Template for Reporting Results of Biomarker Testing of Specimens From Patients With Carcinoma of the Colon and Rectum

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For the Members of the Cancer Biomarker Reporting Workgroup, College of American Pathologists
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CAP Colon and Rectum Biomarker Template Revision History

**Version Code**
The definition of the version code can be found at www.cap.org/cancerprotocols.

**Version:** ColonBiomarkers 1.2.0.0

**Summary of Changes**
The following changes have been made since the October 2013 release.

**RESULTS**

**Microsatellite Instability**
Format for percentage reporting was changed and reference was added for National Cancer Institute (NCI) markers.

**Loci Testing**
An option for “Not performed” was added.

**KRAS Mutational Analysis**
Reference to “wild type KRAS allele” was removed. “Mutation not stated” was revised to “mutation, not otherwise specified.” Reporting option for “Gln61Leu (CAA>CCA)” was added.

**NRAS Mutational Analysis**

**BRAF Expression (by immunohistochemistry)**
Sections were added.

**BRAF Mutational Analysis**
Reference to “wild type BRAF allele” was removed. Formatting for reporting “BRAF V600E (c.1799T>A) mutation” and “Other BRAF mutation” was revised.

**PIK3CA Mutational Analysis**
Reference to “wild type PIK3CA allele” was removed.

**PTEN Mutational Analysis**
Reference to “wild type PTEN allele” was removed.

**METHODS**

**KRAS Mutational Analysis**
Options for specifying applicable codons were removed.

**NRAS Mutational Analysis**
Section was added.

**PTEN Expression and Mutational Analysis**
Options added to specify IHC antibody and ISH probe used in testing.
Completion of the template is the responsibility of the laboratory performing the biomarker testing and/or providing the interpretation. When both testing and interpretation are performed elsewhere (e.g., a reference laboratory), synoptic reporting of the results by the laboratory submitting the tissue for testing is also encouraged to ensure that all information is included in the patient’s medical record and thus readily available to the treating clinical team.

**COLON AND RECTUM**

Select a single response unless otherwise indicated.

*Note:* Use of this template is optional.

**RESULTS**

*Immunohistochemistry (IHC) Testing for Mismatch Repair (MMR) Proteins (select all that apply)*

(Note A)

+ ___ MLH1
  + ___ Intact nuclear expression
  + ___ Loss of nuclear expression
  + ___ Cannot be determined (explain): _____________________
+ ___ MSH2
  + ___ Intact nuclear expression
  + ___ Loss of nuclear expression
  + ___ Cannot be determined (explain): _____________________
+ ___ MSH6
  + ___ Intact nuclear expression
  + ___ Loss of nuclear expression
  + ___ Cannot be determined (explain): _____________________
+ ___ PMS2
  + ___ Intact nuclear expression
  + ___ Loss of nuclear expression
  + ___ Cannot be determined (explain): _____________________
+ ___ Background nonneoplastic tissue/internal control with intact nuclear expression

*IHC Interpretation*

+ ___ No loss of nuclear expression of MMR proteins: low probability of microsatellite instability-high (MSI-H)*
+ ___ Loss of nuclear expression of MLH1 and PMS2: testing for methylation of the MLH1 promoter and/or mutation of BRAF is indicated (the presence of a BRAF V600E mutation and/or MLH1 methylation suggests that the tumor is sporadic and germline evaluation is probably not indicated; absence of both MLH1 methylation and of BRAF V600E mutation suggests the possibility of Lynch syndrome, and sequencing and/or large deletion/duplication testing of germline MLH1 may be indicated)*

*Data elements preceded by this symbol are not required.*
 Loss of nuclear expression of MSH2 and MSH6: high probability of Lynch syndrome (sequencing and/or large deletion/duplication testing of germline MSH2 may be indicated, and, if negative, sequencing and/or large deletion/duplication testing of germline MSH6 may be indicated)*

 Loss of nuclear expression of MSH6 only: high probability of Lynch syndrome (sequencing and/or large deletion/duplication testing of germline MSH6 may be indicated)*

 Loss of nuclear expression of PMS2 only: high probability of Lynch syndrome (sequencing and/or large deletion/duplication testing of germline PMS2 may be indicated)*

* There are exceptions to the above IHC interpretations. These results should not be considered in isolation, and clinical correlation with genetic counseling is recommended to assess the need for germline testing.

