INCIDENTAL GERMLINE FINDINGS IN SOLID TUMOR PROFILES

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DISCLAIMER

I am a full-time employee of Caris Life Sciences—a commercial tumor profiling laboratory.

OBJECTIVES

The Basics: Identify biomarkers and their associated characteristics on a solid tumor profile report that could indicate incidental germline findings.

- How to navigate a tumor profile report
- Definitions: biomarker, tumor molecular burden, % tumor nuclei, allele frequency
- Which tumor profile findings warrant germline follow-up?

Intermediate Cases: Examine actual somatic tumor profile cases and germline outcomes.

- Use context clues to guess if a variant detected by a tumor profile is germline or not.

Advanced Case: Formulate a plan for follow-up when tumor profile and germline results show discrepancies.

SCENARIO

Tumor board is presenting the solid tumor profile results of a 55 yo female with colorectal cancer and a 58 yo male with a metastatic prostate tumor.

THE BASICS

A biomarker is an umbrella term used to describe *something* measured in the tumor that tells us *something* about the tumor at a given moment.

CRC Biomarker	Association
RAS mutations (KRAS/NRAS/HRAS)	Will not benefit from anti-EGFR treatment
BRAF V600E	Poor prognosis
Her2 (ERBB2) amplification	Poor prognosis
Fusions (NTRK/ALK/ROS1/RET)	Clinical trial eligibility
MSI, TMB, PD-L1	Sensitive to immunotherapy

Companion diagnostics (CDx) inform the use of personalized treatment options for advanced cancer patients by identifying FDA-approved treatment options that may be appropriate for treatment based on the unique drivers of their individual cancer.

MORE BIOMARKERS

Advanced Prostate Biomarker	Association
BRCA1, BRCA2	PARP inhibitor
PTEN	Anti-androgen resistance, poor survival; AKT inhibitor clinical trial
TP53 and RB1	Poor survival; divergent neuroendocrine differentiation (more aggressive AR-independent disease)
AR amplification	Anti-androgen resistance
TMPRSS2:ERG fusion	Expected in prostate cancer
DNA damage repair genes: ATM, PALB2, FANCA, RAD51D, CHEK2, CDK12	Clinical trial for PARP inhibitor
MSI, TMB, PD-L1	Sensitive to immunotherapy

Incidental or Secondary findings in tumor profiles usually refer to germline or chimeric results

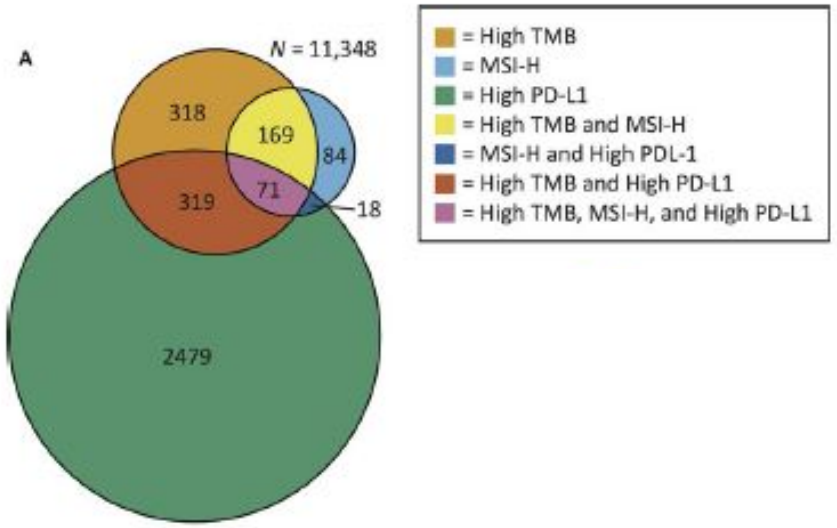
PD-L1, MSI AND TMB

These are *complementary* biomarkers for patients who may benefit from immune checkpoint inhibitors (anti-PD-1 Therapy):

IHC for **PD-L1** (programmed death-ligand 1) expression is the standard of care

MSI (microsatellite instability) is a genomic signature of deficient mismatch repair. It involves the gain/loss of repeats in microsatellite regions or from epigenetic changes.

TMB (tumor mutational burden) quantifies the amount of somatic mutations per Mb there are in a tumor. If the TMB is high, then the immune system is more likely to recognize the tumor.



Microsatellite instability status determined by next-generation sequencing and compared with PD-L1and tumor mutational burden in 11,348 patients

PMID: 29436178
Cancer Med 2018 Mar;7(3):746-756. doi: 10.1002/cam4.1372.

ABOUT THE TEST FoundationOne®CDx is the first and only FDA-Approved comprehensive companion diagnostic for all solid tumors.

