CARIS® ASSURE

Final Report

Patient
Name:
Date of Birth:
Sex:
Case Number: TN25-
Diagnosis: Non-small cell carcinoma

Specimen Information
Primary Tumor Site: Lung, NOS
Specimen Site:
Specimen ID:
Specimen Collected:
Test Report Date:

Ordered By

Results with Therapy Associations

Biomarker	Results	Therapy Association		Biomarker Level*
EGFR	Pathogenic Variant Exon 21 | p.L858R	BENEFIT	osimertinib	Level 2
EGFR T790M	Pathogenic Variant Exon 20 | p.T790M	BENEFIT	datopotamab deruxtecan	Level 2
			osimertinib	Level 2
			afatinib plus cetuximab (combination)	Level 3
		LACK OF BENEFIT	erlotinib, gefitinib	Level 2
			afatinib, dacomitinib	Level 3

*Level 1: Companion diagnostic (CDx); Level 2: Strong evidence of clinical significance or endorsed by clinical guidelines; Level 3: Potential clinical significance.

Tumor Associated Findings

Biomarker	Protein Change	DNA Change	Variant Frequency	Interpretation
EGFR	**L858R**	**c.2573T>G**	**0.5 %**	**Pathogenic Variant**
EGFR T790M		**c.2369C>T**	**0.3 %**	**Pathogenic Variant**

Other Results

BLOOD TMB (mut/Mb) : 1

For a list of mutations informing TMB, see appendix

MICROSATELLITE INSTABILITY : Not Detected

TUMOR FRACTION : 0.5 %

Analysis included these guideline-recommended biomarkers: ALK, BRAF, EGFR (T790M, etc.), ERBB2 (HER2), KRAS, MET, NTRK1/2/3, RET, ROS1

Incidental Findings* (Pathogenic & Likely Pathogenic Variants)

Incidental Germline Variants

Biomarker	Protein Change	DNA Change	Variant Frequency	Zygosity	Interpretation
None Detected					

Incidental Findings continued on the next page. >

Clonal Hematopoiesis (CH)

Biomarker	Protein Change	DNA Change	Variant Frequency	Interpretation
ATM	-	c.8987+1G>A	0.3 %	Pathogenic Variant

*Incidental findings section reports variants characterized as non-tumor derived. These results are not a replacement for comprehensive germline testing. Incidental germline pathogenic alteration in ACMG-recognized & additional selected cancer genes are reported (see reportable gene list). Negative results do not imply the patient does not harbor a germline mutation. CH refers to mutations in cancer-associated genes in white blood cells (WBC) and not of solid tumor origin. Incidental CH variants are reported but may not comprehensively detect all CH variants. These mutations occur naturally and increase with age or may be smoking- or therapy-related. Although CH is considered a benign state, there is a risk of progression to hematological malignancy and thus appropriate clinical correlation is recommended. Variants characterized as unknown origin, if reported, are likely characterized as high-level CH variants, potential mosaic or germline origin. Categorization of incidental pathogenic and likely pathogenic variants are based on the observed allele frequency in the buffy coat, such that, for the majority of cases: ≥ 30% is germline; 20%-30% is unknown origin; ≤20% is clonal hematopoiesis, though some rare exceptions may exist.

Pharmacogenomics (PGx) – Variants associated with response to anticancer therapies

PGx Gene: DPYD

Gene	DNA Change	Protein Change	Zygosity	CPIC Defined Phenotype	Variant Activity Score
DPYD	No Variants Detected				
DPYD Total Activity Score: 2.0					
DPYD Phenotype:	NORMAL METABOLIZER				

