BELAY DIAGNOSTICS

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

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

Ascent™ 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:**Asc-Pos-GBM	**Institution:**Belay Diagnostics **Referring Physician:**Provider Test

RESULT SUMMARY

CLINICALLY SIGNIFICANT VARIANTS - DETECTED

Comments
EGFR amplification (included in focal gain of chr7p11.2) and chr7 gain/chr10 loss are characteristic findings in IDH-wildtype glioblastoma (see Actionability Summary). Clinical correlation is required.

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

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

VARIANTS OF UNKNOWN SIGNIFICANCE (Tier 3)

Ascent™ - Aneuploidy Variants of Unknown Significance	
chr1q Gain	chr16q Loss

ACTIONABILITY SUMMARY

FDA / NCCN Therapies for the Patient's Tumor Type (Tier 1A)
None

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

Prognostic Implications per NCCN
None

Diagnostic Implications per WHO		
Biomarker	**Diseases**	**Note**

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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.
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).

CLINICAL TRIALS / INVESTIGATIONAL THERAPIES

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***EGFR* Amplification**

Therapy	Clinical Trial	Location/Sponsor	Quick Access
CART-EGFR-IL13Ra2	**NCT07544992** (Phase 1) Locoregional Administration of Genetically Engineered Cells (EGFR/IL13Ra2 Pool-CAR T Cells) for the Treatment of Recurrent or Progressive High-Grade Gliomas	Duarte, California City of Hope Medical Center bbadie@coh.org	https://genomoncology.wetrials.com/v1/NCT07544992
CARv3-TEAM-E T-cells	**NCT05660369** (Phase 1) CARv3-TEAM-E T Cells in Glioblastoma	Boston, Massachusetts Marcela V. Maus, M.D.,Ph.D. carteamingbm@mgb.org	https://genomoncology.wetrials.com/v1/NCT05660369
ERAS-801 +/- surgery	**NCT07089641** (Phase 1) *FDA Fast Track* ERAS-801 for the Treatment of Resectable and Progressive or Recurrent or Elderly Newly Diagnosed IDH Wildtype Grade IV Glioblastoma or Gliosarcoma With an EGFR Amplification or Mutation, ERAS801-SARG Trial	Los Angeles, California Jonsson Comprehensive Cancer Center pnghiemphu@mednet.ucla.edu	https://genomoncology.wetrials.com/v1/NCT07089641
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 + Chr7 gain / Chr10 loss**

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

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TIER 1A THERAPY DETAILS

None

TEST DETAILS

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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

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.

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 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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