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

Companion Diagnostic (CDx) Associated Findings

GENOMIC FINDINGS DETECTED	FDA-APPROVED THERAPEUTIC OPTIONS
KRAS wildtype (codons 12 & 13)	Erbitux® (Cetuximab)
KRAS/NRAS wildtype (codons 12, 13, 59, 61, 117, & 146 in exons 2, 3, & 4)	Vectibix® (Panitumumab)
Tumor Mutational Burden (TMB) ≥ 10 Muts/Mb	Keytruda® (Pembrolizumab)

OTHER ALTERATIONS & BIOMARKERS IDENTIFIED

Results reported in this section are not prescriptive or conclusive for labeled use of any specific therapeutic product. See *professional services* section for additional information.

Microsatellite status MSI-High §
Tumor Mutational Burden 47 Muts/Mb §
ASXL1 G646fs*12
ATR I774fs*5
BCOR E623*
BCORL1 P1681fs*20
BRCA2 T3033fs*29
CREBBP R1446C
CTNNB1 R449C
CUL3 R733*
ERBB3 V104M
HNF1A P291fs*51
MAP2K1 (MEK1) K57N
MLH1 Q445*
MLH1 E663D
MLL2 A2205fs*59
MLL2 P648fs*283
MSH3 K383fs*32
MSH6 R361H
MSH6 F1088fs*5
PDGFRB V823I
SPEN A2251fs*102
SPEN R806fs*14
SPEN I1052fs*40

§ Refer to appendix for limitation statements related to detection of any copy number alterations, gene rearrangements, LOH, MSI, or TMB results in this section.

Please refer to appendix for Explanation of Clinical Significance Classification and for variants of unknown significance (VUS).

Note: The intended use (IU) statement and claims made on this sample report may not be up to date. For the latest version of the FoundationOne CDx claims and IU, please see the current label: www.foundationmedicine.com/f1cdx

ABOUT THE TEST FoundationOne®CDx is the first FDA-approved broad companion diagnostic for solid tumors.

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FoundationOne®CDx (F1CDx) is a qualitative next generation sequencing based in vitro diagnostic test that uses targeted high throughput hybridization-based capture technology for detection of substitutions, insertion and deletion alterations (indels), and copy number alterations (CNAs) in 324 genes and select gene rearrangements, as well as genomic signatures including microsatellite instability (MSI) and tumor mutational burden (TMB) using DNA isolated from formalin-fixed, paraffin embedded (FFPE) tumor tissue specimens. The test is intended as a companion diagnostic to identify patients who may benefit from treatment with the targeted therapies listed in Table 1 in accordance with the approved therapeutic product labeling. Additionally, F1CDx is intended to provide tumor mutation profiling to be used by qualified health care professionals in accordance with professional guidelines in oncology for patients with solid malignant neoplasms. Genomic findings other than those listed in Table 1 are not prescriptive or conclusive for labeled use of any specific therapeutic product.

The test is also used for detection of genomic loss of heterozygosity (LOH) from formalin-fixed, paraffin-embedded (FFPE) ovarian tumor tissue. Positive homologous recombination deficiency (HRD) status (F1CDx HRD defined as tBRCA-positive and/or LOH high) in ovarian cancer patients is associated with improved progression-free survival (PFS) from Rubraca (rucaparib) maintenance therapy in accordance with the Rubraca product label.

The F1CDx assay is performed at Foundation Medicine, Inc. sites located in Cambridge, MA and Morrisville, NC.