DPYD Genotyping Recommendations, Pratt, VM, et al. 2024 J Mol Diagn 26(10):851-863. CPIC Guideline for Fluoropyrimidines and DPYD: cpicpgx.org/guidelines/guideline-for-fluoropyrimidines-and-DPYD/ DPD, Dihydropyrimidine Dehydrogenase, encoded by the gene DPYD, is involved in reversible reduction of uracil and thymine as well as the chemotherapeutic drug 5-fluorouracil (5-FU). Individuals with decreased DPD activity are less able to break down fluoropyrimidines to inactive metabolites, thereby increasing exposure to active drug moieties. Compromised DPD activity increases an individual's risk of experiencing potentially life-threatening fluorouracil toxicity, including bone marrow suppression, gastrointestinal toxicity, and neurotoxicity. DPYD Phenotypes are defined as: (1) normal metabolizer, total activity score of 2, (2) intermediate metabolizer, total activity score of 1-1.5, (3) poor metabolizer, total activity score 0-0.5.

TRACK Table

Biomarker	Tissue	Plasma 1
EGFR L858R	Detected	0.5 %
EGFR T790M	Not Detected	0.3 %
TP53 R248L	Detected	Not Detected
Tissue TMB	13 mut/MB	Not Assessed
Blood TMB	Not Assessed	1 mut/MB
Microsatellite Instability	Not Detected	Not Detected
Tumor Fraction	Not Assessed	0.5 %

TRACK = Therapy Response and Clonal Kinetics

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Predicted Human Leukocyte Antigen (HLA) Genotype Results

The predicted HLA genotype results are summarized in the table below. Please note that this assay is not a substitute for histocompatibility testing. It is recommended to confirm the patient's HLA type with an appropriate assay.

Gene	Method	Analyte	Genotype
		MHC CLASS I	
HLA-A	Seq	gDNA	A*02:01 , A*02:01
HLA-B	Seq	gDNA	B*35:01 , B*13:02
HLA-C	Seq	gDNA	C*06:02 , C*04:01

gDNA = genomic DNA extracted from the buffy coat
HLA genotypes with only one allele are either homozygous or have loss-of-heterozygosity at that position.

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Specimen Information

Specimen ID:

Specimen Collected:

Specimen Received:

Testing Initiated:

Test Ordered: Caris Assure

Gross Description:

Pathologic Diagnosis:

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Clinical Trials Connector™

The Clinical Trials Connector lists agents that are matched to available clinical trials according to biomarker status. In some instances, older-generation agents may still be relevant in the context of new combination strategies and, therefore, will still appear on this report.

Therapeutic agents listed below may or may not be currently FDA approved for the tumor type tested.

Please see https://clinicaltrials.gov/ for more information.

TARGETED THERAPY CLINICAL TRIALS (31)

Drug Class	Biomarker	Analyte	Investigational Agent(s)
EGFR TKIs (26)	EGFR	DNA-Tumor	WSD0922-FU, osimertinib, TAS6417, aumolertinib, furmonertinib, lazertinib
	EGFR T790M	DNA-Tumor	
Multi-HER-targeted therapy (5)	EGFR T790M	DNA-Tumor	afatinib, BAY2927088, neratinib

( ) = represents the total number of clinical trials identified by the Clinical Trials Connector for the provided drug class or table.

The Clinical Trials Connector may include trials that enroll patients with additional screening of molecular alterations. In some instances, only specific gene variants may be eligible.

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Caris MPI, Inc. d/b/a Caris Life Sciences is certified under the Clinical Laboratory Improvement Amendments (CLIA) as qualified to perform high complexity clinical laboratory testing. Individual assays that are available through Caris molecular profiling include both Laboratory Developed Tests (LDT) and U.S. Food and Drug Administration (FDA) approved or cleared tests. The LDTs were developed, and their performance characteristics determined by Caris.

The LDTs have not been cleared or approved by the U.S. Food and Drug Administration. The FDA has determined that clearance or approval is not necessary for certain laboratory developed tests. Caris LDTs are used for clinical purposes. They are not investigational or for research. Caris' CLIA certification number is located at the bottom of each page of this report.

