OncoSHARE™ by OncoDNA

Результат OncoSELECT

Дані пацієнта
Дата народження:

Стать:

Клінічні дані:
Клінічний діагноз:

Гістологічний діагноз:

Зразок:
Локалізація первинної пухлини:

Дата забору:

КЛЮЧОВІ ГЕНЕТИЧНІ ЗМІНИ*
не виявлено

ГЕНОМНІ ПОКАЗНИКИ
не виявлено

ДОДАТКОВІ МАРКЕРИ
не виявлено

Клінічно релевантні гени, у яких не виявлено змін, придатних для таргетної терапії**
не виявлено

*Наведені тільки патогенні і ймовірно патогенні варіанти. Повний список виявлених варіантів наведено в оригіналі репорту.
**Специфічний для певного типу раку

ТЕРАПІЇ, АСОЦІЙОВАНІ З КЛІНІЧНОЮ КОРИСТЮ
не виявлено

РЕЗЮМЕ ДОСЛІДЖЕННЯ

Патогенних або ймовірно патогенних варіантів не виявлено.
Примітка: Звертаємо увагу, що наведені висновки ґрунтуються на аналізі циркулюючої пухлинної ДНК (ctDNA), виділеної з наданого зразка крові (рідинна біопсія). На підставі цього аналізу неможливо гарантувати наявність у крові ctDNA, що несе мутацію, раніше виявлену у зразку солідної пухлини.

ТЕРАПІЇ (ПОВНИЙ ПЕРЕЛІК)
не виявлено

ОПИС ВАРІАНТІВ ІЗ ТЕРАПЕВТИЧНИМ ЗНАЧЕННЯМ

Ген	Категор.	Екзон	Частота/№ копій	Зміна ДНК	Зміна білку	Терапевтичні препарати	
						ген	пацієнт
TP53	SNV	8	39.26%	NM_000546.6:c.818G>A	p.(R273H)	-	-

Біологічний вплив: Варіант R273H патогенний; підвищує проліферацію та виживаність клітин, сприяє міграції та інвазії. Дослідження на лабораторних мишах показали більшу частоту карцином.
Терапевтичний вплив (Tier III): Немає затверджених таргетних терапій; часто асоціюється з резистентністю до хіміотерапії; досліджуються нові підходи та WEE1-інгібітори.
Випадкове виявлення: Не пов’язаний зі спадковими захворюваннями.

СПИСОК ПРЕПАРАТІВ ТЕРАПІЇ (ПОВНИЙ СПИСОК)

Ген	Категор.	Частота/ № копій	Зміна ДНК	Зміна білку	Біол. Категор.	Терап. Категор.	Потенц. Спадк.	Глибина прочит.
TP53	SNV	39.26%	NM_000546.6:c.818G>A	p.(R273H)	Патогенний	Tier III	Hi	2797
ARID1A	DEL	1.10%	NM_006015.6:c.3999_4001del	p.(Q1334del)	VUS	Tier III	Hi	3362
ARID1A	SNV	0.32%	NM_006015.6:c.4003C>G	p.(R1335G)	VUS	Tier III	Hi	3087
CDKN2A	SNV	0.69%	NM_001195132.2:c.35C>T	p.(S12L)	VUS	Tier III	Hi	2879
CHEK2	SNV	0.48%	NM_001005735.2:c.420G>C	p.(R140S)	VUS	Tier III	Hi	2892
CHEK2	SNV	0.60%	NM_001005735.2:c.410G>C	p.(R137T)	VUS	Tier III	Hi	2999
CHEK2	SNV	0.43%	NM_001005735.2:c.397_399delinsGTA	p.(S133V)	VUS	Tier III	Hi	3747
DICER1	SNV	0.39%	NM_001291628.2:c.4263T>G	p.(D1421E)	VUS	Tier III	Hi	2844
MET	SNV	0.29%	NM_000245.4:c.2584-1G>A	-	VUS	Tier III	Hi	2095
NF1	SNV	0.26%	NM_001042492.3:c.1094C>T	p.(S365L)	VUS	Tier III	Hi	1162
PALB2	SNV	47.73%	NM_024675.4:c.2653C>A	p.(P885T)	VUS	Tier III	Hi	2160
PTEN	SNV	1.18%	NM_000314.8:c.804C>A	p.(D268E)	VUS	Tier III	Hi	1016
PTEN	SNV	1.34%	NM_000314.8:c.810G>T	p.(M270I)	VUS	Tier III	Hi	1048
PTEN	SNV	0.75%	NM_000314.8:c.814C>T	p.(H272Y)	VUS	Tier III	Hi	1065
TERT	SNV	1.15%	NM_198253.3:c.-79-1858	-	VUS	Tier III	Hi	1397

СПИСОК ПРЕПАРАТІВ ТЕРАПІЇ (ПОВНИЙ СПИСОК)