TABLE 1: COMPANION DIAGNOSTIC INDICATIONS

INDICATION	BIOMARKER	THERAPY
Non-small cell lung cancer (NSCLC)	*EGFR* exon 19 deletions and *EGFR* exon 21 L858R alterations	Gilotrif® (Afatinib), Iressa® (Gefitinib), Tagrisso® (Osimertinib), or Tarceva® (Erlotinib)
	EGFR exon 20 T790M alterations	Tagrisso® (Osimertinib)
	ALK rearrangements	Alecensa® (Alectinib), Alunbrig® (Brigatinib), Xalkori® (Crizotinib), or Zykadia® (Ceritinib)
	BRAF V600E	Tafinlar® (Dabrafenib) in combination with Mekinist® (Trametinib)
	MET single nucleotide variants (SNVs) and indels that lead to *MET* exon 14 skipping	Tabrecta® (Capmatinib)
Melanoma	*BRAF* V600E	BRAF Inhibitor Approved by FDA*
	BRAF V600E and V600K	Mekinist® (Trametinib) or BRAF/MEK Inhibitor Combinations Approved by FDA*
Breast cancer	*ERBB2* (HER2) amplification	Herceptin® (Trastuzumab), Kadcyla® (Ado-trastuzumab emtansine), or Perjeta® (Pertuzumab)
	PIK3CA C420R, E542K, E545A, E545D [1635G>T only], E545G, E545K, Q546E, Q546R, H1047L, H1047R, and H1047Y alterations	Piqray® (Alpelisib)
Colorectal cancer	*KRAS* wild-type (absence of mutations in codons 12 and 13)	Erbitux® (Cetuximab)
	KRAS wild-type (absence of mutations in exons 2, 3, and 4) and *NRAS* wild type (absence of mutations in exons 2, 3, and 4)	Vectibix® (Panitumumab)
Ovarian cancer	*BRCA1/2* alterations	Lynparza® (Olaparib) or Rubraca® (Rucaparib)
Cholangiocarcinoma	*FGFR2* fusions and select rearrangements	Pemazyre® (Pemigatinib) or Truseltiq™ (Infigratinib)
Prostate cancer	Homologous Recombination Repair (*HRR*) gene (*BRCA1*, *BRCA2*, *ATM*, *BARD1*, *BRIP1*, *CDK12*, *CHEK1*, *CHEK2*, *FANCL*, *PALB2*, *RAD51B*, *RAD51C*, *RAD51D* and *RAD54L*) alterations	Lynparza® (Olaparib)
Solid Tumors	*TMB* ≥ 10 mutations per megabase	Keytruda® (Pembrolizumab)
	NTRK1/2/3 fusions	Vitrakvi® (Larotrectinib)

**For the most current information about the therapeutic products in this group, go to: https://www.fda.gov/medicaldevices/productsandmedicalprocedures/invitrodiagnostics/ucm301431.htm*

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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	PHYSICIAN	SPECIMEN
DISEASE Colon adenocarcinoma (CRC)	ORDERING PHYSICIAN	SPECIMEN SITE
NAME	MEDICAL FACILITY	SPECIMEN ID
DATE OF BIRTH	ADDITIONAL RECIPIENT	SPECIMEN TYPE
SEX	MEDICAL FACILITY ID	DATE OF COLLECTION
MEDICAL RECORD #	PATHOLOGIST	SPECIMEN RECEIVED

Biomarker Findings

Microsatellite status - MSI-High
Tumor Mutational Burden - 47 Muts/Mb

Genomic Findings

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

KRAS wildtype
NRAS wildtype
BRCA2 T3033fs*29
MAP2K1 (MEK1) K57N
CTNNB1 R449C - subclonal†
ERBB3 V104M
PDGFRB V823I - subclonal†
ASXL1 G646fs*12
ATR I774fs*5
BCOR E623*
BCORL1 P1681fs*20 - subclonal†
CREBBP R1446C
CUL3 R733* - subclonal†
HNF1A P291fs*51
MLH1 E663D, Q445*
MLL2 A2205fs*59, P648fs*283
MSH3 K383fs*32
MSH6 F1088fs*5, R361H - subclonal†
SPEN A2251fs*102 - subclonal, R806fs*14, I1052fs*40†

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

† See About the Test in appendix for details.