**Microsatellite Instability (MSI) (Note A)**

 MSI - stable (MSS)

 MSI - low (MSI-L)

 - 1% - 29% of the National Cancer Institute (NCI) or mononucleotide markers exhibit instability
 - 1 of the NCI or mononucleotide markers exhibit instability
 - Other (specify): _______________________

 MSI - high (MSI-H)

 - ≥30% of the NCI or mononucleotide markers exhibit instability
 - 2 or more of the NCI or mononucleotide markers exhibit instability
 - Other (specify): _______________________

 MSI - indeterminate

**Loci Testing**

 Mononucleotide panel

 - BAT-25
   - Stable
   - Unstable
   - Cannot be determined (explain): _______________________
   - Not performed

 - BAT-26
   - Stable
   - Unstable
   - Cannot be determined (explain): _______________________
   - Not performed

 - NR-21
   - Stable
   - Unstable
   - Cannot be determined (explain): _______________________
   - Not performed

 - NR-24
   - Stable
   - Unstable
   - Cannot be determined (explain): _______________________
   - Not performed

 - Mono-27
   - Stable
   - Unstable
   - Cannot be determined (explain): _______________________
   - Not performed

 Data elements preceded by this symbol are not required.
+ NCI panel
  + BAT-25
    + Stable
    + Unstable
    + Cannot be determined (explain): __________________________
    + Not performed
  + BAT-26
    + Stable
    + Unstable
    + Cannot be determined (explain): __________________________
    + Not performed
  + D2S123
    + Stable
    + Unstable
    + Cannot be determined (explain): __________________________
    + Not performed
  + D5S346
    + Stable
    + Unstable
    + Cannot be determined (explain): __________________________
    + Not performed
  + D17S250
    + Stable
    + Unstable
    + Cannot be determined (explain): __________________________
    + Not performed
  + Other (specify): __________________________
    + Stable
    + Unstable
    + Cannot be determined (explain): __________________________

+ MLH1 Promoter Methylation Analysis (Note B)
  + MLH1 promoter hypermethylation present
  + MLH1 promoter hypermethylation absent
  + Cannot be determined (explain): __________________________

+ KRAS Mutational Analysis (Note C)
  + No mutation detected
  + Mutation identified (select all that apply)
    + Codon 12
      + Gly12Asp (GGT>GAT)
      + Gly12Val (GGT>GTT)
      + Gly12Cys (GGT>TGT)
      + Gly12Ser (GGT>AGT)
      + Gly12Ala (GGT>GCT)
      + Gly12Arg (GGT>CGT)
      + Codon 12 mutation, not otherwise specified
      + Other codon 12 mutation (specify): __________________________
    + Codon 13
      + Gly13Asp (GGC>GAC)
      + Gly13Arg (GGC>CGC)
      + Gly13Cys (GGC>TGC)
+ Gly13Ala (GGC>GCC)
+ Gly13Val (GGC>GTC)
+ Codon 13 mutation, not otherwise specified
+ Other codon 13 mutation (specify): ______________
+ Codon 61
+ Gln61Leu (CAA>CTA)
+ Gln61His (CAA>CAC)
+ Codon 61 mutation, not otherwise specified
+ Other codon 61 mutation (specify): ______________
+ Codon 146
+ Ala146Thr (G436A) (GCA>ACA)
+ Codon 146 mutation, not otherwise specified
+ Other codon 146 mutation (specify): ______________
+ Other codon (specify): ______________
+ Cannot be determined (explain): ______________

+ NRAS Mutational Analysis (Note C)
+ No mutation detected
+ Mutation identified (select all that apply)
+ Codon 12
  + Gly12Asp (GGT>GAT)
  + Gly12Val (GGT>GTT)
  + Gly12Cys (GGT>TGT)
  + Gly12Ser (GGT>AGT)
  + Gly12Ala (GGT>GCT)
  + Gly12Arg (GGT>CGT)
  + Codon 12 mutation, not otherwise specified
  + Other codon 12 mutation (specify): ______________
+ Codon 13
  + Specific codon 13 mutation (specify): ______________
  + Codon 13 mutation, not otherwise specified
+ Codon 61
  + Gln61Lys (CAA>AAA)
  + Gln61Arg (CAA>CGA)
  + Codon 61 mutation, not otherwise specified
  + Other codon 61 mutation (specify): ______________
  + Other codon (specify): ______________
+ Cannot be determined (explain): ______________

+BRAF Expression (by immunohistochemistry) (Note B)
+ Positive cytoplasmic expression
+ Negative for cytoplasmic expression
+ Cannot be determined (explain): ______________

