BELAY DIAGNOSTICS

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

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:**Oligodendroglioma **CSF Volume Received:**6 mL	**Type:**CSF **Collected:**11/03/2026 **Received:**11/04/2026 **Specimen ID:**SumAscVan-Pos-Oligo	**Institution:**Belay Diagnostics **Referring Physician:**Provider Test

RESULT SUMMARY

CLINICALLY SIGNIFICANT VARIANTS - DETECTED

Comments
IDH1 R132H and 1p/19q codeletion are characteristic findings in oligodendroglioma with *CIC* variants and *MGMT* promoter methylation also frequently observed in this tumor type (see Actionability Summary). 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
CIC p.Q1178* c.3532C>T	62.2%	No	Yes	No
IDH1 p.R132H c.395G>A	29.7%	Yes	Yes	Yes

Summit™ 2.0 - Copy Number Variants

None

Summit™ 2.0 - Fusion Variants

None

Summit™ 2.0 - Biomarkers

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

Ascent™ - Chromosome Arm Level Loss or Gain

Alteration	Type of Relevant Genes	Actionability Summary		
		FDA/NCCN Therapies Associated	Prognostic /Diagnostic Guidelines	Clinical Trial Options
1p/19q Codeletion	Fully Contained: *AKT2*, *CCNE1*, *CIC*, *ERCC1*, *ERCC2*, *FUBP1*, *MYCL* , *NRAS*	Yes	Yes	Yes

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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

ADGRA2 S456N	*FANCA* H1417D	*PBRM1* P1476S	*POLE* G1305R	*XIAP* P257A
AXIN1 A311S	*IDH1* R222C	*PGR* F358S	*RECQL4* H362R	
AXIN2 A684V	*KDM5A* R336G	*PIK3C2G* V1413A	*SDHA* S2L	
DAXX N140S	*MET* R143Q	*PIK3CB* E554G	*TET1* V2128I	

Summit™ 2.0 - Gene Level CNVs

None

Summit™ 2.0 - Fusions

None

Ascent™ - Aneuploidy Variants of Unknown Significance

chr8p Gain	chr11q Loss	chr16q Gain

ACTIONABILITY SUMMARY

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

Biomarker	Therapies	Setting	Source(s)
1p/19q Codeletion / *IDH1* R132H / *MGMT* Promoter Methylation Positive	alkylating agent	Unspecified	NCCN
IDH1 R132H	ivosidenib; vorasidenib	Recurrent or progressive	NCCN

FDA / NCCN Therapies with Resistance / Decreased Response (Tier 1A)

None

Prognostic Implications per NCCN

Biomarker	Prognostic Association	Diseases	Note
1p/19q Codeletion	Favorable	Glioma	For glioma, codeletion of 1p and 19q confers a favorable prognosis.
IDH1 R132H	Favorable	Glioma	For glioma, IDH1 or 2 mutations are associated with a relatively favorable prognosis.
MGMT Promoter Methylation Positive	Favorable	Glioma	MGMT promoter methylation confers a survival advantage in glioblastoma.