Report Highlights

- Targeted therapies with **NCCN categories of evidence** in this tumor type: **Cetuximab** (p. 17), **Dostarlimab** (p. 17), **Nivolumab** (p. 18), **Nivolumab + Ipilimumab** (p. 19), **Panitumumab** (p. 19), **Pembrolizumab** (p. 20)
- Evidence-matched **clinical trial options** based on this patient's genomic findings: (p. 26)
- Variants with **prognostic implications** for this tumor type that may impact treatment decisions: **Microsatellite status MSI-High** (p. 4), ***MLH1* E663D, Q445*** (p. 14), ***MSH6* F1088fs*5, R361H** (p. 16)
- Variants in select cancer susceptibility genes to consider for possible **follow-up germline testing** in the appropriate clinical context: ***BRCA2* T3033fs*29** (p. 7), ***MLH1* E663D, Q445*** (p. 14), ***MSH6* F1088fs*5** (p. 16)
- Variants that may represent **clonal hematopoiesis** and may originate from non-tumor sources: ***ASXL1* G646fs*12** (p. 11), ***MLL2* A2205fs*59, P648fs*283** (p. 15)

BIOMARKER FINDINGS	THERAPIES WITH CLINICAL RELEVANCE (IN PATIENT'S TUMOR TYPE)		THERAPIES WITH CLINICAL RELEVANCE (IN OTHER TUMOR TYPE)
Microsatellite status - MSI-High	Dostarlimab	2A	Atezolizumab
	Nivolumab	2A	Avelumab
	Nivolumab + Ipilimumab	2A	Cemiplimab
	Pembrolizumab	2A	Durvalumab
10 Trials see p. 26			
Tumor Mutational Burden - 47 Muts/Mb	Dostarlimab		Atezolizumab
	Nivolumab		Avelumab
	Nivolumab + Ipilimumab		Cemiplimab
	Pembrolizumab		Durvalumab
10 Trials see p. 28			

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GENOMIC FINDINGS	THERAPIES WITH CLINICAL RELEVANCE (IN PATIENT'S TUMOR TYPE)	THERAPIES WITH CLINICAL RELEVANCE (IN OTHER TUMOR TYPE)
KRAS - wildtype	Cetuximab 2A	none
0 Trials	Panitumumab 2A	
NRAS - wildtype	Cetuximab 2A	none
0 Trials	Panitumumab 2A	
BRCA2 - T3033fs*29	none	Niraparib
		Olaparib
		Rucaparib
10 Trials see p. 30		Talazoparib
MAP2K1 (MEK1) - K57N	none	Selumetinib
10 Trials see p. 34		Trametinib
CTNNB1 - R449C - subclonal	none	none
5 Trials see p. 32		
ERBB3 - V104M	none	none
1 Trial see p. 33		
PDGFRB - V823I - subclonal	none	none
4 Trials see p. 36		

NCCN category

VARIANTS TO CONSIDER FOR FOLLOW-UP GERMLINE TESTING IN SELECT CANCER SUSCEPTIBILITY GENES

Findings below have been previously reported as pathogenic germline in the ClinVar genomic database and were detected at an allele frequency of >10%. See appendix for details.

BRCA2 - T3033fs*29 ..... p. 7 ***MSH6*** - F1088fs*5 ..... p. 16

MLH1 - E663D, Q445* ..... p. 14

This report does not indicate whether variants listed above are germline or somatic in this patient. In the appropriate clinical context, follow-up germline testing would be needed to determine whether a finding is germline or somatic.

VARIANTS THAT MAY REPRESENT CLONAL HEMATOPOIESIS (CH)

Genomic findings below may include nontumor somatic alterations, such as CH. The efficacy of targeting such nontumor somatic alterations is unknown. This content should be interpreted based on clinical context. Refer to appendix for additional information on CH.

ASXL1 - G646fs*12 ..... p. 11 ***MLL2*** - A2205fs*59, P648fs*283 ..... p. 15

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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.