+BRAF Mutational Analysis (Note B)
+ No mutations detected
+ BRAF V600E (c.1799T>A) mutation
+ Other BRAF mutation (specify): ______________
+ Cannot be determined (explain): ______________

+ Data elements preceded by this symbol are not required.
+ PIK3CA Mutational Analysis (Note D)
+ ___ No mutations detected
+ ___ Exon 9 mutation present (specify): _________________
+ ___ Exon 20 mutation present (specify): _________________
+ ___ Cannot be determined (explain): _______________________

+ PTEN Expression (by immunohistochemistry) (Note E)
+ ___ Positive cytoplasmic and/or nuclear expression
+ ___ Negative for cytoplasmic and nuclear expression
+ ___ Cannot be determined (explain): _______________________

+ PTEN Mutational Analysis
+ ___ No mutation detected
+ ___ Exon 1-9 mutation present (specify): _________________
+ ___ Cannot be determined (explain): _______________________

+ Multiparameter Gene Expression/Protein Expression Assay
  + Specify type: ____________________________
  + Results:
    + ___ Low risk
    + ___ Moderate risk
    + ___ High risk
    + Recurrence score: ______

+ METHODS

+ Dissection Method(s) (select all that apply) (Note F)
  + ___ Laser capture microdissection
    + Specify test name#: ____________________________
  + ___ Manual under microscopic observation
    + Specify test name#: ____________________________
  + ___ Manual without microscopic observation
    + Specify test name#: ____________________________
  + ___ Cored from block
    + Specify test name#: ____________________________
  + ___ Whole tissue section (no tumor enrichment procedure employed)
    + Specify test name#: ____________________________
  # If more than 1 dissection method used, please specify which test was associated with each selected dissection method.

+ Microsatellite Instability (MSI)
  + Number of MSI markers tested (specify): ______

+ Cellularity
  + Percent tumor cells present in specimen: _____ %

+ Whole Genome or Exome Sequencing
  + ___ Whole genome sequencing (specify): ____________________________
  + ___ Whole exome sequencing (specify): ____________________________

+ Data elements preceded by this symbol are not required.
+ MLH1 Promoter Methylation

+ Testing Method
+ ___ Methylation-specific real-time polymerase chain reaction (PCR)
+ ___ Other (specify): ______________________________

+ KRAS Mutational Analysis

+ Codons Assessed (select all that apply)
+ ___ 12
+ ___ 13
+ ___ 61
+ ___ 146

+ Testing Method(s) (select all that apply)
+ ___ Direct Sanger sequencing
+ ___ Pyrosequencing
+ ___ High-resolution melting analysis
+ ___ PCR, allele-specific hybridization
+ ___ Real-time PCR
+ ___ Other (specify): ______________________________

* Please specify in Comments section if different testing methods are used for different codons.

+ NRAS Mutational Analysis

+ Codons Assessed (select all that apply)
+ ___ 12
+ ___ 13
+ ___ 61

+ Testing Method(s) (select all that apply)
+ ___ Direct Sanger sequencing
+ ___ Pyrosequencing
+ ___ High-resolution melting analysis
+ ___ PCR, allele-specific hybridization
+ ___ Real-time PCR
+ ___ Other (specify): ______________________________

* Please specify in Comments section if different testing methods are used for different codons.

+ BRAF Mutational Analysis

+ Mutations Assessed (select all that apply)
+ ___ V600E
+ ___ Other BRAF V600 mutation (specify): ________________________________
+ ___ Other (specify): ________________________________
+**Testing Method (select all that apply)**
+ ___ Direct Sanger sequencing
+ ___ PCR, allele-specific hybridization
+ ___ Pyrosequencing
+ ___ Real-time PCR
+ ___ Immunohistochemistry for V600E gene product
+ ___ Other (specify): __________________________

+ **PIK3CA Mutational Analysis**

+ **Testing Method**
+ ___ Direct Sanger sequencing
+ ___ Other (specify): __________________________

+ **PTEN Expression and Mutational Analysis**

+ **Testing Method (select all that apply)**
+ ___ Immunohistochemistry (specify antibody): __________________________
+ ___ In situ hybridization (specify probe): __________________________
+ ___ Direct Sanger sequencing
+ ___ Duplication/deletion testing (MLPA)
+ ___ Other (specify): __________________________

+ **COMMENT(S)**

____________________________________________________________

____________________________________________________________

Note: Fixative type, time to fixation (cold ischemia time), and time of fixation should be reported if applicable in this template or in the original pathology report.