Diagnostic Implications per WHO

Biomarker	Diseases	Note

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1p/19q Codeletion, *IDH1* R132H	Central Nervous System Neoplasm	The co-occurrence of IDH mutation and the combined whole-arm deletion of 1p and 19q defines "oligodendroglioma, IDH-mutant and 1p/19q-codeleted", which is a WHO grade 2 or grade 3 diffusely infiltrating glioma. IDH1 c.395G>A p.R132H is the most frequent mutation in IDH-mutant gliomas, affecting 83-91% of cases (PMID:18985363; PMID:19246647; PMID:19228619).
CIC Q1178*	Central Nervous System Neoplasm	Among central nervous system neoplasms, mutations of CIC, located in chromosome band 19q13.2, are frequent in IDH-mutant and 1p/19q-codeleted oligodendrogliomas (PMID:21817013; PMID:22072542), with large-scale sequencing studies reporting CIC mutations in as many as 70% of oligodendrogliomas (PMID:26061751; PMID: 26061753). CIC mutations in oligodendrogliomas are hemizygous and include almost equal proportions of nonsense or truncating mutations and recurrent missense mutations. Additionally, The combined loss of CIC and FUBP1 protein expression, as a surrogate marker of CIC and FUBP1 nonsense or truncating mutations, has been associated with a shorter time to recurrence in patients with 1p/19q-codeleted oligodendroglioma (PMID:24030748).
IDH1 R132H	Central Nervous System Neoplasm	Among central nervous system neoplasms, an IDH1 codon 132 missense is an essential diagnostic criterion for astrocytoma, IDH-mutant and oligodendroglioma, IDH-mutant and 1p/19q-codeleted. In the case of diffuse pediatric-type high-grade glioma, H3-wildtype and IDH-wildtype, absence of IDH1 mutation is an essential diagnostic criterion. In the case of IDH-wildtype glioblastoma, absence of immunoreactivity for IDH1 p.R132H is sufficient (i.e. without further sequencing) to diagnose IDH-wildtype glioblastoma in a patient aged >= 55 years at diagnosis who has a histologically classic glioblastoma not located in midline structures and no history of a pre-existing lower-grade glioma (PMID: 27157931). Patients aged < 55 years, or patients with a history of lower-grade glioma and/or whose tumors show immunohistochemical loss of nuclear ATRX expression, negative IDH1 p.R132H immunostaining should be followed by DNA sequencing for less common IDH1 or IDH2 mutations. Angiocentric gliomas lack mutations in IDH1 (PMID: 22445362). Chordoid gliomas have lacked accompanying pathogenic alterations in genes characteristic of other brain tumor entities (e.g. IDH1, IDH2, H3-3A, H3C2 [HIST1H3B], FGFR1, BRAF, NF1, CDKN2A, TP53). IDH mutation (either IDH1 p.R132 or IDH2 p.R172) is not compatible with the diagnosis of ganglioglioma. No mutations in IDH1 have been reported to date in diffuse leptomeningeal glioneuronal tumors. In the case of pituicytoma, granular cell tumor of the sellar region, and spindle cell oncocytoma, IDH1 p.R132H mutation is absent (PMID: 23887161).
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).

CLINICAL TRIALS / INVESTIGATIONAL THERAPIES

***IDH1* R132H**			
Therapy	**Clinical Trial**	**Location/Sponsor**	**Quick Access**
olutasidenib	**NCT07486713** (Phase 4) Olutasidenib DDI Study in Patients With IDH1 Mutation Positive Malignancies	Orange, California Rigel Pharmaceuticals kpatel@rigel.com	https://genomoncology.wetrials.com/v1/NCT07486713
pembrolizumab + olaparib + temozolomide	**NCT05188508** (Phase 2) Pembrolizumab, Olaparib, and Temozolomide for People With Glioma	Hartford, Connecticut Memorial Sloan Kettering Cancer Center schaffl@mskcc.org	https://genomoncology.wetrials.com/v1/NCT05188508
valproic acid + simvastatin + irinotecan	**NCT07831369** (Phase 2) VISTA: Valproic Acid, Irinotecan, and Simvastatin for Recurrent Glioma	Fairfax, Virginia Inova Health Care Services keary.jane't@inova.org	https://genomoncology.wetrials.com/v1/NCT07831369
***IDH1* R132H + 1p/19q Codeletion**			

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Therapy	Clinical Trial	Location/Sponsor	Quick Access
safusidenib	NCT05303519 (Phase 3) *FDA Fast Track* SIGMA (Safusidenib in IDH1 Mutant Glioma Maintenance)	Birmingham, Alabama Nuvation Bio Inc. ClinicalTrials@nuvationbio.com	https://genomoncology.wetrials.com/v1/NCT05303519
safusidenib	NCT07703436 (Phase 2) *FDA Fast Track* Safety and Efficacy Study of Safusidenib in Participants With IDH1-Mutant Glioma Who Discontinued Vorasidenib Treatment Due to Progressive Disease	Los Angeles, California Nuvation Bio Inc. ClinicalTrials@nuvationbio.com	https://genomoncology.wetrials.com/v1/NCT07703436

