

**Patient**  
Name: Mary Smith  
Patient ID: H23/028662  
Sex at birth: Female  
Date of birth: Nov 20, 1987

**Physician**  
Name: John Doe  
Institution: Heritage Medical Center  
Contact: +82-10-0000-0000  
Address: 1600 Amphitheatre Parkway  
Mountain View, CA 94043

**Specimen**  
Specimen ID: C346399(9)  
Specimen type: FFPE  
Collected: Nov 20, 2023 14:10  
Received: Nov 24, 2023  
Normal sample:  
Matched blood  
Normal obtained: Nov 20, 2023 20:20  
Primary site: Breast, left  
Sampling site: Breast, left  
Diagnosis:  
Breast cancer, triple negative

**Test Information**  
Test name:  
Target-enhanced whole genome analysis and interpretation  
Quality: Satisfactory  
Tumor Proportion: 48%  
  
Sequencing mean depth  
Tumor (WGS): 48.2x  
Tumor (target): 523.2x  
Normal (WGS): 25.2x

KEY FINDINGS SUMMARY - POTENTIALLY ACTIONABLE\*

Therapeutics, in current diagnosis

Somatic alteration		
BRCA1 rearrangement	Talazoparib Olaparib	Evidence B Evidence B
	Potential clinical trials: Matched**	
Germline alteration		
RAD51C rearrangement	Potential clinical trials: Matched**	

Therapeutics, In other indications

Somatic alteration	
BRCA1 rearrangement	Rucaparib Niraparib Olaparib Olaparib + Bevacizumab (+ 3 more options)
Genomic instability*** HR deficiency	Rucaparib Niraparib

Cancer-related germline alteration

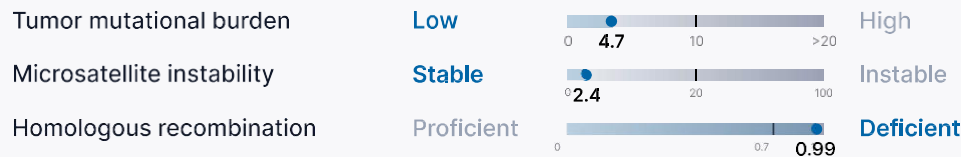
RAD51C rearrangement	Genetic counseling may be beneficial.
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\*The prescribing information for the FDA-approved therapeutic option may not include the associated alterations and it should be noted this information does not pertain to pediatric indications. Therapeutic options with evidence level A or B according to the ASCO/CAP guideline, are reported.

\*\* See details in following pages.

\*\*\* While the TMB/MSI/HRD scores have been validated, this test is not FDA-approved as a companion diagnostic for therapeutic selection, including Anti-PD-1, and PARP inhibitor treatments.

Genomic instability



Somatic driver alteration

Point mutation	
CDKN2A p.P81R	Missense variant
ATR p.W1471*	Stop-gain
PPARG p.A414P	Missense variant
ROS1 p.V1261L	Missense variant

Somatic driver alteration, cont'd

Structural variation	
<b>PTEN</b>	Rearrangement (Disruption)
<b>BRIP1</b>	Rearrangement (Disruption)
<b>ARID1A</b>	Rearrangement (Disruption)
<b>BRCA1</b>	Rearrangement (Disruption)
<b>KMT2C</b>	Rearrangement (Disruption)
Copy number variation	
Gain	<b>CALR, LYL1, PRKACA</b>

SAMPLE

KEY FINDINGS - POTENTIALLY ACTIONABLE

Therapeutics, in current diagnosis

Alteration	Treatment	Predicted response*	Evidence level
BRCA1 rearrangement somatic alteration	Talazoparib	Sensitive	B
	Olaparib	Sensitive	B

The prescribing information for the FDA-approved therapeutic option may not include the associated alteration and it should be noted this information does not pertain to pediatric indications. Therapeutic options with evidence level A or B according to the ASCO/CAP guideline, are reported.  
\*The Predictive Response Type reports treatment response likelihood across five categories: decreased response, predictive-resistant, predictive-sensitive, resistant, and sensitive.