The information presented in the Clinical Trials Connector™ section of this report, if applicable, is compiled from sources believed to be reliable and current. However, the accuracy and completeness of the information provided herein cannot be guaranteed. The clinical trials information present in the biomarker description was compiled from www.clinicaltrials.gov. The contents are to be used only as a guide, and health care providers should employ their best comprehensive judgment in interpreting this information for a particular patient. Specific eligibility criteria for each clinical trial should be reviewed as additional inclusion criteria may apply.

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Final Report

Mutational Analysis by Tumor and Germline Next Generation Sequencing

BLOOD TUMOR MUTATIONAL BURDEN

Mutations / Megabase: 1

MICROSATELLITE INSTABILITY

Result: Not Detected

Gene	Analyte	Variant Interpretation	Chromosomal Position	Protein Alteration	Exon	DNA Alteration	Variant Frequency %	Transcript ID
EGFR	DNA-Tumor	Pathogenic Variant	chr7 | 55191822	p.L858R	21	c.2573T>G	0.53	NM_005228.4

Interpretation: The oncogenic p.L858R mutation was detected in EGFR exon 21.

EGFR or epidermal growth factor receptor, is a transmembrane receptor tyrosine kinase belonging to the ErbB family of receptors. Upon ligand binding, the activated receptor triggers a series of intracellular pathways (Ras/MAPK, PI3K/Akt, JAK-STAT) that result in cell proliferation, migration and adhesion. EGFR mutations have been observed in 20-25% of non-small cell lung cancer (NSCLC), 10% of endometrial and peritoneal cancers. Somatic gain-of-function EGFR mutations, including in-frame deletions in exon 19 or point mutations in exon 21, confer sensitivity to first- and second-generation tyrosine kinase inhibitors (TKIs), whereas the secondary mutation, T790M in exon 20, confers reduced response. Germline mutations and polymorphisms of EGFR have been associated with familial lung adenocarcinomas.

Gene	Analyte	Variant Interpretation	Chromosomal Position	Protein Alteration	Exon	DNA Alteration	Variant Frequency %	Transcript ID
EGFR T790M	DNA-Tumor	Pathogenic Variant	chr7 | 55181378	p.T790M	20	c.2369C>T	0.26	NM_005228.4

Interpretation: The p.T790M mutation is associated with acquired resistance to first and second-generation EGFR-targeted tyrosine kinase inhibitors.

EGFR or epidermal growth factor receptor, is a transmembrane receptor tyrosine kinase belonging to the ErbB family of receptors. Upon ligand binding, the activated receptor triggers a series of intracellular pathways (Ras/MAPK, PI3K/Akt, JAK-STAT) that result in cell proliferation, migration and adhesion. EGFR mutations have been observed in 20-25% of non-small cell lung cancer (NSCLC), 10% of endometrial and peritoneal cancers. Somatic gain-of-function EGFR mutations, including in-frame deletions in exon 19 or point mutations in exon 21, confer sensitivity to first- and second-generation tyrosine kinase inhibitors (TKIs), whereas the secondary mutation, T790M in exon 20, confers reduced response. Germline mutations and polymorphisms of EGFR have been associated with familial lung adenocarcinomas.

NGS Methods:

Next-generation sequencing analysis of DNA and RNA extracted from plasma and white blood cells (buffy coat) was performed using Illumina NovaSeq 6000 sequencers. A hybrid pull-down panel of baits was used to capture DNA and reverse-transcribed RNA from >20,000 genes. Sequence data was analyzed using a custom bioinformatics pipeline based on genome assembly GRCh38.p13 to detect variants as well as the characterization of the source of genomic findings (e.g. somatic tumor, germline, or non-tumor derived mutations that result from clonal hematopoiesis). The ACMG SF v3.0 (Miller, et al. (2021) Genet Med 23(8):1381-1390) guidelines were followed for reporting of alterations identified in cancer-associated genes determined to have originated from the patient's germline. This assay cannot detect all likely pathogenic or pathogenic germline alterations nor all carriers of germline cancer predisposition alterations. A positive germline or suspected germline result is an indication that the patient may be predisposed to cancer and patients may consider further independent testing, consult with their physician, or obtain genetic counseling.