IDH1 R132H + _MGMT_ Promoter Methylation Positive

Therapy	Clinical Trial	Location/Sponsor	Quick Access
temozolomide; dasatinib + quercetin; fisetin; fisetin + temozolomide; dasatinib + quercetin + temozolomide; LMP744; TLPO	NCT07025226 (Early Phase 1) Medication Combinations of Dasatinib, Quercetin, Fisetin, Temozolomide, LMP744, and Autologous TLPO Vaccine for the Treatment of Previously Treated Glioma With Residual Disease	Rochester, Minnesota Mayo Clinic mayocliniccancerstudies@mayo.edu	https://genomoncology.wetrials.com/v1/NCT07025226

MGMT Promoter Methylation Positive

Therapy	Clinical Trial	Location/Sponsor	Quick Access
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

TIER 1A THERAPY DETAILS

1p/19q Codeletion

Therapy	Approval / Guideline Summary	Underlying Evidence
alkylating agent	Per NCCN, the 1/19 codeletion in glioma is predictive of response to alkylating systemic therapy with or without RT (Category 2A).	The NCCN guideline for alkylating agents was supported by two trials: Study 26951 (NCT00002840; PMID: 23071237) and RTOG 9402 (NCT00002569; PMID: 23071247). Data from the randomized, phase-III trial, Study 26951 demonstrated that adjuvant RT/PCV compared with RT alone improved, OS (NR vs. 112 mo., HR = 0.56) in patients with newly diagnosed anaplastic oligodendroglial tumors with 1p/19q codeletion (n = 80). The secondary endpoint was PFS (157 mo. vs. 50 mo., HR = 0.73). Data from the open-label, randomized, phase-III trial RTOG 9402 demonstrated that PCV/RT, compared with RT alone, improved median survival (14.7 yr. v 7.3 yr.; HR = 0.59; P = 0.03) in patients with anaplastic oligodendrogliomas with 1p/19q codeletion. For those with noncodeleted tumors, there was no difference in median survival by treatment arm (2.6 yr. v, 2.7 yr.; HR = 0.85; P = 0.39).

IDH1 R132H

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Therapy	Approval / Guideline Summary	Underlying Evidence
alkylating agent	Per NCCN, IDH1 or IDH2 mutations are associated with a survival benefit for glioma treated with alkylating systemic therapy (Category 2A).	The NCCN guideline for alkylating agents was supported by data from a retrospective analysis at Pitie-Salpetriere Hospital (PMID: 20975057). Clinical data demonstrated that patients with IDH mutations (n = 132/189), compared to those with no IDH mutation (n = 57/189), had prolonged OS (136.5 mo. vs. 83.9 mo.; P = 0.002) in low-grade gliomas when treated up-front with temozolomide.
ivosidenib	NCCN recommended for recurrent or progressive oligodendroglioma and astrocytoma with IDH1 mutations.	The NCCN guideline for ivosidenib is supported by data from the open-label phase-I trial NCT02073994 (PMID: 32530764). The trial demonstrated that ivosidenib (n = 35) conferred an ORR of 2.9%, with 1 partial response, in patients with non-enhancing, advanced glioma with an IDH1 mutation. Stable disease was achieved in 85.7% (30/35) of IDH1-mutated, non-enhancing glioma, compared with 45.2% (14/31) with IDH1-mutated, enhancing glioma. The secondary endpoint was median PFS (13.6 mos. and 1.4 mos. for non-enhancing and enhancing glioma, respectively).
vorasidenib	NCCN recommended for recurrent or progressive oligodendroglioma or astrocytoma with an IDH1 or IDH2 mutation (Preferred intervention).	