All reported gene sequence variations should be identified following the recommendations of the Human Genome Variation Society (www.hgvs.org/mutnomen/; accessed February 10, 2015).
Explanatory Notes

A. Mismatch Repair Testing: Microsatellite instability and Immunohistochemistry

Detection of defective mismatch repair in colorectal carcinomas is important for detection of Lynch syndrome (hereditary nonpolyposis colorectal cancer syndrome [HNPCC]), which accounts for approximately 2% to 3% of all colorectal carcinomas and has clinical implications for treatment of the affected patient and family members. Microsatellite instability (MSI) testing can be used to cost-effectively screen colorectal cancer patients for possible Lynch syndrome. Patients with a microsatellite instability-high (MSI-H) phenotype that indicates mismatch repair deficiency in their cancer may have a germline mutation in one of several DNA mismatch repair (MMR) genes (eg, MLH1, MSH2, MSH6, or PMS2) or an altered EPCAM (TACSTD1) gene. After appropriate genetic counseling, patients may want to consider testing to identify the causative heritable abnormality. An MSI-H phenotype is more frequently observed in sporadic colorectal cancer (about 15% of cases) due to somatic abnormalities, usually hypermethylation of the MLH1 gene promoter. The specificity of MSI testing can be increased by using it primarily on at-risk populations, such as colorectal cancer patients younger than 50 years, or patients with a strong family history of Lynch-associated tumors (eg, colorectal, endometrial, gastric, or upper urinary tract urothelial carcinoma), but with sacrifice of sensitivity, since a sizeable minority of cases lacks these clinical characteristics.

MSI testing of tumor DNA is generally performed with at least 5 microsatellite markers, generally mononucleotide or dinucleotide repeat markers. In 1998, a National Institutes of Health consensus panel proposed that laboratories use a 5-marker panel consisting of 3 dinucleotide and 2 mononucleotide repeats for MSI testing. Recent data suggests that dinucleotide repeats may have lower sensitivity and specificity for identifying tumors with an MSI-H phenotype. As a consequence, there has been a move towards including more mononucleotides and fewer dinucleotides in MSI testing panels. Many laboratories now use a commercially available kit for MSI testing that utilizes 5 mononucleotide markers.

MSI testing is frequently done in conjunction with immunohistochemical (IHC) testing for DNA MMR protein expression (ie, MLH1, MSH2, MSH6, and PMS expression). If DNA MMR IHC has not been performed, this testing should be recommended for any case that shows an MSI-H phenotype, because this information will help identify the gene that is most likely to have a germline mutation (eg, a patient whose tumor shows loss of MSH2 and MSH6 expression, but retention of MLH1 and PMS2 expression, is likely to have an MSH2 germline mutation). If the results of DNA MMR IHC and MSI testing are discordant (eg, MSI-H phenotype with normal IHC or abnormal IHC with MSS phenotype), then the laboratory should make sure that the same sample was used for MSI and IHC testing and that there was no sample mix-up. However, MSI-H may not occur in colorectal cancers of patients with germline MSH6 mutation. Intact expression of all 4 proteins indicates that MMR enzymes tested are intact but does not entirely exclude Lynch syndrome, as approximately 5% of families may have a missense mutation (especially in MLH1) that can lead to a nonfunctional protein with retained antigenicity. Defects in lesser-known MMR enzymes may also lead to a similar result, but this situation is rare.

Any positive reaction in the nuclei of tumor cells is considered as intact expression (normal), and it is common for intact staining to be somewhat patchy. An interpretation of expression loss in tumor cells should be made only if a positive reaction is seen in internal control cells, such as the nuclei of stromal, inflammatory, or nonneoplastic epithelial cells. Loss of expression of MLH1 may be due to Lynch syndrome or methylation of the MLH1 promoter region (as occurs in sporadic MSI colorectal carcinoma). Genetic testing is ultimately required for this distinction, although a specific BRAF gene mutation (V600E) is present in many sporadic cases, but not familial cancers. Loss of MSH2 expression strongly suggests Lynch syndrome. PMS2 loss is often associated with loss of MLH1 and is only independently meaningful if MLH1 is intact. MSH6 is similarly related to MSH2. One should also keep in mind that nucleolar staining or complete loss of MSH6 staining has been described in colorectal cancer.
cases with prior radiation or chemotherapy, and a significant reduction of MSH6 staining has been described in a small percentage of colorectal carcinomas with somatic mutations of the coding region microsatellites of the MSH6 gene in MLH1/PMS2-deficient carcinomas.5