Clinical trial

Alteration	ID / phase	Title
BRCA1 rearrangement	NCT04890613 Phase I	Study of CX-5461 in Patients With Solid Tumours and BRCA1/2, PALB2 or Homologous Recombination Deficiency (HRD) Mutation
RAD51C rearrangement	NCT05340413 Phase II	Predicting Olaparib Sensitivity in Patients With Unresectable Locally Advanced/Metastatic HER2-negative Breast Cancer With BRCA1, BRCA2, PALB2, RAD51C or RAD51D Mutations or RAD51-foci Low

A list of clinical trials, recruiting as of November 25, 2023, in the United States, was provided, taking into account the patient's genomic findings and cancer type. See <https://clinicaltrials.gov/> for more detailed and real-time information.

Therapeutics, in other indications

Alteration	Treatment	Predicted response*	Evidence level**
BRCA1 rearrangement	Rucaparib	Sensitive	A
	Niraparib	Sensitive	A
	Olaparib	Sensitive	A
	Olaparib + Bevacizumab	Sensitive	A
	Talazoparib + Enzalutamide	Sensitive	A
	Niraparib + Abiraterone Acetate + Predisone	Sensitive	A
	Olaparib + Abiraterone + Predisolone	Sensitive	A
Homologous recombination deficiency***	Rucaparib	Sensitive	A
	Niraparib	Sensitive	A

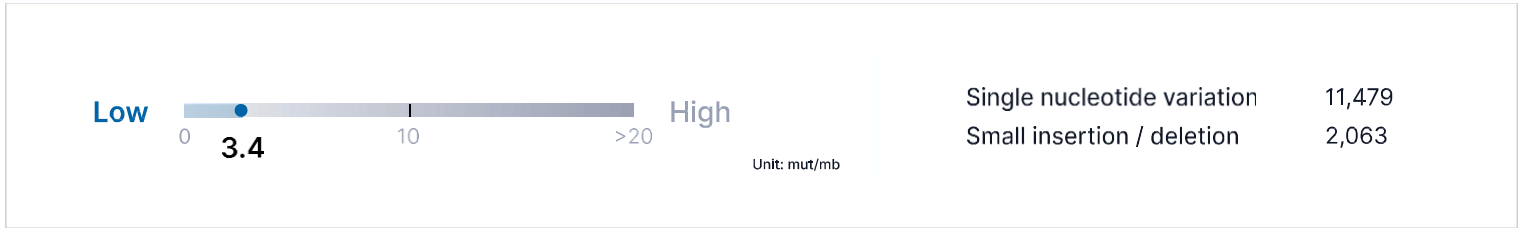
\*The Predictive Response Type reports treatment response likelihood across five categories: decreased response, predictive-resistant, predictive-sensitive, resistant, and sensitive.  
\*\*The Evidence levels are derived on the assumption that the patient's cancer type matches the drug indication, according to the ASCO/CAP guidelines.  
\*\*\*While the TMB/MSI/HRD scores have been validated, this test is not FDA-approved as a companion diagnostic for therapeutic selection, including Anti-PD-1, and PARP inhibitor treatments. The evidence level is associated with results from FDA-approved companion diagnostic tests.

Cancer-related germline alteration

Alteration	Type	Interpretation	Genotype	Note
RAD51C rearrangement	SV (DEL)	Pathogenic	Heterozygous	Loss of heterozygosity in cancer

