL genes**
  -Chromosomal structural analysis shows +7, -10, -11, +12 (CDK4 and MDM2 amplification), 14q-, and 20p+
  -Increased EGFR and VEGFA mRNA
  -No evidence of microsatellite instability
  -Tumor Mutation Burden Low: 5 Mut/Mb
  -Homologous recombination deficiency (HRD): Negative
  -No evidence of IDH1/2, H3 K27M, PTEN and TP53 mutations
  -No evidence of EGFR gene amplification
  -No evidence of CDKN2A/B gene deletion

-EBV viral RNA: Not detected
-HPV viral RNA : Not detected
-HLA Genotyping:
-HLA-A: A*02:844-A*02:05
-HLA-B: B*55:01-B*50:01
-HLA-C: C*06:02-C*01:02
-MGMT promoter methylation: Not detected
-MGMT mRNA expression: slightly above average.

-The findings are consistent with glioblastoma, IDH-wild type, CNS WHO grade 4.

-PIK3R1 mutation suggests possible response to mTOR inhibitors.

-Mutations in FANCA gene suggest possible response to PARP inhibitors.

-MDM2 amplification suggests targeting with nutlin-3a and its class of compounds.

-IGF1R mutation suggests possible response to IGF1R inhibitors (Mecasermin, Masoprocol, Linsitinib …).

Tumor Heterogeneity

There is an abnormal clone with FANCM, PIK3R1, FANCA, TERT, IGF1R, DST, and CNTRL mutations.

Expression

Increased EGFR and VEGFA mRNA

Diagnostic Implications

The findings are consistent with glioblastoma, IDH-wild type, CNS WHO grade 4.

Levels 2, 3 & 4 (Standard of Care and Clinical/Biological Evidence)

PIK3R1	PI3K, AKT, MTOR inhibitors
FANCA	DNA cross-linking agents such as diepoxybutane (DEB) and mitomycin C (MMC)
IGF1R	IFG1-R Inhibitor

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 MET14 deletion or EGFR Viii -No evidence of ERBB2 (HER2) amplification

Test Description:

This is a comprehensive molecular profile which uses next generation sequencing (NGS), fragment length analysis and Sanger Sequencing testing to identify molecular abnormalities (including SNVs, INDELS, CNVs, Fusions, TMB, MSI, HRD, EBV, and HPV) in DNA of 434 genes and RNA in 1600 genes implicated in solid tumors. Whenever possible, clinical relevance and implications of detected abnormalities are described below.

Biological relevance of detected Alterations

- FANCM. The Fanconi anemia complementation group (FANC) currently includes FANCA, FANCB, FANCC, FANCD1 (also called BRCA2), FANCD2 FANCE, FANCF, FANCG, FANCI, FANCJ (also called BRIP1), FANCL, FANCM and FANCN (also called PALB2). The previously defined group FANCH is the same as FANCA. Fanconi anemia is a genetically heterogeneous recessive disorder characterized by cytogenetic instability, hypersensitivity to DNA crosslinking agents, increased chromosomal breakage, and defective DNA repair. The members of the Fanconi anemia complementation group do not share sequence similarity; they are related by their assembly into a common nuclear protein complex. This gene encodes the protein for complementation group M. Alternative splicing results in multiple transcript variants. [provided by RefSeq, Apr 2015]

- PIK3R1. Phosphatidylinositol 3-kinase phosphorylates the inositol ring of phosphatidylinositol at the 3-prime position. The enzyme comprises a 110 kD catalytic subunit and a regulatory subunit of either 85, 55, or 50 kD. This gene encodes the 85 kD regulatory subunit. Phosphatidylinositol 3-kinase plays an important role in the metabolic actions of insulin, and a mutation in this gene has been associated with insulin resistance. Alternative splicing of this gene results in four transcript variants encoding different isoforms. [provided by RefSeq, Jun 2011]

