## Patient

**Name:** YOUNG, ALEXANDER T

**Sex:** Male
**Case Number:**
**Diagnosis:** Adenocarcinoma, NOS

## Specimen Information

**Primary Tumor Site:** Rectum, NOS
**Specimen Site:** Rectum, NOS
**Specimen ID:**
**Specimen Collected:** 27-Mar-2024
**Test Report Date:** 03-Jun-2026

## Results with Therapy Associations

| BIOMARKER | METHOD | ANALYTE | RESULT | THERAPY ASSOCIATION | | BIOMARKER LEVEL* |
|---|---|---|---|---|---|---|
| **KRAS** | **Seq** | **DNA-Tumor** | **Mutation Not Detected** | **BENEFIT** | **panitumumab** | **Level 1** |
| | | | | | **cetuximab** | **Level 2** |
| **NRAS** | **Seq** | **DNA-Tumor** | **Mutation Not Detected** | **BENEFIT** | **panitumumab** | **Level 1** |
| | | | | | **cetuximab** | **Level 2** |
| BRAF | Seq | DNA-Tumor | Mutation Not Detected | BENEFIT | cetuximab, panitumumab# | Level 2 |

* Biomarker reporting classification - Level 1 – MI Cancer Seek™ or other companion diagnostic (CDx) performed as part of professional services; Level 2 – Strong evidence of clinical significance or is endorsed by standard clinical guidelines; Level 3 – Potential clinical significance. Bolded benefit therapies, if present, highlight the most clinically significant findings.

CARIS | FOLFIRSTai

**Result:** **INCREASED BENEFIT to FOLFOX + bevacizumab# in first-line metastatic CRC**

See Page 2 for important details about clinical data regarding Caris FOLFIRSTai™

## Important Note

This content is provided as part of the professional services. For MI Cancer Seek™ results, see CDx Associated Findings.

This specimen is a superficial biopsy with at least intramucosal carcinoma. Foci with definite invasion are rare. Testing is performed on the intramucosal carcinoma. Results should be interpreted with caution as the findings may not be indicative of those in invasive or metastatic carcinoma.

# In the first-line setting of metastatic colorectal cancer (mCRC), wild-type RAS status guides selection of EGFR-monoclonal antibody-based therapy, including cetuximab or panitumumab, which is given in combination with a chemotherapy backbone. The Caris FOLFIRSTai™ validation studies only evaluated the predictive performance in patients who received bevacizumab and did not include patients who received initial cetuximab or panitumumab-based treatments. Therefore, at this time, there are no data addressing the impact of substituting an EGFR-monoclonal antibody for bevacizumab with the FOLFOX chemotherapy backbone.

## Cancer-Type Relevant Biomarkers

| Biomarker | Method | Analyte | Result |
|---|---|---|---|
| MSI | Seq | DNA-Tumor | Stable |
| NTRK1/2/3 | Seq | RNA-Tumor | Fusion Not Detected |
| RET | Seq | RNA-Tumor | Fusion Not Detected |

| Biomarker | Method | Analyte | Result |
|---|---|---|---|
| Tumor Mutational Burden | Seq | DNA-Tumor | Low, 5 mut/Mb |
| BRAF | Seq | RNA-Tumor | Fusion Not Detected |

***(continued on next page)***

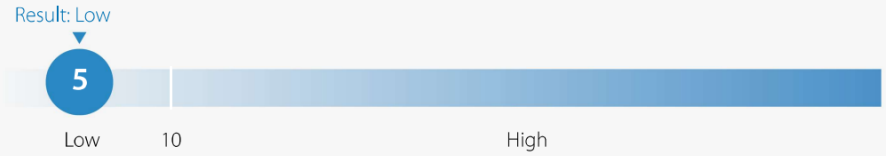
The selection of any, all, or none of the matched therapies resides solely with the discretion of the treating physician. Decisions on patient care and treatment must be based on the independent medical judgment of the treating physician, taking into consideration all available information concerning the patient's condition, the FDA prescribing information for any therapeutic, and in accordance with the applicable standard of care. Whether or not a particular patient will benefit from a selected therapy is based on many factors and can vary significantly. All trademarks and registered trademarks are the property of their respective owners.

## Cancer-Type Relevant Biomarkers *(continued)*

| Biomarker | Method | Analyte | Result |
|---|---|---|---|
| EGFR | CNA-Seq | DNA-Tumor | Amplification Not Detected |
| | Seq | DNA-Tumor | Mutation Not Detected |
| ERBB2 (Her2/Neu) | CNA-Seq | DNA-Tumor | Amplification Not Detected |

| Biomarker | Method | Analyte | Result |
|---|---|---|---|
| NF1 | Seq | DNA-Tumor | Mutation Not Detected |
| PIK3CA | Seq | DNA-Tumor | Mutation Not Detected |
| POLE | Seq | DNA-Tumor | Mutation Not Detected |

## 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 - 5% of tested genomic segments exhibited LOH (assay threshold is ≥ 16%) |



CARIS® FOLFIRSTai **Result:** **INCREASED BENEFIT to FOLFOX + bevacizumab in first-line metastatic CRC**

Intended Use and Result Interpretation:

**To determine the sequencing of therapy for patients who are not being considered for FOLFOXIRI:**

This patient may achieve improved results by receiving FOLFOX + bevacizumab as their initial regimen.

**As an adjustment to frontline FOLFOXIRI following toxicity:**

This patient may achieve improved results by removing the irinotecan portion of their regimen.

Caris FOLFIRSTai™ is a molecular signature that predicts relative benefit from FOLFOX + bevacizumab therapy given as the first-line treatment in metastatic colorectal cancer patients. The signature was developed using Caris Molecular Intelligence sequencing data and an artificial intelligence algorithm. The signature was validated using two independent data sets, as reported in Abraham, J.P., D.B. Spetzler, et al. (2021). "Clinical Validation of a Machine-learning-derived Signature Predictive of Outcomes from First-line Oxaliplatin-based Chemotherapy in Advanced Colorectal Cancer" Clin Cancer Res 27 (4): 1174-1183.

412 manually curated cases with real world evidence (insurance claims, electronic medical records and death registries):

**Median Overall Survival difference between the increased benefit arm and the decreased benefit arm: 17.5 months**

149 cases analyzed retrospectively from the randomized, prospective Phase III TRIBE2 study:

**Median Overall Survival difference between the increased benefit arm and the decreased benefit arm: 6.0 months**

All patients in the validation studies above had stage IV CRC and received FOLFOX + bevacizumab.

Any therapeutic decision should be based on the physician's judgement considering all of the patient's clinical conditions. Please see the Appendix of this report for Caris FOLFIRSTai™ methodology.

