Co-Directors : Aurélie Driss-Corbin / Bénédicte Roquebert
Medical Pathologists

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M. Billet-Delecourt	G. Herbreteau	M. Perret	S. Trombert		
J. Bourgain	A. Ganon	A. Receveur	M. Valduga		

TESTARABIA
TEST
DOB :25.07.1982 Sex : M
File n° : **25T4252345**

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INTERNE GB

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Transmitted by :	LABM TEST7 TEST TEST		
Your references :			$TEST7
Registered :	11.07.2025	Printed on :	11.07.2025
Copies sent to :	Laboratory		

SAMPLE DESCRIPTION

Sample : 11.07.2025 FFPE tissue

Cancer type:	**CNS tumor**
Histological type:	**Glioma**
FFPE block ID:	**25H256389**
Type of sample:	**Surgical sample**
Tumor status:	**Primary tumor**
Anatomical site:	**left temporal lesion**
Percentage of malignant cells:	**90%**
Initial pathology assessment:	**Dr A.... P.....**
Assessment of tumor infiltration:	**Dr A... P....**

Validé par : Dr. M-Magdelaine Coude

THERANOSTIC MUTATIONS (DNAseq NGS)

Sample : 11.07.2025 FFPE tissue

IDH1 gene:	**IDH1 R132C** **[c.394C>T ; p.(Arg132Cys)]** **VAF = 37%**
IDH2 gene:	**No mutation**
Other oncogenic mutations:	**TP53 Y205N** **[c.613T>A ; p.(Tyr205Asn)]** **VAF = 82%**
Non-covered regions:	**None**

Validé par : Dr. M-Magdelaine Coude

UNBALANCED CHROMOSOMAL REARRANGEMENTS (shallow WGS)

Sample : 11.07.2025 FFPE tissue

Chromosomes 1 and 19:

Validé par : Dr. M-Magdelaine Coude

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UNBALANCED CHROMOSOMAL REARRANGEMENTS (shallow WGS)

Sample : 11.07.2025 FFPE tissue

Chromosomes 7 and 10: **No gain of chromosome 7 or loss of chromosome 10**

CDKN2A gene: **PRESENCE OF A CDKN2A DELETION, PROBABLY HOMOZYGOUS**

Other chromosomal rearrangements: **none**

Mean depth: **0.8X**

Validé par : Dr. M-Magdelaine Coude

CONCLUSION

Highlights:

- Presence of an IDH1 R132C activating mutation.
- Presence of a TP53 loss-of-function mutation.
- Presence of a 1p/19q co-deletion.
- Presence of a CDKN2A deletion, probably homozygous.

This tumor genotype leads to the diagnosis of IDH-mutated and 1p/19q-co-deleted oligodendroglioma [1].
The CDKN2A homozygous deletion is indicative of a grade 3 tumor [1].

Targeted therapies:

	Thérapies ciblées
Approved in this indication	None
To discuss in Molecular Tumor Board	ESCAT I-A: - Vorasidenib (IDH1/IDH2 inhibitor) [2]
With a resistance factor	None
Non-eligible treatment (no target)	None

Bibliography:

1. Louis et al. Neuro Oncol. 2021 - PMID: 34185076
2. Mellinghoff et al. N Engl J Med. 2023 - PMID: 37272516

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CONCLUSION

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- ESCAT I: demonstrated clinical benefit (I-A: on prospective randomized trial; I-B: on prospective non-randomized trial; I-C: on basket trial)
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- ESCAT III: clinical benefit in another context (III-A: in another tumor type, or on case reports; III-B: for an alteration having the same effect on a gene as an ESCAT I alteration)
- ESCAT IV: pre-clinical antitumor activity (IV-A: in pre-clinical in vivo or in vitro models; IV-B: predicted in silico)
- ESCAT V: proven antitumor activity, no clinical benefit in prospective trial
(Mateo et al. Ann Oncol. 2018. PMID: 30137196)