- FANCA. The Fanconi anemia complementation group (FANC) currently includes FANCA, FANCB, FANCC, FANCD1 (also called BRCA2), FANCD2 FANCE, FANCF, FANCG, FANCI, FANCJ (also called BRIP1), FANCL, FANCM and FANCN (also called PALB2). The previously defined group FANCH is the same as FANCA. Fanconi anemia is a genetically heterogeneous recessive disorder characterized by cytogenetic instability, hypersensitivity to DNA crosslinking agents, increased chromosomal breakage, and defective DNA repair. The members of the Fanconi anemia complementation group do not share sequence similarity; they are related by their assembly into a common nuclear protein complex. This gene encodes the protein for complementation group A. Alternative splicing results in multiple transcript variants encoding different isoforms. Mutations in this gene are the most common cause of Fanconi anemia. [provided by RefSeq, Jul 2008]

- 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]

- IGF1R. This receptor binds insulin-like growth factor with a high affinity. It has tyrosine kinase activity. The insulin-like growth factor I receptor plays a critical role in transformation events. Cleavage of the precursor generates alpha and beta subunits. It is highly overexpressed in most malignant tissues where it functions as an anti-apoptotic agent by enhancing cell survival. Alternatively spliced transcript variants encoding distinct isoforms have been found for this gene. [provided by RefSeq, May 2014]

- DST. This gene encodes a member of the plakin protein family of adhesion junction plaque proteins. Multiple alternatively spliced transcript variants encoding distinct isoforms have been found for this gene, but the full-length nature of some variants has not been defined. It has been reported that some isoforms are expressed in neural and muscle tissue, anchoring neural intermediate filaments to the actin cytoskeleton, and some isoforms are expressed in epithelial tissue, anchoring keratin-containing intermediate filaments to hemidesmosomes. Consistent with the expression, mice defective for this gene show skin blistering and neurodegeneration. [provided by RefSeq, Mar 2010]

- CNTRL. This gene encodes a centrosomal protein required for the centrosome to function as a microtubule organizing center. The gene product is also associated with centrosome maturation. One version of stem cell myeloproliferative disorder is the result of a reciprocal translocation between chromosomes 8 and 9, with the breakpoint associated with fibroblast growth factor receptor 1 and centrosomal protein 1. [provided by RefSeq, Jul 2008]

Drug Information

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.

Linsitinib

Linsitinib is a small molecule being investigated for use/treatment in cancer/tumors (unspecified) and solid tumors. Linsitinib is an inhibitor of the insulin-like growth factor 1 receptor (IGF-1R) with potential antineoplastic activity. IGF-1R inhibitor OSI-906 selectively inhibits IGF-1R, which may result in the inhibition of tumor cell proliferation and the induction of tumor cell apoptosis.

IGF-1R stimulates proliferation, enables onogenic transformation, and suppresses apoptosis. Inhibitors of IGF-1R are expected to have broad utility in oncology since the over-expression of IGF-1R and/or its ligands or the down-regulation of ligand binding proteins occurs in numerous human malignancies including lung, colon, breast, prostate, brain and skin cancers. In addition, signaling through the IGF system has been implicated in protecting tumor cells from apoptosis induced by anti-cancer treatments such as cytotoxic agents and EGFR inhibitors.

Potential Clinical Trials

Trial URL	Status	Title	Disease	Drug	Sites
https://classic.clinicaltrials.gov/show/NCT04747145	Recruiting	Analyzing Pulsed Reduced Dose Radiotherapy in Upfront Glioblastoma	Glioblastoma	Radiation Concurrent Chemotherapy (Temozolomide) Adjuvant Chemotherapy (Temozolomide)	Medical College of Wisconsin, Milwaukee, Wisconsin, United States
https://classic.clinicaltrials.gov/show/NCT02685605	Recruiting	Intraoperative Radiotherapy in Newly Diagnosed Glioblastoma Multiforme	Glioblastoma	Standard surgery Intraoperative radiotherapy Radiochemotherapy Temozolomide	Barrow Neurological Institute (SJHMC), Phoenix, Arizona, United States Stritch School of Medicine Loyola University, Maywood, Illinois, United States Long Island Jewish Medical Center, North Shore University Hospital, Lake Success, New York, United States
https://classic.clinicaltrials.gov/show/NCT05183204	Recruiting	Paxalisib With a High Fat, Low Carb Diet and Metformin for Glioblastoma	Glioblastoma	Paxalisib Metformin Ketogenic Diet	Weill Cornell Medicine, New York, New York, United States
https://classic.clinicaltrials.gov/show/NCT04752813	Recruiting	A Study of BPM31510 With Vitamin K1 in Subjects With Newly Diagnosed Glioblastoma (GB)	Glioblastoma	BPM31510 Vitamin K1 Temozolomide (TMZ) Radiation	Cedars-Sinai Medical Center, Los Angeles, California, United States Stanford University Cancer Center, Palo Alto, California, United States Sarcoma Oncology Research Center, Santa Monica, California, United States

