BELAY DIAGNOSTICS

Date: 11/10/2026
Order ID: SumAscVan-Pos-GBM

1375 W. Fulton Street, Suite 530
Chicago, IL 60607
Email: contact@belaydiagnostics.com
Phone: (331) 320-0155 | Fax: (800) 501-9246

Summit™ 2.0 + Ascent™ + Vantage™ Report

Patient Information	Provisional Diagnosis	Specimen	Physician Information
Name: John Smith **DOB:**01/01/1990 **Sex Assigned at Birth:**Male **MRN:**11xx22xx33	**Diagnosis:**Glioblastoma **CSF Volume Received:**6 mL	**Type:**CSF **Collected:**11/03/2026 **Received:**11/04/2026 **Specimen ID:**SumAscVan-Pos-GBM	**Institution:**Belay Diagnostics **Referring Physician:**Provider Test

RESULT SUMMARY

CLINICALLY SIGNIFICANT VARIANTS - DETECTED

Comments

TERT promoter variants, *EGFR* amplification (included in chr7p gain), and chr7 gain/chr10 loss are characteristic findings in IDH-wildtype glioblastoma with additional *EGFR* alterations, *PIK3CA* activating variants, *CDK4* and *MDM2* amplifications, and *MGMT* promoter methylation also frequently observed in this tumor type (see Actionability Summary, PMID: 8504413, 37509518, 24893890, 24120142, 37446288). A high level of chromosomal loss and gain as observed in this specimen indicates chromosomal instability (PMID: 38924459). Clinical correlation is required.

CLINICALLY SIGNIFICANT ALTERATION DETAILS (Tier 1 or 2 per AMP/ASCO/CAP)

Summit™ 2.0 - SNV, MNV, Indel Variants

Alteration	VAF	Actionability Summary		
		FDA/NCCN Therapies Associated	Prognostic/Diagnostic Guidelines	Clinical Trial Options
EGFR p.P772_V774dup c.2314_2322dup	12.3%	No	Yes	Yes
PIK3CA p.H1047R c.3140A>G	19.6%	No	Yes	Yes
TERT c.-146C>T	18.4%	No	Yes	Yes

Summit™ 2.0 - Copy Number Variants

Alteration	Location	Fold Change	Actionability Summary		
			FDA/NCCN Therapies Associated	Prognostic/Diagnostic Guidelines	Clinical Trial Options
CDK4 Amplification	chr12	6.19	No	No	Yes
MDM2 Amplification	chr12	6.97	No	Yes	No

Summit™ 2.0 - Fusion Variants

None

Summit™ 2.0 - Biomarkers

Tumor Mutation Burden (TMB)			Microsatellite Instability (MSI)	
Not Detected	Low	High	Stable	High

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Ascent™ - Chromosome Arm Level Loss or Gain

Alteration	Type of Relevant Genes	Actionability Summary		
		FDA/NCCN Therapies Associated	Prognostic /Diagnostic Guidelines	Clinical Trial Options
Chr7 gain / Chr10 loss	Fully Contained: *GATA3*, *FGF8*, *FGFR2*, *PTEN*, *RET*, *SUFU*, *EGFR*, *BRAF*, *CDK6*, *MET*, *SMO*	No	Yes	Yes
chr7p11.2 Gain	Fully Contained: *EGFR*	No	Yes	Yes

Vantage™ - *MGMT* Promoter Methylation

Status	Guidelines	Actionability Summary		
		FDA/NCCN Therapies Associated	Prognostic/Diagnostic Guidelines	Clinical Trial Options
Methylated	NCCN	Yes	Yes	Yes

VARIANTS OF UNKNOWN SIGNIFICANCE (Tier 3)

Summit™ 2.0 - SNV/MNVs/Indels

ARID1B E74K	*GNA11* S267fs	*MEN1* S512L	*PDGFRB* Q972K	*SPTA1* N2165K
AXIN2 M210T	*GNA11* S267_L271delinsRA	*MTOR* S1418_I1419insSF	*PGR* G38W	*SPTA1* I756T
CBFB R3C	*IKZF1* R386_P390del	*MYB* S743P	*POLD1* D854N	*ZBTB2* C395*
EP300 E1937K	*KDM5A* R568H	*NOTCH3* A132P	*PRDM1* K773N	
EPHA5 V426I	*LATS2* P454L	*NTRK1* R780Q	*SMC1A* P746_D750delinsF	
FGF23 P172L	*LRP1B* R353S	*PAX5* F360_P363delinsL	*SPEN* A124V	