***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
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
DNAJB1 NM_006145.1
DNMT1 NM_001130823.1
DNMT3A NM_022552.4
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
H2BC5 NM_021063.3
H3C1 NM_003529.2
H3C2 NM_003537.3
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
MYC NM_002467.4+
MYCL NM_001033082.2+
MYCN NM_005378.4+
MYD88 NM_002468.4+
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
NKX2-1 NM_001079668.2
NKX3-1 NM_006167.3
NOTCH1 NM_017617.3
NOTCH2 NM_024408.3
NOTCH3 NM_000435.2
NOTCH4 NM_004557.3
NPM1 NM_002520.6
NRAS NM_002524.4+
NRG1 NM_013964.3+
COP1 NM_022457.5
RHEB NM_005614.3
RHOA NM_001664.2
RICTOR NM_152756.3+
RIT1 NM_006912.5
RNF43 NM_017763.4
ROS1 NM_002944.2^
RPS6KA4 NM_003942.2
RPS6KB1 NM_003161.3+
RPS6KB2 NM_003952.2
RPTOR NM_020761.2
RUNX1 NM_001754.4
RUNX1T1 NM_175635.2
RYBP NM_012234.5
SDHA NM_004168.2
SDHAF2 NM_017841.2
SDHB NM_003000.2
SDHC NM_003001.3
SDHD NM_003002.3
SETBP1 NM_015559.2
SETD2 NM_014159.6+
SF3B1 NM_012433.2
SH2B3 NM_005475.2
SH2D1A NM_002351.4
SHQ1 NM_018130.2
SLIT2 NM_004787.1