Interpretive content on this page and subsequent pages is provided as a professional service, and is not reviewed or approved by the FDA.

PATIENT

DISEASE Colon adenocarcinoma (CRC)
NAME
DATE OF BIRTH
SEX
MEDICAL RECORD #

PHYSICIAN

ORDERING PHYSICIAN
MEDICAL FACILITY
ADDITIONAL RECIPIENT
MEDICAL FACILITY ID
PATHOLOGIST

SPECIMEN

SPECIMEN SITE
SPECIMEN ID
SPECIMEN TYPE
DATE OF COLLECTION
SPECIMEN RECEIVED

Biomarker Findings

Microsatellite status - MSI-High
Tumor Mutational Burden - 35 Muts/Mb

Genomic Findings

For a complete list of the genes assayed, please refer to the Appendix.

KRAS **wildtype**
NRAS **wildtype**
NTRK1 TPM3-NTRK1 fusion
ATM R3047*
PALB2 M296fs*1
CTNNB1 W383R
RNF43 G659fs*41
SUFU A25fs*23
ASXL1 G645fs*58, S1335fs*115
BAP1 I191fs*2
CDH1 S70fs*13, P127fs*41
CIC P1597fs*23
FAM123B E370fs*8
MLL2 P2354fs*30
TP53 R273C

3 Disease relevant genes with no reportable alterations: *BRAF, KRAS, NRAS*

15 Therapies with Clinical Benefit
0 Therapies with Lack of Response
47 Clinical Trials

BIOMARKER FINDINGS

Microsatellite status - MSI-High

10 Trials *see p. 19*

Tumor Mutational Burden - 35 Muts/Mb

10 Trials *see p. 21*

THERAPIES WITH CLINICAL BENEFIT (IN PATIENT'S TUMOR TYPE)		THERAPIES WITH CLINICAL BENEFIT (IN OTHER TUMOR TYPE)
Nivolumab	2A	Atezolizumab
Pembrolizumab	2A	Avelumab
		Cemiplimab
		Durvalumab
Nivolumab		Atezolizumab
Pembrolizumab		Avelumab
		Cemiplimab
		Durvalumab

EUROPEAN SOCIETY FOR MEDICAL ONCOLOGY

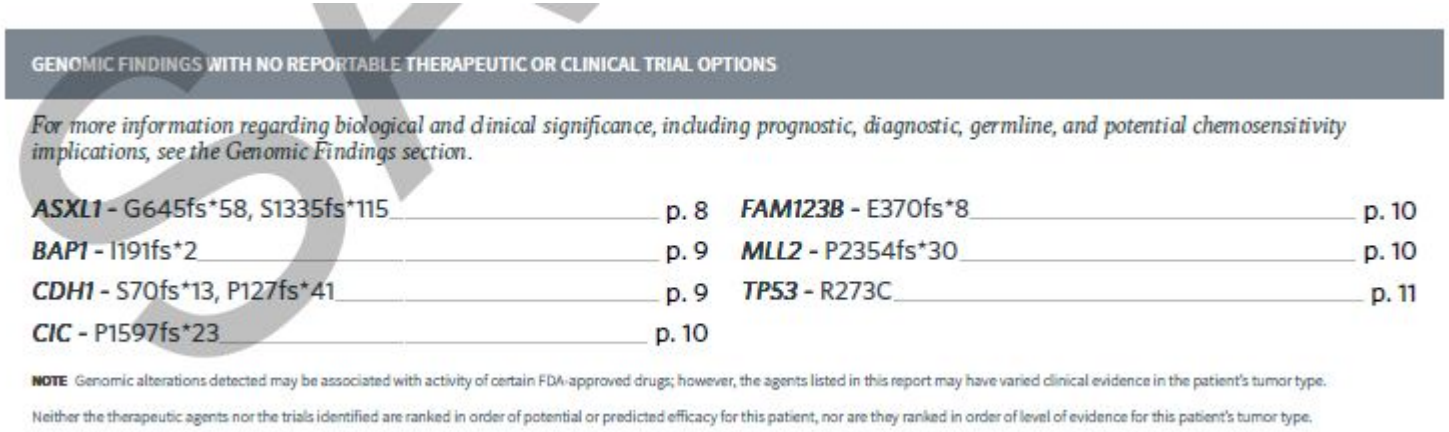
Box 1. Recommendations for genes to be included for germline-focussed analysis and triggering of germline sample laboratory confirmation

	Any tumour type		Associated tumour type only
Tumour arising any age	*BRCA1*	*RAD51C*	*FLCN*
	BRCA2	*RAD51D*	*FH*
	BRIP1	*RET*	*BAP1*
	MLH1	*SDHA*	*POLE*
	MSH2	*SDHAF2*	
	MSH6	*SDHB*	
	PALB2	*SDHC*	
	PMS2	*SDHD*	
	VHL[a]	*TSC2*	
		MUTYH[b]	
Tumour arising age <30 only	*RB1*		*TP53*[c]
	APC		*NF1*

[a]Renal tumours to be excluded.
[b]*MUTYH* should be included for germline-focussed tumour analysis but reporting and germline follow-up testing should only be performed on detection of two pathogenic variants.
[c]Brain tumours to be excluded.