Summit™ 2.0 - Gene Level CNVs

None

Summit™ 2.0 - Fusions

None

Ascent™ - Aneuploidy Variants of Unknown Significance

chr1q Gain	chr6p Gain	chr16q Loss
chr4q Loss	chr6q Loss	chr22q Loss

ACTIONABILITY SUMMARY

FDA / NCCN Therapies for the Patient's Tumor Type (Tier 1A)

Biomarker	Therapies	Setting	Source(s)
MGMT Promoter Methylation Positive	alkylating agent	Unspecified	NCCN

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FDA / NCCN Therapies with Resistance / Decreased Response (Tier 1A)
None

Prognostic Implications per NCCN

Biomarker	Prognostic Association	Diseases	Note
MGMT Promoter Methylation Positive	Favorable	Glioma	MGMT promoter methylation confers a survival advantage in glioblastoma.
TERT c.-146C>T	Unfavorable	Glioma	In the absence of an IDH mutation, TERT mutations in diffusely infiltrative gliomas are associated with reduced overall survival compared to gliomas lacking TERT mutations.

Diagnostic Implications per WHO

Biomarker	Diseases	Note
EGFR Amplification	Central Nervous System Neoplasm	EGFR amplification is a diagnostic criterion for glioblastoma, IDH-wildtype. Overall, about 60% of these tumors show evidence of EGFR amplification, mutation, rearrangement, or altered splicing (PMID: 24120142). The most frequent of these alterations is EGFR amplification (PMID: 1374522), which occurs in about 40% of all IDH-wildtype glioblastomas (PMID: 24120142; PMID: 30187121). Gain of EGFR is also present in ~50% of cases of the RTK2 subtype of diffuse pediatric-type high-grade glioma, H3-wildtype and IDH-wildtype. EGFR amplification has also been identified in hemangioblastomas, and a small subset of MN1-altered astroblastomas.
EGFR P772_V774dup	Central Nervous System Neoplasm	EGFR mutations and fusions have been observed in several central nervous system neoplasms. In glioblastoma, IDH-wildtype, about 60% of tumors show evidence of EGFR amplification, mutation, rearrangement, or altered splicing (PMID:24120142), with mutations/fusions often co-occuring with amplification. EGFR gene fusions are observed in 6-13% of cases, typically as part of complex rearrangements at chromosome band 7p11.2 (PMID: 24120142). Fusion partners are mostly neighboring genes of EGFR (e.g. SEPTIN14, PSPH, SEC61G, SDK1). EGFR mutations also define an EGFR-mutant sub-type of diffuse midline glioma, H3 K27-altered. Within EGFR-mutant DMG, most tumors harbor small in-frame insertions/duplications within exon 20, which encodes the intracellular tyrosine kinase domain, whereas others harbor missense mutations in exons encoding parts of the extracellular domain, most commonly p.A289T or p.A289V. EGFR mutations have also been observed in diffuse pediatric-type high-grade glioma, H3-wildtype and IDH-wildtype.
MDM2 Amplification	Central Nervous System Neoplasm	Among central nervous system neoplasms, amplification of MDM2 has been observed in glioblastoma, IDH-wildtype, where MDM2 or MDM4 (inhibitors of p53) are amplified in about 15% of cases (PMID: 24120142). Suppression of apoptosis via genetic dysregulation of the p53 pathway occurs in nearly 90% of glioblastomas (PMID: 24120142). MDM2 amplification is also observed in other CNS neoplasms, such as diffuse hemispheric glioma, H3 G34-mutant.
MGMT Promoter Methylation Positive	Central Nervous System Neoplasm	Among central nervous system neoplasms, MGMT promoter methylation is detectable in the majority of oligodendrogliomas (PMID: 15455350). The MGMT gene encodes a DNA repair protein (PMID: 24071851) and is transcriptionally silenced by promoter methylation in approximately 40-50% of IDH-wildtype glioblastomas (PMID: 24120142; PMID: 10029064; PMID: 15758010; PMID: 22294349). MGMT promoter methylation in glioblastoma is a strong predictive marker of response to alkylating agents such as temozolomide and is associated with longer overall survival (PMID: 15758010; PMID: 22877848; PMID: 22578793; PMID: 