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Blood TMB Variants

Biomarker	Protein Change	Biomarker	Protein Change	Biomarker	Protein Change	Biomarker	Protein Change
AMER3	R519fs	GP1BA	S441 _T453dup	MUC19	T3905A	SEMA4G	G766dup
BAHCC1	D998fs	INSM1	G186fs	MYO16	S1590fs	SF3B2	P105fs
CELSR2	S2284R	ISYNA1	R247fs	MYO5B	V683L	SYN2	S48W
CIC	Q1593fs	KLHDC7B	A596fs	PRB3	P114 _P115 ins21	TBC1D17	A607fs
EMX2	P79fs	MAP3K10	S783fs	PRDM16	L925fs	TMEM8B	A20fs
EVL	P189dup	MUC19	K3901E	PRDM9	W675R	UTF1	A217fs

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Genes Reported:

This test assays for variants in >20,000 genes (exome). To provide a succinct report, clinical reporting focuses on ~290 genes with known clinical associations with cancer therapies, prognosis, or etiology. Of note, calculation of blood tumor mutational burden (bTMB) utilizes the entire exome. Alterations reported include single nucleotide variants (SNVs) and insertion/deletions (indels) in all ~300 genes and reports other variant types in certain genes as indicated by the following: incidental germline variants (**bolded**), fusion events (🌀), amplifications (▲) and deletions (●).

