Clinical Policy Title: Familial polyposis gene testing

Clinical Policy Number: 02.01.08

Effective Date: December 1, 2013
Initial Review Date: August 21, 2013
Most Recent Review Date: August 17, 2016
Next Review Date: August 2017

Related policies:

CP# 02.01.10 Colaris® testing for Lynch syndrome

ABOUT THIS POLICY: AmeriHealth Caritas Pennsylvania has developed clinical policies to assist with making coverage determinations. AmeriHealth Caritas Pennsylvania’s clinical policies are based on guidelines from established industry sources, such as the Centers for Medicare & Medicaid Services (CMS), state regulatory agencies, the American Medical Association (AMA), medical specialty professional societies, and peer-reviewed professional literature. These clinical policies along with other sources, such as plan benefits and state and federal laws and regulatory requirements, including any state- or plan-specific definition of “medically necessary,” and the specific facts of the particular situation are considered by AmeriHealth Caritas Pennsylvania when making coverage determinations. In the event of conflict between this clinical policy and plan benefits and/or state or federal laws and/or regulatory requirements, the plan benefits and/or state and federal laws and/or regulatory requirements shall control. AmeriHealth Caritas Pennsylvania’s clinical policies are for informational purposes only and not intended as medical advice or to direct treatment. Physicians and other health care providers are solely responsible for the treatment decisions for their patients. AmeriHealth Caritas Pennsylvania’s clinical policies are reflective of evidence-based medicine at the time of review. As medical science evolves, AmeriHealth Caritas Pennsylvania will update its clinical policies as necessary. AmeriHealth Caritas Pennsylvania’s clinical policies are not guarantees of payment.

Coverage policy

AmeriHealth Caritas Pennsylvania considers the use of genetic testing for familial polyposis genes for the purpose of identifying members at risk for familial adenomatous polyposis (FAP), attenuated FAP (AFAP), colorectal cancer and MLH1-related Lynch syndrome to be clinically proven and, therefore, medical necessary when the following criteria are met:

- Members with greater than 20 adenomatous colonic polyps during their lifetime.
- Members who have a first- or second-degree relative diagnosed with FAP or AFAP.
- Members have a first- or second-degree relative with a known FAP or AFAP gene mutation or adenomatous polyposis coli (APC gene).

Limitations:

All other uses of familial polyposis gene testing are not medically necessary, including:

- MutY homologue (MYH) mutation testing, considered experimental and investigational for members of a polyposis family with clear autosomal dominant inheritance or for any other indications because its effectiveness for indications other than the ones listed above has not been established.
• **APC** genetic testing, considered experimental and investigational for all other indications (e.g., desmoid tumor) because its effectiveness for indications other than the ones listed above has not been established.

Note: The following CPT/HCPCS code is not listed in the Pennsylvania Medicaid fee schedule:

81406 - MYH associated polyposis, full gene sequence

**Alternative covered services:**

Standard diagnostic cancer screening tests are medically necessary as preventive services for members age 50 years and older when the following tests are recommended by their physician:

- Colonoscopy (considered medically necessary every 10 years for persons at average risk).
- Double contrast barium enema (DCBE) (considered medically necessary every five years for persons at average risk).
- Sigmoidoscopy (considered medically necessary every five years for persons at average risk).

**Background**

**Disease characteristics:**

APC-associated polyposis conditions include FAP, AFAP, Gardner syndrome and Turcot syndrome.

FAP is a colon cancer predisposition syndrome in which hundreds to thousands of precancerous colonic polyps develop, beginning, on average, at age 16 years (range seven to 36 years). By age 35 years, 95 percent of individuals with FAP have polyps; without colectomy, colon cancer is inevitable. The mean age of colon cancer diagnosis in untreated individuals is 39 years (range 34 – 43 years). Extracolonic manifestations are variably present and include polyps of the gastric fundus and duodenum, osteomas, dental anomalies, congenital hypertrophy of the retinal pigment epithelium (CHRPE), soft tissue tumors, desmoid tumors and associated cancers.

AFAP is characterized by a significant risk for colon cancer but fewer colonic polyps (average of 30), more proximally located polyps and diagnosis of colon cancer at a later age; management may be substantially different.