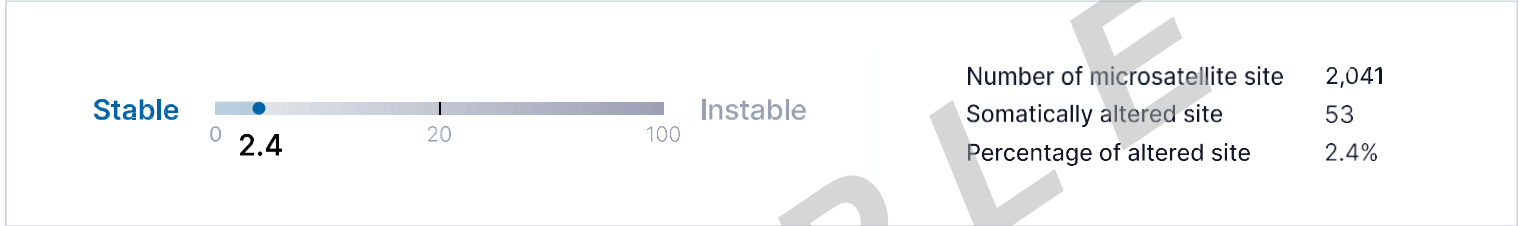
GENOMIC INSTABILITY

Tumor mutational burden



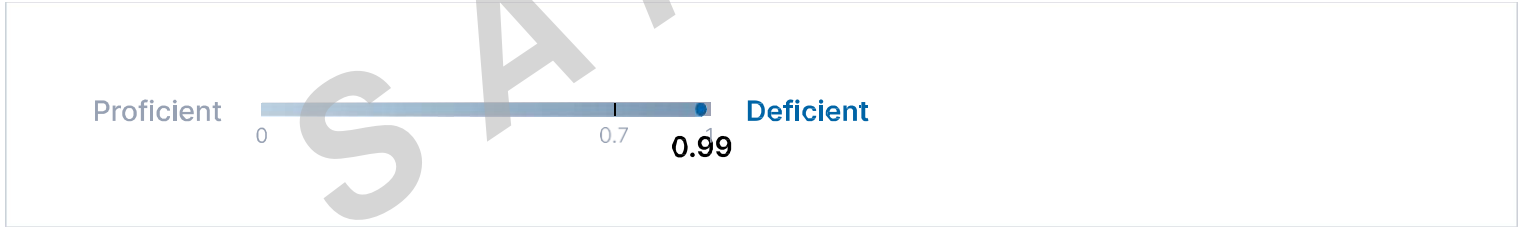
The Tumor Mutational Burden (TMB) score represents the number of mutations per Mb across the whole genome of the tumor. It is calculated by summing all the number of somatic SNVs and indels divided by the effective genome size (~2.9Gb). A tumor is considered to have a high TMB if the score is > 10mut/mb.

Microsatellite status



The microsatellite instability (MSI) score represents the number of somatic insertions and deletions per Mb in microsatellite regions across the whole genome of the tumor. A tumor is considered microsatellite stable (MSS) if the score is < 20, and MSI-High if > 20.

Homologous recombination deficiency score



The homologous recombination deficiency (HRD) score is calculated by our proprietary algorithm. Tumors with HRD score greater than or equal to 0.7 are considered HR-deficient.

The sum of loss of heterozygosity(LOH), telomeric allelic imbalance(TAI), and large-scale state transitions (LST) scores, is 56.

SOMATIC DRIVER ALTERATION

Point mutation

Alteration*	Type	Functional effect	Loss of heterozygosity	Variant allele frequency, (cancer cell fraction**)
CDKN2A p.Pro81Arg	Missense variant	Tumor suppressor	Positive	78%, (99%)
ATR p.Trp1471Ter	Stop-gain	Tumor suppressor	Negative	26%, (92%)
PPARG p.Ala414Pro	Missense variant	Tumor suppressor	Positive	52%, (90%)
ROS1 p.Val1261Leu	Missense variant	Oncogene	Not applicable	34%, (89%)

\*See the appendix to check the detail genome coordinate information of alterations.

\*\* See the test information to check the definition of cancer cell fraction

Structural variation

Alteration*	Breakpoint 1	Breakpoint 2	Functional effect	Loss of heterozygosity	Cancer cell fraction**
PTEN	PTEN (NM_00314) intron 2/8 (+)	SWP70 (NM_015055) Intron 10/11(+)	Tumor suppressor	Negative	99%
BRIP1	BRIP1 (NM_032043) Intron 17/19 (-)	chr17:61,656,237 intergenic region (+)	Tumor suppressor	Positive	99%
ARID1A	ARID1A (NM_006015) Intron 1/19 (+)	chr22:30,223,968 intergenic region (+)	Tumor suppressor	Positive	98%
BRCA1	BRCA1 (NM_007294) Intron 8/22 (-)	NBR1 (NM_005899) Intron 5/20 (+)	Tumor suppressor	Positive	98%
KMT2C	KMT2C (NM_170606) Intron 3/58 (-)	chr2:120,606,494 intergenic region (+)	Tumor suppressor	Negative	72%

\*See the appendix to check the detail genome coordinate information of alterations.

\*\* See the test information to check the definition of cancer cell fraction

Copy number variation

Gene	Location	Functional effect	Consequence*	Copy number
CALR	chr19	Oncogene	Amplification	12
LYL1	chr19	Oncogene	Amplification	11
PRKACA	chr19	Oncogene	Amplification	11

\*Gene amplification indicates that the gene's copy number is a minimum of five higher than the average ploidy level.