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BIOMARKER

Microsatellite status

RESULT
MSI-High

POTENTIAL TREATMENT STRATEGIES

— Targeted Therapies —

On the basis of prospective clinical evidence in multiple solid tumor types, microsatellite instability (MSI) and associated increased tumor mutational burden (TMB)[1-2] may predict sensitivity to immune checkpoint inhibitors, including the approved PD-1-targeting agents cemiplimab, dostarlimab, nivolumab (alone or in combination with ipilimumab), and pembrolizumab[3-8] and PD-L1-targeting agents atezolizumab, avelumab, and durvalumab[9-11] . Pembrolizumab therapy resulted in a significantly higher ORR in MSI-H CRC compared with MSS CRC (40% vs. 0%)[6]. Similarly, a clinical study of nivolumab, alone or in combination with ipilimumab, in patients with CRC reported a significantly higher response rate in patients with tumors with high MSI than those without[3-4]. An earlier case study reported that nivolumab therapy resulted in a complete response in a patient with MSI-H CRC[5]. A Phase 1b trial of atezolizumab combined with bevacizumab reported PRs for 40% (4/10) of patients with MSI-H CRC[9].

— Nontargeted Approaches —

MSI has not been found to be a predictive biomarker for combination chemotherapy regimens, including FOLFOX[12-13] and FOLFIRI[14-15]. MSI and deficient MMR are associated with lack of benefit of postsurgical fluorouracil (FU)-based adjuvant therapy[16-17] but may predict benefit from irinotecan chemotherapy[18].

FREQUENCY & PROGNOSIS

MSI-H colorectal cancers (CRCs) make up 10–15% of CRC cases[2,19-22]. For patients with Stage 2 colorectal cancer, deficient DNA mismatch repair (dMMR) and MSI-High status are associated with better prognosis (NCCN Colon Cancer Guidelines, v3.2021); however, the prognostic impact for patients with more advanced cancer is less clear[23-24]. Multiple studies have shown that MSI-H CRCs have a better prognosis than MSI-low (MSI-L) or microsatellite stable (MSS) tumors[19,25-31]. MSI-H CRCs are associated with certain pathologic and molecular features, including poor differentiation, right-sided and mucinous tumors, increased numbers of tumor infiltrating lymphocytes, diploidy, and a relatively high frequency of BRAF mutations[20-21,32].

FINDING SUMMARY

Microsatellite instability (MSI) is a condition of genetic hypermutability that generates excessive amounts of short insertion/deletion mutations in the genome; it generally occurs at microsatellite DNA sequences and is caused by a deficiency in DNA mismatch repair (MMR) in the tumor[21]. Defective MMR and consequent MSI occur as a result of genetic or epigenetic inactivation of one of the MMR pathway proteins, primarily MLH1, MSH2, MSH6, or PMS2[21,33-34]. This sample has a high level of MSI, equivalent to the clinical definition of an MSI-high (MSI-H) tumor: one with mutations in >30% of microsatellite markers[20,32,35]. MSI-H status indicates high-level deficiency in MMR and typically correlates with loss of expression of at least one, and often two, MMR family proteins[20-21,32,34].

POTENTIAL GERMLINE IMPLICATIONS

While approximately 80% of MSI-H tumors arise due to somatic inactivation of an MMR pathway protein, about 20% arise due to germline mutations in one of the MMR genes[21], which are associated with a condition known as Lynch syndrome (also known as hereditary nonpolyposis colorectal cancer or HNPCC)[36]. Lynch syndrome leads to an increased risk of colorectal, endometrial, gastric, and other cancers[36-38] and has an estimated prevalence in the general population ranging from 1:600 to 1:2000[39-41]. Therefore, in the appropriate clinical context, germline testing of MLH1, MSH2, MSH6, and PMS2 is recommended.