## Genes Tested with Pathogenic or Likely Pathogenic Alterations

| Gene | Method | Analyte | Variant Interpretation | Protein Alteration | Exon | DNA Alteration | Variant Frequency % |
|---|---|---|---|---|---|---|---|
| APC | Seq | DNA-Tumor | Pathogenic Variant | p.S1411fs | 16 | c.4233delT | 23 |
| | Seq | DNA-Tumor | Pathogenic Variant | p.V450fs | 11 | c.1349delT | 28 |
| PTEN | Seq | DNA-Tumor | Pathogenic Variant | p.R335* | 8 | c.1003C>T | 34 |
| SMARCA4 | Seq | DNA-Tumor | Likely Pathogenic Variant | p.D1177Y | 26 | c.3529G>T | 29 |
| SOX9 | Seq | DNA-Tumor | Pathogenic Variant | c.432-2A>T | 2 | c.432-2A>T | 78 |

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.

## Genes Tested with Variants of Uncertain Significance

| Gene | Method | Analyte | Variant Interpretation | Protein Alteration | Exon | DNA Alteration | Variant Frequency % |
|---|---|---|---|---|---|---|---|
| BRCA2 | Seq | DNA-Tumor | Variant of Uncertain Significance | p.W194R | 7 | c.580T>C | 34 |
| KIT | Seq | DNA-Tumor | Variant of Uncertain Significance | p.I403V | 7 | c.1207A>G | 46 |

Additional Variants of Uncertain Significance can be found in the MI Portal.

## Predicted Human Leukocyte Antigen (HLA) Genotype Results

The predicted HLA genotype results are summarized in the table below. Please note that HLA typing via tumor tissue sequencing is not a substitute for histocompatibility testing performed using peripheral blood. It is recommended to confirm the patient's HLA type with an appropriate assay.

| Gene | Method | Analyte | Genotype |
|---|---|---|---|
| MHC CLASS I | | | |
| HLA-A | Seq | DNA-Tumor | A*02:01, A*02:01 |
| HLA-B | Seq | DNA-Tumor | B*07:02, B*44:02 |
| HLA-C | Seq | DNA-Tumor | C*03:04, C*05:01 |

HLA genotypes with only one allele are either homozygous or have loss-of-heterozygosity at that position.

| Biomarker | Result | Lost Allele | Interpretation |
|---|---|---|---|
| HLA-A LOH | Negative | N/A | HLA-A loss of heterozygosity (LOH) is not detected |

## Genes Tested with Indeterminate Results by Tumor DNA Sequencing

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| ATP6AP2 | COL2A1 | DACH1 | MDH2 | PLCB4 | PRDM6 | PRKD1 | RASA1 | RXRA | SMARCA2 | | |

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

# Genes Tested with Intermediate CNA Results by Tumor DNA Sequencing

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| AURKA | GNAS | | | | | | | | | | |

The results in this report were curated to represent biomarkers most relevant for the submitted cancer type. These include results important for therapeutic decision-making, as well as notable alterations in other biomarkers known to be involved in oncogenesis. Additional results, including genes with normal findings, variants of uncertain significance, or unclassified alterations can be found in the MI Portal at **miportal.carismolecularintelligence.com.** If you do not have an MI Portal account, or need assistance accessing it, please contact Caris Customer Support at (888) 979-8669.

©2026 Caris Life Sciences. All rights reserved.

# Notes of Significance

**SEE APPENDIX FOR DETAILS**

CDx reports are generated through automated bioinformatics processing of a pre-defined list of genes and variants. Pathogenic or likely pathogenic variants identified outside the FDA-approved list will not be included in the CDx report. In addition, downgrading of variants approved by board-eligible/board-certified geneticists and pathologists may have occurred through manual variant annotation carried out by Caris' professional services. To ensure optimal coverage, multiple sequencing runs may be performed for a given case. This may account for some discrepancies observed between CDx reports and professional services.

Clinical Trials Connector ™ opportunities based on biomarker expression: 6 Targeted Therapies. See page 6 for details.

**Please Note:** This specimen is a superficial biopsy with at least intramucosal carcinoma. Foci with definite invasion are rare. Testing is performed on the intramucosal carcinoma. Results should be interpreted with caution as the findings may not be indicative of those in invasive or metastatic carcinoma.

# Specimen Information

**Specimen ID:**

**Specimen Collected:** 03/27/2024

**Specimen Received:** 05/20/2026

**Testing Initiated:** 05/30/2026

**Test Ordered*:** MI Cancer Seek™
* If the submitted specimen is inadequate, only a subset of the ordered testing may be reported.

**Gross Description:** 30 (A1-A30) Tissue Biopsy Slide unstained - Client from UCLA AP Referral Sendout Services - Westwood, Los Angeles, CA, with the corresponding pathology report labeled

**Dissection Information:** Molecular testing of this specimen was performed after harvesting of targeted tissues with an approved manual microdissection technique. Candidate slides were examined under a microscope and areas containing tumor cells (and separately normal cells, when necessary for testing) were circled. A laboratory technician harvested targeted tissues for extraction from the marked areas using a dissection microscope.

**H&E Tumor Assessment:**
Professional Component Performed: - - Location 5, Paradise Valley, AZ. Ahmed Bedeir, MD, Lab Director.

Ahmed Bedeir

Electronic Signature
Ahmed Bedeir, MD
05/30/2026

# Clinical Trials Connector™

The Clinical Trials Connector identifies clinical trials by matching reported biomarker status and tumor type to studies listed on ClinicalTrials.gov using internally curated matching logic. Trials listed below are within ~250 miles of the ordering physician's location and were active at the time this report was generated. Trial identification relies on information on http://clinicaltrials.gov/ and may be affected by missing or inaccurate trial information. Therefore, results may not include every potentially relevant study and some listed trials may not apply to an individual patient. For access to the entire list of trials matched based on biomarker and tumor type status, or to query the clinical trials connector at a later time point, visit: https://ctc.caris.ai/public/D2uuvF8K.