19805672; PMID: 25655102; PMID: 24068788; PMID: 25035291; PMID: 24912512; PMID: 30782343), with more than 90% of long-term survivors harboring MGMT promoter methylation (PMID: 19269895). A higher frequency of methylation (>75%) is also associated with gliomas exhibiting the glioma CpG island methylator phenotype (G-CIMP), characteristic of IDH-mutant tumors (PMID: 22810491; PMID: 24120142; PMID: 23209033). Although H3 G34-mutant diffuse hemispheric gliomas display widespread DNA hypomethylation, MGMT is often methylated and may be associated with longer overall survival in the absence of oncogene amplifications (PMID: 23079654; PMID: 28966033; PMID: 30101054; PMID: 26482474). In high-grade astrocytoma with piloid features, a methylated MGMT promoter was reported in 46% of tumors, though no association with outcome was observed, and treatment data were unavailable (PMID: 29564591). In primary diffuse large B-cell lymphoma of the CNS, MGMT promoter methylation is observed in approximately 52% of cases and may have therapeutic implications, as a subset of elderly patients responded to temozolomide monotherapy (PMID: 16858686; PMID: 9546285; PMID: 15327516; PMID: 19494841; PMID: 19841864). In pituitary adenoma/pituitary neuroendocrine tumor (PitNET), MGMT protein expression appears inversely related to temozolomide response; however, promoter methylation status does not correlate with treatment outcomes (PMID: 29046323; PMID: 29330228; PMID: 20668043).
PIK3CA H1047R	Central Nervous System Neoplasm	Among central nervous system neoplasms, detection of PIK3CA mutations combined with suggestive histopathological features can be essential for the diagnosis of conventional meningioma. The oncogenic potential of PIK3CA mutations has been demonstrated in several tumor types (PMID:32120136), and PIK3CA mutations activate several proliferation-associated signaling pathways in meningiomas (PMID:25146167). Detection of a co-occuring PIK3CA and/or NF1 mutation with an FGFR1 mutation is a desirable diagnostic criterion in the diagnosis of rosette-forming glioneuronal tumors. In IDH-wildtype glioblastomas, mutations of PIK3CA, PIK3R1, or other PI3K pathway genes are observed in 25-30% of cases (PMID:24120142). In the H3.1 or H3.2 p.K28M (K27M)-mutant glioma subtype, target canonical cancer-associated pathways have subtype-specific enrichment and include PI3K or MAPK (PIK3CA, PIK3R1, PTEN). Among IDH-mutant and 1p/19q-codeleted oligodendrogliomas, PIK3CA is a less commonly mutated gene (PMID:26061751; PMID:25848751).
TERT c.-146 C>T	Central Nervous System Neoplasm	Among central nervous system neoplasms, TERT promoter mutation is an essential diagnostic criteria for glioblastoma, IDH-wildtype. In oligodendroglioma, IDH-mutant and 1p/19q-codeleted, TERT promoter mutation is desirable for the diagnosis. Additionally, TERT promoter mutation is also observed in diffuse midline glioma, H3 K27-altered (PMID: 28966033; PMID: 29763623), diffuse pediatric-type high-grade glioma, H3-wildtype and IDH-wildtype (~64% of cases) (PMID: 2840133), high-grade astrocytoma with piloid features (2 [3%] of 74, both C228T mutations) (PMID:29564591), medulloblastoma, SHH-activated and TP53-wildtype, and meningioma. In pleomorphic xanthoastrocytoma, TERT promoter mutation and less frequently TERT amplifications have been identified (PMID: 24154961; PMID: 30240866; PMID: 30051528; PMID: 32619305).
Chr7 gain / Chr10 loss	Central Nervous System Neoplasm	Among central nervous system neoplasms, whole chromosome 7 gain (trisomy 7) and whole chromosome 10 loss (monosomy 10) is one of the essential diagnostic criteria for glioblastoma, IDH-wildtype (WHO grade 4). +7/-10 is the most frequent numerical chromosome alteration in glioblastoma (PMID: 30187121), and the sensitivity and specificity for the diagnosis of IDH-wildtype glioblastoma were reported as 59% and 98% for +7/−10 (PMID: 30187121).