Validé par : Dr. M-Magdelaine Coude

TECHNICAL INFORMATION

DNA extraction : Maxwell RSC FFPE Plus DNA kit (AS1720)
RNA extraction : Maxwell RSC RNA FFE kit (AS1440)
RNA retrotranscription: Thermofisher SuperScript IV Vilo kit
Enrichment by capture (Twist Custom panel)
Sequencing: paired-end Element Biosciences AVITI (2x150bp)
Bioinformatics analysis: Life&Soft hg38 reference genome

DNAseq method:

Panel : AKT1 ex 4 (NM_001382430.1); ALK ex 5, 8 15, 18-25, 29 (NM_004304.5); ATRX (NM_000489.6); BRAF ex 11, 15 (NM_ 004333.6); BRCA1 (NM_007294.4); BRCA2(NM_000059.4); CDK4 ex2 (NM_000075.4); CDK6 (NM_ 001145306. 2); CDKN2A (NM_000077.2); CTNNB1 ex 3 (NM_001904. 4); EGFR ex 18-21 (NM_005228.5); ERBB2 ex 8, 17-21 (NM_004448.4); ERBB3 (NM_001982.4); ESR1 ex 1, 5-8 (NM _000125.4); FGFR1 ex 12, 14 (NM_023110.3); FGFR2 ex 7, 9, 12 (NM_000141.5); FGFR3 ex 7, 9, 14,18 (NM_ 000142.5); FOXL2 ex 1 (NM_023067. 4); GNA11 ex 5 (NM_ 002067.5); GNAQ ex 5 (NM_002072.5); GNAS ex 8-9 (NM_000516. 4); H3C2 (NM_003537.4); H3C3 (NM_003531.3); H3F3A ex 2 (NM_ 002107.7); H3F3B ex 2 (NM_ 005324.5); HRAS ex 2-4 (NM_ 005343.4); IDH1 ex 4 (NM_005896. 4); IDH2 ex 4-5 (NM_002168.4); KEAP1 (NM_203500.2); KIT ex 2, 8-15, 17-21 (NM_000222.3); KRAS ex 2-4 (NM_033360.4); MAP2K1 ex 2-3, (NM_002755.4); MET ex 14+ introns, 19 (NM_000245.4); NRAS ex 2-4 (NM_ 002524.5); PDGFRA ex 3-7, 10, 12-15, 18-19, 23 (NM_ 006206. 6); PIK3CA ex 2-3, 5, 8, 10, 21 (NM_006218.4); POLD1 ex 4-12 (NM _002691.4); POLE ex 9-14 (NM_006231.4); PTEN (NM_000314.8); RET ex 10-11, 13, 15-16 (NM_020975.6); SMARCA4 (NM_003072.5); STK11 (NM_000455.4); TERT promoteur (NM_198253.3); TP53 (NM_ 000546.6)
Quality criteria for each region: 100% coverage, at a minimum depth depending on tumor infiltration: >337X (20% malignant cells) ; >223X (30%) ; >166X (40%) ; >131X (50%) ; >108X (60%) ; >92X (70%) ; >80X (80%) ; >70X (90%) Detection of substitutions and small insertion-deletions.
Limit of detection = 2% VAF
Benign variants and variants of unknown significance are not reported.
BRCA1 and BRCA2 genes are investigated on prescription only.

sWGS method:

Shallow Whole Genome Sequencing from unenriched libraries (collected before capture).
Sequencing: paired-end Element Biosciences AVITI (2x150bp) Bioinformatics analysis: Life&Soft. Identification of unbalanced chromosomal rearrangements using the Control-FREEC tool.
Quality criteria: mean depth > 0.5X

Validé par : Dr. M-Magdelaine Coude

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Autorisation de diffusion
Dr. Aurélie Driss Corbin

A. Driss Corbin

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Genetic Biologists
D. Trost

TESTARABIA
TEST
DOB :25.07.1982 Sex : M
File n° : **25T4253546**

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BRAF, V600E MUTATION SCREENING

Sample : 11.07.2025 FFPE tissue

Result :

Mutation BRAF V600E :
POSITIVE detection

Mutation Allele Frequency : 28.0 %

Validated by : Dr. M-Magdelaine Coude

CONCLUSION

Detection of BRAF V600E mutation in this sample.