B. MLH1 Promoter Hypermethylation Analysis and BRAF Mutational Analysis
Defective mismatch repair in sporadic colorectal cancer is most often due to inactivation of the MLH1 gene promoter by hypermethylation (epigenetic silencing). The V600E mutation of the BRAF gene may be present in up to 70% of tumors with hypermethylation of the MLH1 promoter. In colorectal cancer, this mutation has been associated with a limited clinical response to epidermal growth factor receptor (EGFR) targeted therapies (cetuximab or panitumumab). Analysis for somatic mutations in the V600E hot spot in BRAF may also be indicated for tumors that show MSI-H, as this mutation has been found in sporadic MSI-H tumors, but not in Lynch-associated cancers with MLH1 or MSH2 mutations.6 BRAF V600E mutations have been described in probands with monoallelic PMS2 mutations.7 Direct testing of MLH1 promoter hypermethylation and/or the use of BRAF V600E mutational analysis prior to germline genetic testing in patients with MSI-H tumors and loss of MLH1 by IHC may be a cost-effective means of identifying patients with sporadic tumors for whom further testing is not indicated.8

C. RAS Mutational Analysis
The presence of a KRAS mutation has been shown to be associated with lack of clinical response to therapies targeted at EGFR, such as cetuximab and panitumumab.10 While clinical guidelines for KRAS mutational analysis are evolving, current provisional recommendations from the American Society of Clinical Oncology are that all patients with stage IV colorectal carcinoma who are candidates for anti-EGFR antibody therapy should have their tumor tested for KRAS mutations.11 Anti-EGFR antibody therapy is not recommended for patients whose tumors show mutations in KRAS codon 12, 13, or 61, but data on codon 146 are currently insufficient. A recent study has shown that NRAS mutation, like KRAS mutation, has influence on response to anti-EGFR therapy.12 Although more studies are needed, these findings may lead to broad KRAS and NRAS panels to include codons 12, 13, 61, and 146 of both genes.

D. PIK3CA Mutational Analysis
PIK3CA mutations activate the PI3K-PTEN-AKT pathway that is downstream from both the EGFR and the RAS-RAF-MAPK pathways. PIK3CA mutation and subsequent activation of the AKT pathway has been shown to play an important role in colorectal carcinogenesis and have been associated with KRAS mutation and microsatellite instability.14 PIK3CA mutation has further been associated with poor survival in resectable stage I to III colon cancer, with the adverse effect of PIK3CA mutation potentially limited to patients with KRAS wild-type tumors.15 PIK3CA mutations have been associated with resistance to anti-EGFR therapy in several studies,16,17 but not in others.18 The reasons for the discrepancy are not clear. Mutations of exons 1, 9, and 20 of the PIK3CA gene represent ≥95% of known mutations.

A European consortium recently suggested that only PIK3CA exon 20 mutations are associated with a lack of cetuximab activity in KRAS wild-type tumors and with a shorter median progression-free survival and overall survival.17 By contrast, exon 9 PIK3CA mutations are associated with KRAS mutations and do not have an independent effect on cetuximab efficacy.17 More studies are needed to establish the prognostic and predictive roles of PIK3CA exon-9 and exon-20 mutations.

E. PTEN Mutational Analysis
The role of PTEN loss in colorectal cancer prognosis and therapy is unclear. It has been suggested that loss of PTEN expression, as determined by immunohistochemistry, is associated with lack of benefit from cetuximab in metastatic colorectal cancer.19-22 Loss of PTEN has been found to co-occur with KRAS, BRAF, and PIK3CA mutations.19,22 The recorded frequency of loss of PTEN expression varies from 19% to 36%, with some studies reporting an effect on response rate and survival, whereas others found an effect only on progression-free or overall survival. Moreover, data on the loss of PTEN expression are not
concordant in primary and metastatic tissues. There is currently no standardized method for PTEN expression analysis by immunohistochemistry.

F. Dissection Method
Please denote the manner in which the tissue was dissected and specify the biomarker test only if different dissection methods are used for different tests.

1. Laser capture microdissection (LCM): Use of a laser-equipped microscope to isolate and retrieve specific cells of interest from a histopathologic region of interest.
2. Manual under microscopic observation: hematoxylin and eosin (H&E) slide is examined under a light microscope and marked by a pathologist for subsequent tumor dissection and retrieval.
3. Manual without microscopic observation: H&E slide is examined without a microscope and marked by a pathologist for subsequent tumor dissection and retrieval.
4. Cored from block: Area of interest is cored from a paraffin-embedded tissue block.
5. Whole tissue section: No tumor enrichment procedure employed for tissue retrieval.

References