Ген	Категор.	Частота/ № копій	Зміна ДНК	Зміна білку	Біол. Категор.	Терап. Категор.	Потенц. Спадк.	Глибина прочит.
TERT	SNV	0.52%	NM_198253.3:c.-79-1803G>A	-	VUS	Tier III	Hi	2709
TERT	SNV	0.88%	NM_198253.3:c.-79-2658A>C	-	VUS	Tier III	Hi	1025
TERT	SNV	0.26%	NM_198253.3:c.-79-1852C>T	-	VUS	Tier III	Hi	1557
TERT	SNV	1.14%	NM_198253.3:c.-79-2693T>C	-	VUS	Tier III	Hi	703
TERT	SNV	1.71%	NM_198253.3:c.-79-2741T>A	-	VUS	Tier III	Hi	821
TERT	SNV	0.42%	NM_198253.3:c.-79-2755A>G	-	VUS	Tier III	Hi	959
TERT	SNV	1.83%	NM_198253.3:c.-79-2770G>C	-	VUS	Tier III	Hi	1149
TERT	SNV	0.64%	NM_198253.3:c.-79-2777A>G	-	VUS	Tier III	Hi	1256
TERT	SNV	0.57%	NM_198253.3:c.-79-2788T>C	-	VUS	Tier III	Hi	1409
TERT	SNV	1.49%	NM_198253.3:c.-79-2793T>C	-	VUS	Tier III	Hi	1541
TERT	SNV	0.90%	NM_198253.3:c.-79-2794G>A	-	VUS	Tier III	Hi	1553
TERT	SNV	0.55%	NM_198253.3:c.-79-2777A>G	-	VUS	Tier III	Hi	1651
TERT	SNV	0.63%	NM_198253.3:c.-79-2788T>C	-	VUS	Tier III	Hi	1915
TERT	SNV	0.73%	NM_198253.3:c.-79-2793T>C	-	VUS	Tier III	Hi	1931
TERT	SNV	0.98%	NM_198253.3:c.-79-2794G>A	-	VUS	Tier III	Hi	713

OncoSHARE™ by OncoDNA

OncoSELECT Analysis Report

Patient
ID:
Date of Birth:

Sex:
Cancer Type:

Clinical
Medical Doctor:

Clinical Diagnosis:

Sample
Primary Tumor Site:
Collection Date:

KEY GENOMIC ALTERATIONS*

None

GENOMIC SIGNATURES

None

ADDITIONAL BIOMARKERS

None

RELEVANT GENES WITH NO ACTIONABLE ALTERATIONS**

None

* Only variants classified as pathogenic and likely pathogenic are reported here. The full list of identified variants is available in the report.

** Cancer type specific

THERAPIES ASSOCIATED WITH CLINICAL BENEFIT

None

CLINICAL TRIALS

None

COMPREHENSIVE SUMMARY

We didn't identify any pathogenic nor likely pathogenic variant.

Rmk: Please note that our conclusions are based on the analysis of the ctDNA extracted from the blood biopsy provided. Based on this analysis, we cannot guarantee that ctDNA carrying a mutation previously detected in the solid tumor is released into the blood.

THERAPIES (FULL LIST)

None

ACTIONABLE VARIANTS DESCRIPTION

Gene	Cat.	Exon	Var. Freq. / Copy Nb	cDNA	AA	Drugs related	
						to gene	to patient
TP53	SNV	8	39.26%	NM_000546.6:c.818G>A	p.(R273H)		

BIOLOGICAL IMPACT: PATHOGENIC
This variant significantly alters the target DNA sequence without causing structural distortions, thus maintaining the ability to bind DNA. In vivo studies in mice with an engineered R273H variant in the endogenous locus (p53R273H/−) demonstrated no difference in survival as compared to p53−/− mice. However, the p53R273H/− mice develop more carcinomas. It remains unclear, how R273H p53 mediates broad chemoresistance or resistance to a particular class of DNA damaging agents. Interaction of R273H p53 with MRE11 prevents the DNA damage protein complex from being recruited, thereby promoting genomic instability and contributing to the oncogenic phenotype. The biological impacts of this vari-

ant seems to increase proliferation and survival. (PMID: 15607980 ; PMID: 19815500). It has also been reported that this variant results in decreased activation of Tp53 target gene expression, and also confers a gain of function to Tp53, resulting in aberrant transcriptional activation and increased cell migration (PMID:22114072; PMID:14743206). Interaction of this variant and BCAR1 in the nucleus has been reported to play an important role in enhancing cancer cell invasion *in vitro* (PMID: 33144694).