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CLINICAL TRIALS / INVESTIGATIONAL THERAPIES

CDK4 Amplification + *CDK6* Amplification			
Therapy	**Clinical Trial**	**Location/Sponsor**	**Quick Access**
abemaciclib	NCT03310879 (Phase 2) Study of the CDK4/6 Inhibitor Abemaciclib in Solid Tumors Harboring Genetic Alterations in Genes Encoding D-Type Cyclins or Amplification of CDK4 or CDK6	Boston, Massachusetts Dana-Farber Cancer Institute geoffrey_shapiro@dfci.harvard.edu	https://genomoncology.wetrials.com/v1/NCT03310879
palbociclib	NCT02896335 (Phase 2) Palbociclib and Pembrolizumab In Central Nervous System Metastases	Boston, Massachusetts Massachusetts General Hospital Nwang6@mgb.org	https://genomoncology.wetrials.com/v1/NCT02896335

CDK4 Amplification + *CDK6* Amplification + *PIK3CA* H1047R + *PTEN* Loss			
Therapy	**Clinical Trial**	**Location/Sponsor**	**Quick Access**
everolimus + ribociclib	NCT05843253 (Phase 2) Study of Ribociclib and Everolimus in HGG and DIPG or Ribociclib and Temozolomide in DHG, H3G34-mutant	Washington D.C., District of Columbia Nationwide Children's Hospital kelsey.troyer@nationwidechildrens.org	https://genomoncology.wetrials.com/v1/NCT05843253

EGFR Amplification + *EGFR* P772_V774dup			
Therapy	**Clinical Trial**	**Location/Sponsor**	**Quick Access**
verteporfin	NCT04590664 (Phase 1/Phase 2) Verteporfin for the Treatment of Recurrent High Grade EGFR-Mutated Glioblastoma	Atlanta, Georgia Emory University william.l.read@emory.edu	https://genomoncology.wetrials.com/v1/NCT04590664

EGFR Amplification + *Monosomy* 10 + *TERT* c.-146C>T + *Trisomy* 7			
Therapy	**Clinical Trial**	**Location/Sponsor**	**Quick Access**
bevacizumab + temozolomide + radiation therapy; temozolomide + radiation therapy	NCT05271240 (Phase 3) Repeated Superselective Intraarterial Cerebral Infusion (SIACI) of Bevacizumab With Temozolomide and Radiation Compared to Temozolomide and Radiation Alone in Newly Diagnosed GBM	New York, New York Northwell Health jboockvar@northwell.edu	https://genomoncology.wetrials.com/v1/NCT05271240

EGFR P772_V774dup			

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Therapy	Clinical Trial	Location/Sponsor	Quick Access
BDTX-1535 + temozolomide	NCT07326566 (Phase 2) Study of Silevertinib With Temozolomide for the Treatment of Newly Diagnosed GBM With Unmethylated MGMT and EGFRvIII	Birmingham, Alabama Black Diamond Therapeutics, Inc. blackdiamondtx@careboxhealth.com	https://genomoncology.wetrials.com/v1/NCT07326566

***MET* Amplification**			
Therapy	**Clinical Trial**	**Location/Sponsor**	**Quick Access**
VERT-002	NCT06669117 (Phase 1/Phase 2) FIH Trial of VERT-002 in Patients With Locally Advanced or Metastatic Solid Tumors With MET Alterations	Washington, District of Columbia Pierre Fabre Medicament yuhua.wang@pierre-fabre.com	https://genomoncology.wetrials.com/v1/NCT06669117
telisotuzumab adizutecan	NCT07196644 (Phase 2) A Study to Assess Adverse Events and Change in Disease Activity in Participants 12 Years of Age or Older With Locally Advanced or Metastatic Solid Tumors That Harbor MET Amplification Receiving Intravenously Infused Telisotuzumab Adizutecan	Beverly Hills, California AbbVie abbvieclinicaltrials@abbvie.com	https://genomoncology.wetrials.com/v1/NCT07196644

***MGMT* Promoter Methylation Positive**			
Therapy	**Clinical Trial**	**Location/Sponsor**	**Quick Access**
MT-125 + temozolomide + radiation therapy	NCT07604285 (Phase 1/Phase 2) A Phase 1/2 Study of Safety and Tolerability of MT-125 With Chemoradiation in Patients With Newly Diagnosed MGMT Methylated Glioblastoma	Phoenix, Arizona Myosin Therapeutics Inc.	https://genomoncology.wetrials.com/v1/NCT07604285
selinexor + temozolomide	NCT05432804 (Phase 1/Phase 2) Testing the Addition of an Anti-cancer Drug, Selinexor, to the Usual Chemotherapy Treatment (Temozolomide) for Brain Tumors That Have Returned After Previous Treatment	Sacramento, California National Cancer Institute (NCI)	https://genomoncology.wetrials.com/v1/NCT05432804
tuvusertib + temozolomide	NCT05691491 (Phase 1/Phase 2) Testing the Combination of the Anti-Cancer Drugs Temozolomide and M1774 to Evaluate Their Safety and Effectiveness	Irvine, California National Cancer Institute (NCI)	https://genomoncology.wetrials.com/v1/NCT05691491

