Satviki Patidar

AGE: 6 years | **Gender:** Female | **Coupon/UID:** NA / NA

Diagnosis: Glioma

MEDGENOME

MGM1229 : [Combo] Advanced Glioma Panel (FISH, NGS & MGMT Methylation)

Report Details		Specimen Information		Ordering Clinician	
Sample ID / Order ID:	9121184 / 1288741	**Specimen Site:**	Brain	**Clinician:**	Dr. Roopesh Kumar
Collection Date:	NA	**Specimen Received:**	FFPE Tissue Blocks [2]	**Affiliation:**	MGM Healthcare Pvt Ltd - Chennai
Date Received:	7th May 2025	**Specimen Tested:**	1294/25 B	**Serviced By:**	NA
Report Date & Time:	23rd May 2025 17:33 PM	**Tumor Content (%):**	55	**Report Status:**	Final

Clinical Summary: Diffuse low grade glioma with oligoid features

TEST RESULT SUMMARY

Next Generation Sequencing (NGS) Results

POSITIVE

Gene	Findings	Gene	Findings
ALK	Not Detected	*ATRX*	Not Detected
BRAF	**FUSION**	*EGFR*	Not Detected
FGFR1	Not Detected	*FGFR2*	Not Detected
FGFR3	Not Detected	*H3-3A*	Not Detected
IDH1	Not Detected	*IDH2*	Not Detected
MET	Not Detected	*MYB*	Not Detected
MYBL1	Not Detected	*NTRK1*	Not Detected
NTRK2	Not Detected	*NTRK3*	Not Detected
RET	Not Detected	*ROS1*	Not Detected
TERT	Not Detected	*TP53*	Not Detected

Please refer to the complete variant details in the result table in page 2.

Next Generation Sequencing (NGS) Test Result

Result - POSITIVE
CLINICALLY RELEVANT VARIANT/S DETECTED

AMP Classification ^	CDS variant details	Interpretation	Treatment Recommendations	$Treatment Response
***DBN1/BRAF* (FUSION)** Total Read depth- 63x				
Tier I	NA	Oncogenic	Tovorafenib	Effective

^Refer to Glossary section for the classification criteria details.
$Drug Approvals are based on US-FDA Guidelines. Kindly refer to local guidelines if required.

ADDITIONAL BIOMARKERS DETECTED

This section provides information about variants that do not have any therapeutic value. However, these variants may or may not have a likely oncogenic effect.

No other biomarkers that warrants to be reported was detected

ACTIONABLE BIOMARKER DETAILS

DBN1/BRAF (FUSION)

Gene Fusion: *DBN1*:*BRAF*	**5'Gene** *DBN1*	**3'Gene:** *BRAF*
Total Read Depth: 63x	**Ensemble Gene ID:** ENST00000309007	**Ensemble Gene ID:** ENST00000644969
	Exon/Intron: E:12	**Exon/Intron:** E:12
	5'Chromosome Breakpoint (hg38): chr5:177459145:-	**3'Chromosome Breakpoint (hg38):** chr7:140781706:-

Gene Summary 1: The protein encoded by *DBN1* is a cytoplasmic actin-binding protein thought to play a role in the process of neuronal growth. It is a member of the drebrin family of proteins that are developmentally regulated in the brain. A decrease in the amount of this protein in the brain has been implicated as a possible contributing factor in the pathogenesis of memory disturbance in Alzheimer's disease. At least two alternative splice variants encoding different protein isoforms have been described for *DBN1*.

Gene Summary 2: *BRAF* encodes a protein belonging to the RAF family of serine/threonine protein kinases. This protein plays a role in regulating the MAP kinase/ERK signaling pathway, which affects cell division, differentiation, and secretion. Mutations in *BRAF*, most commonly the V600E mutation, are the most frequently identified cancer-causing mutations in melanoma, and have been identified in various other cancers as well, including non-Hodgkin lymphoma, colorectal cancer, thyroid carcinoma, non-small cell lung carcinoma, hairy cell leukemia and adenocarcinoma of lung. Mutations in *BRAF* are also associated with cardiofaciocutaneous, Noonan, and Costello syndromes, which exhibit overlapping phenotypes. A pseudogene of *BRAF* has been identified on the X chromosome.