Test description : Droplet Digital PCR, Biorad QX200- Kit BRAF V600E analytical sensitivity 1%

Validated by : Dr. M-Magdelaine Coude

Autorisation de diffusion
Dr. Aurélie Driss Corbin

A. Driss Corbin

Complete File

In compliance with the French regulation, your biological specimen will be discarded, except for human Genetics and unless you notify otherwise by written to the Customer Department, or it will be anonymized to may be used or transferred, for scientific or quality control purposes

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DOB :25.07.1982 Sex : M
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Registered :	11.07.2025	Printed on :	11.07.2025
Copies sent to :	Laboratory		

SAMPLE DESCRIPTION

Sample : 11.07.2025 FFPE tissue

Cancer type:	**CNS tumor**
Histological type:	**Glioma**
FFPE block ID:	**25H256389**
Type of sample:	**Surgical sample**
Tumor status:	**Primary tumor**
Anatomical site:	**left temporal lesion**
Percentage of malignant cells:	**90%**
Initial pathology assessment:	**Dr A.... P.....**
Assessment of tumor infiltration:	**Dr A... P....**

Validé par : Dr. M-Magdelaine Coude

THERANOSTIC MUTATIONS (DNAseq NGS)

Sample : 11.07.2025 FFPE tissue

IDH1 gene:	**IDH1 R132C** **[c.394C>T ; p.(Arg132Cys)]** **VAF = 37%**
IDH2 gene:	**No mutation**
Other oncogenic mutations:	**TP53 Y205N** **[c.613T>A ; p.(Tyr205Asn)]** **VAF = 82%**
Non-covered regions:	**None**

Validé par : Dr. M-Magdelaine Coude

UNBALANCED CHROMOSOMAL REARRANGEMENTS (shallow WGS)

Sample : 11.07.2025 FFPE tissue

Chromosomes 1 and 19:

Validé par : Dr. M-Magdelaine Coude

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UNBALANCED CHROMOSOMAL REARRANGEMENTS (shallow WGS)

Sample : 11.07.2025 FFPE tissue

Chromosomes 7 and 10: **No gain of chromosome 7 or loss of chromosome 10**

CDKN2A gene: **PRESENCE OF A CDKN2A DELETION, PROBABLY HOMOZYGOUS**

Other chromosomal rearrangements: **none**

Mean depth: **0.8X**

Validé par : Dr. M-Magdelaine Coude

CONCLUSION

Highlights:

- Presence of an IDH1 R132C activating mutation.
- Presence of a TP53 loss-of-function mutation.
- Presence of a 1p/19q co-deletion.
- Presence of a CDKN2A deletion, probably homozygous.

This tumor genotype leads to the diagnosis of IDH-mutated and 1p/19q-co-deleted oligodendroglioma [1].
The CDKN2A homozygous deletion is indicative of a grade 3 tumor [1].

Targeted therapies:

	Thérapies ciblées
Approved in this indication	None
To discuss in Molecular Tumor Board	ESCAT I-A: - Vorasidenib (IDH1/IDH2 inhibitor) [2]
With a resistance factor	None
Non-eligible treatment (no target)	None

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A. Driss Corbin

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DOB :25.07.1982 Sex : M
File n° : **25T4253782**

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GRANDE-BRETAGNE

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Registered :	11.07.2025	Printed on :	11.07.2025
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SAMPLE DESCRIPTION

Sample : 11.07.2025 FFPE tissue

Cancer type:	**CNS tumor**
Histological type:	**Glioma**
FFPE block ID:	**25H256389**
Type of sample:	**Surgical sample**
Tumor status:	**Primary tumor**
Anatomical site:	**left temporal lesion**
Percentage of malignant cells:	**90%**
Initial pathology assessment:	**Dr A.... P.....**
Assessment of tumor infiltration:	**Dr A... P....**

Validé par : Dr. M-Magdelaine Coude

THERANOSTIC MUTATIONS (DNAseq NGS)

Sample : 11.07.2025 FFPE tissue

IDH1 gene:	**IDH1 R132C** **[c.394C>T ; p.(Arg132Cys)]** **VAF = 37%**
IDH2 gene:	**No mutation**
Other oncogenic mutations:	**TP53 Y205N** **[c.613T>A ; p.(Tyr205Asn)]** **VAF = 82%**
Non-covered regions:	**None**