Gardner syndrome is characterized by colonic polyposis typical of FAP, together with osteomas and soft tissue tumors. Turcot syndrome is the association of colonic polyposis and central nervous system (CNS) tumors. Differences in phenotype may relate to the location of the mutation within APC.

**Diagnosis/testing:**

APC-associated polyposis conditions are caused by mutations in APC. The diagnosis relies primarily on clinical findings. Molecular genetic testing of APC detects disease-causing mutations in up to 90 percent of individuals with typical FAP. Molecular genetic testing is most often used in the early diagnosis of at-risk family members, as well as in confirming the diagnosis of FAP or AFAP in individuals with equivocal findings (e.g., < 100 adenomatous polyps).

**Management/Treatment of manifestations:**
Colectomy is advised when more than 20 or 30 adenomas or multiple adenomas with advanced histology have occurred. Nonsteroidal anti-inflammatory drugs (NSAIDs), especially sulindac, have caused regression of adenomas in FAP and decreased the number of polyps requiring ablation in the remaining rectum of persons with a subtotal colectomy. Endoscopic or surgical removal of duodenal adenomas is considered if polyps exhibit villous change or severe dysplasia, exceed one centimeter in diameter, or cause symptoms. Osteomas may be removed for cosmetic reasons. Desmoid tumors may be surgically excised or treated with NSAIDs, anti-estrogens, cytotoxic chemotherapy or radiation.

**Surveillance:**

Screening for hepatoblastoma by liver ultrasound and measurement of serum alpha-fetoprotein concentration (until age 5 years); sigmoidoscopy or colonoscopy beginning at age 10 to 12 years; annual colonoscopy once polyps are detected until colectomy; esophagogastroduodenoscopy by age 25 years or prior to colon surgery; small bowel X-ray or CT when duodenal adenomas are detected; and regular physical examinations, including thyroid palpation.

**Evaluation of relatives at risk:**

Molecular genetic testing for early identification of at-risk family members improves diagnostic certainty and reduces the need for costly screening procedures in those at-risk family members who have not inherited the disease-causing mutation

**Genetic counseling:**

APC-associated polyposis conditions are inherited in individuals who have an affected parent. Offspring of an affected individual are at a 50 percent risk of inheriting the disease-causing mutation in APC. Prenatal testing and pre-implantation genetic diagnosis are possible if a disease-causing mutation has been identified in an affected family member.

Lynch syndrome, caused by germ-line mutation in a mismatch repair gene and associated with tumors exhibiting microsatellite instability (MIS), is characterized by an increased risk of colon cancer and cancers of the endometrium, ovary, stomach, small intestine, hepatobiliary tract, urinary tract, brain and skin. In individuals with Lynch syndrome, the following lifetime risks for cancer are seen: 52 percent – 82 percent for colorectal cancer (mean age at diagnosis 44 – 61 years); 25 percent – 60 percent for endometrial cancer in women (mean age at diagnosis 48 – 62 years); 6 percent – 13 percent for gastric cancer (mean age at diagnosis 56 years); and 4 percent – 12 percent for ovarian cancer (mean age at diagnosis 42.5 years; approximately 30 percent are diagnosed before age 40 years). The risk for other Lynch syndrome-related cancers is lower, though substantially increased over general population rates.

**Searches**

AmeriHealth Caritas Pennsylvania searched PubMed and the databases of:

- UK National Health Services Centre for Reviews and Dissemination.
- Agency for Healthcare Research and Quality’s National Guideline Clearinghouse and other evidence-based practice centers.
- The Centers for Medicare & Medicaid Services (CMS).
We conducted searches on July 14, 2016 using the terms “genetic testing, familial polyposis” and “colon cancer.”

We included:

- **Systematic reviews**, which pool results from multiple studies to achieve larger sample sizes and greater precision of effect estimation than in smaller primary studies. Systematic reviews use predetermined transparent methods to minimize bias, effectively treating the review as a scientific endeavor, and are thus rated highest in evidence-grading hierarchies.
- **Guidelines based on systematic reviews.**
- **Economic analyses**, such as cost-effectiveness, and benefit or utility studies (but not simple cost studies), reporting both costs and outcomes — sometimes referred to as efficiency studies — which also rank near the top of evidence hierarchies.