MUTATIONAL SIGNATURE

Major single base substitution signatures

Signature	Count	Proportion	Etiology
SBS3	5925	52.3%	Component of HRD phenotype
SBS5	3548	31.3%	Aging, presumable (clock-like signature)

The mutational signature analysis is primarily based on the COSMIC mutational signatures of SNVs.



TEST INFORMATION

1. The CancerVision test is a target-enhanced whole-genome assay that pairs a tumor with its matched normal, that provides a list of detected single nucleotide variants (SNVs), multiple nucleotide variants (MNVs), small insertions and deletions (indels), copy number alterations (CNAs), and structural variations (SVs) in tumor tissue, along with an analysis report of mutational signature, tumor mutational burden (TMB), microsatellite instability (MSI), and homologous recombination deficiency (HRD).
2. The genomic DNA is extracted from the patient's normal and tumor samples via the ThermoFisher KingFisher Apex and prepared using the Watchmaker Genomics enzymatic library preparation which includes end repair, purification, adapter ligation and PCR amplification. A portion of the library is hybridized to targeted probes. The libraries are sequenced using the Illumina NovaSeq X+. For whole-genome sequencing, the mean genome-wide read-depth is 40x for tumor and 20x for its matched-normal sample (deduplicated unique reads). For target-enhanced panel sequencing, on-target read-depth is 500x on average. The gene list is provided at the end of this section.
3. The sequence data are analyzed using various validated bioinformatics tools and a custom data-processing pipeline that have been established by a team of experts in genomics and engineering and powered by AI algorithms. For formalin-fixed paraffin-embedded (FFPE) specimens, AI-powered data correction algorithms developed by Inocras are used to mitigate data-quality issues commonly encountered in FFPE-derived samples. GRCh38 is used for human reference genome.
4. Each tumor's cancer-specific mutations are then queried against a proprietary gene-drug database based on peer-reviewed literature to identify potential therapeutic associations; however, this information should be considered in conjunction with other clinical and diagnostic findings.
5. Potential actionable findings encompass the identification of on-label drugs pertinent to the patient's reported diagnosis, detection of off-label drugs associated with genomic findings not approved for the patient's disease state, alignment with clinical trials (accessible via clinicaltrials.gov), and the identification of likely or pathogenic germline findings.
6. Tumor cellularity is a proportion of tumor cells in the specimen of whole-genome sequencing. Somatic mutations may be under-detected when tumor cellularity is low. Samples with a tumor content of less than 20% may have reduced sensitivity, potentially leading to false negative results.
7. SNVs and indels whose consequences are predicted to activate oncogenes and reported as hotspot mutations by COSMIC were categorized as oncogenic mutations. Those whose consequences cause loss of function of tumor suppressor genes (TSGs) were classified as TSG-disrupting mutations.
8. CNAs that amplify oncogenes more than five copies above the average ploidy were classified as oncogene amplification, whereas those that delete both copies of TSGs were categorized as biallelic deletion of TSGs.
9. SVs that generate known fusion oncogenes or disrupt TSGs were classified as driver events. SVs that produce known fusion oncogenes by connecting two independent genes were classified as fusion oncogene-generating SVs. SVs that alter the arrangement of exons of TSGs were classified as TSG-disrupting SVs.
10. The term 'indirect fusion' describes a condition in which the pair of genes subject to fusion are not directly connected but are linked through another DNA component. It is important to exercise caution in interpreting the results, as the efficacy of therapeutic agents may vary in cancer tissues containing indirect fusion findings compared to those with conventional fusion alterations.
11. Variant allele frequency (VAF) is the fraction of variant-supporting reads among total sequencing reads and is dependent on tumor cellularities. Cancer cell fraction accounts for contamination by normal cells, providing a more accurate estimate of the mutation's prevalence and its clonality.
12. The TMB score represents the number of mutations per Mb across the whole genome of the tumor. It is calculated by summing all the number of somatic SNVs and indels divided by the effective genome size (2.9Gb). A tumor is considered to have a high TMB if the score is > 10mut/ mb. While the TMB score has been validated, it is not FDA approved as a companion diagnostic for therapeutic selection, such as Anti-PD-1 treatments.
13. The MSI score represents the number of somatic insertions and deletions per Mb in microsatellite regions across the whole genome of the tumor. A tumor is considered microsatellite stable (MSS) if the score is < 20, and MSI-High if > 20. While the MSI score has been validated, it is not FDA approved as a companion diagnostic for therapeutic selection, such as Anti-PD-1 treatments.
14. The HRD score is calculated by our proprietary algorithm. Tumors with HRD score greater than or equal to 0.7 are considered HR deficient. While the HRD score has been validated, it is not FDA approved as a companion diagnostic for therapeutic selection, such as PARP inhibitor treatments.
15. A lack of a variant call does not necessarily indicate the absence of a variant, as technical limitations may restrict data acquisition in certain genetic regions. Additionally, it is possible that the sample contains a mutation below our established limit of detection (1% allele frequency in hotspots, 5% in other regions), or in a gene excluded by our assay. Alterations present in repetitive or high GC content region may not be detected. The inherent DNA fragmentation, damage, and background noise in FFPE samples can reduce the sensitivity and specificity of copy number alterations (amplifications/deletions) and structural variations.