Annals of Oncology 30: 1221–1231, 2019 doi:10.1093/annonc/mdz136 Published online 3 May 2019

CHECK THE APPENDIX AND THE PORTAL



GENOMIC FINDINGS WITH NO REPORTABLE THERAPEUTIC OR CLINICAL TRIAL OPTIONS

For more information regarding biological and clinical significance, including prognostic, diagnostic, germline, and potential chemosensitivity implications, see the Genomic Findings section.

NOTE Genomic alterations detected may be associated with activity of certain FDA-approved drugs; however, the agents listed in this report may have varied clinical evidence in the patient's tumor type.

Neither the therapeutic agents nor the trials identified are ranked in order of potential or predicted efficacy for this patient, nor are they ranked in order of level of evidence for this patient's tumor type.

If you plan to order germline testing, you will often need to provide the c. nomenclature and transcript ID from the somatic lab.

TUMOR NUCLEI AND VARIANT ALLELE FREQUENCY

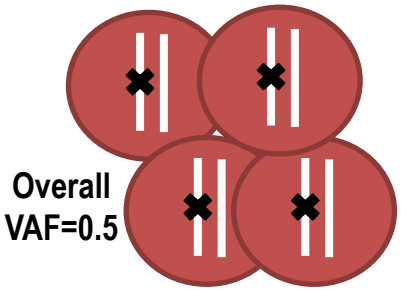
Tumor nuclei %

- aka Tumor cellularity
- the estimated percentage of neoplastic cells in the sample

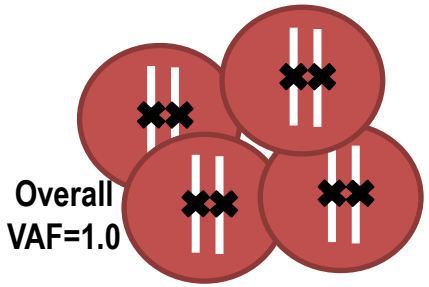
Variant allele frequency (%) –

- aka VAF, variant allele fraction, mutation allele frequency
- Percentage of sequence reads of a given DNA variant divided by the overall coverage at that locus
- **Interpret with caution**

Ideally, VAF of a germline mutation is 0.5 or 1.0



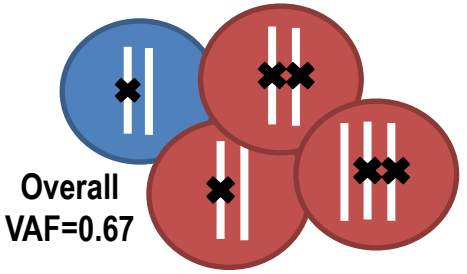
No LOH*



LOH

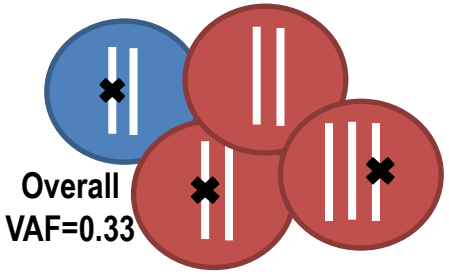
However…

VAF is usually between 0.5-1.0

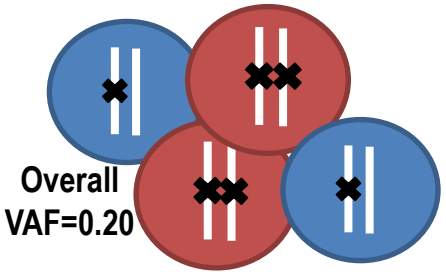


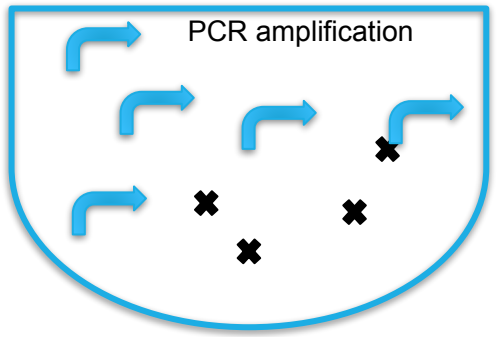
Normal admixture, aneuploidy and intratumoral heterogeniety or LOH

Furthermore, VAF of a germline mutation can be <0.5



Loss of mutant allele in proportion of tumor cells





Preferential amplification of wildtype allele

*Loss of heterozygosity = common genetic event in cancer, somatic loss of wild-type allele in hereditary cancer

Slide compliments of Dana Farengo-Clark, MS, CGC

Metastatic prostate cancer

Tumor specimen:
Lymph node, left inguinal
Chicago Cancer Center
#ABC 123, C2
Collected 3/08/2019
~~Received 3/18/2019~~
Tumor Percentage: 70%

Normal specimen:
Blood
Collected 3/20/2019
Received 3/22/2019

Notes

The tumor shows loss of heterozygosity in TP53.