https://classic.clinicaltrials.gov/show/NCT05083754	Recruiting	Carmustine Wafer in Combination With Retifanlimab and Radiation With/Without Temozolomide in Subjects With Glioblastoma	Glioblastoma	Retifanlimab Temozolomide Radiation Therapy	Johns Hopkins Medical Institution, Baltimore, Maryland, United States

Detailed Results

Single Nucleotide Variant (SNV) and Insertions-Deletions (INDELS)

Gene name	Hgvsp	Hgvsc	Aminoacids	Codons	Consequence	Allele frequency	Read depth	Predicted effect on protein
FANCM	NP_065988.1:p.Leu557Val	NM_020937.2:c.1669C>G	L/V	Ctt/Gtt	missense_variant	51.17	213	deleterious (0)
PIK3R1	0	NM_181523.2:c.1746-2delA	0	0	splice_acceptor_variant	50.0	110	0
FANCA	NP_000126.2:p.Met989Leu	NM_000135.2:c.2965A>T	M/L	Atg/Ttg	missense_variant	45.55	180	tolerated (1)
TERT	0	NM_198253.2:c.-124C>T	0	0	missense_variant_promoter	41.17	136	0
IGF1R	NP_000866.1:p.Met1245Ile	NM_000875.3:c.3735G>C	M/I	atG/atC	missense_variant	36.78	261	deleterious (0.01)
DST (RNA)	NP_001138241.1:p.Arg84Ter	NM_001144769.2:c.250C>T	R/*	Cga/Tga	stop_gained	40.32	630	0
CNTRL (RNA)	NP_008949.4:p.Leu1401TyrfsTer3	NM_007018.4:c.4194_4195delTG	VE/VX	gtTGag/gtag	frameshift_variant	38.57	70	0

Methodology and Test Background

This is a next generation sequencing (NGS) test that analyzes DNA for abnormalities in 434 genes that are reported to be altered in various types of tumors. Nucleic acid is isolated from paraffin-embedded tissue. 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 50 nucleotides at the 5' and 3' ends of each coding exon. Our sequencing method has a typical sensitivity of 3% for detecting common specific mutations and 5% for other mutations. MSI status is inferred by interrogating all available genomic microsatellites covered. Tumor mutational burden (TMB) is measured by counting all non-synonymous 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 based on lung carcinoma analysis. The cut off for other types of tumors is not well established at this time. Performance of the assay may vary dependent on the quantity and quality of nucleic acid, sample preparation and sample age. The assay is designed to detect significant gene amplification and deletion in addition to various single nucleotide variations (SNV) and indels.

In addition to DNA analysis, targeted RNA NGS analysis is performed. This is a next generation sequencing (NGS) test that analyzes targeted RNA on 1,600 genes implicated in solid tumors. It is based on hybrid capture of targeted RNA. Duplicates are excluded for levels measurements. While the major focus of the analysis is the detection of fusion mRNA, mutations in the expressed RNA of the analyzed genes are also analyzed and reported. mRNA expression levels are evaluated, and only significant high expression of specific genes are relatively reported. CD274 (PD-L1) mRNA levels are reported when they are significantly elevated. All detect fusion transcripts are reported. 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. The sensitivity of this assay in detecting fusion mRNA is between 1% and 5%. This assay is not designed to detect minimal residual disease and should be used for diagnosis. For optimal results neoplastic cells should be >30% of the analyzed cells.

The Universal Human Reference (UHR) RNA is used as control.