**Findings**

AFAP is a phenotypically distinct entity, presenting with fewer than 100 adenomas. Multiple colorectal adenomas can also be caused by mutations in the human MutY homologue (MYH) gene, in an autosomal recessive condition referred to as MYH-associated polyposis (MAP). Endoscopic screening of FAP probands and relatives is advocated as early as age 10 – 12 years to reduce the occurrence of colorectal cancer. Colectomy remains the optimal prophylactic treatment, while the choice of procedure (subtotal vs. proctocolectomy) is still controversial. Along with identifying better chemopreventive agents, optimizing screening of extracolonic cancers and applying new radiological and endoscopic technology to the diagnosis and management of extracolonic features are the major challenges for the future.

The prevalence data reported from national registries include all of the APC-associated polyposis conditions (except possibly some cases of attenuated FAP); reported prevalence is 2.29 to 3.2 per 100,000 individuals (Burn et al. 1991, Jarvinen 1992, Bülow et al. 1996). AFAP is likely underdiagnosed, given the lower number of colonic polyps and lower risk for colorectal cancer compared to classic FAP (Neklason et al. 2008). APC-associated polyposis conditions historically accounted for about 0.5 percent of all colorectal cancers; this figure is declining as more at-risk family members undergo successful treatment following early polyp detection and prophylactic colectomy.

Diagnosis is based on a suggestive family history, clinical findings, and large bowel endoscopy or full colonoscopy. Whenever possible, the clinical diagnosis should be confirmed by genetic testing. When the APC mutation in the family has been identified, genetic testing of all first-degree relatives should be performed. Presymptomatic, prenatal (amniocentesis and chorionic villous sampling) and even preimplantation genetic testing is possible. Referral to a geneticist or genetic counselor is mandatory. Differential diagnoses include other disorders causing multiple polyps (such as Peutz-Jeghers syndrome, familial juvenile polyps or hyperplastic polyposis, hereditary mixed polyposis syndromes, and Lynch syndrome). Cancer prevention and maintaining a good quality of life are the main goals of management and regular and systematic follow-up, and supportive care should be offered to all patients.

**Policy updates:**

**July 2015:**
### Summary of clinical evidence:

<table>
<thead>
<tr>
<th>Citation</th>
<th>Content, Methods, Recommendations</th>
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| American Society of Clinical Oncology (ASCO) (2003) | **Key points:** Statement regarding genetic testing for cancer susceptibility. ASCO recommends genetic counseling and testing be offered when (ASCOP, 2003; Robson, et al., 2010):  
  - The individual has personal or family history and the features suggestive of a genetic cancer susceptibility condition.  
  - The genetic test can be adequately interpreted.  
  - The test results will aid in diagnosis or influence the medical or surgical management of the patient or family members at hereditary risk of cancer. |
| American Gastroenterological Association (AGA) (2001) | **Key points:** AGA suggests genetic testing for Lynch syndrome should be offered to first-degree relatives of individuals with a known inherited sequence variant in an MMR gene. Furthermore, genetic testing should also be offered to those without a known familial sequence variant in an MMR gene, but who do match one of the first three components of the revised Bethesda criteria (note that there is some confusion in this report between the original and revised Bethesda guidelines):  
  - Individuals with cancer in families that meet the Amsterdam criteria.  
  - Individuals with two HNPCC-related tumors, including synchronous and metachronous colorectal cancer (CRC) or associated extra colonic cancer (endometrium, ovarian, gastric, hepatobiliary or small bowel cancer, or transitional cell carcinoma of the renal pelvis or ureter).  
  - Individuals with CRC and a first-degree relative with CRC or HNPCC-related extra colonic cancer or a colorectal adenoma; one of the cancers diagnosed at < 45 years of age, and the adenoma diagnosed at < 40 years of age.  
The AGA also suggests an alternative approach: performing MSI testing on any patient who meets the revised Bethesda guidelines, followed by genetic testing for the DNA MMR sequence variants in those patients found to be MSI-H. |
| National Comprehensive Cancer Network (NCCN) (2008) | **Key points:** NCCN guidelines include a flowchart for CRC screening for Lynch syndrome. The following are risk factors for Lynch syndrome in the extended family pedigree:  
  - Autosomal dominant inheritance pattern.  
  - Colon cancer or other Lynch syndrome-related cancers in first-or second-degree family member.  
  - Colon cancer at < 50 years of age.  
  - Endometrial cancer at < 50 years of age.  
  - Multiple primaries, including colorectal, endometrial, ovarian, duodenal/small bowel, gastric, ureteral/renal pelvis, sebaceous adenomas or carcinomas, hepatobiliary/pancreas, brain tumors (particularly glioblastoma).  
  - Right-sided colon cancer predominance and/or MSI histology.  
For more information regarding outcomes following a risk assessment, consult the... |
NCCN guideline.