16. The Predictive Response Type reports treatment response likelihood across five categories: decreased response, predictive-resistant, predictive-sensitive, resistant, and sensitive. A decreased response implies diminished efficacy compared to alternative therapies or molecular profiles, without meeting resistance criteria. Predictive-Resistant indicates literature-backed resistance of a variant or pathway to therapy, potentially including resistance. Predictive-Sensitive suggests literature-supported sensitivity of an unspecified variant or pathway to therapy. Resistant denotes explicit resistance of a molecular profile to therapy due to biochemical mechanisms, while Sensitive indicates literature-supported therapy sensitivity. Efficacy evidence levels are determined based on AMP/CAP/ASCO criteria.

Gene list (target)

The list below is for target-enhanced panel sequencing.

- A-B  
ABL1, ABL2, ABRAXAS1, ACVR1, ACVR1B, ADGRA2, AKT1, AKT2, AKT3, ALK, ALOX12B, AMER1, ANKRD11, ANKRD26, APC, AR, ARAF, ARFRP1, ARID1A, ARID1B, ARID2, ARID5B, ASXL1, ASXL2, ATM, ATR, ATRX, AURKA, AURKB, AXIN1, AXIN2, AXL, B2M, BAP1, BARD1, BBC3, BCL10, BCL2, BCL2L1, BCL2L11, BCL2L2, BCL6, BCOR, BCORL1, BCR, BIRC3, BLM, BMPRIA, BRAF, BRCA1, BRCA2, BRD4, BRIP1, BTG1, BTK
- C-D  
CALR, CARD11, CASP8, CBFB, CBL, CCN6, CCND1, CCND2, CCND3, CCNE1, CD274, CD276, CD74, CD79A, CD79B, CDC73, CDH1, CDK12, CDK4, CDK6, CDK8, CDKN1A, CDKN1B, CDKN2A, CDKN2B, CDKN2C, CEBPA, CENPA, CHD2, CHD4, CHEK1, CHEK2, CIC, COP1, CREBBP, CRKL, CRLF2, CSF1R, CSF3R, CSNK1A1, CTCF, CTLA4, CTNNA1, CTNNB1, CUL3, CUX1, CXCR4, CYLD, DAXX, DCUN1D1, DDR2, DDX41, DHX15, DICER1, DIS3, DNAJB1, DNMT1, DNMT3A, DNMT3B, DOT1L
- E-F  
E2F3, EED, EGFL7, EGFR, EIF1AX, EIF4A2, EIF4E, ELOC, EML4, EMSY, EP300, EPCAM, EPHA3, EPHA5, EPHA7, EPHB1, ERBB2, ERBB3, ERBB4, ERCC1, ERCC2, ERCC3, ERCC4, ERCC5, ERG, ERFF1, ESR1, ETS1, ETV1, ETV4, ETV5, ETV6, EWSR1, EZH2, FAM46C, FANCA, FANCC, FANCD2, FANCE, FANCF, FANCG, FANCI, FANCL, FAS, FAT1, FBXW7, FGF1, FGF10, FGF14, FGF19, FGF2, FGF23, FGF3, FGF4, FGF5, FGF6, FGF7, FGF8, FGF9, FGFR1, FGFR2, FGFR3, FGFR4, FH, FLCN, FLI1, FLT1, FLT3, FLT4, FOXA1, FOXL2, FOXO1, FOXP1, FRS2, FUBP1, FYN
- G-H  