THERAPEUTICAL IMPACT: TIER III

At present, there are no approved targeted therapies for TP53 mutations, despite their high prevalence in cancer. Moreover, since many conventional anticancer agents (for example cisplatin and doxorubicin) induce DNA damage that triggers a p53 response, variant of TP53 is often associated with enhanced resistance to conventional chemotherapy (PMID:8673929, PMID:10102818). Therefore, the clinical benefit of chemotherapies might not be as strong as expected.

About 8% of TP53 mutations are nonsense mutations that lead to the expression of a truncated and inactive p53 protein. Currently, different approaches to restore nonsense TP53 mutations are being developed. The most promising one consists in inducing readthrough of premature stop codons in nonsense mutants. Aminoglycosides such as gentamicin and G418 have been shown to induce readthrough of the c.637C>T (p.R213*) mutant p53 and expression of full–length p53 (PMID:21149266). However, clinical use of these drugs is limited by their toxicity. Other new therapies are being developed to target tumors expressing mutant p53, such as ALT 801 and adenovirus–p53 transduced dendritic cell vaccine (PMID:21994418; PMID:24387333).

Further, TP53 mutations have been shown to be sensitive to WEE1 inhibitors (PMID:25125259, http://ascopubs.org/doi/abs/10.1200/jco.2015.33.15_suppl.5506).

INCIDENTAL FINDING

This variant has not been associated with any inherited disease.

DETECTED VARIANTS LIST

Gene	Cat.	Var. Freq. / Copy Nb	cDNA	AA	Biological Impact	Therapeutical Impact	Incidental Findings	Depth
TP53	SNV	39.26%	NM_000546.6:c.818G>A	p.(R273H)	Pathogenic	Tier III	NO	2797
ARID1A	DEL	1.10%	NM_006015.6:c.3999_4001del	p.(Q1334del)	VUS	Tier III	NO	3362
ARID1A	SNV	0.32%	NM_006015.6:c.4003C>G	p.(R1335G)	VUS	Tier III	NO	3087
CDKN2A	SNV	0.69%	NM_001195132.2:c.35C>T	p.(S12L)	VUS	Tier III	NO	2879
CHEK2	SNV	0.48%	NM_001005735.2:c.420G>C	p.(R140S)	VUS	Tier III	NO	2892
CHEK2	SNV	0.60%	NM_001005735.2:c.410G>C	p.(R137T)	VUS	Tier III	NO	2999
CHEK2	SNV	0.43%	NM_001005735.2:c.397_399delinsGTA	p.(S133V)	VUS	Tier III	NO	3747
DICER1	SNV	0.39%	NM_001291628.2:c.4263T>G	p.(D1421E)	VUS	Tier III	NO	2844
MET	SNV	0.29%	NM_000245.4:c.2584-1G>A	-	VUS	Tier III	NO	2095

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Gene	Cat.	Var. Freq. / Copy Nb	cDNA	AA	Biological Impact	Therapeutical Impact	Incidental Findings	Depth
NF1	SNV	0.26%	NM_001042492.3:c.1094C>T	p.(S365L)	VUS	Tier III	NO	1162
PALB2	SNV	47.73%	NM_024675.4:c.2653C>A	p.(P885T)	VUS	Tier III	NO	2160
PTEN	SNV	1.18%	NM_000314.8:c.804C>A	p.(D268E)	VUS	Tier III	NO	1016
PTEN	SNV	1.34%	NM_000314.8:c.810G>T	p.(M270I)	VUS	Tier III	NO	1048
PTEN	SNV	0.75%	NM_000314.8:c.814C>T	p.(H272Y)	VUS	Tier III	NO	1065
TERT	SNV	1.15%	NM_198253.3:c.-79-1858T>C	-	VUS	Tier III	NO	1397
TERT	SNV	0.52%	NM_198253.3:c.-79-1803G>A	-	VUS	Tier III	NO	2709
TERT	SNV	0.88%	NM_198253.3:c.-79-2658A>C	-	VUS	Tier III	NO	1025
TERT	SNV	0.26%	NM_198253.3:c.-79-1852C>T	-	VUS	Tier III	NO	1557
TERT	SNV	1.14%	NM_198253.3:c.-79-2693T>C	-	VUS	Tier III	NO	703
TERT	SNV	1.71%	NM_198253.3:c.-79-2741T>A	-	VUS	Tier III	NO	821
TERT	SNV	0.42%	NM_198253.3:c.-79-2755A>G	-	VUS	Tier III	NO	959
TERT	SNV	1.83%	NM_198253.3:c.-79-2770G>C	-	VUS	Tier III	NO	1149
TERT	SNV	0.64%	NM_198253.3:c.-79-2777A>G	-	VUS	Tier III	NO	1256
TERT	SNV	0.57%	NM_198253.3:c.-79-2788T>C	-	VUS	Tier III	NO	1409
TERT	SNV	1.49%	NM_198253.3:c.-79-2793T>C	-	VUS	Tier III	NO	1541
TERT	SNV	0.90%	NM_198253.3:c.-79-2794G>A	-	VUS	Tier III	NO	1553