The NCCN recommends that genetic testing for individuals in whom a familial sequence variant is not known should initially focus on the **MSH2** and **MLH1** genes, followed by testing of the **MSH6** and **PMS2** genes if no sequence variant is found.

<table>
<thead>
<tr>
<th>American College of Gastroenterology (ACG) (2000)</th>
<th>Key points:</th>
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<tr>
<td>Genetic testing and management of Lynch syndrome, familial adenomatous polyposis (FAP), attenuated familial adenomatous polyposis (AFAP), <strong>MUTYH</strong>-associated polyposis (MAP), Peutz–Jeghers syndrome, juvenile polyposis syndrome, Cowden syndrome, serrated (hyperplastic) polyposis syndrome, hereditary pancreatic cancer, and hereditary gastric cancer.</td>
<td>The revised guidelines indicate the following for Lynch syndrome based on weak evidence from clinical trials:</td>
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<td>• Patients who meet the revised Bethesda criteria should undergo MSI and/or IHC testing of their tumor or a family member's tumor for MMR proteins.</td>
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<td>• Patients who test positive can be offered genetic testing to detect MMR gene sequence variants.</td>
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<td>Those with positive genetic testing, or at risk when genetic testing is unsuccessful in an affected probe and, should undergo colonoscopy every two years beginning at 20 to 25 years of age, until 40 years of age, then annually.</td>
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<table>
<thead>
<tr>
<th>American College of Gastroenterology (ACG) (2015)</th>
<th>Key points:</th>
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<tr>
<td>• Systematic and focused family history of cancer and premalignant conditions is the first step and sufficient to screen for ~ 10–15 percent of patients who may need more detailed risk assessment by more extensive family history assessment, genetic counseling, and genetic testing. The underlying genetic etiologies for several syndromes are now well established, and the array of cancer susceptibility genes is continually expanding.</td>
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<td>• Genetic testing is widely available and should be part of standard of care of patients at increased risk for a hereditary cancer syndrome. Mutation carriers and at-risk individuals require intensive surveillance, possibly prophylactic surgery, and family counseling, and management needs to be individualized based on the syndrome under consideration, as well as the specifics of the family history at hand.</td>
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<tr>
<td>• There is a dire need of organized collaborative international efforts to study benefits of surveillance and surgical strategies in patients with these relatively rare syndromes in order to be able to offer truly evidence-based management recommendations.</td>
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**Glossary**

**Autosomal** — Any of the chromosomes other than the sex-determining chromosomes (i.e., the X and Y) or the genes on these chromosomes.

**Congenital** — Present from birth, but not necessarily genetic.

**Consultand** — The individual (not necessarily affected) who presents for genetic counseling and through whom a family with an inherited disorder comes to medical attention.

**Deletion** — Absence of a segment of DNA; may be as small as a single base or as large as one or more genes.

**DNA (deoxyribonucleic acid)** — The molecule which encodes the genes responsible for the structure and function of an organism and allows for transmission of genetic information from one generation to the next.
**Familial adenomatous polyposis** — Familial adenomatous polyposis (FAP) is an inherited disorder characterized by cancer of the large intestine (colon) and rectum. People with the classic type of familial adenomatous polyposis may begin to develop multiple noncancerous (benign) growths (polyps) in the colon as early as their teenage years. Unless the colon is removed, these polyps will become malignant (cancerous). The average age at which an individual develops colon cancer in classic familial adenomatous polyposis is 39 years. Some people have a variant of the disorder, called attenuated familial adenomatous polyposis, in which polyp growth is delayed. The average age of colorectal cancer onset for attenuated familial adenomatous polyposis is 55 years.

**Familial** — A phenotype that occurs in more than one family member; may have genetic or non-genetic etiology.

**Family history** — The genetic relationships and medical history of a family; when represented in diagram form using standardized symbols and terminology, usually referred to as a pedigree.

**Family-specific mutation** — In a family, the sequence alteration observed that causes or predisposes members to a particular disease; the mutation may be rare or common.