GABRA6, GATA1, GATA2, GATA3, GATA4, GATA6, GEN1, GID4, GLI1, GNA11, GNA13, GNAQ, GNAS, GPS2, GREM1, GRIN2A, GRM3, GSK3B, H1-2, H2BC5, H3-3A, H3-3B, H3-4, H3-5, H3C1, H3C10, H3C11, H3C12, H3C13, H3C14, H3C15, H3C2, H3C3, H3C4, H3C6, H3C7, H3C8, HGF, HLA-A, HLA-B, HLA-C, HNF1A, HNRNPK, HOXB13, HRAS, HSD3B1, HSP90AA1
- I-M  
ICOSLG, ID3, IDH1, IDH2, IFNGR1, IGF1, IGF1R, IGF2, IKBKE, IKZF1, IL10, IL7R, INHA, INHBA, INPP4A, INPP4B, INSR, IRF2, IRF4, IRS1, IRS2, JAK1, JAK2, JAK3, JUN, KAT6A, KDM5A, KDM5C, KDM6A, KDR, KEAP1, KEL, KIF5B, KIT, KLF4, KLHL6, KMT2A, KMT2B, KMT2C, KMT2D, KRAS, LAMP1, LATS1, LATS2, LMO1, LRP1B, LYN, LZTR1, MAGI2, MALT1, MAP2K1, MAP2K2, MAP2K4, MAP3K1, MAP3K13, MAP3K14, MAP3K4, MAPK1, MAPK3, MAX, MCL1, MDC1, MDM2, MDM4, MED12, MEF2B, MEN1, MET, MGA, MITF, MLH1, MLLT3, MPL, MRE11, MSH2, MSH3, MSH6, MST1, MST1R, MTOR, MUTYH, MYB, MYC, MYCL, MYCL1, MYCN, MYD88, MYOD1
- N-P  
NAB2, NBN, NCOA3, NCOR1, NEGR1, NF1, NF2, NFE2L2, NFKBIA, NKX2-1, NKX3-1, NOTCH1, NOTCH2, NOTCH3, NOTCH4, NPM1, NRAS, NRG1, NSD1, NTRK1, NTRK2, NTRK3, NUP93, NUTM1, PAK1, PAK3, PAK5, PALB2, PARP1, PAX3, PAX5, PAX7, PAX8, PBRM1, PDCD1, PDCD1LG2, PDGFRA, PDGFRB, PDK1, PDPK1, PGR, PHF6, PHOX2B, PIK3C2B, PIK3C2G, PIK3C3, PIK3CA, PIK3CB, PIK3CD, PIK3CG, PIK3R1, PIK3R2, PIK3R3, PIM1, PLCG2, PLK2, PMAIP1, PMS1, PMS2, PNRC1, POLD1, POLE, PPARG, PPM1D, PPP2R1A, PPP2R2A, PPP6C, PRDM1, PREX2, PRKAR1A, PRKCI, PRKDC, PRKN, PRSS8, PTCH1, PTEN, PTPN11, PTPRD, PTPRS, PTPRT
- Q-R  
QKI, RAB35, RAC1, RAD21, RAD50, RAD51, RAD51B, RAD51C, RAD51D, RAD52, RAD54L, RAF1, RANBP2, RARA, RASA1, RB1, RBM10, RECQL4, REL, RET, RHEB, RHOA, RICTOR, RIT1, RNF43, ROS1, RPS6KA4, RPS6KB1, RPS6KB2, RPTOR, RUNX1, RUNX1T1, RYBP
- S-T  
SDHA, SDHAF2, SDHB, SDHC, SDHD, SETBP1, SETD2, SF3B1, SH2B3, SH2D1A, SHQ1, SLC7A8, SLIT2, SLX4, SMAD2, SMAD3, SMAD4, SMARCA4, SMARCB1, SMARCD1, SMC1A, SMC3, SMO, SNCAIP, SOCS1, SOX10, SOX17, SOX2, SOX9, SPEN, SPOP, SPTA1, SRC, SRSF2, STAG1, STAG2, STAT3, STAT4, STAT5A, STAT5B, STK11, STK40, STT3A, SUFU, SUZ12, SYK, TAF1, TBX3, TCF3, TCF7L2, TENT5C, TERC, TERT, TET1, TET2, TFE3, TFRC, TGFB1, TGFB2, TMEM127, TMPRSS2, TNFAIP3, TNFRSF14, TOP1, TOP2A, TP53, TP63, TRAF2, TRAF7, TSC1, TSC2, TSHR,
- U-Z  
U2AF1, VEGFA, VHL, VTCN1, WT1, XIAP, XPO1, XRCC2, YAP1, YES1, ZBTB2, ZBTB7A, ZFH3, ZNF2, ZNF217, ZNF703, ZRSR2