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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
CD74 NM_001025159.2^
CD79A NM_001783.3
CD79B NM_000626.2+
CDC73 NM_024529.4
CDH1 NM_004360.3+
CDK12 NM_016507.2
CDK4 NM_000075.3+
CDK6 NM_001259.6+
CDK8 NM_001260.1
CDKN1A NM_000389.4
CDKN1B NM_004064.3
CDKN2A NM_000077.4+
CDKN2B NM_004936.3+
CDKN2C NM_001262.2
CEBPA NM_004364.3
CENPA NM_001809.3
CHD2 NM_001271.3
CHD4 NM_001273.2
CHEK1 NM_001114122.2+
CHEK2 NM_007194.3+
CIC NM_015125.3+
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+
FGF7 NM_002009.3+
FGF8 NM_033163.3+
FGF9 NM_002010.2+
FGFR1 NM_023110.2+
FGFR2 NM_000141.4^+
FGFR3 NM_000142.4^+
FGFR4 NM_213647.1+
FH NM_000143.3
FLCN NM_144997.5
FLI1 NM_002017.4
FLT1 NM_002019.4
FLT3 NM_004119.2
FLT4 NM_182925.4
FOXA1 NM_004496.3
FOXL2 NM_023067.3
FOXO1 NM_002015.3
FOXP1 NM_032682.5
FRS2 NM_001278351.1
FUBP1 NM_003902.3+
FYN NM_002037.5
GABRA6 NM_000811.2
GATA1 NM_002049.3
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+
LAMP1 NM_005561.3+
LATS1 NM_004690.3
LATS2 NM_014572.2
LMO1 NM_002315.2
LRP1B NM_018557.2
LYN NM_002350.3
LZTR1 NM_006767.3
MAGI2 NM_012301.3
MALT1 NM_006785.3
MAP2K1 NM_002755.3
MAP2K2 NM_030662.3
MAP2K4 NM_003010.3
MAP3K1 NM_005921.1
MAP3K13 NM_004721.4
MAP3K14 NM_003954.3
MAP3K4 NM_005922.2
MAPK1 NM_002745.4
MAPK3 NM_002746.2
MAX NM_002382.4
MCL1 NM_021960.4
MDC1 NM_014641.2
MDM2 NM_002392.5+
MDM4 NM_002393.4+
MED12 NM_005120.2
MEF2B NM_001145785.1
MEN1 NM_130799.2
MET NM_000245.2+
NSD1 NM_022455.4
NTRK1 NM_002529.3^
NTRK2 NM_006180.3^
NTRK3 NM_001012338.2
NUP93 NM_014669.4
NUTM1 NM_175741.1^
PAK1 NM_001128620.1
PAK3 NM_002578.3
PAK5 NM_020341.3
PALB2 NM_024675.3
PRKN NM_004562.2
PARP1 NM_001618.3
PAX3 NM_181457.3^
PAX5 NM_016734.2
PAX7 NM_001135254.1
PAX8 NM_013953.3^
PBRM1 NM_018313.4
PDCD1 NM_005018.2
PDCD1LG2 NM_025239.3
PDGFRA NM_006206.4+
PDGFRB NM_002609.3+
PDK1 NM_001278549.1
PDPK1 NM_002613.4
PGR NM_000926.4
PHF6 NM_032458.2
PHOX2B NM_003924.3
PIK3C2B NM_002646.3
PIK3C2G NM_004570.4
PIK3C3 NM_002647.2
PIK3CA NM_006218.2+
PIK3CB NM_006219.2+
PIK3CD NM_005026.3
PIK3CG NM_002649.2
PIK3R1 NM_181523.2
PIK3R2 NM_005027.3
PIK3R3 NM_003629.3
PIM1 NM_002648.3
PLCG2 NM_002661.3
PLK2 NM_006622.3
PMAIP1 NM_021127.2
PMS1 NM_000534.4
PMS2 NM_000535.5
PNRC1 NM_006813.2
POLD1 NM_001256849.1
POLE NM_006231.2
PPARG NM_138712.3^
PPM1D NM_003620.3
PPP2R1A NM_014225.5
PPP2R2A NM_001177591.1
PPP6C NM_001123355.1
PRDM1 NM_001198.3
PREX2 NM_024870.2
PRKAR1A NM_212472.2
PRKCI NM_002740.5
PRKDC NM_006904.6
PRSS8 NM_002773.3
PTCH1 NM_000264.3+
PTEN NM_000314.4+
PTPN11 NM_002834.3
PTPRD NM_002839.3
PTPRS NM_002850.3
PTPRT NM_133170.3
SLX4 NM_032444.2
SMAD2 NM_005901.5
SMAD3 NM_005902.3
SMAD4 NM_005359.5+
SMARCA4 NM_001128849.1+
SMARCB1 NM_003073.3+
SMARCD1 NM_003076.4
SMC1A NM_006306.3
SMC3 NM_005445.3
SMO NM_005631.4+
SNCAIP NM_005460.2
SOCS1 NM_003745.1
SOX10 NM_006941.3
SOX17 NM_022454.3
SOX2 NM_003106.3
SOX9 NM_000346.3
SPEN NM_015001.2
SPOP NM_001007228.1
SPTA1 NM_003126.2
SRC NM_198291.2
SRSF2 NM_003016.4
STAG1 NM_005862.2
STAG2 NM_001042749.1
STAT3 NM_139276.2
STAT4 NM_003151.3
STAT5A NM_003152.3
STAT5B NM_012448.3
STK11 NM_000455.4
STK40 NM_032017.1
SUFU NM_016169.3+
SUZ12 NM_015355.2
SYK NM_003177.5
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
TFE3 NM_006521.4^
TFRC NM_003234.2+
TGFBR1 NM_004612.2
TGFBR2 NM_001024847.2
TMEM127 NM_017849.3
TMPRSS2 NM_001135099.1^
TNFAIP3 NM_006290.3
TNFRSF14 NM_003820.2
TOP1 NM_003286.2
TOP2A NM_001067.3
TP53 NM_000546.5+
TP63 NM_003722.4
TRAF2 NM_021138.3
TRAF7 NM_032271.2+
TSC1 NM_000368.4
TSC2 NM_000548.3
TSHR NM_000369.2
U2AF1 NM_006758.2
VEGFA NM_001025366.2
VHL NM_000551.3+
VTCN1 NM_024626.3
CCN6 NM_003880.3