| Biomarker Directed Clinical Trials | | | | | |
|---|---|---|---|---|---|
| **Biomarker(s)** | **Investigational Agent(s)** | **NCT ID** | **Study Title** | **Trial Phase** | **Locations** |
| APC | FOG-001 | NCT05919264 | FOG-001 in Locally Advanced or Metastatic Solid Tumors | PHASE 1/2 | California (2) |
| PTEN | AMXI-5001:Dose Escalation Phase I | NCT04503265 | A Trial of AMXI-5001 for Treatment in Patients With Advanced Malignancies | PHASE 1/2 | California (1) |
| | nab-sirolimus | NCT05997017 | Trial of Nab-Sirolimus in Combination With Letrozole in Patients With Advanced or Recurrent Endometrioid Endometrial Cancer | PHASE 2 | Nevada (1) |
| | ETX-636 dose escalation | NCT06993844 | Phase 1/2 Study of ETX-636 in Participants With Advanced Solid Tumors | PHASE 1/2 | California (1) |
| | inavolisib | NCT04929223 | A Study Evaluating the Safety and Efficacy of Targeted Therapies in Subpopulations of Patients With Metastatic Colorectal Cancer (INTRINSIC) | PHASE 1 | California (1) |
| SMARCA4 | LY4050784 | NCT06561685 | A Study of LY4050784 in Participants With Advanced or Metastatic Solid Tumors | PHASE 1 | California (1) |

# Disclaimer

Decisions on patient care and treatment must be based on the independent medical judgment of the treating physician, taking into consideration all available information concerning the patient's condition, prescribing information for any therapeutic, and in accordance with the applicable standard of care. Drug associations provided in this report do not guarantee that any particular agent will be effective for the treatment of any patient or for any particular condition. Caris Life Sciences® expressly disclaims and makes no representation or warranty whatsoever relating, directly or indirectly, to the performance of services, including any information provided and/or conclusions drawn from therapies that are included or omitted from this report. Whether or not a particular patient will benefit from a selected therapy is based on many factors and can vary significantly. The selection of therapy, if any, resides solely in the discretion of the treating physician and the tests should not be considered a companion diagnostic.

Caris MPI, Inc. d/b/a Caris Life Sciences is certified under the Clinical Laboratory Improvement Amendments (CLIA) as qualified to perform high complexity clinical laboratory testing, including all Caris molecular profiling assays. Individual assays that are available through Caris molecular profiling include both Laboratory Developed Tests (LDT) and U.S. Food and Drug Administration (FDA) approved or cleared tests. In addition, certain tests have been CE-marked as a general IVD under the In Vitro Diagnostic Directive (IVDD) 98/79/EC. Offered LDTs were developed and their performance characteristics determined by Caris. Certain tests have not been cleared or approved by the FDA. Caris LDTs are used for clinical purposes. They are not investigational or for research. Caris' CLIA certification number is located at the bottom of each page of this report.

The information presented in the Clinical Trials Connector™ section of this report, if applicable, is compiled from sources believed to be reliable and current. However, the accuracy and completeness of the information provided herein cannot be guaranteed. The clinical trials information present in the biomarker description was compiled from www.clinicaltrials.gov. The contents are to be used only as a guide, and health care providers should employ their best comprehensive judgment in interpreting this information for a particular patient. Specific eligibility criteria for each clinical trial should be reviewed as additional inclusion criteria may apply.

All materials, documents, data, data software, information and/or inventions supplied to customers by or on behalf of Caris or created by either party relating to the services shall be and remain the sole and exclusive property of Caris. Customer shall not use or disclose the information provided by Caris through the services or related reports except in connection with the treatment of the patient for whom the services were ordered and shall not use such property for, or disseminate such property to, any third parties without expressed written consent from Caris. Customer shall deliver all such property to Caris immediately upon demand or upon Caris ceasing to provide the services. The technical and professional component of all testing was performed at the laboratory location displayed in the footer unless otherwise notated in the report.

Caris molecular testing is subject to Caris' intellectual property. Patent www.CarisLifeSciences.com/ip.

Professional Component Performed: - - Location 5, Paradise Valley, AZ. Ahmed Bedeir, MD, Lab Director.



Electronic Signature
Ahmed Bedeir, MD
03-Jun-2026

CARIS | MI CANCER SEEK

## Patient

**Name:** YOUNG, ALEXANDER T

**Sex:** Male
**Case Number:**
**CDx Report Generated:** 06/03/2026

## Specimen Information

**Diagnosis and Tumor Type:** Adenocarcinoma, NOS, Rectum, NOS
**Specimen Site:** Rectum, NOS
**Specimen ID:**
**Specimen Type:** Formalin-fixed paraffin embedded
**Specimen Collected:** 03/27/2024

## CDx Associated Findings

| Genomic Findings Detected | FDA-approved Therapeutic Options |
|---|---|
| ***KRAS Wild-Type (Exons 2, 3, 4)***<br>***NRAS Wild-Type (Exons 2, 3, 4)*** | VECTIBIX® (panitumumab) |
| ***BRAF V600E*** | Not Detected |
| ***Microsatellite Instability (MSI)*** | Not MSI-H |

## Tumor Profiling Results

MI Cancer Seek is FDA-approved to provide tumor mutation profiling results for previously diagnosed oncology patients with solid tumors.

## Other Alterations and Biomarkers Identified

Results reported in this section are not prescriptive or conclusive for labeled use of any specific therapeutic product.
Confirmation of tumor mutation status using an FDA-approved CDx test is needed for therapeutic use.

| | | | |
|---|---|---|---|
| ***Tumor Mutational Burden (TMB)*** | 5 mut/Mb | ***PTEN*** | R335* |
| ***APC*** | S1411fs<br>V450fs | ***SMARCA4*** | D1177Y |

CARIS | MI CANCER SEEK

# MI Cancer Seek™

## Intended Use:

MI Cancer Seek is a next-generation sequencing (NGS) based in vitro diagnostic (IVD) device using total nucleic acid (TNA) isolated from formalin-fixed paraffin embedded (FFPE) tumor tissue specimens for the detection of single nucleotide variants (SNVs) and insertions and deletions (indels) in 228 genes, microsatellite instability (MSI), tumor mutational burden (TMB) in patients with previously diagnosed solid tumors, and copy number amplification (CNA) in one gene in patients with breast cancer.

MI Cancer Seek is intended as a companion diagnostic to identify patients who may benefit from treatment with the targeted therapies listed in Table 1 below, in accordance with the approved therapeutic product labeling.

Additionally, MI Cancer Seek is intended to provide tumor mutational profiling to be used by qualified healthcare professionals in accordance with professional oncology guidelines for cancer patients with previously diagnosed 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.