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BIOMARKER

Tumor Mutational Burden

RESULT
47 Muts/Mb

POTENTIAL TREATMENT STRATEGIES

— Targeted Therapies —

On the basis of clinical evidence in solid tumors, increased TMB may be associated with greater sensitivity to immunotherapeutic agents, including anti-PD-L1[42-44], anti-PD-1 therapies[42-45], and combination nivolumab and ipilimumab[46-51]. In multiple pan-tumor studies, higher TMB has been reported to be associated with increased ORR and OS from treatment with immune checkpoint inhibitors[42-45,52]. Higher TMB was found to be significantly associated with improved OS upon immune checkpoint inhibitor treatment for patients with 9 types of advanced tumors[42]. Analyses across several solid tumor types reported that patients with higher TMB (defined as ≥16-20 Muts/Mb) achieved greater clinical benefit from PD-1 or PD-L1-targeting monotherapy, compared with patients with higher TMB treated with chemotherapy[53] or those with lower TMB treated with PD-1 or PD-L1-targeting agents[43]. However, the KEYNOTE 158 trial of pembrolizumab monotherapy for patients with solid tumors found significant improvement in ORR for patients with TMB ≥10 Muts/Mb (based on this assay or others) compared to those with TMB <10 Muts/Mb, in a large cohort that included multiple tumor types; similar findings were observed in the KEYNOTE 028 and 012 trials[45,52]. Together, these studies suggest that patients with TMB ≥10 Muts/Mb may derive clinical benefit from PD-1 or PD-L1 inhibitors. In CRC specifically, a retrospective analysis of immune checkpoint inhibitor efficacy reported significantly improved OS for patients with tumors harboring TMB ≥9.8 Muts/MB compared with those with tumors with TMB <9.8 Muts/Mb (~ equivalency <12 Muts/Mb as measured by this assay)[42]. Another retrospective study reported that a TMB ≥12 Muts/Mb cutoff identifies >99% of MSI-High CRC cases but only 3% of MSS cases, indicating the utility of this cutoff for identification of patients with CRC likely to benefit from treatment with immune checkpoint inhibitors[54].

FREQUENCY & PROGNOSIS

Elevated tumor mutational burden (TMB) has been reported in 8-25% of colorectal cancer (CRC) samples[22,55-56]. Multiple studies have reported that up to 90% of hypermutated CRC cases exhibit high levels of microsatellite instability (MSI-H) and mismatch repair deficiency (MMR-D)[22,55]. Increased TMB is significantly associated with MSI-H and MMR-D, with studies reporting that 100% of MSI-H CRCs harbor elevated TMB and conversely that 100% of tumors with low TMB harbor intact MMR[55]. A subset of CRCs that harbor increased TMB but not MSI-H are driven by mutations in POLE, which leads to an "ultramutated" phenotype with especially high TMB[22,55]. Tumors with increased TMB harbor BRAF V600E mutations more frequently than those with low TMB[22,55], whereas TMB-low tumors more frequently harbor mutations in TP53 and APC[22]. In a study for 61 patients with metastatic, microsatellite stable (MSS) CRC treated with best standard of care, plasma TMB scores ≥28 muts/Mb (approximately 14 muts/Mb as measured by this assay) were associated with reduced OS as compared with plasma TMB scores <28 muts/Mb (3.0 vs. 5.3 months, HR 0.76, p=0.007), whereas tissue TMB was not found to be prognostic in this population[57].

FINDING SUMMARY

Tumor mutation burden (TMB, also known as mutation load) is a measure of the number of somatic protein-coding base substitution and insertion/deletion mutations occurring in a tumor specimen. TMB is affected by a variety of causes, including exposure to mutagens such as ultraviolet light in melanoma[58-59] and cigarette smoke in lung cancer[7,60], treatment with temozolomide-based chemotherapy in glioma[61-62], mutations in the proofreading domains of DNA polymerases encoded by the POLE and POLD1 genes[22,63-66], and microsatellite instability (MSI)[22,63,66]. This sample harbors a TMB level that may be associated with sensitivity to PD-1- or PD-L1-targeting immune checkpoint inhibitors, alone or in combination with other agents[42,52,54].

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