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. This poor coverage is mainly due to high GC content with inherited problem in obtaining adequate coverage. 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 contains 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)*

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	AURKB	C15ORF41	CEBPA	DNMT3A	FANCC	FLT3	GRIN2A	IRF2	LMO1	MSH6	NTRK2	POT1	RARA	SF3B1	STAT6	TSHR
ABL1	AURKC	CALR	CHD2	DOT1L	FANCD2	FLT4	GRM3	IRF4	LPIN2	MTOR	NTRK3	PPM1D	RB1	SLIT2	STK11	U2AF1
ABL2	AXIN1	CARD11	CHD4	EED	FANCE	FOXL2	GSK3B	IRS2	LRP1B	MUTYH	NUP93	PPP2R1A	RBBP6	SLX4	SUFU	U2AF2
ACD	AXIN2	CBFB	CHEK1	EGFR	FANCF	FOXP1	GSKIP	JAGN1	LYN	MVK	PAK3	PRDM1	RBM10	SMAD2	SUZ12	VEGFA
ACVR1B	AXL	CBL	CHEK2	EGLN1	FANCG	FRS2	H3F3A	JAK1	LYST	MYC	PALB2	PREX2	RBM8A	SMAD3	SYK	VHL
ADA	B2M	CBLB	CIC	ELANE	FANCI	FUBP1	HAX1	JAK2	LZTR1	MYCL	PARK2	PRKAR1A	RET	SMAD4	TAF1	WAS
AK2	BAP1	CBLC	CREBBP	EP300	FANCL	G6PC3	HGF	JAK3	MAGI2	MYCN	PAX5	PRKCI	RHEB	SMAD9	TAL1	WHSC1
AKT1	BARD1	CCND1	CRKL	EPAS1	FANCM	GABRA6	HIST1H3B	JUN	MAP2K1	MYD88	PBRM1	PRKDC	RHOA	SMAD9L	TBX3	WISP3
AKT2	BCL2	CCND2	CRLF2	EPCAM	FAS	GALNT12	HNF1A	KAT6A	MAP2K2	NBN	PDCD1LG2	PRSS1	RICTOR	SMARCA4	TCF3	WT1
AKT3	BCL2L1	CCND3	CSF1R	EPHA3	FAT1	GATA1	HOXA11	KDM5A	MAP2K4	NF1	PDGFRA	PRSS8	RIT1	SMARCB1	TCIRG1	XPO1
ALK	BCL2L2	CCNE1	CSF3R	EPHA5	FBXW7	GATA2	HOXB13	KDM5C	MAP3K1	NF2	PDGFRB	PSTPIP1	RNF168	SMC1A	TERC	XRCC2
AMER1	BCL6	CD274	CTC1	EPHA7	FGF10	GATA3	HRAS	KDM6A	MAP3K14	NFE2L2	PDK1	PTCH1	RNF43	SMC3	TERF1	XRCC3
ANKRD26	BCOR	CD79A	CTCF	EPHB1	FGF14	GATA4	HSD3B1	KDR	MAPK1	NFKBIA	PHF6	PTEN	ROS1	SMO	TERF2	ZBTB2
APC	BCORL1	CD79B	CTNNA1	ERBB2	FGF19	GATA6	HSP90AA1	KEAP1	MCL1	NHP2	PIK3C2B	PTPN11	RPTOR	SNCAIP	TERF2IP	ZNF217
AR	BCR	CDAN1	CTNNB1	ERBB3	FGF23	GEN1	ID3	KEL	MDM2	NKX2-1	PIK3CA	QKI	RTEL1	SOCS1	TERT	ZNF703
ARAF	BIRC3	CDC73	CUL3	ERBB4	FGF3	GFI1	IDH1	KIF23	MDM4	NLRP3	PIK3CB	RAB27A	RUNX1	SOX10	TET2	ZRSR2
ARFRP1	BLM	CDH1	CUX1	ERCC4	FGF4	GFI1B	IDH2	KIT	MED12	NME1	PIK3CG	RAC1	RUNX1T1	SOX2	TGFBR2	-
ARID1A	BMPR1A	CDK12	CXCR4	ERG	FGF6	GID4	IGF1R	KLF1	MEF2B	NOP10	PIK3R1	RAD21	SBDS	SOX9	TNFAIP3	-
ARID1B	BRAF	CDK4	CYLD	ERRFI1	FGFR1	GLI1	IGF2	KLHL6	MEFV	NOTCH1	PIK3R2	RAD50	SBF2	SPEN	TNFRSF14	-
ARID2	BRCA1	CDK6	DAXX	ESR1	FGFR2	GLI2	IKBKE	KLLN	MEN1	NOTCH2	PIM1	RAD51	SDHA	SPOP	TNFRSF1A	-
ASXL1	BRCA2	CDK8	DDR2	ETV6	FGFR3	GNA11	IKZF1	KMT2A	MET	NOTCH3	PLCG1	RAD51B	SDHB	SPTA1	TOP1	-
ATG2B	BRD4	CDKN1A	DDX11	EXO1	FGFR4	GNA13	IKZF3	KMT2B	MITF	NPM1	PLCG2	RAD51C	SDHC	SRC	TOP2A	-
ATM	BRIP1	CDKN1B	DDX41	EZH2	FH	GNAQ	IL2RG	KMT2C	MLH1	NRAS	PMS1	RAD51D	SDHD	SRSF2	TP53	-
ATR	BTG1	CDKN2A	DICER1	FAM175A	FLCN	GNAS	IL7R	KMT2D	MPL	NROB1	PMS2	RAD54L	SEC23B	STAG2	TRAF3	-