***PIK3CA* H1047R**			
Therapy	**Clinical Trial**	**Location/Sponsor**	**Quick Access**

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ETX-636	NCT06993844 (Phase 1/Phase 2) Phase 1/2 Study of ETX-636 in Participants With Advanced Solid Tumors	Newport Beach, California Ensem Therapeutics janaki.parameswaran@Ensemtx.com	https://genomoncology.wetrials.com/v1/NCT06993844
REC-7735	NCT07819136 (Phase 1/Phase 2) REC-7735 in Participants With Solid Tumors	Grand Rapids, Michigan Recursion Pharmaceuticals Inc. clinicaltrials@recursionpharma.com	https://genomoncology.wetrials.com/v1/NCT07819136

***PTEN* Loss**			
Therapy	**Clinical Trial**	**Location/Sponsor**	**Quick Access**
EIS-12656; EIS-12656 + olaparib; EIS-12656 + fam-trastuzumab deruxtecan	NCT06525298 (Phase 1/Phase 2) EIS-12656 as Single Agent and in Combination in Patients With Specified Solid Tumors	Houston, Texas Eisbach Bio GmbH adrian@eisbach.bio	https://genomoncology.wetrials.com/v1/NCT06525298
dostarlimab + niraparib	NCT05700721 (Phase 2) Phase II Trial of the PARP Inhibitor Niraparib and PD-1 Inhibitor Dostarlimab in Patients With Advanced Cancers With Active Progressing Brain Metastases (STARLET)	Houston, Texas M.D. Anderson Cancer Center tyap@mdanderson.org	https://genomoncology.wetrials.com/v1/NCT05700721

TIER 1A THERAPY DETAILS

***MGMT* Promoter Methylation Positive**		
Therapy	**Approval / Guideline Summary**	**Underlying Evidence**
alkylating agent	Per NCCN, methylation of the MGMT promoter in glioma silences MGMT, making the tumor more sensitive to treatment with alkylating agents (Category 2A).	The NCCN guideline for alkylating agents was supported by data from a retrospective analysis of the MGMT promoter in tumor DNA by a methylation-specific polymerase chain reaction at the University Hospital of Navarre (PMID: 11070098). Clinical data demonstrated methylation of the promoter was positively correlated with the clinical response and with overall and disease-free survival; 63% (n = 12/19) of the patients with methylated tumors had a partial or complete response to carmustine, as compared with 4%; P < 0.001 (n = 1/ 28) patients with unmethylated tumors. Additionally, the median time to the progression of disease was 21 mo. for methylated gliomas vs. 8 mo. for unmethylated glioma; P < 0.001.

TEST DETAILS

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PANEL CONTENT AND REPORTING TRANSCRIPTS				
ABL1 NM_005157.4^ *ABL2* NM_007314.3	*DNAJB1* NM_006145.1 *DNMT1* NM_001130823.1 *DNMT3A* NM_022552.4	*H2BC5* NM_021063.3 *H3C1* NM_003529.2 *H3C2* NM_003537.3	*MYC* NM_002467.4+ *MYCL* NM_001033082.2+	*COP1* NM_022457.5 *RHEB* NM_005614.3 *RHOA* NM_001664.2