GENOMIC VARIANTS

Somatic - Potentially Actionable

Variant Allele Fraction

TP53	p.R196* Stop gain - LOF	61.4%
AR	Copy number gain	
CDKN2A	Copy number loss	
TMPRSS2 - ERG	Chromosomal rearrangement	

Somatic - Biologically Relevant

CDKN2B	Copy number loss

Germline - Pathogenic / Likely Pathogenic

No pathogenic variants were found in the limited set of genes on which we report.

IMMUNOTHERAPY MARKERS

Tumor Mutational Burden

2.1 m/MB — 40th percentile

Microsatellite Instability Status

Stable | Equivocal | High

VARIANTS OF UNKNOWN SIGNIFICANCE

Somatic	Mutation effect	Variant allele fraction
TMPRSS2	c.1339_1424del p.C447fs Frameshift NM_001135099	38.0%
EGF	c.1153G>C p.G385R Missense variant NM_001963	37.3%
FGFR4	c.2273G>A p.R758H Missense variant NM_002011	33.3%
FGFR4	c.1985T>C p.F662S Missense variant NM_002011	29.4%
BCL11B	c.2098G>A p.A700T Missense variant NM_138576	26.5%

Germline	Mutation effect	Condition
BMPR1A	c.1433G>A p.R478H Missense variant chr10:88683223 NM_004329	Juvenile polyposis

LOW COVERAGE REGIONS

FLT4	GFRA2	NOTCH1	PDPK1	TAF1

QUESTIONS TO ASK THE LAB

What biomarkers are included in the test?

For DNA NGS, how does gene coverage compare to a typical germline lab?

If paired with germline testing, is confirmation testing needed?

Where is the tumor nuclei %, VAF, c. and transcript ID?

Get the appendix and supplementary materials.

Check the online version of the report.

IS IT GERMLINE?

You be the judge!

72 yo woman with lobular breast cancer

Reason for Referral: **MALIGNANT NEOPLASM OF UPPER-OUTER QUADRANT OF LEFT FEMALE BREAST**

Results Summary

2 Clinically Significant Variants Detected[§]	**CDH1** E35*; **PIK3CA** E542K
0 Alterations Detected by FISH	NONE detected
Immuno-Oncology Biomarkers	**Microsatellite Instability:** MSI - Stable (MSS); **PD-L1 SP142:** EXPRESSED; **Tumor Mutation Burden:** Low
Additional Studies	**Pan-TRK:** EXPRESSED
Pertinent Negatives	NO abnormalities detected in the following genes: BRCA1, BRCA2, ERBB2, ESR1

Interpretation

- The expression of PD-L1 suggests response to immunotherapy with anti-PD-1 or anti-PD-L1, which are FDA-approved for diverse solid tumor types.
- Pan-TRK by IHC: Expressed. Refer to separate results for reflex to NTRK NGS Fusion Profile.

[§] See full list of genes tested in Biomarkers Evaluated section at end of report.

DETAILS

Gene name	Variant	Amino Acid Change	Nucleotide Change	Consequence	Mutant Allele Frequency (%)	Read Depth
PIK3CA	E542K	p.E542K	NM_006218.4: c.1624G>A	Missense	46.0	4939
CDH1	E35*	p.E35*	NM_004360.5: c.103G>T	Stop gained	56.5	4712

GERMLINE RESUI

Test performed

Sequence analysis of the 9 genes listed in the Genes Analyzed section.

- Invitae Breast Cancer STAT Panel
- Add-on ATM Gene
- Add-on CHEK2 Gene

RESULT: NEGATIVE

About this test

This diagnostic test evaluates 9 gene(s) for variants (genetic changes) that are associated with genetic disorders. Diagnostic genetic testing, when combined with family history and other medical results, may provide information to clarify individual risk, support a clinical diagnosis, and assist with the development of a personalized treatment and management strategy.

GENE	TRANSCRIPT
ATM	NM_000051.3
BRCA1	NM_007294.3
BRCA2	NM_000059.3

GENE	TRANSCRIPT
CDH1	NM_004360.3
CHEK2	NM_007194.3
PALB2	NM_024675.3

GENE	TRANSCRIPT
PTEN	NM_000314.4
STK11	NM_000455.4
TP53	NM_000546.5

Previous analysis performed at a different laboratory reportedly identified a variant in CDH1 c.103G>T (p.E35*), in this individual's tumor testing. This variant was not detected in the submitted sample.