Table 1. MI Cancer Seek Companion Diagnostic Indications

| INDICATION | BIOMARKER | THERAPY |
|---|---|---|
| Breast Cancer | PIK3CA (C420R, E542K, E545A, E545D [1635G>T only], E545G, E545K, Q546E, Q546R, H1047L, H1047R, H1047Y) | PIQRAY® (alpelisib) |
| Colorectal Cancer (CRC) | 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) |
| | BRAF V600E | BRAFTOVI® (encorafenib) in combination with ERBITUX® (cetuximab) |
| Melanoma | BRAF V600E | BRAF Inhibitors approved by FDA* |
| | BRAF V600E or BRAF V600K | MEKINIST® (trametinib) or BRAF/MEK Inhibitor Combinations approved by FDA* |
| Non-small cell lung cancer (NSCLC) | EGFR exon 19 deletions and exon 21 L858R alterations | EGFR Tyrosine Kinase Inhibitors approved by FDA* |
| Solid Tumors | MSI-H | KEYTRUDA® (pembrolizumab), JEMPERLI® (dostarlimab-gxly) |
| Endometrial Carcinoma | Not MSI-H | KEYTRUDA® (pembrolizumab) in combination with LENVIMA® (lenvatinib) |

* For the most current information about the therapeutic products in this group, go to:

www.fda.gov/medical-devices/in-vitro-diagnostics/list-cleared-or-approved-companion-diagnostic-devices-in-vitro-and-imaging-tools#Group_Labeling

MI Cancer Seek™ is a single-site assay performed at Caris Life Sciences, Phoenix, AZ.

## Contraindications:

There are no known contraindications.

## Warnings and Precautions:

Biopsy may pose a risk to the patient when archival tissue is not available for use with the assay. The patient's physician should determine whether the patient is a candidate for biopsy.

## Limitations:

- For in vitro diagnostic use.
- CAUTION: Federal law restricts this device to sale by or on the order of a physician.
- The acceptable preparation method for MI Cancer Seek CDx specimens is FFPE. Other preparations have not been evaluated.
- The test is designed to report out somatic variants and is not intended to report germline variants.
- MI Cancer Seek requires a minimum tumor percentage of 20% for detection of alterations, with tumor content enrichment recommended for specimens with tumor percentage lower than 20%.

©2026 Caris Life Sciences. All rights reserved.

- Genomic findings other than those listed in the Companion Diagnostic Indications table are not prescriptive or conclusive for labeled use of any specific therapeutic product. Confirmation of tumor mutation status using an FDA-approved CDx test is needed for therapeutic use.
- A negative result does not rule out the presence of a mutation below the limits of detection of the assay.
- MI Cancer Seek is only approved for use with Caris Life Sciences pre-qualified Illumina NovaSeq 6000 instruments.
- The test is intended to be performed on specific serial number-controlled instruments by Caris Life Sciences.
- MI Cancer Seek does not report TMB for values lower than 3 mut/Mb as the accuracy of TMB values below 3 mut/Mb are unreliable.
- Decisions on patient care and treatment must be based on the independent medical judgment of the treating physician, taking into consideration all applicable information concerning the patient's condition, such as patient and family history, physical examinations, information from other diagnostic tests, and patient preferences, in accordance with the standard of care in a given community.
- Based on the low positive percent agreement (PPA) in the accuracy study for ERBB2 copy number amplifications (CNAs) in breast cancer patients, this alteration may not be detected. Additional clinical investigation to confirm the presence of ERBB2 CNAs in the breast cancer patient's tumor with another FDA approved or cleared test is strongly recommended.
- Patients with breast cancer whose specimens have intermediate ERBB2 CNA status should be tested with another FDA approved or cleared test to ascertain ERRB2 CNA status in their tumor.
- Test may have reduced sensitivity and may yield false negative results in samples where necrotic tissue is >15%, melanin is >5%, fat cells are >10%.

## Test Principle:

MI Cancer Seek is a single-site assay performed at Caris Life Sciences located at 4610 South 44th Place, Phoenix, AZ 85040. The test includes reagents, software, and procedures for testing of total nucleic acid (TNA) from formalin fixed paraffin embedded (FFPE) tumor tissue. The test uses a custom bait panel to measure all coding regions of the exome to detect and report SNVs and indels within 228 genes outlined in Table 2 across solid tumors and amplifications in ERBB2 in patients with breast cancer only. The test also detects MSI (determined from 3,210 genes) and whole exome based TMB in patients with solid tumors.

**Table 2. MI Cancer Seek Reportable Gene List for SNVs and indels**

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| ABL1 | BARD1 | CDH1 | EP300 | FAT1 | H3F3B | KMT2D | MSH3 | NTRK2 | PRKAR1A | SDHA | STAT3 |
| ACVR1 | BCL2 | CDK12 | EPHA2 | FBXW7 | HIST1H3B | KRAS | MSH6 | NTRK3 | PRKDC | SDHAF2 | STK11 |
| AIP | BCL9 | CDK4 | ERBB2 | FGFR1 | HNF1A | LZTR1 | MTOR | PALB2 | PTCH1 | SDHB | SUFU |
| AKT1 | BCOR | CDKN1B | ERBB3 | FGFR2 | HOXB13 | MAP2K1 | MUTYH | PBRM1 | PTEN | SDHC | TCF7L2 |
| AKT2 | BLM | CDKN2A | ERBB4 | FGFR3 | HRAS | MAP2K2 | MYC | PDGFRA | PTPN11 | SDHD | TERT |
| AKT3 | BMPR1A | CHEK1 | ERCC2 | FGFR4 | IDH1 | MAP2K4 | MYCN | PDGFRB | RAC1 | SETD2 | TET2 |
| ALK | BRAF | CHEK2 | ESR1 | FH | IDH2 | MAP3K1 | MYD88 | PIK3CA | RAD50 | SF3B1 | TMEM127 |
| AMER1 | BRCA1 | CIC | EZH2 | FLCN | IRF4 | MAPK1 | NBN | PIK3CB | RAD51B | SMAD2 | TNFAIP3 |
| APC | BRCA2 | CREBBP | FANCA | FLT1 | JAK1 | MAX | NF1 | PIK3R1 | RAD51C | SMAD4 | TNFRSF14 |
| AR | BRIP1 | CSF1R | FANCB | FLT3 | JAK2 | MED12 | NF2 | PIK3R2 | RAD51D | SMARCA4 | TP53 |
| ARAF | BTK | CTCF | FANCC | FOXA1 | JAK3 | MEF2B | NFE2L2 | PIM1 | RAD54L | SMARCB1 | TRAF7 |
| ARID1A | CALR | CTNNA1 | FANCD2 | FOXL2 | KDM5C | MEN1 | NFKBIA | PMS2 | RAF1 | SMARCE1 | TSC1 |
| ARID2 | CARD11 | CTNNB1 | FANCE | FUBP1 | KDM6A | MET | NOTCH1 | POLD1 | RASA1 | SMO | TSC2 |
| ASXL1 | CBFB | CXCR4 | FANCF | GATA3 | KDR | MITF | NPM1 | POLE | RB1 | SOCS1 | U2AF1 |
| ATM | CCND1 | CYLD | FANCG | GNA11 | KEAP1 | MLH1 | NRAS | POT1 | RET | SOS1 | VHL |
| ATRX | CCND2 | DDR2 | FANCI | GNA13 | KIT | MLH3 | NSD1 | PPP2R1A | RHOA | SPEN | WRN |
| AXIN2 | CCND3 | DICER1 | FANCL | GNAQ | KLF4 | MPL | NSD2 | PPP2R2A | RNF43 | SPOP | WT1 |
| B2M | CD79B | DNMT3A | FANCM | GNAS | KMT2A | MRE11 | NTHL1 | PRDM1 | ROS1 | SRC | XPO1 |
| BAP1 | CDC73 | EGFR | FAS | H3F3A | KMT2C | MSH2 | NTRK1 | PRKACA | RUNX1 | STAG2 | XRCC1 |

©2026 Caris Life Sciences. All rights reserved.

