Lab Director : Aurélie Driss-Corbin
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Genetic Biologists
D. Trost

PATIENT_DEMO
ONCO
DOB :01.01.1970 Sex : M
File n° : **26V2109195**

Transmitted by :	LABM TEST7 TEST TEST		
Your references :			$TEST7
Registered :	10.11.2026	Printed on :	10.11.2026
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LABM TEST7 TEST TEST
INTERNE GB

GRANDE-BRETAGNE

SAMPLE DESCRIPTION

Sample : 01.01.2026 FFPE tissue Ambient

Cancer type:
Lung cancer
Histological type:
Adenocarcinoma
Clinical details:
Progressive disease with osimertinib
FFPE block ID:
123456789
Type of sample:
Biopsy
Tumor status:
Primary tumor
Anatomical site:
Left upper lobe
Percentage of malignant cells:
50%
Initial pathology assessment:
DR ANNA PATHE
Assessment of tumor infiltration:
DR ANNA PATHE

Validated by : Dr. Guillaume Herbreteau

THERANOSTIC MUTATIONS (DNAseq NGS)

EGFR gene:
EGFR exon 19 E746_A750del
[c.2235_2249del ; p.(Glu746_Ala750del)]
VAF = 58%

EGFR T790M
[c.2369C>T ; p.(Thr790Met)]
VAF = 43%

BRAF gene:
No mutation

KRAS gene:
No mutation

ERBB2 gene:
No mutation

MET gene:
No mutation

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SELAFA CERBA - 10/12 av. Roland Moréno - ZAC des Epineaux - 95740 Frépillon - France - REGISTRATION N°95.9 -Tel : 33 1 34 40 20 20 - FAX : 33 1 34 40 21 29
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GRANDE-BRETAGNE

STK11 gene:
No mutation

KEAP1 gene:
No mutation

TP53 gene:
TP53 R148C
[c.428C>T ; p.(Arg148Cys)]
VAF = 54%

Other oncogenic mutations:
None

Gene fusions (on DNA):
No detectable ALK, ROS1 or RET fusions (the analysis targets only the most frequent fusions)

Somatic copy number alterations:
MET gene amplification

Non-covered regions:
None

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GENE FUSIONS AND SPLICING DEFECTS (NGS RNAseq)

Fusions and splicing defects:
No gene fusion or splicing defect

Validated by : Dr. Guillaume Herbreteau

CONCLUSION

Highlights:
- Presence of the EGFR activating mutation (exon 19 deletion) and the EGFR T790M resistance mutation identified at diagnosis.
- Presence of a MET amplification.
- Presence of a TP53 loss-of-function mutation.

Tumor genotype consistent with that observed on the left upper lobe biopsy performed on January 1, 2024.

Targeted therapies:

Approved in this indication
ESCAT I-A:
- Amivantamab + carboplatin + pemetrexed [1]

To discuss in Molecular Tumor Board
None

With a resistance factor
REDUCED SENSITIVITY:
- 3rd gen. EGFR inhibitors : MET amplification [2,3]

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GRANDE-BRETAGNE

RESISTANCE:
- 1st and 2nd gen. EGFR inhibitors : EGFR T790M mutation, MET amplification [2,3]

Non-eligible treatment (no target)
- ALK inhibitors
- BRAF inhibitors
- MET inhibitors
- ROS1 inhibitors
- Selective RET inhibitors

Bibliography:
1. Passaro et al. Ann Oncol. 2024 - PMID: 37879444
2. Leonetti et al. Br J Cancer. 2019 - PMID: 31564718
3. Huang et al. Acta Pharm Sin B. 2015 - PMID: 26579470

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)

Validated by : Dr. Guillaume Herbreteau

TECHNICAL INFORMATION

DNA extraction: Maxwell HT FFPE Plus, on Hamilton robot
or 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
Limit of detection = 2% VAF

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.

RNAseq method:
Panel : ALK, BRAF, EGFR, ERBB2, FGFR1, FGFR2, FGFR3, KRAS, MAML2, MET (exon 14 skipping), NRG1, NTRK1, NTRK2, NTRK3, PIK3CA, PPARG, RET, ROS1 (all RefSeq transcripts)
Quality criteria: > 2 million reads.
Detection of gene fusions and exon skipping.
Variant screening is not performed.

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