GENETIC COUNSELING FOR BIOMARKERS

Is the patient personally motivated to have germline testing OR are you screening tumor profiles to find appropriate candidate for testing?

Is the variant expected to in this tumor?

COSMIC: https://cancer.sanger.ac.uk/cosmic/browse/tissue

Is the personal and family medical history consistent?

70 yo female with ovarian cancer, TN% 40

Genomic Signatures

Biomarker	Method	Analyte	Result
Microsatellite Instability (MSI)	Seq	DNA-Tumor	Stable
Tumor Mutational Burden (TMB)	Seq	DNA-Tumor	
Genomic Loss of Heterozygosity (LOH)	Seq	DNA-Tumor	High - 18% of tested genomic segments exhibited LOH (assay threshold is ≥ 16%)

Genes Tested with Pathogenic or Likely Pathogenic Alterations

Gene	Method	Analyte	Variant Interpretation	Protein Alteration	Exon	DNA Alteration	Variant Frequency %
BRCA2	Seq	DNA-Tumor	Pathogenic Variant	p.S599fs	10	c.1796delC	76
MAP2K4	Seq	DNA-Tumor	Pathogenic Variant	c.1086+1G>A	10	c.1086+1G>A	51
TP53	Seq	DNA-Tumor	Pathogenic Variant	p.E286K	8	c.856G>A	53

Unclassified alterations for DNA sequencing can be found in the MI Portal.
Formal nucleotide nomenclature and gene reference sequences can be found in the Appendix of this report.

Immunohistochemistry Results

Biomarker	Result	Biomarker	Result
ER	Positive | 2+, 75%	PD-L1 (22c3)	Positive, CPS: 3
MLH1	Positive | 1+, 100%	PMS2	Positive | 2+, 100%
MSH2	Positive | 2+, 100%	PR	Negative | 1+, 3%
MSH6	Positive | 2+, 100%		

Management Tool - BRACAnalysis CDx®
BRCA1 and *BRCA2* Analysis

GENETIC RESULT: POSITIVE - CLINICALLY SIGNIFICANT MUTATION IDENTIFIED

Note: "CLINICALLY SIGNIFICANT," as defined in this report, is a genetic change that is associated with the potential to alter medical intervention.

GENE	MUTATION	THIS GENETIC TEST RESULT IS ASSOCIATED WITH THE FOLLOWING CANCER RISKS:
BRCA2	**c.1796del (p.Ser599Phefs*15)** Heterozygous	**HIGH RISK: Female Breast, Ovarian, Pancreatic** **ELEVATED RISK: Melanoma**

53 yo female with pancreatic cancer, TN% 20

Genes Tested With Mutations/Alterations

Gene	Method	Analyte	Variant Interpretation	Protein Alteration	Exon	DNA Alteration	Variant Frequency %
ARID1A	Seq	DNA-Tumor	Pathogenic Variant	p.E1803*	20	c.5407G>T	18
CDKN2A	Seq	DNA-Tumor	Pathogenic Variant	p.R58*	2	c.172C>T	20
CTNNB1	Seq	DNA-Tumor	Likely Pathogenic Variant	p.R661Q	13	c.1982G>A	16
CYLD	Seq	DNA-Tumor	Pathogenic Variant	p.Q455*	10	c.1363C>T	19
ERBB2 (Her2/ Neu)	Seq	DNA-Tumor	Pathogenic Variant	p.R678Q	17	c.2033G>A	17
JAK2	Seq	DNA-Tumor	Pathogenic Variant	p.R803*	18	c.2407C>T	18
KRAS	Seq	DNA-Tumor	Pathogenic Variant	p.G12V	2	c.35G>T	12
MSH6	Seq	DNA-Tumor	Variant of Uncertain Significance	p.S459F	4	c.1376C>T	17
	Seq	DNA-Tumor	Pathogenic Variant	p.F1088fs	5	c.3261delC	19
	Seq	DNA-Tumor	Pathogenic Variant	p.A1320fs	9	c.3959_3962delCAAG	38
NFE2L2	Seq	DNA-Tumor	Likely Pathogenic Variant	p.G81C	2	c.241G>T	20
PALB2	Seq	DNA-Tumor	Variant of Uncertain Significance	p.L453I	4	c.1357C>A	18
PIK3CA	Seq	DNA-Tumor	Pathogenic Variant	p.Q546H	10	c.1638G>T	17
POLE	Seq	DNA-Tumor	Likely Benign Variant	p.S1827L	40	c.5480C>T	14
PTEN	Seq	DNA-Tumor	Pathogenic Variant	c.1027-1G>T	9	c.1027-1G>T	16
RET	Seq	DNA-Tumor	Variant of Uncertain Significance	p.A641T	11	c.1921G>A	16