**Family-specific mutation analysis** — Testing for the specific disease-causing mutation(s) previously identified in a family member. Note: Family-specific mutation analysis is different from targeted mutation analysis.

**First-degree relative** — Any relative one meiosis away from a particular individual in a family (i.e., parent, sibling and offspring).

**Gene** — The basic unit of heredity, consisting of a segment of DNA arranged in a linear manner along a chromosome. A gene codes for a specific protein or segment of protein leading to a particular characteristic or function.

**Gene therapy** — Experimental treatment of a genetic disorder by replacing, supplementing or manipulating the expression of abnormal genes with normally functioning genes.

**Gene transfer** — The transfer of genetic material, ranging from a small segment of DNA to the entire genome, from a human cell to another type of cell in culture to study the frequency with which known genetic markers are transferred together to the recipient genome; used to determine the physical proximity of genetic markers in the human genome; also used to study gene expression and regulation.

**Genetic counseling** — A process, involving an individual or family, comprising evaluation to confirm, diagnose or exclude a genetic condition, malformation syndrome or isolated birth defect; discussion of natural history and the role of heredity; identification of medical management issues; calculation and communication of genetic risks; provision of or referral for psychosocial support.

**Genetic predisposition (also, genetic susceptibility)** — Increased susceptibility to a particular disease due to the presence of one or more gene mutations associated with an increased risk for the disease and/or a family history that indicates an increased risk for the disease.
**Genome** — The complete DNA sequence, containing all genetic information and supporting proteins, in the chromosomes of an individual or species.

**Genotype** — The genetic constitution of an organism or cell; also refers to the specific set of alleles inherited at a locus.

**Genotype-phenotype correlation** — The association between the presence of a certain mutation or mutations (genotype) and the resulting pattern of abnormalities (phenotype).

**Homologous chromosomes (also, homologs)** — The two chromosomes from a particular pair, normally one inherited from the mother and one from the father, containing the same genetic loci in the same order.

**Locus** — The physical site or location of a specific gene on a chromosome.

**Lynch syndrome** — Often called hereditary nonpolyposis colorectal cancer (HNPCC), Lynch syndrome is an inherited disorder that increases the risk of many types of cancer, particularly cancers of the colon (large intestine) and rectum, collectively referred to as colorectal cancer. People with Lynch syndrome also have an increased risk of cancers of the stomach, small intestine, liver, gallbladder ducts, upper urinary tract, brain and skin. Additionally, women with this disorder have a high risk of cancer of the ovaries and lining of the uterus (the endometrium). People with Lynch syndrome may occasionally have noncancerous (benign) growths (polyps) in the colon. In individuals with this disorder, colon polyps occur earlier but not in greater numbers than they do in the general population.

**MLH1 gene** — The official name of this gene is “mutL homolog 1, colon cancer, nonpolyposis type 2 (E. coli).” MLH1 is the gene’s official symbol. The MLH1 gene provides instructions for making a protein that plays an essential role in DNA repair. This protein helps fix mistakes made when DNA is copied (DNA replication) in preparation for cell division. The MLH1 protein joins with another protein called PMS2 (produced from the PMS2 gene) to form a protein complex. This complex coordinates the activities of other proteins that repair mistakes made during DNA replication. The repairs are made by removing a section of DNA that contains mistakes and replacing the section with a corrected DNA sequence. The MLH1 gene is a member of a set of genes known as the mismatch repair (MMR) genes. The MLH1 gene is also known by other names, listed below:

- COCA2.
- FCC2.
- hMLH1.
- HNPCC.
- HNPCC2.
- MLH1_HUMAN.
- mutL (E. coli) homolog 1 (colon cancer, nonpolyposis type 2).
- MutL protein homolog 1.

**MSH2 gene** — The official name of this gene is “mutS homolog 2, colon cancer, nonpolyposis type 1 (E. coli).” MSH2 is the gene's official symbol. The MSH2 gene provides instructions for making a protein that plays an essential role in DNA repair. This protein helps fix mistakes made when DNA is copied (DNA replication) in preparation for cell division. The MSH2 protein joins with one of two other proteins, MSH6 or MSH3 (each produced from a different gene), to form a protein complex. This complex identifies locations on the DNA where mistakes have been made during DNA replication. Another group
of proteins, the MLH1-PMS2 protein complex, then repairs the errors. The MSH2 gene is a member of a set of genes known as the mismatch repair (MMR) genes. The MSH2 gene is also known by other names, listed below.