ATRX	BTK	CDKN2B	DKC1	FAM46C	FLI1	GPR124	INHBA	KRAS	MRE11A	NSD1	POLD1	RAF1	SETBP1	STAT3	TSC1	-
AURKA	C11orf40	CDKN2C	DNM2	FANCA	FLT1	GREM1	INPP4B	LIG4	MSH2	NTRK1	POLE	RANBP2	SETD2	STAT4	TSC2	-

RNA Fusions/Expression

Fusion/Expression													
ABL1	BCL2	CBFB	ERG	FGFR2	FOXO1	IKZF3	MAP3K1	NTRK1	NUP98	PICALM	RHOA	SS18	TCF3
AKT3	BCL6	CIC	ETV6	FGFR3	FUS	JAK2	MECOM	NTRK2	PDGFRA	PML	ROS2	STAT6	TFG
ALK	BRAF	CREBBP	EWSR1	FIP1L1	GLI1	KIAA1549	MYC	NTRK3	PDGFRB	RARA	RUNX1	TAFG	YWHAE
BCL1	CAMTA1	EGFR	FGFR1	FLAG1	HMGA2	KMT2A	NOTCH1	NUP214	PD-L1	RET	RUNX1T1	TAL1	

Reference

1. Molecular targeted therapy of glioblastoma. Le Rhun E, Preusser M, Roth P, Reardon DA, van den Bent M, Wen P, Reifenberger G, Weller M. Le Rhun E, et al. Cancer Treat Rev. 2019 Nov;80:101896. doi: 10.1016/j.ctrv.2019.101896. Epub 2019 Sep 11. Cancer Treat Rev. 2019. PMID: 31541850

2. Glioblastoma and other malignant gliomas: a clinical review. Omuro A, DeAngelis LM. Omuro A, et al. JAMA. 2013 Nov 6;310(17):1842-50. doi: 10.1001/jama.2013.280319. JAMA. 2013. PMID: 24193082

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5. Progress and prospect in tumor treating fields treatment of glioblastoma. Liu S, Shi W, Zhao Q, Zheng Z, Liu Z, Meng L, Dong L, Jiang X. Liu S, et al. Biomed Pharmacother. 2021 Sep;141:111810. doi: 10.1016/j.biopha.2021.111810. Epub 2021 Jun 30. Biomed Pharmacother. 2021. PMID: 34214730

Electronic Signature

The test (sample processing, sequencing and data generation) was performed at Genomic Testing Cooperative, LCA, Genomic Testing Cooperative, LCA, 175 Technology Drive, Suite 100, Irvine, CA 92618. Medical Director Maher Albitar, M.D. Analysis of the data was performed by Genomic Testing Cooperative, LCA, 175 Technology Drive, Suite 100, Irvine, CA 92618. 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.