Patient ID	Patient Name		Birth Date	Sex	Age
SA00178385	TESTING VALIDATION, MCCRC ABNORMAL		1966-02-28	M	59
Order Number	Client Order Number	Ordering Physician	Report Notes		
SA00178385	SA00178385	CLIENT,CLIENT			
Account Information		Collected			
C7028846 DLMP Rochester		01 Dec 2025 10:00			

MayoComplete CRC Panel

Result

MCR

Provided diagnosis: colorectal adenocarcinoma involving omentum

Microsatellite Instability (MSI) status: Stable (MSS)

The following CLINICALLY RELEVANT VARIANT was detected:
Gene: BRAF
DNA Change: c.1781A>G (Exon 15)
Amino Acid Change: p.D594G (Asp594Gly)
Variant Allele Frequency: 3%

No other reportable sequence variants were detected within the analyzed regions of the tested gene(s) listed in the method description.

Interpretation

MCR

MSI: Stable (MSS)
NO evidence of microsatellite instability suggesting the presence of normal DNA mismatch repair function within the tumor. Current data suggest that advanced stage solid tumors with intact mismatch repair (MSS) are less likely to be responsive to treatment with immunotherapies such as anti-PD-1 therapies [PMID 28596308, PMID 29355075].

The presence of intact mismatch repair (MSS) is considered to be an unfavorable prognostic factor for patients with colorectal cancer [PMID: 15659508].

These results decrease the likelihood but do not eliminate the possibility that this individual has Lynch syndrome. These results do not rule out the possibility that this individual's tumor is due to an inherited defect in another gene not involved in DNA mismatch repair. A significant fraction of clinically defined Lynch syndrome cases (30% or more) do not have defective DNA mismatch repair as the underlying genetic basis of their disease. Additionally, we cannot rule out the possibility that this tumor could represent a sporadic occurrence. If there is a strong personal or family history of Lynch syndrome related cancers for this patient or if this individual has multiple tumors, consider microsatellite instability (MSI) and immunohistochemical staining (IHC) on a different tumor to further evaluate the possible role of defective DNA mismatch repair for this individual or family. A genetic consult may be of benefit.

1) BRAF c.1781A>G (p.D594G) (Exon 15)

BRAF encodes the signaling protein Braf, which is downstream of Ras and activates the MAPK pathway. Braf signaling is critically involved in the processes of cell division and differentiation. BRAF activating mutations occur predominantly at a single location (V600E). BRAF activating mutations or amplification have been reported to result in uncontrolled cell growth and tumorigenesis [PMID:12068308, PMID:21447722].

BRAF mutations have been reported in 11–12% of colon samples [COSMIC, cBioPortal for Cancer Genomics].

Current data suggest that the efficacy of EGFR-targeted therapies alone in colorectal cancer is limited to patients with tumors lacking BRAF V600E mutations [PMID 23666916, PMID 19001320].

There are currently no known clinically approved therapies that specifically target BRAF non-V600 mutations.

Additional Information

MCR

CLINICAL TRIALS

Possible clinical trials of benefit for this patient can be found at the following sites:
1) ClinicalTrials.gov: www.clinicaltrials.gov/ct2/search/advanced
2) Mayo Clinic: www.mayo.edu/research/clinical-trials/ 3) National Cancer Institute: www.cancer.gov/clinicaltrials/search

REFERENCE TRANSCRIPT

Sequence variant nomenclature is based on the following RefSeq accession number (build GRCh37 (hg19)): BRAF NM_004333.

Performing Site Legend

Code	Laboratory	Address	Lab Director	CLIA Certificate
MCR	Mayo Clinic Laboratories - Rochester Main Campus	200 First Street SW, Rochester, MN 55905	Nikola Baumann Ph.D.	24D0404292

MAYO CLINIC LABORATORIES

Patient ID	Patient Name		Birth Date	Sex	Age
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C7028846 DLMP Rochester		01 Dec 2025 10:00			

Specimen

MCR

Tissue, Tumor

Tissue ID

MCR

13245

Method

MCR

Microscopic examination is performed by a pathologist to identify areas of tumor for enrichment by macrodissection.

Next generation sequencing is performed to test for the presence of microsatellite instability and a mutation within all coding regions and exon/intron boundaries of the following 13 targeted genes: APC, BRAF, HRAS, KRAS, MLH1, MSH2, MSH6, NRAS, PIK3CA, PIK3R1, PMS2, POLE and PTEN.

Variant nomenclature is based on build GRCh37 (hg19). For details about gene transcripts (RefSeq accession numbers), specific targeted regions of each gene, and additional information on this test, see www.mayocliniclabs.com (Test ID MCCRC).

Disclaimer

1 MCR

This test cannot differentiate between somatic and germline alterations. Additional testing may be necessary to clarify the significance of results if there is a potential hereditary risk.

DNA variants of uncertain significance may be identified.

A negative result does not rule out the presence of a variant that may be present but below the limits of detection of this assay. The analytical sensitivity of this assay for sequence reportable alterations is 5% mutant allele frequency with a minimum coverage of 500X in a sample with at least 20% tumor content.

Point mutations and small insertion/deletion mutations will be detected in the APC, BRAF, HRAS, KRAS, MLH1, MSH2, MSH6, NRAS, PIK3CA, PIK3R1, PMS2, POLE and PTEN genes only. This test may detect single exon deletions but does not detect multi-exon deletions, duplications, larger-scale genomic copy number variants, copy neutral loss of heterozygosity (LOH), or epigenetic modifications such as promoter methylation. DELINS of 1,000 bp or less are detectable with at least 50 or more supporting reads.

This test cannot reliably determine if a variant identified in PMS2 exons 11–15 originated from PMS2 or the highly homologous pseudogene PMS2CL. In the instance that a reportable variant is detected in PMS2 exons 11–15, additional testing will be recommended in the patient report.

Variant allele frequency (VAF) is the percentage of sequencing reads supporting a specific variant divided by the total sequencing reads at that position. In somatic testing, VAF should be interpreted in the context of several factors including, but not limited to: tumor purity/heterogeneity/copy number status (ploidy, gains/losses, loss of heterozygosity) and sequencing artifact/misalignment [PMID: 27144058, PMID: 29769535].

Rare polymorphisms may be present that could lead to false-negative or false-positive results.

The presence or absence of a variant may not be predictive of response to therapy in all patients.

Test results should be interpreted in the context of clinical, tumor sampling, histopathological, and other laboratory data. If results obtained do not match other clinical or laboratory findings, contact the laboratory for discussion. Misinterpretation of results may occur if the information provided is inaccurate and/or incomplete.

Reliable results are dependent on adequate specimen collection and processing. This test has been validated on cytology slides and formalin-fixed, paraffin-embedded tissues; other types of fixatives are discouraged. Improper treatment of tissues, such as decalcification, may cause PCR failure.

Released By

MCR

Benjamin R. Kipp, Ph.D.

Received: 02 Dec 2025 09:48 **Reported:** 03 Dec 2025 13:33

Performing Site Legend

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MCR	Mayo Clinic Laboratories - Rochester Main Campus	200 First Street SW, Rochester, MN 55905	Nikola Baumann Ph.D.	24D0404292

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Laboratory Notes

[1] This test was developed and its performance characteristics determined by Mayo Clinic in a manner consistent with CLIA requirements. This test has not been cleared or approved by the U.S. Food and Drug Administration.

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