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ACVR1 NM_001105.4
ACVR1B NM_020328.3
AKT1 NM_001014432.1+
AKT2 NM_001626.4+
AKT3 NM_005465.4
ALK NM_004304.4^+
ANKRD11 NM_001256182.1
ANKRD26 NM_014915.2
APC NM_000038.5+
AR NM_000044.3+
ARAF NM_001654.4
ARFRP1 NM_003224.4
ARID1A NM_006015.4
ARID1B NM_020732.3
ARID2 NM_152641.2
ARID5B NM_032199.2
ASXL1 NM_015338.5
ASXL2 NM_018263.4
ATM NM_000051.3+
ATR NM_001184.3
ATRX NM_000489.3+
AURKA NM_198433.1
AURKB NM_004217.3
AXIN1 NM_003502.3
AXIN2 NM_004655.3
AXL NM_021913.4
B2M NM_004048.2
BAP1 NM_004656.3
BARD1 NM_000465.2
BBC3 NM_001127240.2
BCL10 NM_003921.4
BCL2 NM_000633.2
BCL2L1 NM_138578.1
BCL2L11 NM_001204108.1
BCL2L2 NM_001199839.1
BCL6 NM_001706.4
BCOR NM_001123385.1
BCORL1 NM_021946.4
BCR NM_004327.3^
BIRC3 NM_001165.4
BLM NM_000057.2
BMPR1A NM_004329.2
BRAF NM_004333.4^+
BRCA1 NM_007294.3+
BRCA2 NM_000059.3+
BRD4 NM_058243.2
BRIP1 NM_032043.2
BTG1 NM_001731.2
BTK NM_000061.2
EMSY NM_020193.3
CALR NM_004343.3
CARD11 NM_032415.4
CASP8 NM_001228.4
CBFB NM_001755.2
CBL NM_005188.3
CCND1 NM_053056.2+
CCND2 NM_001759.3
CCND3 NM_001760.3+
CCNE1 NM_001238.2+
CD274 NM_014143.3
CD276 NM_001024736.1
DNMT3B NM_006892.3
DOT1L NM_032482.2
E2F3 NM_001949.4
EED NM_003797.3
EGFL7 NM_016215.4
EGFR NM_005228.3^+
EIF1AX NM_001412.3
EIF4A2 NM_001967.3
EIF4E NM_001130679.1
EML4 NM_019063.3
EP300 NM_001429.3
EPCAM NM_002354.2
EPHA3 NM_005233.5
EPHA5 NM_004439.5
EPHA7 NM_004440.3
EPHB1 NM_004441.4
ERBB2 NM_004448.2+
ERBB3 NM_001982.3+
ERBB4 NM_005235.2
ERCC1 NM_001983.3+
ERCC2 NM_000400.3+
ERCC3 NM_000122.1
ERCC4 NM_005236.2
ERCC5 NM_000123.3
ERG NM_001136154.1
ERRFI1 NM_018948.3
ESR1 NM_001122742.1+
ETS1 NM_001143820.1
ETV1 NM_004956.4^
ETV4 NM_001079675.2^
ETV5 NM_004454.2
ETV6 NM_001987.4^
EWSR1 NM_013986.3^
EZH2 NM_004456.4
AMER1 NM_152424.3
ABRAXAS1 NM_139076.2
TENT5C NM_017709.3
FANCA NM_000135.2
FANCC NM_000136.2
FANCD2 NM_033084.3
FANCE NM_021922.2
FANCF NM_022725.3
FANCG NM_004629.1
FANCI NM_001113378.1
FANCL NM_001114636.1
FAS NM_000043.4
FAT1 NM_005245.3
FBXW7 NM_033632.3+
FGF1 NM_001144934.1+
FGF10 NM_004465.1+
FGF14 NM_175929.2+
FGF19 NM_005117.2+
FGF2 NM_002006.4+
FGF23 NM_020638.2+
FGF3 NM_005247.2+
FGF4 NM_002007.2+
FGF5 NM_004464.3+
FGF6 NM_020996.1+
H3C3 NM_003531.2+
H3C4 NM_003530.4
H3C6 NM_003532.2
H3C7 NM_021018.2
H3C8 NM_003534.2
H3C10 NM_003536.2
H3C11 NM_003533.2
H3C12 NM_003535.2
H3C15 NM_001005464.2
H3C14 NM_021059.2
H3C13 NM_001123375.2
H3-4 NM_003493.2
HLA-A NM_002116.7
HLA-B NM_005514.6
HLA-C NM_002117.5
HNF1A NM_000545.5
HNRNPK NM_002140.3
HOXB13 NM_006361.5
HRAS NM_005343.2+
HSD3B1 NM_000862.2
HSP90AA1 NM_001017963.2
ICOSLG NM_015259.4
ID3 NM_002167.4
IDH1 NM_005896.2+
IDH2 NM_002168.2+
IGF1 NM_001111283.1
IGF1R NM_000875.3
IGF2 NM_001127598.1
IKBKE NM_014002.3
IKZF1 NM_006060.4
IL10 NM_000572.2
IL7R NM_002185.3
INHA NM_002191.3
INHBA NM_002192.2
INPP4A NM_001134224.1
INPP4B NM_003866.2
INSR NM_000208.2
IRF2 NM_002199.3
IRF4 NM_002460.3
IRS1 NM_005544.2
IRS2 NM_003749.2
JAK1 NM_002227.2
JAK2 NM_004972.3+
JAK3 NM_000215.3
JUN NM_002228.3
KAT6A NM_006766.3
KDM5A NM_001042603.1
KDM5C NM_004187.3
KDM6A NM_021140.2
KDR NM_002253.2
KEAP1 NM_012289.3
KEL NM_000420.2
KIF5B NM_004521.2
KIT NM_000222.2+
KLF4 NM_004235.4
KLHL6 NM_130446.2
KMT2B NM_014727.1
KMT2C NM_170606.2
KMT2D NM_003482.3
KRAS NM_004985.3+
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LATS2 NM_014572.2
LMO1 NM_002315.2
MYCN NM_005378.4+
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MYOD1 NM_002478.4
NAB2 NM_005967.3^
NBN NM_002485.4
NCOA3 NM_181659.2
NCOR1 NM_006311.3
NEGR1 NM_173808.2
NF1 NM_001042492.2+
NF2 NM_000268.3+
NFE2L2 NM_006164.4+
NFKBIA NM_020529.2
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NOTCH4 NM_004557.3
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NRAS NM_002524.4+
NRG1 NM_013964.3+
NSD1 NM_022455.4
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NUTM1 NM_175741.1^
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TBX3 NM_016569.3
ELOC NM_005648.3
TCF3 NM_003200.3
TCF7L2 NM_030756.4
TERC
TERT NM_198253.2+
TET1 NM_030625.2
TET2 NM_001127208.2