ABL1	CALR	DDR2	FGFR3 🌀	KMT2A	NKX2-1	PRKACA	**SMARCA4**
ACD	CARD11	DGCR8	FGFR4	KMT2C	NOTCH1	**PRKAR1A**	**SMARCB1**
ACVR1	CASP8	**DICER1**	**FH**	KRAS ▲	NOTCH2	PRKD1	**SMARCE1**
ACVR1B	CBFB	DNMT3A	**FLCN**	**LZTR1**	NOTCH3	PRKDC	SMO
AIP	CBL	EED	FLT1	MAP2K1 (MEK1)	NPM1	**PTCH1**	SOCS1
AJUBA	CCDC6	**EGFR** ▲	FLT3	MAP2K2 (MEK2)	NRAS	**PTEN** ●	SOS1
AKT1	CCND1 ▲	EIF1AX	FLT4	MAP2K4	NRG1 🌀	PTPN11	SOX9
AKT2 ▲	CCNE1 ▲	ELF3	FOXA1	MAP3K1	NSD1	PTPRD	SPEN
AKT3	CCND2	ELOC	FOXL2	MAPK1	NSD2	RABL3	SPOP
ALK 🌀	CCND3 ▲	EP300	FUBP1	**MAX**	NT5C2	RAC1	SRC
AMER1	CD274 ●	EPHA2	GATA3	MDH2	**NTHL1**	RAD50	STAG2
AR	CD79B	ERBB2 ▲	GNA11	MDM2 ▲	NTRK1 🌀	RAD51B	STAT3
APC	**CDC73**	ERBB3	GNA13	MED12	NTRK2 🌀	**RAD51C**	STAT5B
ARAF	**CDH1**	ERBB4	GNAQ	MEF2B	NTRK3 🌀	**RAD51D**	**STK11**
ARHGAP35	CDK12	ERCC2	GNAS	**MEN1**	NYNRIN	RAD54L	**SUFU**
ARID1A	**CDK4** ▲	ERCC5 ●	GRIN2A	**MET** ▲	**PALB2**	RAF1	SUZ12
ARID2	CDK6 ▲	ERCC6 ●	GRM3	MGA	PARP1	RASA1	TCF7L2
ASXL1	CDKN1A	ERG 🌀	H3F3A	**MITF**	PBRM1	**RB1**	TERT
ATM	**CDKN1B**	ESR1	H3F3B	**MLH1**	**PDGFRA**	RBM10	TET2
ATP6AP1	**CDKN2A** ●	EXT1	HIST1H3B	MLH3	PDGFRB	**RET** ▲ 🌀	TGFBR1
ATP6AP2	CDKN2B ●	EZH2	HNF1A	MPL	PHOX2B	RHEB	TGFBR2
ATR	CEBPA	FANCA	**HOXB13**	MRE11	PIK3CA ▲	RHOA	**TMEM127**
ATRX	CHEK1	FANCB	HRAS	**MSH2**	PIK3CB	RIT1	TNFAIP3
AXIN1	**CHEK2**	FANCC	IDH1	**MSH3**	PIK3R1	RNF43	TNFRSF14
AXIN2	CIC	FANCD2	IDH2	**MSH6**	PIK3R2	ROS1 🌀	**TP53**
B2M	CNOT3	FANCE	IGF1R ▲	MTAP ●	PIM1	RRAS2	TRAF3
BAP1	COL2A1	FANCF	IRF4	MTOR	PLCB4	RUNX1	TRAF7
BARD1	CREBBP	FANCG	JAK1	**MUTYH**	**PMS2**	**SDHA**	TRIM28
BCL2	CSF1R	FANCI	JAK2 ●	MYC ▲	**POLD1**	**SDHAF2**	TRRAP
BCL9	CSF3R	FANCL	JAK3	MYCN	**POLE**	**SDHB**	**TSC1**
BCOR	CTCF	FANCM	KBTBD4	MYD88	**POT1**	**SDHC**	**TSC2**
BLM	**CTNNA1**	FAS	KDM5C	MYOD1	PPARG	**SDHD**	U2AF1
BMPR1A	CTNNB1	FAT1	KDM6A	NBN	PPM1D	SETD2	**VHL**
BRAF 🌀	CUL3	FBXW7	KDR	**NF1**	PPP2R1A	SF3B1	WRN
BRCA1	CXCR4	FGF3 ▲	KEAP1	**NF2**	PPP2R2A	SMAD2	**WT1**
BRCA2	CYLD	FGF4 ▲	**KIT**	NFE2L2	PPP6C	**SMAD4**	XPO1
BRIP1	CYSLTR2	FGFR1 ▲ 🌀	KLF4	NFKBIA	PRDM1	SMARCA2	XRCC1
BTK	DACH1	FGFR2 ▲ 🌀	KMT2D	NFKBIE	PREX2		

Additional Biomarkers:

Blood Tumor Mutational Burden (bTMB, mutations/Mb)
Microsatellite Instability
Human Leukocyte Antigen (HLA) genotypes for HLA-A, HLA-B and HLA-C
Tumor Fraction
Pharmacogenomics (PGx) (when applicable): DPYD, Tier 1 variants reported (NM_000110.4, c.1905+1G>A, c.1679T>G, c.[1129-5923C>G ; 1236G>A] (Only c.1236G>A is directly tested. c.1129-5923C>G causes reduced DPYD function and has not been reported to occur without c.1236G>A in CIS; false negative results are possible, but would be extremely rare), c.557A>G, c.868A>G, c.2279C>T, c.2846A>T)