Clinical and Therapeutic Relevance: The serine/threonine-protein kinase *BRAF* activates the RAS/MAPK signaling pathway to promote cell proliferation and survival. *BRAF* fusion proteins lead to constitutive *BRAF* kinase activity that activates downstream signaling. **The type II RAF inhibitor tovorafenib (tak-580) is indicated for patients 6 months of age and older with relapsed or refractory pLGG harboring a *BRAF* fusion or rearrangement.**The approval was based on a phase II clinical trial (FIREFLY-1, n=77) pediatric LGG patients treated with tovorafenib showed an overall response rate (ORR) of 69% (10 complete response (CR), 31 partial response (PR)) for BRAF-fusion-positive patients, the majority of which harbored the *KIAA1549-BRAF* fusion. *BRAF* fusion proteins lead to constitutive BRAF kinase activity that activates downstream signaling. The tumors of two patients with *BRAF*-mutated melanoma did not respond to treatment with vemurafenib or ulixertinib, respectively. Melanoma xenograft mouse models carrying *BRAF* fusions were resistant to vemurafenib but sensitive to trametinib. Single case reports have described partial response (PR) of lung adenocarcinoma patients harboring *BRAF*-fusions treated with trametinib. Preclinical data confirmed trametinib sensitivity of cell lines carrying *BRAF*-fusion variants. Lung cancer tumors with activating *EGFR* mutations have been described to acquire *BRAF* fusion mutations upon disease progression on osimertinib treatment, indicating a potential resistance mechanism. Introduction of *BRAF* fusion genes into lung cancer cell lines confers resistance to osimertinib. In a case report of a lung adenocarcinoma patient with an activating *EGFR* mutation, the *BRAF-MKRN1* fusion was detected upon developing osimertinib resistance. The patient was treated with the triple combination of osimertinib, trametinib, and dabrafenib and achieved a PR. One spindle cell sarcoma patient with a *SEPT7-BRAF* fusion achieved a PR with trametinib treatment. Another patient with a spindle cell sarcoma harboring an *SNX8-BRAF* fusion was first treated with trametinib and achieved a complete response (CR) with 20 months progression-free survival (PFS). Upon progression, the patient was treated with tovorafenib and also achieved CR with more than 18 months PFS. In a cohort study of 70 pediatric low-grade glioma patients, 30 *BRAF* fusion, 19 *BRAF* V600E mutant, and 21 wild-type were identified. *BRAF* fusion tumors compared with *BRAF* V600E and wild-type tumors were larger (P=.0022), and had a greater mass effect (P=.0053). *BRAF* fusion and *BRAF* V600E mutant pediatric low-grade gliomas have unique imaging features that can be used to differentiate them from each other and wild-type pediatric low-grade gliomas [PMID: 35863783].

DBN1/BRAF (FUSION)

PubMed References: 37978284, 37978284, 37410972, 36217175, 36153311, 35952324, 35672277, 35543076, 34589930, 34476331, 34167970, 34148767, 30831205, 30254212, 29247021, 28278349, 28092667, 26072686, 35863783

AMP-ASCO-CAP CLASSIFICATION CRITERIA

Genetic test results are reported based on the somatic variant classification recommendations of College of American Pathologists (CAP) /American society for Clinical Oncology (ASCO)/Association of Molecular Pathologists (AMP) [PMID: 27993330] as described in the table below:

Tier	Criteria
Tier I	Variants of strong clinical significance.
Tier II	Variants of potential clinical significance.
Tier III	Variants of unknown clinical significance
Tier IV	Benign or likely benign variants