Immunohistochemistry Results

Biomarker	Result	Biomarker	Result
MLH1	Positive | 2+, 90%	PD-L1 (SP142)	Negative | 0
MSH2	Positive | 1+, 70%	PMS2	Positive | 1+, 70%
MSH6	Negative | 0		

CANCERNEXT EXPANDED RESULT

MSH6	Pathogenic	Heterozygous	A1320Efs*6

Interpretation:

The c.3959_3962delCAAG pathogenic mutation, located in coding exon 9 of the MSH6 gene, results from a deletion of 4 nucleotides at nucleotide positions 3959 to 3962, causing a translational frameshift with a predicted alternate stop codon (p.A1320Efs*6). This mutation has been reported as an Ashkenazi Jewish founder mutation for Lynch syndrome (Raskin L et al. Clin. Genet. 2011 Jun:79:512-22; Goldberg Y et al. Fam. Cancer. 2014 Mar;13:65-73). It has been identified in numerous individuals with Lynch syndrome tumors, including several with tumors demonstrating microsatellite instability and/or absent MSH6 on IHC (Goodfellow PJ et al. Proc. Natl. Acad. Sci. U.S.A. 2003 May;100:5908-13; Hampel H et al. N. Engl. J. Med. 2005 May;352:1851-60; Baglietto L et al. J. Natl. Cancer Inst. 2010 Feb;102:193-201). In addition, the international consortium of childhood constitutional mismatch repair deficiency (CMMRD) reported this deletion in three individuals with CMMRD: one with GI polyposis, one with T-cell lymphoma and GI polyposis, and one with glioblastoma multiforme (Bakry D et al. Eur. J. Cancer. 2014 Mar;50:987-96). In addition to the clinical data presented in the literature, this alteration is expected to result in loss of function by premature protein truncation. As such, this alteration is interpreted as a disease-causing mutation.

MSH6	Seq	DNA-Tumor	Variant of Uncertain Significance	p.S459F	4	c.1376C>T	17
	Seq	DNA-Tumor	Pathogenic Variant	p.F1088fs	5	c.3261delC	19
	Seq	DNA-Tumor	Pathogenic Variant	p.A1320fs	9	c.3959_3962delCAAG	38

74 yo male with prostate cancer

Genomic Signatures

Biomarker	Method	Analyte	Result
Microsatellite Instability (MSI)	Seq	DNA-Tumor	Stable
Tumor Mutational Burden (TMB)	Seq	DNA-Tumor	
Genomic Loss of Heterozygosity (LOH)	Seq	DNA-Tumor	Low - 6% of tested genomic segments exhibited LOH (assay threshold is ≥ 16%)

Genes Tested with Pathogenic or Likely Pathogenic Alterations

Gene	Method	Analyte	Variant Interpretation	Protein Alteration	Exon	DNA Alteration	Variant Frequency %
AR	Seq	RNA-Tumor	V7 Detected	-	-	-	-
MITF	Seq	DNA-Tumor	Pathogenic Variant	p.E318K	9	c.952G>A	58
TP53	Seq	DNA-Tumor	Pathogenic Variant	p.C242F	7	c.725G>T	87

Unclassified alterations for DNA and RNA sequencing can be found in the MI Portal.
Formal nucleotide nomenclature and gene reference sequences can be found in the Appendix of this report.
Variants of Uncertain Significance can be found in the MI Portal.

Genes Tested with Indeterminate Results by Tumor DNA Sequencing

HDAC1	NFE2L2	NPM1	PIK3CB	PRKACA	PTPN11	RB1					

Genes in this table were ruled indeterminate due to low coverage for some or all exons.

One Pathogenic variant identified in MITF. MITF is associated with autosomal dominant cutaneous malignant melanoma and Waardenburg syndrome.

Additional Variant(s) of Uncertain Significance identified.

GENE	VARIANT	ZYGOSITY	VARIANT CLASSIFICATION
MITF	c.952G>A (p.Glu318Lys)	heterozygous	PATHOGENIC
TP53	c.749C>T (p.Pro250Leu)	possibly mosaic	Uncertain Significance

About this test

This diagnostic test evaluates 85 gene(s) for variants (genetic changes) that are associated with genetic disorders. Diagnostic genetic testing, when combined with family history and other medical results, may provide information to clarify individual risk, support a clinical diagnosis, and assist with the development of a personalized treatment and management strategy.

