MEDICAL POLICY – 12.04.111
Gene Expression Profiling and Protein Biomarkers for Prostate Cancer Management

BCBSA Ref. Policy: 2.04.111
Effective Date: Feb. 1, 2018
Last Revised: Jan. 23, 2018
Replaces: 2.04.111

RELATED MEDICAL POLICIES:
12.04.33 Genetic and Protein Biomarkers for the Diagnosis and Cancer Risk Assessment of Prostate Cancer
12.04.54 Gene Expression-Based Testing for Cancers of Unknown Primary

Select a hyperlink below to be directed to that section.

POLICY CRITERIA | CODING | RELATED INFORMATION
EVIDENCE REVIEW | REFERENCES | HISTORY

∞ Clicking this icon returns you to the hyperlinks menu above.

Introduction

Different types of treatment and follow up screening may be needed to treat men with prostate cancer. Some doctors feel that doing genetic testing and looking for specific protein “biomarkers” in the man’s body may help to choose which treatments will be most effective in treating his prostate cancer. Medical studies have not shown that doing genetic testing and looking at biomarkers are helpful in deciding how to manage a patient with prostate cancer. For this reason, doing genetic testing and looking at protein biomarkers are considered to be unproven (investigational).

Note: The Introduction section is for your general knowledge and is not to be taken as policy coverage criteria. The rest of the policy uses specific words and concepts familiar to medical professionals. It is intended for providers. A provider can be a person, such as a doctor, nurse, psychologist, or dentist. A provider also can be a place where medical care is given, like a hospital, clinic, or lab. This policy informs them about when a service may be covered.

Policy Coverage Criteria
Testing

Gene expression analysis and protein biomarker testing

Investigational

Use of gene expression analysis (e.g., Decipher, Oncotype DX Prostate, Prolaris) and protein biomarker testing (e.g., Promark) to guide management of prostate cancer is considered investigational in all situations.

Coding

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0005U</td>
<td>Oncology (prostate) gene expression profile by real-time RT-PCR of 3 genes (ERG, PCA3, and SPDEF), urine, algorithm reported as risk score</td>
</tr>
<tr>
<td>81479</td>
<td>Unlisted molecular pathology procedure</td>
</tr>
<tr>
<td>81551</td>
<td>Oncology (prostate), promoter methylation profiling by real-time PCR of 3 genes (GSTP1, APC, RASSF1), utilizing formalin-fixed paraffin-embedded tissue, algorithm reported as a likelihood of prostate cancer detection on repeat biopsy (new code effective 1/1/18)</td>
</tr>
<tr>
<td>81599</td>
<td>Unlisted multianalyte assay with algorithmic analysis (MAAA)</td>
</tr>
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</table>

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Related Information

N/A

Evidence Review
Description

Gene expression profile analysis and protein biomarkers have been proposed as a means to risk-stratify patients with prostate cancer to guide treatment decisions. These tests are intended to be used either on prostate needle-biopsy tissue to guide management decisions for active surveillance or therapeutic intervention, or to guide radiotherapy after radical prostatectomy.

Background

Prostate Cancer

Prostate cancer is the second most common noncutaneous cancer diagnosed among men in the United States. According to the National Cancer Institute, approximately 161,000 new cases are expected to be diagnosed in the United States in 2017 and more than 26,000 deaths will occur. Autopsy studies in the era before the availability of prostate-specific antigen (PSA) screening have identified incidental cancerous foci in 30% of men 50 years of age, with incidence reaching 75% at age 80 years.

Localized prostate cancers may clinically appear very similar at diagnosis. However, they often exhibit diverse risk of progression that may not be captured by clinical risk categories (eg, D’Amico criteria) or prognostic tools based on clinical findings, including PSA titers, Gleason grade, or tumor stage. In studies of conservative management, the risk of localized disease progression based on prostate cancer–specific survival rates at 10 years may range from 15% to 20%, to perhaps 27% at 20-year follow-up. Among older men (ages ≥70 years) with low-risk disease, comorbidities typically supervene as a cause of death; these men will die with prostate cancer present, rather than from the cancer itself. Other very similar appearing low-risk tumors may unexpectedly progress rapidly, quickly disseminating and becoming incurable.