DISCLAIMER

- **Decisions regarding treatment action plans should not be solely based on these test results. These findings are highly recommended to be correlated with the patient's clinical, pathological, radiological and family history for decisions on diagnosis, prognosis, or therapeutics.**
- The therapy information provided in this report is based on FDA approved drugs data, NCCN guidelines, peer reviewed published literature, standard clinical databases, and strength of biomarker results till date. These therapies may or may not be suitable/beneficial to a particular patient. This clinical report summarises potentially effective medications, potentially ineffective medications, and medications that may pose a higher risk of adverse reactions by mapping the patient's genetic alterations to the biomedical reference information. The report may also provide prognostic and diagnostic biomarkers detected or shown for the given disease context.The treatment recommendations for the variants classified in Tier II are not provided.
- The clinical trials information provided in this report is compiled from www.clinicaltrials.gov as per currently available data, however completeness of information provided herein cannot be guaranteed. This information should only be used as a guide and specific eligibility criteria should be reviewed thoroughly for the concerned patient. MedGenome Labs does not guarantee or promise an enrolment in any clinical trials.
- The identification of a genomic biomarker does not necessarily imply pharmacological effectiveness or ineffectiveness. The medications identified by the treating physician may or may not be suitable for use on a particular patient. Thus, the clinical report does not guarantee that any particular agent will be effective in the treatment of any particular condition. Also, the absence of a treatment option does not determine the effectiveness or predict an ineffective or safety-relevant effect of a medication selected by the treating physician.
- The classification and clinically relevant information for the reported variants is based on peer-reviewed publications, public clinical databases, medical guidelines (NCCN, ASCO, AMP) or other publicly available information and it has been ensured that the information provided is up to date at the time of report generated, however continuous updates may happen in public domains. Also, the classification of variants can change based on the updated literature evidence. Re-analysis of the results can be requested at additional cost.
- This test is performed on the patient's tumor sample without a paired blood sample; therefore, it may include variations which may be of germline origin. However, this test is designed and validated for the detection and reporting of somatic genomic variants only and does not discriminate between germline and somatic variants. If clinically warranted, appropriate germline testing and genetic counselling for the patient should be considered for further evaluation.
- Due to poor quality of FFPE tissue blocks, the QC parameters for extracted RNA may not pass to proceed further with the testing, therefore there is a possibility of assay failure at various steps (RNA QC, Library QC, Bioinformatics QC) or compromised results that include low gene coverage and low variant depth. However, sample status in such scenarios shall be sent through mail to the ordering clinician.
- This test has been validated at MedGenome Labs and the limit of detection (LOD) of allele fraction for SNVs and InDels is ≥5% and for fusions is ≥10 spanning reads. However, the report may include, at the discretion of laboratory director, the variants with lower allele burden (3-5%) having strong or potential clinical significance or those have been reported earlier in the patient. Variants with <1% allele

fraction and variants of uncertain significance with <5% allele fraction are not routinely reported. However, possibility of false negative or false positive below the limit of detection of this assay cannot be ruled out.

- Large deletions and deep intronic variations are not detected in this assay.
- Copy Number Variations (CNVs) are based on the RNA expression data using a CNV prediction model developed with control samples. Hence, the chromosome coordinates and size of the CNV can not be determined. It is recommended to confirm the CNVs by alternate methods, such as FISH as the sensitivity of NGS for detecting CNVs is not 100%.
- **Additional case specific disclaimer : None**

TEST DESCRIPTION

The MedGenome's Glioma gene panel is a high throughput next-generation sequencing assay that may provide treatment benefit to the patients. This panel covers key genes related to diagnosis, prognosis and treatment of gliomas (Refer to gene list).

TEST METHODOLOGY

Sample type: FFPE Specimen; A histopathologic review is performed to determine the tumor content in the FFPE block/curls.

Extraction and Library Preparation: Tumor nucleic acid is extracted from FFPE (Formalin fixed) tissue block and used to perform targeted gene capture using a custom hybrid capture kit.

Sequencing: The QC passed libraries are sequenced to a minimum depth of 250X on validated Illumina sequencing platform.

Data Analysis:The sequences are processed using a customized and validated analysis pipeline designed to accurately detect all classes of genomic alterations (SNVs, InDels, CNVs and Fusions).