FDA Evidence Levels:

Genomic findings other than those listed in the Intended Use are not prescriptive or conclusive for labeled use of any specif ic therapeutic product. Test results should be interpreted in the context of pathological evaluation of tumors, treatment history, clinical findings, and other laboratory data. The test report includes genomic findings reported in the following levels (Table 3).

Table 3. Classification Criteria for FDA Evidence Levels

| LEVEL | CRITERIA |
|---|---|
| Level 1 | Biomarker is FDA-approved as a companion diagnostic as part of MI Cancer Seek™ |
| Level 2 | Cancer alterations that are well-established with strong clinical evidence that the clinician must know according to professional consensus guidelines in the specific tumor type. |
| Level 3 | Cancer alterations with potential clinical significance, e.g., biomarkers deemed useful for directing patients to a clinical trial or simply for informational purposes.<br>i. Clinical data such as case reports, single or several case series, or Phase I/II clinical trial data that support the utility of specific biomarker alteration to direct a patient to clinical trials, or<br>ii Pre-clinical and/or in vitro studies provide structural analysis of the mutation, fusion, or isoform to confirm pathogenicity (tumor-promoting), sensitivity, or resistance through colony forming assays, growth inhibition or drug sensitivity assays, etc. |

 ©2026 Caris Life Sciences. All rights reserved. 

# Gene Expression

| Gene | Percentile in Cancer Type | Gene | Percentile in Cancer Type | Gene | Percentile in Cancer Type |
|---|---|---|---|---|---|
| ADAM9 | 23 | EREG | 75 | NTRK1 | 63 |
| ADORA2A | 24 | FGFR2 | 60 | NTRK2 | 26 |
| ALK | 65 | FGFR3 | 96 | NTRK3 | 48 |
| APC | 29 | FN1 | 4 | PDCD1 | 60 |
| AREG | 57 | FOLR1 | 37 | PDCD1LG2 | 48 |
| ATM | 42 | GNAS | 34 | PIK3CA | 18 |
| BRAF | 86 | HRAS | 78 | PIWIL1 | 70 |
| BRCA1 | 79 | IGF1R | 32 | PRAME | 67 |
| BRCA2 | 78 | ITGB6 | 72 | PTEN | 29 |
| BRD4 | 81 | KDM1A | 4 | PVRIG | 40 |
| CCND1 | 90 | KDR | 36 | RB1 | 46 |
| CCNE1 | 55 | KRAS | 72 | RET | 82 |
| CD274 | 48 | LAG3 | 46 | ROR1 | 53 |
| CD276 | 74 | MAGEA4 | 46 | ROR2 | 50 |
| CDH17 | 65 | MDM2 | 73 | ROS1 | 90 |
| CDH6 | 24 | MET | 82 | SRC | 94 |
| CDKN2A | 48 | MGMT | 98 | SSTR2 | 80 |
| CEACAM5 | 78 | MKI67 | 74 | SSTR3 | 82 |
| CLDN18 | 56 | MSLN | 66 | SSTR5 | 80 |
| CLDN4 | 96 | MTAP | 57 | TACSTD2 | 38 |
| CLDN6 | 72 | MTOR | 76 | TGFB1 | 36 |
| CTLA4 | 20 | MUC1 | 88 | TNFRSF1B | 62 |
| DKK1 | 38 | MUC16 | 46 | TOP1 | 66 |
| EGFR | 94 | MYC | 38 | TP53 | 98 |
| EPHA2 | 52 | NECTIN4 | 38 | TSC1 | 24 |
| EPHA5 | 58 | NF1 | 64 | TSC2 | 67 |
| ERBB2 | 60 | NRAS | 80 | VEGFA | 6 |
| ERBB3 | 42 | NRG1 | 92 | XPO1 | 7 |

**Gene Expression of Selected Genes by Whole Transcriptome Sequencing (WTS) Methods:**
Gene expression is derived from whole transcriptome sequencing. Relative expression of genes are calculated as normalized values using Transcripts per Million Molecules or TPM. TPM is presented as a percentile derived by comparison to a distribution of Caris' internal cohort of the tumor-type profiled. Selected genes reported in this section were chosen based on their tumor-type specific relevance for matching to clinical trials, or tumor type subclassification.

# Karyotype




**Karyotyping using Copy Number Analysis by Whole Exome Sequencing (WES) Methods:**
Whole exome sequencing in combination with interrogation of single nucleotide polymorphisms (SNPs) tiled throughout the genome, allows for the identification and visualization of cytogenetic aberrations.

Somatic structural variants like whole or partial chromosome duplications or deletions, are important for cancer development and progression, and may identify clinically actionable alterations.

# Colorectal Cancer Consensus Molecular Subtype (CMS) Classification

| Result |
| --- |
| CMS2 |

Gene expression data was utilized to validate a CMS classifier. Chowdhury S, Xiu J, Ribeiro JR, et al. Consensus molecular subtyping of metastatic colorectal cancer expands biomarker-directed therapeutic benefit for patients with CMS1 and CMS2 tumors. Br J Cancer. 2024;131(8):1328-1339.

Colorectal Cancer (CRC) Consensus Molecular Subtypes was established by an International Consortium described in Guinney J, Dienstmann R, Wang X, et al. The consensus molecular subtypes of colorectal cancer.#Nat Med. 2015;21(11):1350-1356.

Four molecular subtypes of CRC are defined below, though this classification is not recommended for clinical practice (NCCN Guidelines Colon/Rectal 2024).