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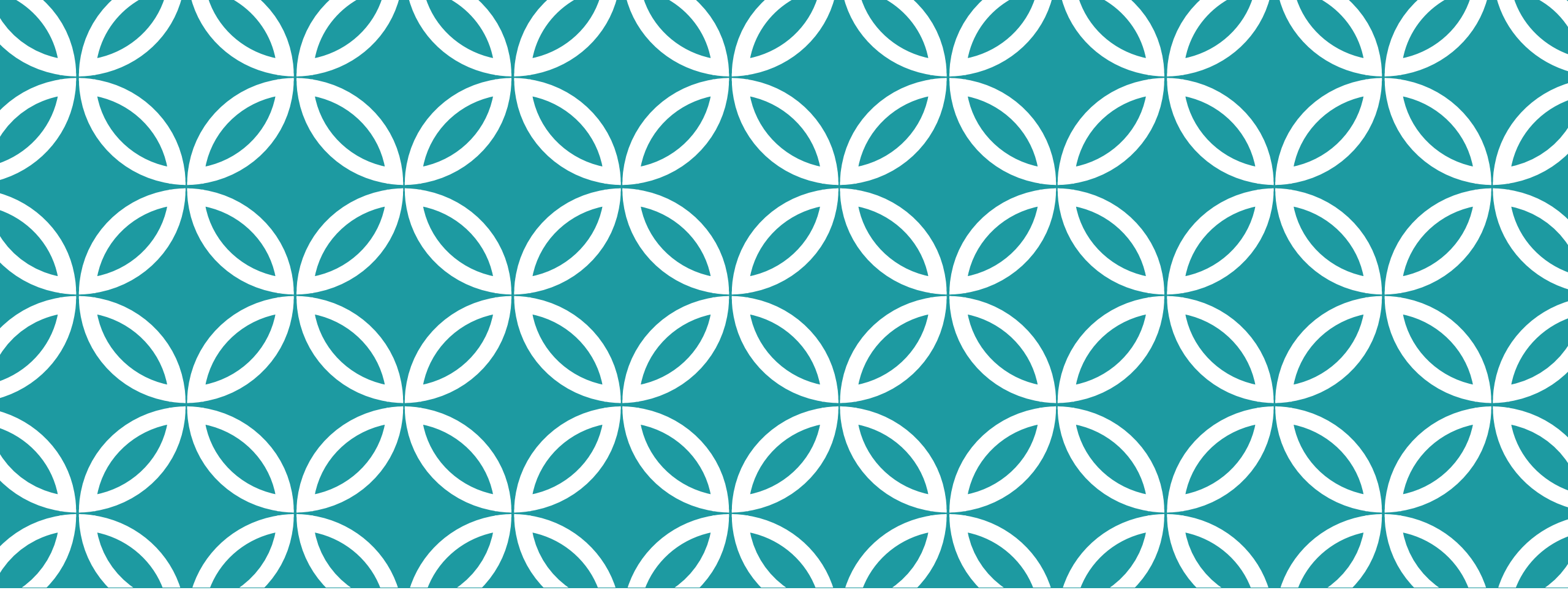
TP53	Seq	DNA-Tumor	Pathogenic Variant	p.C242F	7	c.725G>T	87



TEST DISCRPANCIES

Troubleshooting

FAQS

Why didn't the tumor profiling lab detect a known germline variant?

Can tumor profiling help me resolve whether a low frequency germline variant is due to mosaicism vs CHIP?

Why do the tumor profiling lab and the germline lab have different variant classifications?

TUMOR TESTING LIMITATIONS

NOT INCLUDED

- A tumor profiling lab may not cover all the same exons or deep intronic variants as a germline lab
- Large deletions/duplications may not be detected

LOW Q

- Tumor tissue may be degraded and/or fixative may affect gene coverage
- Tumor heterogeneity and LOH

CHIP VS MOSAICISM

Some low frequency variants detected by germline labs represent clonal hematopoiesis of indeterminate potential (CHIP)

The likelihood of CHIP increases with patient age and advanced stage cancer

Tumor profiling does not replace testing cultured skin fibroblasts.

One Pathogenic variant identified in MITF. MITF is associated with autosomal dominant cutaneous malignant melanoma and Waardenburg syndrome.

Additional Variant(s) of Uncertain Significance identified.