Variant Annotation and Reporting: The variants are annotated using our in-house annotation pipeline. Reportable genomic alterations and fusions are prioritized, classified, and reported based on AMP-ASCO-CAP guidelines [PMID: 27993330] and NCCN guidelines.

Limit of Detection (LOD): The LOD for SNVs and InDels is 5% Variant allele Frequency (VAF) and for fusions is >10 spanning reads.

The transcript used for clinical reporting generally represents the canonical transcript (according to Ensembl release 99 human gene model), which is usually the longest coding transcript with strong/multiple supporting evidence. However, clinically relevant variants annotated in alternate complete coding transcripts could also be reported. Variants annotated on incomplete, and nonsense mediated decay transcripts are not reported.

§This test is developed, and its performance characteristics is determined by MedGenome Labs Ltd.

GENES ANALYSED

SNVs/InDels						
ATRX	*BRAF*	*TP53*	*IDH1*	*IDH2*	*H3-3A*	*TERT*

CNVs
EGFR

FUSIONS							
RET	*NTRK1*	*NTRK2*	*NTRK3*	*FGFR1*	*FGFR2*	*FGFR3*	*BRAF*
MYB	*MYBL1*	*ALK*	*ROS1*	*MET*			

CLINICAL TRIALS

The following trials are potentially best suited for your patient's indication, considering all reported treatment recommendations. See https://clinicaltrials.gov (clinical trials from NCT) or https://trialsearch.who.int (clinical trials from other registries) for more information.

Clinical trials in total : 0 **Trial countries :** IN-India, US-United States

S.No	Title	Phase and ID	Intervention	Disease	Age & Sex
No Clinical Trials.					

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END OF REPORT

Medgenome Labs Ltd
Sy No 94/1C and 94/2, Tower-1, Veerasandra Village, Attibele Hobli, Electronic City, Phase-1, Bangalore, Karnataka, 560100.

DNA TEST REPORT

Full Name	**Satviki Patidar**	Order ID/Sample ID	**1288741/9121184**
Date of Birth / Age	**6 Years**	Gender	**Female**
Parental Sample ID	**NA**	Sample Type	**FFPE Block & Slide**
Referring Clinician	**Dr. Roopesh Kumar** **MGM Healthcare Pvt Ltd - Chennai (Chennai)**	Block No & Tumor content	**1294/25 B /55%**
		Date & time of Sample Receipt	**07-05-2025, 11:04:00**
		Date & time of Report	**14-05-2025, 17:43:50**
Test Requested	**MGMT gene methylation analysis (Temozolomide Resistance) [MGM207]**		

CLINICAL DIAGNOSIS / SYMPTOMS / HISTORY

Glioma

SCOPE

This assay screens for the promoter methylation status of MGMT gene by real-time PCR technology

RESULTS

This tumor sample [Block ID:1294/25] is NEGATIVE for *MGMT* promoter methylation

Assay Information	
Analysis for : **MGMT gene methylation analysis (Temozolomide resistance) [MGM207]**	Method : **Real Time PCR**
Gene : **O6-methylguanine-DNA methyltransferase (MGMT) Promoter region**	Endogenous Control : **Beta actin (ACTB)**
Result Summary	
MGMT Promoter methylation Status	
Negative	

RESULT AND INTERPRETATION

The amplification signal corresponding to methylated DNA was not detected in the provided clinical sample within the detection limits of real time PCR, while the reference gene ACTB was successfully amplified (Table 1), indicating that the *MGMT* promoter region of this sample is not methylated (negative).

TEST INFORMATION

Glioblastoma is the most common and most aggressive malignant primary brain tumor. While occurring in only two to three cases per 100,000 people in Europe and North America, glioblastoma represents 52% of all functional tissue brain tumor cases and 20% of all intracranial tumors. Prognosis for those diagnosed with glioblastoma is poor, with a median survival time of about 14 months. Patients with glioblastoma can be treated with alkylating agents such as Temador (temozolomide). Epigenetic silencing of the MGMT (O6-methylguanine-DNA methyltransferase) DNA-repair gene by promoter methylation compromises DNA repair and has been associated with longer survival in patients with glioblastoma who receive temozolomide. Determination of promoter methylation of the MGMT gene is being included as a relevant factor of the patient molecular profile.