**CMS Subtypes**
(1) CMS1- Microsatellite Instability (MSI-H) Immune, hypermutated, microsatellite instability high, with strong immune activation.
(2) CMS2 - Canonical, epithelial, chromosomal instability, with Wnt and MYC signaling pathway activation
(3) CMS3 - Metabolic, epithelial, metabolic dysregulation
(4) CMS4 - Mesenchymal, TGFB signaling pathway activation, angiogenic and stromal invasive features

# Mutational Analysis by Next-Generation Sequencing (NGS)

| TUMOR MUTATIONAL BURDEN | |
|---|---|
| **Mutations / Megabase** | **Result** |
| 5 | Low |

### TMB

Tumor Mutational Burden (TMB) is defined as the number of somatic non-synonymous mutations per million bases of sequenced DNA in a tumor sample. Tumors with high TMB may increase the number of neoantigens which is hypothesized to increase T-cell reactivity and potential for response to immune checkpoint inhibitors. TMB analysis was performed based on next generation sequencing analysis of genomic DNA isolated from a tumor sample.

| MICROSATELLITE INSTABILITY ANALYSIS | |
|---|---|
| **Test** | **Result** |
| MSI | Stable |

### MSI

Microsatellite instability (MSI) status is a measure of the number of somatic mutations within short, repeated sequences of DNA (microsatellites). MSI-High status can indicate that the tumor has a defect in mismatch repair (MMR) abrogating the ability to correct mistakes during DNA replication. Tumors with MSI-high status may increase the number of neoantigens which is hypothesized to increase T-cell reactivity and potential for response to immune checkpoint inhibitors. Tumor-only microsatellite instability status by NGS (MSI-NGS) is measured by the direct analysis of known microsatellite regions sequenced in the CMI NGS panel.

| GENOMIC LOSS OF HETEROZYGOSITY | |
|---|---|
| **Test** | **Result** |
| Genomic Loss of Heterozygosity (LOH) | Low - 5% of tested genomic segments exhibited LOH (assay threshold is ≥ 16%) |

### LOH

To calculate genomic loss-of-heterozygosity (LOH), the 22 autosomal chromosomes are split into 552 segments and the LOH of single nucleotide polymorphisms (SNPs) within each segment is calculated. Caris WES data consist of approximately 250k SNPs spread across the genome. SNP alleles with frequencies skewed towards 0 or 100% indicate LOH (heterozygous SNP alleles have a frequency of 50%). The final call of genomic LOH is based on the percentage of all 552 segments with observed LOH.

*Additional Next-Generation Sequencing results continued on the next page. >*

# Mutational Analysis by Next-Generation Sequencing (NGS)

| Gene | Analyte | Variant Interpretation | Protein Alteration | Exon | DNA Alteration | Variant Frequency % | Transcript ID |
|---|---|---|---|---|---|---|---|
| APC | DNA-Tumor | Pathogenic Variant | p.S1411fs | 16 | c.4233delT | 23 | NM_000038.5 |

**Interpretation:** A pathogenic frameshift mutation was detected in APC.

APC or adenomatous polyposis coli is a key tumor suppressor gene that encodes for a large multi-domain protein. This protein exerts its tumor suppressor function in the Wnt/ß-catenin cascade mainly by controlling the degradation of ß-catenin, the central activator of transcription in the Wnt signaling pathway. The Wnt signaling pathway mediates important cellular functions including intercellular adhesion, stabilization of the cytoskeleton, and cell cycle regulation and apoptosis, and it is important in embryonic development and oncogenesis. Mutation in APC results in a truncated protein product with abnormal function, lacking the domains involved in ß-catenin degradation. Somatic mutation in the APC gene can be detected in the majority of colorectal tumors (80%) and it is an early event in colorectal tumorigenesis. APC wild type patients have shown better disease control rate in the metastatic setting when treated with oxaliplatin, while when treated with fluoropyrimidine regimens, APC wild type patients experience more hematological toxicities. APC mutation has also been identified in oral squamous cell carcinoma, gastric cancer as well as hepatoblastoma and may contribute to cancer formation. Germline mutation in APC causes familial adenomatous polyposis, which is an autosomal dominant inherited disease that will inevitably develop to colorectal cancer if left untreated. COX-2 inhibitors including celecoxib may reduce the recurrence of adenomas and incidence of advanced adenomas in individuals with an increased risk of CRC. Turcot syndrome and Gardner's syndrome have also been associated with germline APC defects. Germline mutations of the APC have also been associated with an increased risk of developing desmoid disease, papillary thyroid carcinoma and hepatoblastoma.

| Gene | Analyte | Variant Interpretation | Protein Alteration | Exon | DNA Alteration | Variant Frequency % | Transcript ID |
|---|---|---|---|---|---|---|---|
| APC | DNA-Tumor | Pathogenic Variant | p.V450fs | 11 | c.1349delT | 28 | NM_000038.5 |

**Interpretation:** A pathogenic frameshift mutation was detected in APC.

| Gene | Analyte | Variant Interpretation | Protein Alteration | Exon | DNA Alteration | Variant Frequency % | Transcript ID |
|---|---|---|---|---|---|---|---|
| BRCA2 | DNA-Tumor | Variant of Uncertain Significance | p.W194R | 7 | c.580T>C | 34 | NM_000059.3 |

**Interpretation:** A variant with no known clinical or functional significance was detected in BRCA2.

BRCA2 or breast cancer type 2 susceptibility gene encodes a protein involved in cell growth, cell division, and DNA-damage repair. It is a tumor suppressor gene which plays an important role in mediating double-strand DNA breaks by homologous recombination (HR). Tumors with BRCA2 mutation may be more sensitive to platinum agents and PARP inhibitors.

| Gene | Analyte | Variant Interpretation | Protein Alteration | Exon | DNA Alteration | Variant Frequency % | Transcript ID |
|---|---|---|---|---|---|---|---|
| KIT | DNA-Tumor | Variant of Uncertain Significance | p.I403V | 7 | c.1207A>G | 46 | NM_000222.2 |

**Interpretation:** A rare variant with no known clinical or biological significance was found in KIT.

c-KIT is a receptor tyrosine kinase expressed by hematopoietic stem cells, interstitial cells of cajal (pacemaker cells of the gut) and other cell types. Upon binding of c-KIT to stem cell factor (SCF), receptor dimerization initiates a phosphorylation cascade resulting in proliferation, apoptosis, chemotaxis and adhesion. cKIT mutations have been identified in various cancer types including gastrointestinal stromal tumors (GIST) (up to 85%) and melanoma (chronic sun damage type, acral or mucosal) (20-40%). Germline mutations in c-KIT have been associated with multiple gastrointestinal stromal tumors (GIST) and Piebald trait.