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Date: 11/10/2026
Order ID: SumAscVan-Pos-GBM

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CD74 NM_001025159.2^
CD79A NM_001783.3
CD79B NM_000626.2+
CDC73 NM_024529.4
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CHD4 NM_001273.2
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CRLF2 NM_022148.2
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FGF9 NM_002010.2+
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FLT4 NM_182925.4
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TNFAIP3 NM_006290.3
TNFRSF14 NM_003820.2
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TSHR NM_000369.2
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VHL NM_000551.3+
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CCN6 NM_003880.3
WT1 NM_024426.4
XIAP NM_001167.3
XPO1 NM_003400.3
XRCC2 NM_005431.1
YAP1 NM_001130145.2
YES1 NM_005433.3
ZBTB2 NM_020861.1
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ZFHX3 NM_006885.3
ZNF217 NM_006526.2
ZNF703 NM_025069.1
ZRSR2 NM_005089.3
MTAP NM_002451.3+*

^Summit™ also reports fusion events for this gene
+Summit™ also reports copy number alterations for this gene
*Summit™ only reports copy number alterations for this gene

Aneuploidy (chromosome arm level loss and gain)									
chr1p	chr3p	chr5p	chr7p	chr9p	chr11p	chr13q	chr16q	chr18q	chr20q
chr1q	chr3q	chr5q	chr7q	chr9q	chr11q	chr14q	chr17p	chr19p	chr21q
chr2p	chr4p	chr6p	chr8p	chr10p	chr12p	chr15q	chr17q	chr19q	chr22q
chr2q	chr4q	chr6q	chr8q	chr10q	chr12q	chr16p	chr18p	chr20p	

Methods and Limitations

The Summit™ 2.0 comprehensive genomic profiling (CGP) next-generation sequencing (NGS) test investigates tumor derived nucleic acid extracted from cerebrospinal fluid (CSF) for clinically relevant single/multi nucleotide variants (SNVs, MNVs), insertions and deletions (indels), gene level copy number variants (CNVs), and other biomarkers such as tumor mutation burden (TMB) and microsatellite instability (MSI). Methodology involves evaluation of 520 genes for SNVs, MNVs, Indels, 62 genes for CNVs, 28 genes for fusions, as well as TMB and MSI (PMID: 41595175). The LOD (limit of detection) for SNVs, MNVs and Indels was determined to be 0.3% variant allelic frequency (VAF), for CNVs was determined to be >=2-fold change

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for amplifications and <=0.5-fold change for deletions, and for fusions was determined to be >=2 supporting reads. Reporting thresholds for TMB and MSI are: <10 Mut/Mb (TMB low), >=10 Mut/Mb (TMB high), and when total unstable sites are <30% (MSS) and >=30% (MSI-High). Ascent™ evaluates chromosomal arm level loss/gain (aneuploidy), and focal alterations (gene level amplification/deletion) using >0.1x low pass whole genome sequencing (LP-WGS) (PMID: 37014860). The LOD (limit of detection) for aneuploidy was determined to be log2(*r*) of abs (0.09), and for focal alteration was determined to be seq.mean cutoff of 0.1 for amplification and -0.2 for deletions. Variants are called against the human genome build reference hg19 using Summit™Omics pipeline version 1.5.0, developed at Belay Diagnostics.