GENE	VARIANT	ZYGOSITY	VARIANT CLASSIFICATION
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FILTER	DP	TI	TP53	FC	EXON	AltPos	GT	VF
.	1022	NM_000546.5	TP53	Missense/C242F	7	7577556	0/1	0.873
Benign	853	NM_000546.5	TP53	Missense/P72R	4	7579472	0/1	0.941
lowQ	971	NM_000546.5	TP53	Synonymous/P36=	4	7579579	0/1	0.046

VARIANT CLASSIFICATIONS

Comparison of Somatic and Germline Variant Interpretation in Hereditary Cancer Genes
Emily W. Moody, Jennie Vagher, Whitney Espinel, David Goldgar, Kelsi J. Hagerty and Amanda Gammon
JCO Precision Oncology 2019 :3, 1-8

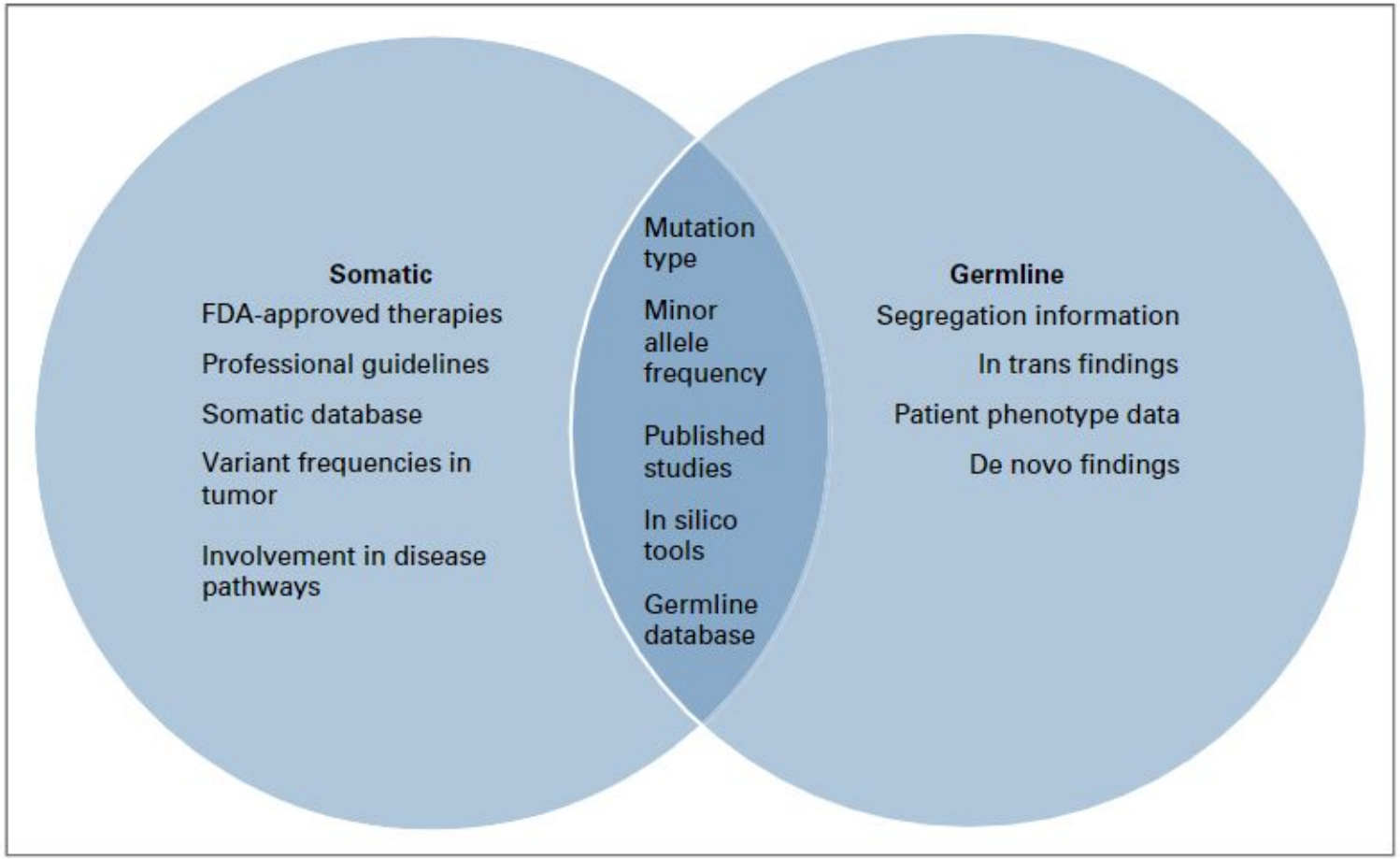

FIG 3. Diagram of lines of evidence published in guidelines for both somatic and germline variant interpretations. FDA, US Food and Drug Administration.

Slide courtesy of Kelly Knickelbein.

VHL R200W

c.598C>T (p.Arg200Trp)

Russian founder mutation that causes a blood clotting condition called Chuvash polycythemia

This variant is not associated with von Hippel Lindau or cancer risks.

TROUBLESHOOTING TIPS

Call the laboratories

- Providing clinical background can help resolve discrepancies
- Be prepared to share laboratory report information
- Talk to the pathology department who sent the tissue

Ask friend (NSGC Cancer SIG Somatic Subcommittee)

- Somaticexpertpanel@gmail.com

THANK YOU!

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