*Additional Next-Generation Sequencing results continued on the next page. >*

# Mutational Analysis by Next-Generation Sequencing (NGS)

| Gene | Analyte | Variant Interpretation | Protein Alteration | Exon | DNA Alteration | Variant Frequency % | Transcript ID |
|---|---|---|---|---|---|---|---|
| PTEN | DNA-Tumor | Pathogenic Variant | p.R335* | 8 | c.1003C>T | 34 | NM_000314.6 |

**Interpretation:** A pathogenic mutation was detected in PTEN. Germline mutations in the PTEN gene are causal for PTEN Hamartoma Tumor Syndrome (Cowden syndrome).

PTEN or phosphatase and tensin homolog is a tumor suppressor gene that prevents cells from proliferating. PTEN is an important mediator in signaling downstream of EGFR, and loss of PTEN gene function/expression due to gene mutations or allele loss is associated with reduced benefit to EGFR-targeted monoclonal antibodies. Mutation in PTEN is found in 5-14% of colorectal cancer and 7% of breast cancer. PTEN mutation leads to loss of function of the encoded phosphatase, and an upregulation of the PIK3CA/AKT pathway. Germline PTEN mutations associate with Cowden disease and Bannayan-Riley-Ruvalcaba syndrome. These dominantly inherited disorders belong to a family of hamartomatous polyposis syndromes which feature multiple tumor-like growths (hamartomas) accompanied by an increased risk of breast carcinoma, follicular carcinoma of the thyroid, glioma, prostate and endometrial cancer. Trichilemmoma, a benign, multifocal neoplasm of the skin is also associated with PTEN germline mutations.

| Gene | Analyte | Variant Interpretation | Protein Alteration | Exon | DNA Alteration | Variant Frequency % | Transcript ID |
|---|---|---|---|---|---|---|---|
| SMARCA4 | DNA-Tumor | Likely Pathogenic Variant | p.D1177Y | 26 | c.3529G>T | 29 | NM_001128844.1 |

**Interpretation:** This SMARCA4 mutation is presumed to be pathogenic due to its recurrence in cancers.

The protein encoded by this gene is a member of the SWI/SNF family of proteins and is similar to the brahma protein of Drosophila. Members of this family have helicase and ATPase activities and are thought to regulate transcription of certain genes by altering the chromatin structure around those genes. In addition, this protein can bind BRCA1, as well as regulate the expression of the tumorigenic protein CD44. Mutations in this gene cause rhabdoid tumor predisposition syndrome type 2.

| Gene | Analyte | Variant Interpretation | Protein Alteration | Exon | DNA Alteration | Variant Frequency % | Transcript ID |
|---|---|---|---|---|---|---|---|
| SOX9 | DNA-Tumor | Pathogenic Variant | c.432-2A>T | 2 | c.432-2A>T | 78 | NM_000346.3 |

**Interpretation:** A pathogenic mutation that disrupts an intron splice site was detected in SOX9.

*Additional Next-Generation Sequencing results continued on the next page. >*

# Mutational Analysis by Next-Generation Sequencing (NGS)

| GENES TESTED WITH INDETERMINATE* RESULTS BY TUMOR DNA SEQUENCING | | | | | |
|---|---|---|---|---|---|
| ATP6AP2 | DACH1 | PLCB4 | PRKD1 | RXRA | |
| COL2A1 | MDH2 | PRDM6 | RASA1 | SMARCA2 | |

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

**For a complete list of genes tested, visit www.CarisMolecularIntelligence.com/profilemenu.**

**NGS Methods**

Direct sequence analysis was performed on genomic DNA isolated from a micro-dissected tumor sample using Illumina NovaSeq 6000 sequencers. A hybrid pull-down panel of baits was used to enrich more than 700 clinically relevant genes along with > 20,000 other genes. Sequence data is analyzed using a customized bioinformatics pipeline to detect sequencing variants, copy number alterations (amplifications and deletions) indels and predicted HLA genotypes, including copy number estimation of HLA LOH. In addition, genomic signatures for tumor mutational burden (TMB), microsatellite instability (MSI), genomic loss-of-heterozygosity (LOH) or HRD-Genomic Scar Score (HRD-GSS), and homologous recombination deficiency (HRD) are reported when applicable. For a complete list of what is covered by the assay, and genes with partial coverage, please contact Caris Customer Support.

©2026 Caris Life Sciences. All rights reserved.

# Copy Number Alterations by Next-Generation Sequencing (NGS)

| GENES TESTED WITH INTERMEDIATE CNA RESULTS | | | | | |
|---|---|---|---|---|---|
| AURKA | GNAS | | | | |

| GENES WITH INDETERMINATE CNA RESULTS | | | | | |
|---|---|---|---|---|---|
| BCL2 | SETBP1 | SMAD2 | SMAD4 | ZNF521 | |

**CNA Methods**

The copy number alteration (CNA) of each exon is determined by a calculation using the average sequencing depth of the sample along with the sequencing depth of each exon and comparing this calculated result to a pre-calibrated value. A complete list of genes for reporting copy number alterations, including amplifications and deletions, is available upon request.

# Caris FOLFIRSTai™ by Next-Generation Sequencing (NGS)

CARIS® FOLFIRSTai **Result:** **INCREASED BENEFIT to FOLFOX + bevacizumab in first-line metastatic CRC**

**Methods**

The Caris FOLFIRSTai™ predictor is comprised of 5,000 machine learning algorithms which are trained to identify patients with metastatic colorectal cancer (mCRC) that may exhibit increased benefit (IB) to first-line FOLFOX + bevacizumab from those that may exhibit decreased benefit (DB). The algorithms use information from the Caris Life Sciences Whole Exome Sequencing panel to make a single aggregated prediction for each patient. The prediction is not a guarantee that IB or DB status will be realized on a regimen of FOLFOX + bevacizumab. The predictor was validated against two blinded, stage IV CRC cohorts: i) 412 cases from a manually curated real world evidence dataset of insurance claims and electronic medical records that received first-line FOLFOX combined with bevacizumab, ii) 149 patients from the TRIBE2 phase III clinical trial (all TRIBE2 patients received bevacizumab in addition to the chemotherapy backbone). In the manually curated real-world-evidence cohort (i), the median predicted IB patient had an overall survival 17.5 months (71%) longer than the median predicted DB with a hazard ratio 0.466 (95% CI: 0.325-0.670, $p < 0.001$). In the TRIBE2 cohort (ii), the median predicted IB patient had an overall survival 6.0 months (32%) longer than the median predicted DB patient with a hazard ratio of 0.629 (95% CI: 0.404-0.981, p=0.04). When the algorithm cannot place a patient's results in either the IB or DB categories, the result is reported as "No Call". In both of the independent, blinded testing sets there was no statistical difference between these no-call cases and the total population with respect to overall survival or hazard. Abraham et al., "Clinical validation of a machine-learning derived signature predictive of outcomes from first-line oxaliplatin-based chemotherapy in patients with advanced colorectal cancer". (December 8, 2020), Clinical Cancer Res., 10.1158/1078-0432.CCR-20-3286.