Validé par : Dr. M-Magdelaine Coude

UNBALANCED CHROMOSOMAL REARRANGEMENTS (shallow WGS)

Sample : 11.07.2025 FFPE tissue

Chromosomes 1 and 19:

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Medical Pathologists

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J. Bourgain	A. Ganon	A. Receveur	M. Valduga		

TESTARABIA
TEST
DOB :25.07.1982 Sex : M
File n° : **25T4253782**

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UNBALANCED CHROMOSOMAL REARRANGEMENTS (shallow WGS)

Sample : 11.07.2025 FFPE tissue

Chromosomes 7 and 10: **No gain of chromosome 7 or loss of chromosome 10**

CDKN2A gene: **PRESENCE OF A CDKN2A DELETION, PROBABLY HOMOZYGOUS**

Other chromosomal rearrangements: **none**

Mean depth: **0.8X**

Validé par : Dr. M-Magdelaine Coude

CONCLUSION

Highlights:

- Presence of an IDH1 R132C activating mutation.
- Presence of a TP53 loss-of-function mutation.
- Presence of a 1p/19q co-deletion.
- Presence of a CDKN2A deletion, probably homozygous.

This tumor genotype leads to the diagnosis of IDH-mutated and 1p/19q-co-deleted oligodendroglioma [1].
The CDKN2A homozygous deletion is indicative of a grade 3 tumor [1].

Targeted therapies:

	Thérapies ciblées
Approved in this indication	None
To discuss in Molecular Tumor Board	ESCAT I-A: - Vorasidenib (IDH1/IDH2 inhibitor) [2]
With a resistance factor	None
Non-eligible treatment (no target)	None

Bibliography:

1. Louis et al. Neuro Oncol. 2021 - PMID: 34185076
2. Mellinghoff et al. N Engl J Med. 2023 - PMID: 37272516

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CONCLUSION

ESCAT scale

The ESCAT classification, established by ESMO, assesses the level of evidence of the clinical benefit of a targeted therapy in the presence of a given molecular target.
- ESCAT I: demonstrated clinical benefit (I-A: on prospective randomized trial; I-B: on prospective non-randomized trial; I-C: on basket trial)
- ESCAT II: proven antitumor activity, clinical benefit unknown (II-A: benefit in retrospective study; II-B: clinical response without survival data in prospective trial)
- ESCAT III: clinical benefit in another context (III-A: in another tumor type, or on case reports; III-B: for an alteration having the same effect on a gene as an ESCAT I alteration)
- ESCAT IV: pre-clinical antitumor activity (IV-A: in pre-clinical in vivo or in vitro models; IV-B: predicted in silico)
- ESCAT V: proven antitumor activity, no clinical benefit in prospective trial
(Mateo et al. Ann Oncol. 2018. PMID: 30137196)

Validé par : Dr. M-Magdelaine Coude

TECHNICAL INFORMATION

DNA extraction : Maxwell RSC FFPE Plus DNA kit (AS1720)
RNA extraction : Maxwell RSC RNA FFE kit (AS1440)
RNA retrotranscription: Thermofisher SuperScript IV Vilo kit
Enrichment by capture (Twist Custom panel)
Sequencing: paired-end Element Biosciences AVITI (2x150bp)
Bioinformatics analysis: Life&Soft hg38 reference genome

DNAseq method:

Panel : AKT1 ex 4 (NM_001382430.1); ALK ex 5, 8 15, 18-25, 29 (NM_004304.5); ATRX (NM_000489.6); BRAF ex 11, 15 (NM_ 004333.6); BRCA1 (NM_007294.4); BRCA2(NM_000059.4); CDK4 ex2 (NM_000075.4); CDK6 (NM_ 001145306. 2); CDKN2A (NM_000077.2); CTNNB1 ex 3 (NM_001904. 4); EGFR ex 18-21 (NM_005228.5); ERBB2 ex 8, 17-21 (NM_004448.4); ERBB3 (NM_001982.4); ESR1 ex 1, 5-8 (NM _000125.4); FGFR1 ex 12, 14 (NM_023110.3); FGFR2 ex 7, 9, 12 (NM_000141.5); FGFR3 ex 7, 9, 14,18 (NM_ 000142.5); FOXL2 ex 1 (NM_023067. 4); GNA11 ex 5 (NM_ 002067.5); GNAQ ex 5 (NM_002072.5); GNAS ex 8-9 (NM_000516. 4); H3C2 (NM_003537.4); H3C3 (NM_003531.3); H3F3A ex 2 (NM_ 002107.7); H3F3B ex 2 (NM_ 005324.5); HRAS ex 2-4 (NM_ 005343.4); IDH1 ex 4 (NM_005896. 4); IDH2 ex 4-5 (NM_002168.4); KEAP1 (NM_203500.2); KIT ex 2, 8-15, 17-21 (NM_000222.3); KRAS ex 2-4 (NM_033360.4); MAP2K1 ex 2-3, (NM_002755.4); MET ex 14+ introns, 19 (NM_000245.4); NRAS ex 2-4 (NM_ 002524.5); PDGFRA ex 3-7, 10, 12-15, 18-19, 23 (NM_ 006206. 6); PIK3CA ex 2-3, 5, 8, 10, 21 (NM_006218.4); POLD1 ex 4-12 (NM _002691.4); POLE ex 9-14 (NM_006231.4); PTEN (NM_000314.8); RET ex 10-11, 13, 15-16 (NM_020975.6); SMARCA4 (NM_003072.5); STK11 (NM_000455.4); TERT promoteur (NM_198253.3); TP53 (NM_ 000546.6)
Quality criteria for each region: 100% coverage, at a minimum depth depending on tumor infiltration: >337X (20% malignant cells) ; >223X (30%) ; >166X (40%) ; >131X (50%) ; >108X (60%) ; >92X (70%) ; >80X (80%) ; >70X (90%) Detection of substitutions and small insertion-deletions.
Limit of detection = 2% VAF
Benign variants and variants of unknown significance are not reported.
BRCA1 and BRCA2 genes are investigated on prescription only.

sWGS method:

Shallow Whole Genome Sequencing from unenriched libraries (collected before capture).
Sequencing: paired-end Element Biosciences AVITI (2x150bp) Bioinformatics analysis: Life&Soft. Identification of unbalanced chromosomal rearrangements using the Control-FREEC tool.
Quality criteria: mean depth > 0.5X

Validé par : Dr. M-Magdelaine Coude

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Autorisation de diffusion
Dr. Aurélie Driss Corbin

A. Driss Corbin

Complete File

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TESTARABIA
TEST
DOB :25.07.1982 Sex : M
File n° : **25T4253883**

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GRANDE-BRETAGNE

Transmitted by :	LABM TEST7 TEST TEST		
Your references :			$TEST7
Registered :	11.07.2025	Printed on :	11.07.2025
Copies sent to :	Laboratory		

Sample : 11.07.2025 FFPE tissue

MGMT PROMOTER METHYLATION

SAMPLE INFORMATION

Type de Cancer :
Brain

Type Histologique
Glioma

Numéro bloc :
25H35265B

Anatomic site:
Surgical specimen

Anatomic site:
Primitive

Percentage of tumor cells: 40 %

Résultat :

Statut Hyperméthylation: Positif

Conclusion et interprétation:
Hypermethylation of the proximal region of the MGMT gene promoter was identified in the sample analyzed. This epigenetic modification leads to impaired repair of DNA alkylations, and is associated with sensitivity to alkylating agents (temozolomide).

Assay: determination of MGMT promoter hypermethylation status. DNA Extraction Maxwell AS1720 / kit Diatech EasyPGX® ready MGMT / EasyPGX® qPCR instrumen. MGMT promoter
is categorized as depending on fusion curve interpretation. Due to the sensitivity of the method being used, methylation status cannot be reliably detected in sample containing less than 30% of tumor DNA.

Validated by : Dr. M-Magdelaine Coude

Autorisation de diffusion
Dr. Aurélie Driss Corbin



Complete File

In compliance with the French regulation, your biological specimen will be discarded, except for human Genetics and unless you notify otherwise by written to the Customer Department, or it will be anonymized to may be used or transferred, for scientific or quality control purposes

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