METHODOLOGY

DNA extracted from FFPE tissue tumor samples was subjected to bisulphite treatment and the bisulphite-modified DNA was used as template for fluorescence-based real time qualitative Methylation-Specific PCR (qMSP). Fluorescence signal will be emitted only when specific primer-probe set detect the methylation region on bisulphite converted DNA. An additional amplification of the ACTB gene is performed as a reference.

DISCLAIMER

- The results of this test are dependent on the tumor content in the tissue sample provided.
- This is not a medical report. It has laboratory test findings that need to be correlated with clinical symptoms and discussed with the referring clinician for any further management.

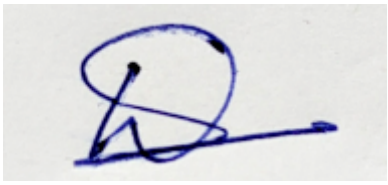
APPENDIX

Table 1: Ct values used for the calculation of the level of methylation of sample and control, showing amplification status of *MGMT* and *ACTB* genes

Sl. No.	Sample	Gene	Ct value
1	Sample (9121184)	MGMT	Undetermined
		ACTB	28.091
2	Control (100% methylated DNA)	MGMT	31.698
		ACTB	27.566
3	Unmethylated Control	MGMT	Undetermined
		ACTB	27.916

REFERENCES

1. Hegi ME, Diserens AC, Gorlia T, Hamou MF, de Tribolet N, Weller M, Kros JM, Hainfellner JA, Mason W, Mariani L, Bromberg JE, Hau P, Mirimanoff RO, Cairncross JG, Janzer RC, Stupp R. MGMT gene silencing and benefit from temozolomide in glioblastoma. N Engl J Med. 2005 Mar 10;352(10):997-1003.
2. Rivera AL1, Pelloski CE, Gilbert MR, Colman H, De La Cruz C, Sulman EP, Bekele BN, Aldape KD. MGMT promoter methylation is predictive of response to radiotherapy and prognostic in the absence of adjuvant alkylating chemotherapy for glioblastoma.Neuro Oncol. 2010 Feb;12(2):116-21. doi: 10.1093/neuonc/nop020. Epub 2009 Dec 14.



Debabrata Mishra

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Dr. Syed Muqlisur Rehman, MD

Molecular Pathologist
KMC Reg No. - 71468

---------- End of Report ----------

MEDGENOME

Sy No 94/1C and 94/2, Tower-1, Veerasandra Village, Attibele Hobli, Electronic City, Phase-1, Bangalore, Karnataka, 560100.

Fluorescent In Situ Hybridisation (FISH) REPORT: FISH for Glioma -1p19q co-deletion (1p3619q13)

Patient Name	Satviki Patidar	**Requesting Clinician**	Dr. Roopesh Kumar
Gender	Female	**Hospital Information**	MGM Healthcare Pvt Ltd - Chennai
Age/Date of Birth	6 Years	**Sample Received**	Two FFPE blocks labelled as 1294/25 A and B. The assay is performed on 1294/25 B.
Sample ID	9121184	**Samples Collected(Date & Time)**	
Order ID(s)	1288741	**Samples Received(Date & Time)**	07-05-2025 11:04 am
Clinical Indication	Low grade Glioma with Oligoid features	**Report Date**	13-05-2025 8:03 pm

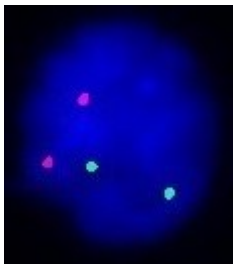
RESULT SUMMARY

Abnormality name	Result	ISCN 2020
1p/19q co-deletion	**Negative**	`nuc ish(TP73x2,ABL2x2)[82/100]/(MANB2x2,CRXx2)[83/100]`

Interpretation:

FISH test is negative for 1p36/19q13 Co-deletion. The observed ratio for 1p36/1q25 is 0.96 and for 19q13/19p13 is 1.0. Kindly correlate with other molecular findings.