©2026 Caris Life Sciences. All rights reserved. 

# Gene Fusion and Transcript Variant Detection by RNA Sequencing

**Whole Transcriptome Sequencing (WTS) Methods**

Gene fusion and variant transcript detection were performed on RNA isolated from a tumor sample using next generation sequencing. The assay also detects fusions occurring at known and novel breakpoints within genes. The genes included in this report represent the subset of genes associated with cancer. The complete list of unclassified alterations is available by request.

# References


| # | Drug | Biomarker | Reference |
|---|---|---|---|
| 1 | cetuximab, panitumumab | BRAF | Di Nicolantonio,F. , A. Bardelli, et. al. (2008). "Wild-type BRAF is required for response to panitumumab or cetuximab in metastatic colorectal cancer." J. Clin. Oncol. 2008 View Citation Online |
| 2 | cetuximab, panitumumab | BRAF | National Comprehensive Cancer Network. NCCN Clinical Practice Guidelines in Oncology. Colon Cancer Version 1.2017. 2017; National Comprehensive Cancer Network. View Citation Online |
| 3 | cetuximab, panitumumab | BRAF | Pietrantonio, F., S. Barni, et al. (2015) "Predictive role of BRAF mutations in patients with advanced colorectal cancer receiving cetuximab and panitumumab: a meta-analysis." Eur J Cancer. 2015 51(5):587-94 View Citation Online |
| 4 | cetuximab, panitumumab | BRAF | Sartore-Bianchi, A., S. Siena, et. al. (2009). "Multi-determinants analysis of molecular alterations for predicting clinical benefit to EGFR-targeted monoclonal antibodies in colorectal cancer." PLoS ONE. 4(10): e7287. doi:10.1371/journal.pone.0007287. View Citation Online |
| 5 | cetuximab, panitumumab | BRAF | Sepulveda, A. R., J.A., Nowak, et al. (2017) "Molecular Biomarkers for the Evaluation of Colorectal Cancer: Guideline From the American Society for Clinical Pathology, College of American Pathologists, Association for Molecular Pathology, and the American Society of Clinical Oncology" J Clin Oncol. J Clin Oncol. 2017 Feb 6:JCO2016719807 View Citation Online |
| 6 | cetuximab, panitumumab | BRAF | Van Cutsem, E., F. Ciardiello, et al. (2011). "Cetuximab plus irinotecan, fluorouracil, and leucovorin as first-line treatment for metastatic colorectal cancer: updated analysis of overall survival according to tumor KRAS and BRAF mutation status."J Clin Oncol.29(15):2011-9. View Citation Online |
| 7 | cetuximab, panitumumab | BRAF, NRAS | De Roock, W., S. Tejpar, et. al. (2010). "Effects of KRAS, BRAF, NRAS, and PIK3CA mutations on the efficacy of cetuximab plus chemotherapy in chemotherapy-refractory metastatic colorectal cancer: a retrospective consortium analysis." Lancet Oncol. 11: 753-62. View Citation Online |
| 8 | cetuximab, panitumumab | KRAS | Amado, R.G., et. al. (2008). "Wild-type KRAS is required for panitumumab efficacy in patients with metastatic colorectal cancer." J. Clin. Oncol. 26:1626-1634. View Citation Online |
| 9 | cetuximab, panitumumab | KRAS | Chen, J., G. Shi, et al. (2012). "Association between KRAS codon 13 mutations and clinical response to anti-EGFR treatment in patients with metastatic colorectal cancer: results from a meta-analysis"Cancer Chemother Pharmacol. 2012 Oct 23. [Epub ahead of print] View Citation Online |
| 10 | cetuximab, panitumumab | KRAS | De Roock, W., Tejpar, S., (2010) Association of KRAS p.G13D mutation with outcome in patients with chemotherapy-refractory metastatic colorectal cancer. Jama:304(16):1012-20. View Citation Online |
| 11 | cetuximab, panitumumab | KRAS | Douillard, J.Y., J. Gansert (2010). "Randomized, phase III trial of panitumumab with infusional fluorouracil, leucovorin, and oxaliplatin (FOLFOX4) versus FOLFOX4 alone as first-line treatment in patients with previously untreated metastatic colorectal cancer: the PRIME study." J. Clin. Oncol. 28:4697-4705. View Citation Online |
| 12 | cetuximab, panitumumab | KRAS | Lievre, A., Laurent-Puge, P., (2008). "KRAS mutations as an independent prognostic factor in patients with advanced colorectal cancer treated with cetuximab". J Clin Oncol, 26(3):374-9. View Citation Online |
| 13 | cetuximab, panitumumab | KRAS | National Comprehensive Cancer Network. NCCN Clinical Practice Guidelines in Oncology. Colon Cancer Version 1.2015; View Citation Online |
| 14 | cetuximab, panitumumab | KRAS | Peeters, M., J. Gansert, et. al. (2013). "Mutant KRAS Codon 12 and 13 Alleles in Patients With Metastatic Colorectal Cancer: Assessment As Prognostic and Predictive Biomarkers of Response to Panitumumab" J. Clin. Oncol. 31:759-765 View Citation Online |
| 15 | cetuximab, panitumumab | KRAS | Tejpar, S., E. Van Cutsem, et al. (2012). ""Association of KRAS G13D Tumor Mutations With Outcome in Patients With Metastatic Colorectal Cancer Treated With First-Line Chemotherapy With or Without Cetuximab.""J Clin Oncol. 30(29):3570-7. View Citation Online |
| 16 | cetuximab, panitumumab | KRAS, NRAS | Douillard, J-Y, S.D. Patterson, et al. (2013). "Panitumumab–FOLFOX4 Treatment and RAS Mutations in Colorectal Cancer" N. Engl. J. Med. 369;11: 1023-1034 View Citation Online |

# References


| # | Drug | Biomarker | Reference |
|---|---|---|---|
| 17 | cetuximab, panitumumab | NRAS | Peeters, M., S.D. Patterson, et al. (2013). "Massively Parallel Tumor Multigene Sequencing to Evaluate Response to Panitumumab in a Randomized Phase III Study of Metastatic Colorectal Cancer. "Clin Cancer Res; 19(7): 1902-1912. View Citation Online |