The Vantage™ *MGMT* Promoter Methylation Assay utilizes a quantitative PCR (qPCR) followed by high-resolution melt analysis (HRM) using the EpiMelt MGMT kit (MethylDetect) after enzymatic conversion (NEBNext Enzymatic Methyl-seq, New England Biolabs) on a portion of the library generated in the Summit™ workflow. Methylated and unmethylated melting temperature peaks are evaluated using the LightCycler® 480 Software v. 1.5.1 (Roche LifeScience). Qualitative results are reported as "Negative - Unmethylated", "Positive - Methylated", or "Indeterminate Results were equivocal". Specimens with results above the validated 25% methylated control are interpreted as "Positive". Specimens with results between unmethylated and methylated control are interpreted as "Indeterminate".

Tertiary analysis is performed using the precision oncology workbench (GenomOncology) based on the joint AMP/ASCO/CAP consensus guidelines for interpretation of sequence variants in cancer (PMID: 27993330). Please reach out to contact@belaydiagnostics.com for additional information or queries.

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Disclaimers

This test was developed, and its performance characteristics determined by Belay Diagnostics Laboratory (CLIA# 14D2302605), which is certified under the Clinical Laboratory Improvement Amendments of 1988 (CLIA) as qualified to perform high complexity testing. This test has not been cleared or approved by the U.S. Food and Drug Administration (FDA). This test may be used for clinical purposes. However, the results of this test do not establish a diagnosis and should not be used alone for diagnosis or patient care decisions or otherwise replace the judgment of a treating physician and must always be interpreted in the context of all relevant clinical and pathological data.

This test is performed only to evaluate for somatic (i.e., tumor-specific) variants within the genes listed and cannot distinguish between germline and somatic alterations with absolute certainty. This test therefore does not report on incidental findings as defined by the American College for Medical Genetics and Genomics (ACMG) (PMID: 37347242). If a germline variant is suspected, follow-up germline testing using non-neoplastic (normal) tissue should be performed by a laboratory permitted to perform germline genetic testing along with genetic counseling. It is possible for a genomic variant to be present yet go undetected by our assay either due to the heterogeneous nature of the specimen or the limits of detection of our assay. Therefore, to the extent a particular genomic variant is not reported, Belay Diagnostics LLC does not guarantee that the variant does not exist in the specimen provided. Likely benign, and benign variants are not reported. For any reported variant of uncertain significance (VUS), if the classification changes, there is no obligation to send out a new report updating this information.

The information presented in the clinical trials and therapeutic sections of this report is compiled from public sources which are continuously updated. While we strive to ensure this information is accurate and complete, we cannot guarantee the accuracy or completeness of this information. This public sourced information is not ranked in order of potential or predicted efficacy and may not be complete. Specific eligibility criteria should be reviewed as applicable. This information may include associations between a genomic variant (or lack of a variant) and one or more therapeutic agents with potential clinical benefit (or lack of clinical benefit), including agents that are being studied in clinical research. The finding of a genomic variant does not necessarily indicate or demonstrate pharmacologic effectiveness (or lack thereof) of any agent or treatment regimen found in public source information. Similarly, the finding of "no clinically significant variant" does not necessarily indicate or demonstrate lack of pharmacologic effectiveness (or lack of effectiveness) of any agent or treatment regimen found in public source information. Belay Diagnostics expressly disclaims, and makes no representation of or warranty of, the accuracy or completeness with respect to the publicly available information included herein or reviewed or collected during creation of this report.

ACTIONABILITY REFERENCES

FDA: U.S. Food & Drug Administration (fda.gov)

NCCN: National Comprehensive Cancer Network® (NCCN®). Referenced with permission from the NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®). © National Comprehensive Cancer Network, Inc. 2024. All rights reserved. The NCCN Guidelines® and illustrations herein may not be reproduced in any form for any purpose without the express written permission of the NCCN. To view the most recent and complete version of the guideline, go online to NCCN.org. NCCN makes no warranties of any kind whatsoever regarding their content, use or application and disclaims any responsibility for their application or use in any way.

WHO: World Health Organization Classification of Tumours online (tumourclassification.iarc.who.int)

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