DETAILED REPORT



Representative image of a cell showing 2 spectrum green and 2 spectrum orange signals of 1q25 and 1p36 loci respectively.

Representative image of a cell showing 2 spectrum green and 2 spectrum orange signals of 19p13 and 19q13 loci respectively

Spectrum Green (G)	**1q25/TP73, 19p13/MANB2**	**Spectrum Orange (O)**	**1p36/ABL2,19q13/CRX**

Loci analyzed	Ratio calculation	Cut off ratio	Observed ratio	Result
1p36/1q25	**Total Orange Signals / Total Green Signals**	**Negative if >0.9** **Positive if <=0.9**	**0.96**	**Negative**
19q13/19p13	**Total Orange Signals / Total Green Signals**	**Negative if >0.9** **Positive if <=0.9**	**1.00**	**Negative**

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Testing methodology: FISH is a molecular cytogenetic technique used to detect the presence or absence and location of specific gene sequences. FISH involves co-denaturation and hybridization of fluorescent labelled specific DNA probes to target DNA sequence in the interphase cells. Paraffin-embedded tissue specimen should be deparaffinized and pretreated to enhance tissue permeability. The excess unbound probe is removed during post hybridization washes. The sample is stained with DAPI a counter-stain to demarcate the nuclei. Each fluorescent labelled probe that hybridizes to region of interest in interphase cells are visualized as signal using suitable optical filters under Epi fluorescent microscope. 100 interphase cells are counted for each probe manually by two readers. Interpretation of results is done based on the signal patterns observed and the results of the test are reported. Appropriate controls are run in each batch along with the patient samples.

Test was performed using Vysis LSI 1p36/1q25 and 19q13/19p13 probe. In a normal cell hybridized with the LSI 1p36 (Spectrum Orange)/1q25 (Spectrum Green) probe, two orange and two green signals will be observed indicative of two intact copies of chromosome 1. In an abnormal cell with a deletion in the 1p36 region fewer than two orange signals will be observed. In a normal cell hybridized with the LSI 19q13 (Spectrum Orange)/19p13 (Spectrum Green) probe, two orange and two green signals will be observed indicative of two intact copies of chromosome 19. In an abnormal cell with a deletion in the 19q13 region fewer than two orange signals will be observed.

Comments: Among the various candidate biomarkers in gliomas, 1p/19q co-deletion in oligodendroglioma probably constitutes the best-characterized and most extensively investigated marker up to date. The 1p/19q co-deletion is a characteristic and early molecular genetic event in oligodendroglial tumors, and 1p/19q co-deleted tumors are associated with a better prognosis and enhanced response to therapy. The presence of 1p deletion and combined 1p and 19q deletion supports a diagnosis of oligodendroglioma which may indicate that the patient may respond to chemotherapy and radiation therapy. The solitary loss of 19q is not predictive to prolonged survival or response to enhanced chemotherapy. A negative result does not exclude a diagnosis of oligodendroglioma or high-grade astrocytoma.

References:

1. Vysis 1p36/1q25 and 19q13/19p13 probe set kit insert.
2. A. Woehrer . Sander, C. et al. "FISH-based detection of 1p19q co-deletion in oligodendroglial tumors: procedures and protocols for neuropathological practice – a publication under the auspices of the Research Committee of the European Confederation of Neuropathological Societies (Euro-CNS)" Clinical Neuropathology,Vol. 30 – No. 2/2011 (47-55)

Disclaimers:

1. This test was developed and its performance characteristics determined by Medgenome.
2. The finding of this test must be correlated with clinical diagnosis.
3. Genetic changes other than those assayed cannot be ruled out on the basis of this testing.

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