- COCA1.
- HNPCC.
- HNPCC1.
- MSH2_HUMAN.
- mutS (E. coli) homolog 2.
- mutS (E. coli) homolog 2 (colon cancer, nonpolyposis type 1).
- mutS homolog 2.

**MSH6 gene** — The official name of this gene is “mutS homolog 6 (E. coli).” MSH6 is the gene's official symbol. The MSH6 gene provides instructions for making a protein that plays an essential role in repairing DNA. This protein helps fix mistakes made when DNA is copied (DNA replication) in preparation for cell division. The MSH6 protein joins with another protein called MSH2 (produced from the MSH2 gene) to form a protein complex. This complex identifies locations on the DNA where mistakes have been made during DNA replication. Another group of proteins, the MLH1-PMS2 protein complex, then repairs the errors. The MSH6 gene is a member of a set of genes known as the mismatch repair (MMR) genes. The MSH6 gene is also known by other names, listed below:

- DNA Mismatch Repair Protein MSH6.
- GTBP.
- GTMBP.
- G/T mismatch-binding protein.
- HNPCC5.
- MSH6_HUMAN.
- MutS-alpha 160 kDa subunit.
- mutS (E. coli) homolog 6.
- mutS homolog 6.

**Molecular genetic testing (also, DNA-based testing, molecular testing, DNA testing)** — Testing that involves the analysis of DNA, either through linkage analysis, sequencing or one of several methods of mutation detection.

**Mutation** — Any alteration in a gene from its natural state; may be disease-causing or a benign, normal variant.

**Mutation scanning** — A process by which a segment of DNA is screened via one of a variety of methods to identify variant gene region(s). Variant regions are further analyzed (by sequence analysis or mutation analysis) to identify the sequence alteration.

**Parent-of-origin studies** — An analysis used to determine whether a particular chromosome or segment of DNA was inherited from an individual's mother or father; helpful in the diagnosis of disorders in which imprinting or uniparental disomy is a possible underlying etiological mechanism.

**Pedigree** — A diagram of the genetic relationships and medical history of a family using standard symbols and terminology.
**Phenotype** — The observable physical and/or biochemical characteristics of the expression of a gene; the clinical presentation of an individual with a particular genotype.

**Proband (also, propositus)** — The affected individual through whom a family with a genetic disorder is ascertained; may or may not be the consultand (the individual presenting for genetic counseling).

**Second-degree relative** — Any relative two meioses away from a particular individual in a pedigree; a relative with whom one quarter of an individual’s genes is shared (i.e., grandparent, grandchild, uncle, aunt, nephew, niece, half-sibling).

**References**

**Professional society guidelines/other:**


**Peer-reviewed references:**


**Clinical trials:**

Searched clinicaltrials.gov on Accessed July 14, 2016.using terms Familial polyposis gene testing and colon cancer | Open Studies; five studies found, one relevant.


**CMS National Coverage Determinations (NCDs):**

No NCDs identified as of the writing of this policy.

**Local Coverage Determinations (LCDs):**

Genetic Testing (L24308). Effective date: December 1, 2006. Revision date: January 1, 2013. Noridian Administrative Services, LLC. Region X (AK, AZ, ID., MT., ND, SD, UT., WA., WY.).

**Commonly submitted codes**

Below are the most commonly submitted codes for the service(s)/item(s) subject to this policy. This is not an exhaustive list of codes. Providers are expected to consult the appropriate coding manuals and bill accordingly.

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<thead>
<tr>
<th>CPT Code</th>
<th>Description</th>
<th>Comment</th>
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<tr>
<td>81201</td>
<td>APC Gene Sequencing</td>
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<tr>
<td>81202</td>
<td>APC Know Familial Variants</td>
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<tr>
<td>81203</td>
<td>APC Gene Deletion or Duplication</td>
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<tr>
<td>81406</td>
<td>Molecular Pathology Procedure Level 7.</td>
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<td>D12.2 – D12.6</td>
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<tr>
<td>Z86.010</td>
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<tr>
<td>Z83.71</td>
<td>Family history of colonic polyps</td>
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