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References

#	Drug	Biomarker	Reference
1	osimertinib	EGFR	Ramalingam, S.S., P.A., Janne, et al. (2017). "Osimertinib As First-Line Treatment of EGFR Mutation-Positive Advanced Non-SmallCell Lung Cancer." J Clin Oncol . doi: 10.1200/JCO.2017.74.7576. View Citation Online
2	osimertinib	EGFR	Soria, J.-C., S.S. Ramalingam, et al. (2017). "Osimertinib in Untreated EGFR-Mutated Advanced Non-Small Cell Lung Cancer." N Eng J Med. doi:10.1056/NEJMoa1713137. View Citation Online
3	afatinib, dacomitinib	EGFR T790M	Sequist, L.V., M. Schuler, et al. (2013). "Phase III Study of Afatinib or Cisplatin Plus Pemetrexed in Patients with Metastatic Lung Adenocarcinoma With EGFR Mutations." J Clin Oncol ahead of print July 1, 2013, doi: 10.1200/JCO.2012.44.2806 View Citation Online
4	afatinib plus cetuximab (combination)	EGFR T790M	Janjigian, Y.Y., W. Pao, et al. (2014) "Dual inhibition of EGFR with afatinib and cetuximab in kinase inhibitor-resistant EGFR-mutant lung cancer with and without T790M mutations." Cancer Discov, 4(9):1036-45. View Citation Online
5	datopotamab deruxtecan	EGFR T790M	Ahn, M., I. Okamoto, et al., (2025). "A pooled analysis of datopotamab deruxtecan in patients with EGFR-mutated NSCLC." J Thorac Oncol. Published online June 12, 2025. View Citation Online
6	datopotamab deruxtecan	EGFR T790M	Sands, J., L. Paz-Ares, et al. (2025). "Datopotamab Deruxtecan in Advanced or Metastatic Non-Small Cell Lung Cancer With Actionable Genomic Alterations: Results From the Phase II TROPION-Lung05 Study." J Clin Oncol 43(10):1254-1265. View Citation Online
7	erlotinib, gefitinib	EGFR T790M	Balak, M.N., W. Pao, et. al. (2006). "Novel D761Y and common secondary T790M mutations in epidermal growth factor receptor-mutant lung adenocarcinomas with acquired resistance to kinase inhibitors." Clin. Cancer Res. 12(21):6494-6501. View Citation Online
8	erlotinib, gefitinib	EGFR T790M	Chen, H.J., Y.L. Wu, et. al. (2009). "Clinicopathologic and molecular features of epidermal growth factor receptor T790M mutation and c-MET amplification in tyrosine kinase inhibitor-resistant Chinese non-small cell lung cancer." Pathol. Oncol. Res. DOI 10.1007/s12253-009-9167-8. View Citation Online
9	erlotinib, gefitinib	EGFR T790M	Lindeman, N.I., M. Ladanyi, et al. (2013) "Molecular testing guideline for selection of lung cancer patients for EGFR and ALK tyrosine kinase inhibitors: guideline from the College of American Pathologists, International Association for the Study of Lung Cancer, and Association for Molecular Pathology." Arch Pathol Lab Med, 137(6):828-60 View Citation Online
10	erlotinib, gefitinib	EGFR T790M	Su, K.Y., P.C. Yang, et. al. (2012). "Pretreatment epidermal growth factor receptor (EGFR) T790M mutation predicts shorter EGFR tyrosine kinase inhibitor response duration in patients with non-small-cell lung cancer." J. Clin. Oncol. 30:433-440. View Citation Online
11	osimertinib	EGFR T790M	Goss, G., T. Mitsudomi, et al. (2016). "Osimertinib for pretreated EGFR Thr790Met-positive advanced non-small-cell lung cancer (AURA2): a multicentre, open-label, single-arm, phase 2 study." Lancet Oncol 17(12):1643-1652. View Citation Online
12	osimertinib	EGFR T790M	Hunger, K.A., T.F. Burns, et al. (2017). "First-Line Osimertinib in Patients with Treatment-Naive Somatic or Germline EGFR T790M-Mutant Metastatic NSCLC." J Thorac Oncol 13(1):e3-35. View Citation Online
13	osimertinib	EGFR T790M	Mok, T.S., AURA3 investigators, et al. (2017) "Osimertinib or Platinum-Pemetrexed in EGFR T790M-Positive Lung Cancer." View Citation Online

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