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BELAY DIAGNOSTICS

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

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CREBBP NM_004380.2	*GATA2* NM_032638.4	*MGA* NM_001164273.1	*QKI* NM_006775.2	*WT1* NM_024426.4
CRKL NM_005207.3	*GATA3* NM_001002295.1+	*MITF* NM_000248.3	*RAB35* NM_006861.6	*XIAP* NM_001167.3
CRLF2 NM_022148.2	*GATA4* NM_002052.3	*MLH1* NM_000249.3	*RAC1* NM_018890.3	*XPO1* NM_003400.3
CSF1R NM_005211.3	*GATA6* NM_005257.4	*KMT2A* NM_001197104.1	*RAD21* NM_006265.2	*XRCC2* NM_005431.1
CSF3R NM_156039.3	*GEN1* NM_182625.3	*MLLT3* NM_004529.2	*RAD50* NM_005732.3	*YAP1* NM_001130145.2
CSNK1A1 NM_001025105.2	*GID4* NM_024052.4	*MPL* NM_005373.2	*RAD51* NM_002875.4	*YES1* NM_005433.3
CTCF NM_006565.3	*GLI1* NM_005269.2	*MRE11* NM_005591.3	*RAD51B* NM_133509.3	*ZBTB2* NM_020861.1
CTLA4 NM_005214.4	*GNA11* NM_002067.2	*MSH2* NM_000251.2	*RAD51C* NM_058216.2	*ZBTB7A* NM_015898.2
CTNNA1 NM_001903.2	*GNA13* NM_006572.4	*MSH3* NM_002439.4	*RAD51D* NM_002878.3	*ZFHX3* NM_006885.3
CTNNB1 NM_001904.3+	*GNAQ* NM_002072.3	*MSH6* NM_000179.2	*RAD52* NM_134424.2	*ZNF217* NM_006526.2
CUL3 NM_003590.4	*GNAS* NM_000516.4+	*MST1* NM_020998.3	*RAD54L* NM_001142548.1	*ZNF703* NM_025069.1
CUX1 NM_181552.3	*ADGRA2* NM_032777.9	*MST1R* NM_002447.2	*RAF1* NM_002880.3+	*ZRSR2* NM_005089.3
CXCR4 NM_003467.2	*GPS2* NM_004489.4	*MTOR* NM_004958.3	*RANBP2* NM_006267.4	*MTAP* NM_002451.3+*
CYLD NM_015247.2	*GREM1* NM_013372.6	*MUTYH* NM_001128425.1	*RARA* NM_000964.3	
DAXX NM_001141970.1	*GRIN2A* NM_000833.3	*MYB* NM_001130173.1	*RASA1* NM_002890.2	
DCUN1D1 NM_020640.2	*GRM3* NM_000840.2		*RB1* NM_000321.2+	
DDR2 NM_001014796.1	*GSK3B* NM_002093.3		*RBM10* NM_005676.4	
DDX41 NM_016222.2	*H3-3A* NM_002107.4+		*RECQL4* NM_004260.3	
DHX15 NM_001358.2	*H3-3B* NM_005324.3		*REL* NM_002908.2	
DICER1 NM_177438.2	*H3-5* NM_001013699.2		*RET* NM_020975.4^+	
DIS3 NM_014953.3	*HGF* NM_000601.4			
	H1-2 NM_005319.3			

^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	

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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 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.

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

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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.

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ACTIONABILITY REFERENCES
FDA: U.S. Food & Drug Administration (fda.gov)
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WHO: World Health Organization Classification of Tumours online (tumourclassification.iarc.who.int)

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