Risk Stratification in Newly Diagnosed Disease

In the United States, most prostate cancers are clinically localized at diagnosis due in part to the widespread use of PSA testing. Clinicopathologic characteristics are used to stratify patients by risk based on the extent of the primary tumor (T category), nearby lymph node involvement (N category), metastasis (M category), PSA level and Gleason score. The National Comprehensive Cancer Network and American Urological Association risk categories for clinically localized prostate cancer are similar, derived from the D’Amico criteria and broadly include low-, intermediate-, or high-risk as follows as well as subcategories within these groups.
• Low: T1-T2a and Gleason score ≤6 grade group 1 and PSA level ≤10 ng/mL;

• Intermediate: T2b-T2c or Gleason score 3+4=7/Gleason grade group 2 or Gleason score 4+3=7/Gleason grade group 3 or PSA level 10-20 ng/mL;

• High: T3a or Gleason score 8/Gleason grade group 4 or Gleason score 9-10/Gleason grade group 5 or PSA level >20 ng/mL.

Risk stratification is combined with patient age, life expectancy, and treatment preferences to make initial therapy decisions.

**Monitoring After Prostatectomy**

Theoretically, all normal prostate tissue and tumor tissue is removed during radical prostatectomy (RP), so the serum level of PSA should be undetectable following RP. Detectable PSA post-RP indicates residual prostate tissue and presumably persistent or recurrent disease. PSA is serially measured following RP to detect early disease recurrence. The National Comprehensive Cancer Network recommends monitoring serum PSA every 6 to 12 months for the first 5 years and annually thereafter. Many recurrences following RP can be successfully treated. The American Urological Association has recommended a biochemical recurrence be defined as a serum PSA of 0.2 ng/mL or higher, which is confirmed by a second determination with a PSA level of 0.2 ng/mL or higher.

**Summary of Evidence**

*Initial Management Decision: Active Surveillance vs Therapeutic Intervention*

For individuals who have low-risk clinically localized prostate cancer who receive Prolaris Oncotype DX Prostate, or ProMark protein biomarker test, the evidence includes studies of analytic validity and studies of clinical validity using archived samples from patients in mixed risk categories. Relevant outcomes include overall survival, disease-specific survival, test accuracy and validity, quality of life, and treatment-related morbidity. The PROTECT trial showed 99% ten-year disease-specific survival in mostly low-risk patients receiving active surveillance. The low mortality rate estimated with tight precision makes it unlikely that a test intended to identify a subgroup of low-risk men with a net benefit from immediate treatment instead of active surveillance would find such a group.
For individuals who have intermediate-risk clinically localized untreated prostate cancer who receive Prolaris, the evidence includes a study of analytic validity and retrospective cohort studies of clinical validity using archived samples in patients of mixed risk categories and a decision-curve analysis providing indirect evidence of clinical utility. Relevant outcomes include overall survival, disease-specific survival, test accuracy and validity, quality of life, and treatment-related morbidity. Evidence has shown that using the Prolaris Cell Cycle Progression score in patients who were managed conservatively after needle biopsy may be helpful. Using the score has shown improved clinical validity or prognostic accuracy for prostate cancer death, and some improvement has been shown in areas under the receiver operating characteristic curve over clinicopathologic risk stratification tools. All validation studies are Simon category C. There is limited indirect evidence for potential clinical utility. The evidence is insufficient to determine the effects of the technology on health outcomes.

For individuals who have low- or intermediate-risk clinically localized untreated prostate cancer who receive Oncotype DX Prostate, the evidence includes 2 studies of analytic validity, case-cohort and retrospective cohort studies of clinical validity using archived samples in patients of mixed risk categories, and a decision-curve analysis examining indirect evidence of clinical utility. Relevant outcomes include overall survival, disease-specific survival, test accuracy and validity, quality of life, and treatment-related morbidity. Evidence for clinical validity and potential clinical utility of Oncotype DX Prostate in patients with clinically localized prostate cancer derives from a study predicting adverse pathology after RP. The validity of using tumor pathology as a surrogate for risk of progression and cancer-specific death is unclear. It is also unclear whether results from an RP population can be generalized to an active surveillance population. All validation studies are Simon category C or D. The evidence is insufficient to determine the effects of the technology on health outcomes.

For individuals who have low- or intermediate-risk clinically localized untreated prostate cancer who receive the ProMark protein biomarker test, the evidence includes a study of analytic validity, a retrospective cohort study of clinical validity using archived samples, and no studies of clinical utility. Relevant outcomes include overall survival, disease-specific survival, test accuracy and validity, quality of life, and treatment-related morbidity. Current evidence does not support improved outcomes with ProMark given that only a single clinical validity study was available. The evidence is insufficient to determine the effects of the technology on health outcomes.

**Management Decision After Radical Prostatectomy**

For individuals who have localized prostate cancer who are treated with RP who receive Prolaris, the evidence includes a study of analytic validity and retrospective cohort studies of clinical
validity using archived samples. Relevant outcomes include overall survival, disease-specific survival, test accuracy and validity, quality of life, and treatment-related morbidity. Evidence of improved clinical validity or prognostic accuracy for prostate cancer death using the Prolaris Cell Cycle Progression score in patients after prostatectomy has shown some improvement in areas under the receiver operating characteristic curve over clinicopathologic risk stratification tools. All validation studies are Simon category C or D. The evidence is insufficient to determine the effects of the technology on health outcomes.

For individuals who have localized prostate cancer who are treated with RP and who receive the Decipher prostate cancer classifier, the evidence includes a study of analytic validity, prospective and retrospective studies of clinical validity using overlapping archived samples, decision-curve analyses examining indirect evidence of clinical utility, and prospective decision-impact studies without pathology or clinical outcomes. Relevant outcomes include overall survival, disease-specific survival, test accuracy and validity, quality of life, and treatment-related morbidity. The clinical validity of the Decipher genomic classifier has been evaluated in samples of patients with high-risk prostate cancer undergoing different interventions following RP. Studies reported some incremental improvement in discrimination. However, it is unclear whether there is consistent improved reclassification—particularly to higher risk categories—or whether the test could be used to predict which men will benefit from radiotherapy. All validation studies are Simon category C or D. The evidence is insufficient to determine the effects of the technology on health outcomes.

Ongoing and Unpublished Clinical Trials

Currently unpublished trials that might influence this review are listed in Table 1.

Table 1. Summary of Key Trials

<table>
<thead>
<tr>
<th>NCT No.</th>
<th>Trial Name</th>
<th>Planned Enrollment</th>
<th>Completion Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ongoing</td>
<td></td>
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<tr>
<td>Prolaris</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>NCT02454595</td>
<td>Registry to Measure the Impact of Adding Genomic Testing</td>
<td>116</td>
<td>May 2017</td>
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<tr>
<td>NCT03152448</td>
<td>Two-Part Prospective Study to Measure Impact of Prolaris® Testing Added to Treatment Decision Following Biopsy in Newly Diagnosed Prostate Cancer Patients to Measure</td>
<td>1500</td>
<td>Jan 2024</td>
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<tr>
<td>NCT No.</td>
<td>Trial Name</td>
<td>Planned Enrollment</td>
<td>Completion Date</td>
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<tr>
<td>NCT02668276</td>
<td>The Impact of a Gene Expression Profile on Treatment Choice and Outcome Among Minority Men Newly Diagnosed With Prostate Cancer: A Randomized Trial</td>
<td>300</td>
<td>Aug 2022</td>
</tr>
<tr>
<td>NCT02080689</td>
<td>Prospective Clinical Utility Study to Assess the Impact of Decipher on Treatment Decisions after Surgery (PRO-IMPACT)</td>
<td>286</td>
<td>Feb 2017</td>
</tr>
<tr>
<td>NCT02609269</td>
<td>Decipher Genomics Resource Information Database (GIRD)</td>
<td>10,000</td>
<td>Dec 2020</td>
</tr>
<tr>
<td>NCT02723734</td>
<td>Validation Study on the Impact of Decipher Testing – VANDAAM Study</td>
<td>240</td>
<td>May 2021</td>
</tr>
<tr>
<td>NCT01350180</td>
<td>Assessing DNA Changes in High Risk Prostate Cancer to Determine Prognosis</td>
<td>132</td>
<td>Dec 2017</td>
</tr>
<tr>
<td>NCT00002874</td>
<td>Radiation Therapy With or Without Bicalutamide in Treating Patients With Stage II, Stage III, or Recurrent Prostate Cancer</td>
<td>840</td>
<td>Aug 2015</td>
</tr>
</tbody>
</table>

NCT: national clinical trial; PTEN: phosphatase and tensin homolog

Practice Guidelines and Position Statements

National Comprehensive Cancer Network

The National Comprehensive Cancer Network (NCCN) guidelines for prostate cancer (v.2.2017)\(^\text{11}\) provide a table of tissue-based tests for prostate cancer prognosis, along with evidence information and Molecular Diagnostic Services Program (MoDX) recommendations (see Table 2). The NCCN panel suggested that men with clinically localized disease may consider these assays, although the panel warned that the utility of these assays has not been fully assessed in randomized clinical trials.
<table>
<thead>
<tr>
<th>Test</th>
<th>Platform</th>
<th>Population</th>
<th>Outcomes</th>
<th>References</th>
<th>MolDX Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Decipher</strong></td>
<td>Whole transcriptome 1.4 RNA expression (44,000 genes) oligonucleotide microarray optimized for FFPE tissue</td>
<td>Post RP, adverse pathology/high-risk features</td>
<td>Metastasis; prostate cancer–specific mortality</td>
<td>Cooperberg 2015&lt;sup&gt;79&lt;/sup&gt; Den 2014&lt;sup&gt;78&lt;/sup&gt; Den 2015&lt;sup&gt;67&lt;/sup&gt; Erho 2013&lt;sup&gt;82&lt;/sup&gt; Karnes 2013&lt;sup&gt;81&lt;/sup&gt; Klein 2015&lt;sup&gt;77&lt;/sup&gt; Prensner 2014&lt;sup&gt;96&lt;/sup&gt; Ross 2014&lt;sup&gt;80&lt;/sup&gt; Ross 2016&lt;sup&gt;83&lt;/sup&gt; Tomlins 2015&lt;sup&gt;97&lt;/sup&gt; Yamoah 2015&lt;sup&gt;98&lt;/sup&gt;</td>
<td>Cover post-RP for: pT2 with positive margins Any pT3 disease Rising PSA (above nadir)</td>
</tr>
<tr>
<td><strong>Ki-67</strong></td>
<td>IHC</td>
<td>Biopsy, intermediate- to high-risk treated with EBRT</td>
<td>Metastasis</td>
<td>Fisher 2013&lt;sup&gt;99&lt;/sup&gt; Khor 2009&lt;sup&gt;100&lt;/sup&gt; Li 2004&lt;sup&gt;101&lt;/sup&gt; Verhoven 2013&lt;sup&gt;102&lt;/sup&gt;</td>
<td>Not recommended</td>
</tr>
<tr>
<td><strong>Oncotype DX</strong></td>
<td>Quantitative RT-PCR for 12 prostate cancer–related genes and 5 housekeeping controls</td>
<td>Biopsy, low- to intermediate-risk treated with RP</td>
<td>Non-organ-confined pT3 or Gleason grade 4 disease on RP</td>
<td>Cullen 2015&lt;sup&gt;55&lt;/sup&gt; Klein 2014&lt;sup&gt;54&lt;/sup&gt;</td>
<td>Cover postbiopsy for NCCN very low risk and low-risk prostate cancer at diagnosis with 10-20 y of life expectancy</td>
</tr>
<tr>
<td><strong>Prolaris</strong></td>
<td>Quantitative RT-PCR for 31 cell cycle–related genes and 15 housekeeping controls</td>
<td>TURP, active surveillance</td>
<td>Prostate cancer–specific mortality</td>
<td>Bishoff 2014&lt;sup&gt;43&lt;/sup&gt; Cooperberg 2013&lt;sup&gt;57&lt;/sup&gt; Cuzick 2011&lt;sup&gt;47&lt;/sup&gt; Cuzick 2012&lt;sup&gt;44&lt;/sup&gt; Cuzick 2015&lt;sup&gt;42&lt;/sup&gt; Freedland 2013&lt;sup&gt;75&lt;/sup&gt;</td>
<td>Cover postbiopsy for NCCN very low risk and low-risk prostate cancer at diagnosis with at least 10 y of life expectancy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Biopsy, active surveillance</td>
<td>Prostate cancer–specific mortality</td>
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<td></td>
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<tr>
<td></td>
<td></td>
<td>Biopsy, localized prostate cancer</td>
<td>Biochemical recurrence; metastasis</td>
<td></td>
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</table>

Table 2. Available Tissue-Based Tests for Prostate Cancer Prognosis<sup>13</sup>
<table>
<thead>
<tr>
<th>Test</th>
<th>Platform</th>
<th>Population</th>
<th>Outcomes</th>
<th>References</th>
<th>MolDX Recommendation</th>
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<tbody>
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<tr>
<td></td>
<td></td>
<td>Biopsy, intermediate-risk, treated with EBRT</td>
<td>Biochemical failure</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>RP, node-negative localized prostate cancer</td>
<td>Biochemical recurrence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Promark</td>
<td>Multiplex immunofluorescent staining of 8 proteins</td>
<td>Biopsy, Gleason grade 3+3 or 3+4</td>
<td>Non-organ-confined pT3 or Gleason 4 disease on RP</td>
<td>Blume-Jensen 2015\textsuperscript{66}</td>
<td>Not reviewed</td>
</tr>
<tr>
<td>PTEN</td>
<td>Fluorescent in situ hybridization or IHC</td>
<td>TURP, active surveillance</td>
<td>Prostate cancer–specific mortality</td>
<td>Cuzick 2013\textsuperscript{103} Lotan 2011\textsuperscript{104} Lotan 2015\textsuperscript{105}</td>
<td>Not recommended</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Biopsy, Gleason grade 3+3</td>
<td>Upgrading to Gleason 4 on RP</td>
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<tr>
<td></td>
<td></td>
<td>RP, high-risk localized disease</td>
<td>Biochemical recurrence</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


**American Urological Association et al**

In 2017, the American Urological Association, American Society for Radiation Oncology, and the Society of Urologic Oncology published joint guidelines on the management of clinically localized prostate cancer.\textsuperscript{34} The guidelines stated that among most low-risk localized prostate cancer patients, genomic biomarkers have not demonstrated a clear role in the selection of active surveillance or in follow-up of patients on active surveillance.
National Institute for Health and Care Excellence

The National Institute for Health and Care Excellence (NICE) updated its guidance on the diagnosis and management of prostate cancer in 2014.\textsuperscript{106} The guidance did not address gene expression profile analysis.

Medicare National Coverage

Palmetto GBA, a local carrier, issued “limited coverage” determinations under the auspices of a Coverage with Data Development mechanism for the following tests (date effective): Prolaris (03/02/15), Decipher (03/02/15), Oncotype DX Prostate (10/05/15), and ProMark (10/10/2016).\textsuperscript{107,108}

Regulatory Status

Clinical laboratories may develop and validate tests in-house and market them as a laboratory service; laboratory-developed tests (LDTs) must meet the general regulatory standards of the Clinical Laboratory Improvement Amendments (CLIA). Prolaris\textsuperscript{®} (Myriad Genetics, Salt Lake City, UT), Oncotype DX\textsuperscript{®} Prostate Genomic Health, Redwood City, CA), and Decipher\textsuperscript{®} gene expression profiling test (GenomeDx Biosciences, Vancouver, BC), and the ProMark™ protein biomarker test (Metamark Genetics, Cambridge, MA) are available under the auspices of CLIA. Laboratories that offer LDTs must be licensed by CLIA for high-complexity testing. To date, the U.S. Food and Drug Administration (FDA) has chosen not to require any regulatory review of this test.

In November 2015, the FDA’s Office of Public Health Strategy and Analysis published a document on public health evidence for FDA oversight of LDTs.\textsuperscript{16} The FDA argued that many tests need more FDA oversight than the regulatory requirements of CLIA. CLIA standards relate to laboratory operations, but do not address inaccuracies or unreliability of specific tests. Prolaris is among the 20 case studies in the document cited as needing FDA oversight. The document asserted that patients are potentially receiving inappropriate prostate cancer care because there is no evidence that results from the test meaningfully improve clinical outcomes.

References


**History**

<table>
<thead>
<tr>
<th>Date</th>
<th>Comments</th>
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<tr>
<td>12/09/13</td>
<td>New Policy. New policy developed with review of the literature through June 2013. Microarray-based gene expression analysis to guide management of prostate cancer is considered investigational in all situations.</td>
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<tr>
<td>01/20/14</td>
<td>Update Related Policies. Add 12.04.54.</td>
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<td>06/16/14</td>
<td>Update Related Policies. Add 12.04.64.</td>
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<td>07/24/14</td>
<td>Update Related Policies. Remove 12.04.91.</td>
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<tr>
<td>01/28/15</td>
<td>Minor update. Rationale updated with revised NCCN Guidelines information regarding Prolaris and Oncotype DX. Reference 39 added; policy statement unchanged.</td>
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<tr>
<td>06/16/15</td>
<td>Update Related Policies. Change title to 12.04.33.</td>
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<tr>
<td>08/11/15</td>
<td>Annual Review. Policy updated with literature review through March 16, 2015; references 24-25 and 40-51 added. Information on Promark and Decipher tests added</td>
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<tr>
<td>Date</td>
<td>Comments</td>
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<tr>
<td>01/12/16</td>
<td>Annual Review. Policy updated with literature review through October 28, 2015; references 1, 3, 28-30, 35, 37, 43, 45, 48, 55, and 59-60 added. Extensive revisions; updated Medicare coverage section. Policy statement unchanged.</td>
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<td>02/09/16</td>
<td>Update Related Policies. Remove 12.04.64 as it is archived.</td>
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<td>02/01/17</td>
<td>Annual Review, approved January 10, 2017. Policy updated with literature review through October 4, 2016; references updated. Appendix deleted and sentence about Categories of Genetic Testing Addressed in This Policy moved to Rationale section. Policy statement unchanged.</td>
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<td>Coding update. Added new CPT code 0005U.</td>
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<tr>
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<td>Coding updated. Added new CPT code 0016U (effective 8/1/17).</td>
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<tr>
<td>10/06/17</td>
<td>Policy moved into new format; no change to policy statements.</td>
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<tr>
<td>01/23/18</td>
<td>Coding update, added CPT code 81551 (new code effective 1/1/18).</td>
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