## Gene list (cancer-related germline genes)

The following is a list of genes that we report when a pathogenic or likely pathogenic germline mutation is found.

ABCC11, ALK, APC, AR, ATM, ATR, AXIN2, BAP1, BARD1, BLM, BMPR1A, BRCA1, BRCA2, BRIP1, BUB1B, CBL, CDC73, CDH1, CDK4, CDKN1B, CDKN2A (p14ARF), CDKN2A (p16INK4a), CEBPA, CHEK2, COL7A1, CTNNA1, CTR, CYLD, DDB2, DICER1, DIS3L2, DKC1, DOCK8, EGFR, ELANE, EPCAM, ERCC1, ERCC2, ERCC3, ERCC4, ERCC5, EXT1, EXT2, FAH, FANCA, FANCC, FANCE, FANCG, FANCI, FANCM, FH, FLCN, GALNT3, GATA2, GBA, GJB2, GPC3, GREM1, HFE, HMBS, HNF1A, HOXB13, HRAS, ITK, JMJD1C, KIT, MAX, MEN1, MET, MITF, MLH1, MSH2, MSH3, MSH6, MTAP, MUTYH (alpha3), MUTYH (alpha5), NBN, NF1, NF2, NTHL1, PALB2, PAX5, PDGFRA, PHOX2B, PMS2, POLD1, POLE, POLH, PRDM9, PRKAR1A, PRSS1, PTCH1, PTEN, PTPN11, RAD51C, RAD51D, RB1, RECQL, RECQL4, RET, RHBDF2, RMRP, RUNX1, SBDS, SDHA, SDHAF2, SDHB, SDHC, SDHD, SERPINA1, SETBP1, SH2B3, SH2D1A, SLC25A13, SMAD4, SMARCA4, SMARCB1, SMARCE1, SOS1, SRGAP1, SRY, STAT3, STK11, SUFU, TERT, TGFBR1, TMEM127, TNFRSF6, TP53, TRIM37, TSC1, TSC2, UROD, VHL, WAS, WRN, WT1, XPA, XPC

## REFERENCES

1. Catalogue of Somatic Mutations in Cancer (COSMIC, cancer.sanger.ac.uk). Tate, J.G, et al. (2019) COSMIC: the catalogue of somatic mutations in cancer. *Nucleic Acids Research* 47(D1):D941-D947 (<https://doi.org/10.1093/nar/gky1015>)

## DISCLAIMER

This report is intended to provide information to the treating physician and is not intended to guarantee or promise the efficacy or usefulness of any particular drug or treatment regimen for any patient. The potential clinical benefit of any drug listed in this report may vary based on a variety of factors, including the patient's specific tumor type and other clinical considerations.

This test was developed, and quality-assured by Inocras. It has not been cleared or approved by the US Food and Drug Administration. The test has been validated as a Laboratory Developed Test per institutional and applicable CLIA regulation (CLIA# 05D2280195) as qualified to perform high complexity clinical laboratory testing. Data interpretations are based on our current understanding of genes and variants as of the report date. When the report does identify variants with therapeutic implications, this does not promise or guarantee that a particular drug or treatment regimen will be effective or helpful in the treatment of disease in any patient, and the selection of any drug for patient treatment is done at the discretion of the treating physician. Genomic alterations should be considered in the context of the patient's history, risk factors and any previous genomic testing.

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For germline alterations, Inocras strongly suggests that the patient receive appropriate genetic counseling to explain the implications of this test result, its residual risks and uncertainties, and the reproductive or medical options it raises for the patient.

## TRACKING INFORMATION

Sample tracking ID: 2401-034.001

Analysis ID: GINS-0000-0000-01AD

Pipeline version: Somatic 1.2.0



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*This is a sample report*

*This includes only select key pages*

SAMPLE