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Gene	Cat.	Var. Freq. / Copy Nb	cDNA	AA	Biological Impact	Therapeutical Impact	Incidental Findings	Depth
TERT	SNV	0.55%	NM_198253.3:c.-79-2800A>G	-	VUS	Tier III	NO	1651
TERT	SNV	0.63%	NM_198253.3:c.-79-2818T>C	-	VUS	Tier III	NO	1915
TERT	SNV	0.73%	NM_198253.3:c.-79-2819A>G	-	VUS	Tier III	NO	1931
TERT	SNV	0.98%	NM_198253.3:c.-79-2689C>G	-	VUS	Tier III	NO	713

CLINICAL FORM

Date informed consent given/signed	Jan, 09 2026
Initial diagnosis date	Aug, 13 2021
Clinical diagnosis	Lung adenocarcinoma
Primary tumour site	Lung
Known metastatic sites	Yes
Date of biopsy/surgery or blood withdrawal	Jan, 09 2026
Sample type(s)	Blood
Histological diagnosis	Lung adenocarcinoma
TNM known ?	Yes
Biomarkers tested	Yes
Is the tissue sample sent for molecular diagnostics the one used for the diagnosis (detailed above) ?	Unmentioned
Sample site	Metastasis
Does patient have comorbidities ?	Yes
Has the patient previously undergone organ cancer surgery?	No
Is patient currently receiving a cancer therapy ?	No
Known previous cancer therapies	Yes
Does the patient have a previous history of cancer?	No
ECOG	-
Smoking status	-
Alcohol consumption	-
Comments	-

PROCESS

IPG is the biggest Belgian anatomopathology laboratory and is among the biggest laboratories of its kind in Europe with headquarters in Gosselies and a large section in Brussels. It has a total workspace of 285 people, among whom medical specialists including 20 pathologists and 8 geneticists, 10 clinical biologists and highly skilled technicians. It was one of the first companies to implement a high degree of integration of anatomic pathology data and molecular genetics. The ability to integrate pathological data and molecular biology is not common and is an asset for the products provided by OncoDNA.

All the technical processes including the pathology QC check are performed by the Institute of Pathology and Genetics (IPG) which is ISO15189 accredited (ISO15189:2012 Medical Laboratories – Requirements for Quality and Competence) since the 6th October 2009 by BELAC, an ILAC MRA signatory. The quality of raw data is validated by OncoDNA before any further interpretation.

OncoDNA is compliant with the Guideline for Good Practices of the International Conference on Harmonization (ICH GCP E6 R2) and certified ISO/IEC 27001:2013 (Requirement for Information Security Management Systems) since the 23rd November 2018 by the European Certification Accredity Body ICTS – International Certifcation Trust Services.

REPORT

Please keep in mind that this summary is not the complete report and is to be printed only for archiving purposes.
For more information, please see the dynamic version of the report displayed on oncoshare.oncokdm.com.
This report has been generated and validated on **January, 27 2026**

DISCLAIMER

Although reports can be kept in the patient's medical file, the reports do not constitute and are not intended to replace independent medical judgment and advice. The information and drug recommendations contained in the reports are intended solely for the general information of the medical doctor. Reports are not to be used "as is" for treatment purposes. The information presented in the reports is not intended to replace professional medical care. The information contained in the reports is neither intended to dictate what constitutes reasonable, appropriate or best care for any given health issue, nor is it intended to be used as a substitute for the independent judgment of the medical doctor for any given health issue. The reports merely constitute one element among all applicable information concerning the patient's condition (such as patient and family history, physical examinations, information from other diagnostic tests, and patient preferences) to assist medical doctors in the determination or adaptation of the patients' medical treatment. Treatment decisions remain the exclusive responsibility of the medical doctor. The medical doctor solely and exclusively decides whether (and to what extent) to take into consideration the reports with respect to his/her patient's treatment.

Consequently, ONCODNA (including any of its subsidiaries or affiliates) assumes no liability whatsoever as to the possible consequences of the decision of the medical doctor to follow or not the (content of) the reports. By accepting the terms and conditions of this service and – where applicable –by signing or otherwise consenting to the ICF, the client, the medical doctor and patient expressly declare and acknowledge having understood and agreed to ONCODNA's exclusion of liability.

As science changes rapidly, our proprietary database is continuously updated. Please note that depending on updates, minor discrepancies may occur and especially when, for various reasons, the reports are republished.

MEDICAL VALIDATION

This report has been reviewed and validated by a certified pathologist in accordance with current clinical quality standards.

The signature below confirms the accuracy of the analysis, the interpretation of results, and their compliance with applicable clinical and regulatory guidelines.

Pathologist's name: Dr. Pierre Lefesvre
Signature: