LCD - Genomic Sequence Analysis Panels in the Treatment of Solid Organ Neoplasms (L37810)

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National Government Services, Inc.	A and B and HHH MAC	14512 - MAC B	J - K	Vermont

LCD Information

Document Information

LCD ID
L37810

LCD Title
Genomic Sequence Analysis Panels in the Treatment of Solid Organ Neoplasms

Proposed LCD in Comment Period
N/A

Source Proposed LCD
DL37810

Original Effective Date
For services performed on or after 04/01/2019

Revision Effective Date
For services performed on or after 04/01/2022

Revision Ending Date
N/A

Retirement Date

AMA CPT / ADA CDT / AHA NUBC Copyright Statement

N/A

Notice Period Start Date
02/10/2022

Notice Period End Date
03/31/2022

CMS National Coverage Policy

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Unless otherwise specified, *italicized* text represents quotation from one or more of the following CMS sources:

Title XVIII of the Social Security Act (SSA):
Section 1862(a)(1)(A) excludes expenses incurred for items or services which are not reasonable and necessary for the diagnosis or treatment of illness or injury or to improve the functioning of a malformed body member.

Section 1833(e) prohibits Medicare payment for any claim which lacks the necessary information to process the claim.

Section 1862(a)(7) excludes routine physical examinations, unless otherwise covered by statute.

CMS Publications:
CMS Publication 100-02, *Medicare Benefit Policy Manual*, Chapter 15, Section 80.1 – Laboratory services must meet applicable requirements of CLIA

CMS Publication 100-04, *Medicare Claims Processing Manual*, Chapter 16, Section 40.7 Billing for Noncovered Clinical Laboratory Tests Section and 120.1 Clarification of the Use of the Term “Screening” or “Screen”

CMS Publication 100-04, *Medicare Claims Processing Manual*, Chapter 30, Section 50 Advance Beneficiary Notice of Noncoverage (ABN)

CMS Publication 100-08, *Medicare Program Integrity Manual*, Chapter 13, Local Coverage Determinations

CMS National Correct Coding Initiative (NCCI) *Policy Manual for Medicare Services*, Chapter 10, Pathology/Laboratory Services, (A) Introduction

CMS Publication 100-02, *Medicare Benefit Policy Manual*, Chapter 15, Section 80.6.5 which describes the Surgical/Cytopathology Exception.

CMS National Correct Coding Initiative (NCCI) *Policy Manual for Medicare Services*, Chapter 10 Pathology/Laboratory Services which addresses reflex testing.

Code of Federal Regulations:
42 CFR, Section 410.32, indicates that diagnostic tests may only be ordered by the treating physician (or other treating practitioner acting within the scope of his or her license and Medicare requirements) who furnishes a

consultation or treats a beneficiary for a specific medical problem and who uses the results in the management of the beneficiary's specific medical problem. Tests not ordered by the physician (or other qualified non-physician provider) who is treating the beneficiary are not reasonable and necessary (see Sec. 411.15(k)(1) of this chapter).

Coverage Guidance

Coverage Indications, Limitations, and/or Medical Necessity

Non-Small Cell Lung Cancer (NSCLC)

Indications and Limitations of Coverage

Genomic Sequential Analysis Panel will be considered reasonable and necessary in the evaluation of tumor tissue in the following clinical circumstances:

- Newly diagnosed patients with advanced (stage IIIB or IV) NSCLC, who are not treatable by resection or radiation with curative intent, and who are suitable candidates for therapy at the time of testing.
- Previously diagnosed patients with advanced (stage IIIB or IV) NSCLC, who have not responded to at least one systemic therapy, or who have progressed following resection. The patient must be a candidate for treatment at the time of the testing.
- Previously diagnosed patients with advanced (stage IIIB or IV) NSCLC, who have been resistant to at least one targeted therapy, are able to undergo tumor tissue biopsy for testing, and who are suitable candidates for additional treatment at the time of testing.

Metastatic Colorectal Cancer (mCRC)

Indications and Limitations of Coverage

Genomic Sequential Analysis Panel will be considered reasonable and necessary when the test is performed in a CLIA-certified laboratory qualified to perform high complexity testing, ordered by a treating physician, and the patient has:

1. metastatic CRC; and
2. is a candidate for intensive chemotherapy with an anti-EGFR biologic agent; and
3. has not had prior RAS/BRAF testing (except after initiation of anti-EGFR therapy with evidence of acquired resistance).

Next-Generation Sequence (NGS) Comprehensive Genomic Profile (CGP) Testing

Indications and Limitations of Coverage

This policy section describes coverage of NGS CGP diagnostic testing for patients with advanced cancer as allowable by a Medicare Administrative Contractor (MAC) under the National Coverage Determination (NCD) 90.2 (1). The policy scope is specific to solid tumors and exclusive of hematologic malignancies (the subject of separate LCD L37606), circulating tumor DNA testing (ctDNA), and other cancer-related uses of NGS, such as germline testing.

CGP is a NGS approach that uses a single assay to assess hundreds of genes including relevant cancer biomarkers, with solid evidentiary support for clinical utility in guidelines and clinical trials. CGP assays include not only individual

genetic variants (single nucleotide variants (SNVs), insertions/deletions (INDELs), copy number alterations (CNAs), structural variants (SVs), and splice-site variants), but also patterns of mutations such as DNA mismatch repair deficiency (dMMR), microsatellite instability (MSI), and total mutational burden (TMB). CGP testing may also include RNA sequencing to detect structural rearrangements, such as fusions/translocations and functional splicing mutations.

CGP NGS testing for patients with advanced cancer is reasonable and necessary only when performed in a CLIA-certified laboratory, when ordered by a treating physician, and when the patient has:

- either recurrent, relapsed, refractory, metastatic, or advanced stages III or IV cancer; and
- not been previously tested with a CGP for the same cancer genetic content; and
- decided to seek further cancer treatment (e.g., therapeutic chemotherapy)

Additionally, the test performed must be able to detect at least the minimum genes and genomic positions required for the identification of clinically supported, FDA-approved therapies. The genes and genomic positions required are listed in Category 1 or 2A of the most current version of the National Comprehensive Cancer Network (NCCN) Biomarkers Compendium (2). Testing assays must be FDA approved/cleared, or if a laboratory developed test (LDT), have a published, peer-reviewed study supporting analytic validity, or certification by a third-party consistent with the New York State Department of Health's Clinical Laboratory Evaluation Program (CLEP) review standards.

Summary of Evidence

Non-Small Cell Lung Cancer (NSCLC)

The American Cancer Society estimates that over 220,000 new cases of lung cancer will be diagnosed in 2015, with over 85% of those cancers being classified as non-small cell lung cancer. Lung cancer represents approximately 13% of all new cancer diagnoses, and approximately 27% of cancer deaths. The estimated 5yr survival rate for all lung cancer patients is 17% and is only 4% for patients with metastatic disease.

Most lung cancers are epithelial in origin, with squamous cell carcinomas, adenocarcinomas, and small cell carcinomas being the predominant histologic types. The first two, squamous and adenocarcinomas, have been traditionally grouped as non-small cell lung cancer (NSCLC). Surgery remains the cornerstone of treatment for early stage NSCLC of either type, however treatment of advanced stage disease is based primarily on drugs. Distinctive response patterns to specific therapeutic drugs have been demonstrated over the past 12 years, necessitating the distinction between squamous cell and adenocarcinoma morphology. Consequently, the most recent WHO guidelines advocate sub-classification of all NSCLC in to a more specific subtype whenever possible. This is typically accomplished by histologic evaluation with support from specific immunohistochemical studies, which are particularly useful in the evaluation of small biopsies.

Adenocarcinomas account for approximately 40% of all lung cancers, and are the most common lung cancer in never- or light smokers. Adenocarcinomas are characterized by glandular differentiation, mucin production, or pneumocyte marker expression. Certain genomic alterations are more commonly found in lung adenocarcinomas (when compared to squamous or small cell carcinomas) and clinical laboratory testing to identify these alterations is important in two respects: First, some mutations are now recognized as "driver mutations," which are essential for tumor cell survival. Inhibition of these mutated proteins results in tumor cell death, making them attractive therapeutic targets. While not all driver mutations have specific therapies at this time, an important corollary of this concept is that with rare exception, such driver mutations are mutually exclusive, i.e. the identification of one driver mutation in a tumor effectively makes the likelihood that another driver mutation is present extremely unlikely.

The second reason clinical laboratory testing for specific driver mutations in lung adenocarcinomas is important is the

association of specific genomic alterations with response to specific drugs. Different driver mutations respond to different targeted therapies, and only genetic testing can clearly identify which mutation is present and, therefore, which treatment should be administered. While some of the genetic alterations that affect response to targeted inhibitors are found in a higher proportion of adenocarcinomas from patients with certain clinical risk factors (i.e., low to no history of tobacco exposure, female gender, young age, Asian ethnicity) , these clinical associations are not sufficiently predictive of mutation status to appropriately determine therapy without genetic testing. Accordingly, professional practice guidelines from CAP-IASLC-AMP, WHO, and NCCN all advise against using smoking history, or other clinical risk factors, to exclude testing patients for specific genomic alterations.

NCCN Guidelines (v 7.2015) for Non-Small Cell Lung Cancer recommend testing all non-squamous NSCLCs (i.e. adenocarcinomas, large cell carcinomas, and NSCLC not otherwise specified) for specific alterations in EGFR and ALK and recommend consideration of such testing in tumors with mixed squamous and adeno histology, and in the rare squamous cell carcinomas in never smokers. The NCCN NSCLC Panel "strongly endorses broader molecular profiling to identify rare driver mutations using multiplex/NGS to ensure that patients receive the most appropriate treatment". In addition to testing for alterations in EGFR and ALK, NCCN Guidelines explicitly recognize the prognostic and predictive value of KRAS mutations as well as alterations in the BRAF, MET, and ROS1 genes to select a therapeutic agent, the use of which may be "off-label" but which also meets Medicare coverage requirements for off-label cancer drugs (CMS Publication Pub 100-02, *Medicare Benefit Policy Manual*, Chapter 15, §50.4.5).

Genomic alterations contribute to the development of non-small cell lung carcinoma. Two of the best studied alterations are EGFR mutations and ALK (e.g., EML4-ALK) gene fusions. EGFR mutations are permissive for the use of oral EGFR inhibitors, such as erlotinib. Similarly, activating fusions of ALK permit treatment with oral ALK inhibitors, such as crizotinib. Gene alterations for which a targeted therapeutic agent is available and the use of which meets Medicare coverage requirements (outside of a clinical trial) are listed in Table 1.

Table 1

Gene	**NCCN Category 1 or 2A Recommended Therapeutic Option**
ALK	crizotinib, ceritinib, and alectinib
EGFR	afatinib, erlotinib hydrochloride, and gefitinib
ROS 1	crizotinib
KRAS	Avoid TKI
BRAF	dabrafenib and vemurafib
MET	crizotinib

Metastatic Colorectal Cancer (mCRC)

Despite improvements in mortality secondary to advances in screening and treatment over the last several decades, CRC is the second leading cause of cancer deaths in the US, with about 50,000 deaths per year, 8% of all cancer deaths (1,2). Approximately 50-60% of patients with CRC develop metastases (usually metachronous, but synchronous in 20-34%), with 80-90% unresectable metastatic liver disease. Twenty percent present with metastatic disease. Today, the median overall survival (OS) for patients with mCRC is approximately 30 months, more than double that of 20 years ago (3).

The level of understanding of the molecular events underlying CRC is far greater than for other common solid tumors. Sporadic disease, in which there is no family history, accounts for approximately 70% of all CRCs (4). Specific germline mutations are responsible for the inherited CRC syndromes, while a stepwise accumulation of somatic mutations is thought to underlie most sporadic cases.

RAS and BRAF Mutations

The activation of the epidermal growth factor receptor (EGFR) signaling cascade is a well-described pathway leading to colon tumorigenesis. The receptor for EGFR is overexpressed in 49-82% of CRC. Cetuximab and panitumumab are monoclonal antibodies directed against EGFR that inhibit downstream signaling pathways, but are only effective in about 10-20% of CRC. Mutations within the RAS and BRAF oncogenes located downstream of EGFR within this pathway lead to its constitutive activation, even if the EGFR is blocked.

KRAS exon 2–4 and NRAS exon 2–4 mutations (RAS mutations) are found in about 50% of mCRC and exclude affected patients from EGFR-directed therapy (5,6). There is good concordance between the primary and synchronous distant metastases (but not lymph node metastases) (7). Besides their negative predictive value, RAS mutations have been independently associated with a worse prognosis in most studies (5,8). Some studies suggest that EGFR inhibition may even be detrimental in patients with RAS-mutant mCRC (9).

Activating BRAF mutations (mostly V600E), mutually exclusive from KRAS mutations, are found in approximately 5-10% of mCRCs, and are associated with poor overall prognosis (10). Evidence increasingly suggests that response to EGFR-targeted agents is unlikely in BRAF V600E patients, even if RAS wild type (11,12).

The most recent TNM staging classification considers RAS and BRAF V600E mutations to have both prognostic (level 1 evidence) and predictive (level 1 evidence) significance (2). In a Provisional Clinical Opinion (PLO), the American Society of Clinical Oncology (ASCO) recommended that all patients who are candidates for anti-EGFR therapy should have their tumor tested for mutations in both KRAS and NRAS exons 2 (codons 12 and 13), 3 (codons 59 and 61), and 4 (codons 117 and 146) (13). Because anti-EGFR agents have no role in the management of stage I-III disease, this genotyping is reserved for metastatic disease.

Mismatch Repair Deficiency (dMMR)

MMR genes are responsible for correcting the ubiquitous nucleotide base mispairings and small insertions or deletions that occur during DNA replication. Cells that are MMR-deficient accumulate DNA errors throughout the genome (14). The dMMR genetic signature is a high number of DNA replication errors (RER+) and high levels of DNA microsatellite instability (MSI-H), defined as expansion or contraction in ≥30 percent of microsatellite loci; microsatellites are short sequences of nucleotide bases that are repeated dozens to hundreds of times within the genome.

The gold standard for assessment of MSI has been concurrent analysis of patient tumor and normal DNA for five mononucleotide microsatellite loci with polymerase chain reaction (PCR) (15). The standard for detecting MMR protein expression status has been immunohistochemistry (IHC) for MLH1, MSH2, PMS2, and MSH6 expression. MMR can also be measured by total mutational burden (TMB) via next generation sequencing (NGS). In sporadic tumors, epigenetic changes (acquired hypermethylation of the promoters of both alleles of MLH1 gene), rather than gene mutations, usually account for defective MMR gene expression (20,21).

MSI-H tumors are associated with longer survival in both Lynch syndrome and sporadic CRC, for unclear reasons (4). However, the prognostic influence of MSI is less clear in patients with metastatic CRC, a population in which the prevalence of MSI-H disease is only 3.5% (16). Emerging data have shown MMR status to predict the clinical benefit of immune checkpoint blockade with pembrolizumab in patients with mCRC (17, 22, 23).

Next-Generation Sequence (NGS) Comprehensive Genomic Profile (CGP) Testing

The pace of discovery in the fields of immunology and cancer biology is rapidly accelerating as understanding the role of the immune system in tumor initiation, progression, and metastasis evolves. Momentum is shifting away from sequential analyte testing toward adoption of ever larger NGS panels, capable of evaluating for all classes of potentially actionable genomic alterations across hundreds of genes simultaneously. Factors include decreasing cost, proliferation of actionable biomarkers, need to detect smaller amounts of DNA variants, FDA approval of tumor-type agnostic predictive biomarkers, and most recently, so-called "pan-mutational signature" biomarkers that, almost by definition, require panels that include hundreds of genes, if not whole exome sequencing (WES). Pan-tumor biomarkers make CGP testing not just a more time, specimen, and cost-effective approach, but necessary from an analytic validity standpoint.

The advent of immune checkpoint inhibitors (ICIs) targeting programmed cell death protein 1 (PD-1) and programmed death ligand 1 (PD-L1) proteins has recently emerged as a pillar of cancer care. ICI immunotherapy, however, can induce unique immune-related side effects and, in a subset of patients, trigger accelerated disease progression, termed hyperprogression (3). Given this variability, determining a proper biomarker to select patients for ICI therapy has become increasingly important. In 2017, the FDA approved the ICI pembrolizumab (anti-PD-1) for treatment of unresectable or metastatic tumors from any tumor histology that exhibits MSI/dMMR, resulting in a hypermutator phenotype, marking the first tumor-type agnostic predictive biomarker approval (4,5). While traditionally measured using conventional, non-NGS methods (IHI, PCR), NGS CGP may detect a larger number of microsatellites with improved detection of MSI-H across different tumor types, as data have demonstrated that the variation in length of microsatellites is tissue specific (6).

TMB has become of increasing interest as another potential ICI immunotherapy biomarker. TMB refers to the total number of nonsynonymous mutations per megabase in the exon coding region of the gene being evaluated in the tumor cell genome. The basis for this approach is the idea that high TMB (TMB-H) leads to more antigenic peptides and enhanced immunogenicity. TMB is theoretically a more direct measure of tumor rejection potential than MSI assays, which detect the presence of microsatellite alterations and do not quantify the mutations themselves. In fact, MSI-H may be associated with immunotherapy response largely because it results in high TMB (7). The most compelling data on the predictive capacity of TMB in the response to ICI immunotherapy come from the multicenter open-label phase II KEYNOTE-158 study, which established a link between TMB-H status (as determined by the FoundationOne CDx assay) and overall response rate with pembrolizumab (8). In June 2020, largely based on these results, the FDA granted accelerated approval to pembrolizumab for the treatment of patients with any unresectable or metastatic TMB-H [≥ 10 mutations/megabase (mut/Mb)] solid tumor by an FDA approved assay, who have progressed on prior therapy and have no alternative treatment options (9).

As it was based on a relatively small single-arm study, the tumor agnostic nature of the pembrolizumab approval has generated considerable debate within the oncology community (3,7,10). While TMB estimated from NGS panels has generally correlated with TMB determined by WES (11), a host of questions related to analytic and clinical validity remains. TMB (a continuous variable) values and thresholds were developed independently in each tumor type, and likely differ across tumor types (12) and platforms (11). As a result, no consistent pan-cancer testing approach has been validated (6). For example, each panel may include different numbers and types of genes, use different sequencing platforms, have different methods of filtering germline mutations, incorporate different mutation types in the quantification of TMB, and use proprietary bioinformatics protocols to calculate TMB (11). Indeed, NCCN states: "there is no consensus on how to measure TMB" (13). Regarding clinical validity, the optimal TMB-H cut-off may vary among cancer types; TMB-H cancers that lack correlation between neoantigen load and CD8 T-cell infiltration may even demonstrate a worse response rate to ICIs (14). The conflicting data highlight the TMB-H tumor type dependency, and caution is needed when extrapolating the FDA labelling to all tumor types (6). Some go further, noting that: "TMB has yet to show reproducibility for a specific ICI or tumor. For these reasons, it is our opinion that FDA tumor-agnostic approval of pembrolizumab when TMB is ≥ 10, showing no significant correlation for overall

survival at the tumor level is flawed" (3). This problem is being actively addressed by The Friends of Cancer Research TMB harmonization project, which is endeavoring "to establish a uniform approach to measure and report TMB across different sequencing panels by harmonizing the definition of TMB, proposing best practices for analytic validation studies, and ensuring consistency of TMB calculation through alignment with a universal reference standard" (11).

Gene fusion is another emerging area exerting pressure for NGS CGP testing, beginning with neutrotrophic tyrosine receptor kinase (NTRK) fusion-positive cancers for TRK inhibitor therapy. Two of these agents (larotrectinib, entrectinib) are approved for treatment of TRK fusion-positive refractory solid tumors, regardless of the site of disease origin, "without a known acquired resistance mutation, that are either metastatic or where surgical resection is likely to result in severe morbidity, and who have no satisfactory alternative treatments or whose cancer has progressed following treatment" (15,16). The presence of a TRK fusion defines a new tissue-agnostic diagnostic category for solid tumors (17). Given the rarity of NTRK gene fusions in most cancers, assays that evaluate exclusively for the presence of NTRK gene fusions are impractical in most cases (17). NGS CGP testing allows the assessment of NTRK1/2/3 gene fusions in conjunction with other tumor-agnostic biomarkers as well as any relevant tumor-specific biomarkers. Although NGS sequencing of either DNA or RNA can detect NTRK gene fusions, multiple technical considerations make RNA-based methods superior.

Other biomarkers best measured using NGS CGP include other fusion genes (ALK, ROS1, RET), and the splicing mutation MET exon 14 skipping variants (METex14) (13). NGS-based testing is the primary method for detection of METex14 skipping events, with RNA-based NGS demonstrating improvement in detection (13).

Analysis of Evidence (Rationale for Determination)

Non-Small Cell Lung Cancer (NSCLC)

In total, there are over 40 single nucleotide or small insertion/deletion variants occurring at numerous specific loci in ten genes. These variants represent potential therapeutic targets and, as therapeutic agents aimed at these targets are proven safe and effective and meet Medicare coverage guidelines, additional genes may be added to the above table. In addition, gene fusions can involve five different genes, and amplification is the significant recognized alteration in at least one gene.

Metastatic Colorectal Cancer (mCRC)

The genetic factors with strong evidence for clinical decision-making (both prognostic and predictive of chemotherapy efficacy) are BRAF and RAS mutations along with MMR status (2). Guidelines from NCCN, the European Society for Medical Oncology (ESMO), as well as a combined guideline from the American Society for Clinical Pathology (ASCP), College of American Pathologists (CAP), Association for Molecular Pathology (AMP), and ASCO consider the following molecular genetic biomarkers necessary for diagnosis and management of mCRC (1,3,18,19). Testing is not necessary for mCRC patients being considered for palliative or hospice care only (19). Re-testing may be indicated after initiation of anti-EGFR treatment if resistance develops (24-26).

Gene Test	Clinical Utility	NCCN Category

Extended RAS Testing	Prognostic and predictive of anti-EGFR efficacy	2A
KRAS/NRAS exon 2, codons 12/13	Prognostic and predictive of anti-EGFR efficacy	2A
KRAS/NRAS exon 3, codons 59/61	Prognostic and predictive of anti-EGFR efficacy	2A
KRAS/NRAS exon 4, codons 117/146	Prognostic and predictive of anti-EGFR efficacy	2A
BRAF (V600E)	Prognostic and predictive of anti-EGFR efficacy	2A
MSI	Prognostic stratifications and risk for Lynch syndrome	2A

Next-Generation Sequence (NGS) Comprehensive Genomic Profile (CGP)Testing

The Association for Molecular Pathology (AMP) and the College of American Pathologists (CAP) published guidelines for validating NGS-based oncology panels and bioinformatics pipelines (18,19), and with the American Society of Clinical Oncology (ASCO), guidelines for interpreting somatic variants (20). Other societal guidelines suggest the use of NGS over other methods for lung cancer (21,22), colorectal cancer (22-24), and melanoma, thyroid, and ovarian cancers, among others (22,25-29).

Nevertheless, the analytic and clinical validation of pan-tumor markers, the only absolute NGS CGP raison d'etre, remain problematic. Current evidence fails to support the use of TMB-H as a biomarker for ICI treatment in all tumor types, particularly with a universal one-size fits all cut-off of 10 mut/Mb. "Future studies should focus both on improving cancer type-specific assessment of TMB from targeted sequencing and cancer type-specific activity of ICIs in TMB-H tumors before broad clinical implementation" (14). In fact, limitations to precision medicine's impact, involving the widespread assumption of clinical utility for wholesale genetic testing, is coming under new scrutiny (31-34). Others claim limitations are related to insufficient implementation (30).

Pan-tumor biomarker considerations aside, NGS CGP testing in advanced cancer is arguably increasingly medically necessary from both a time-effectiveness and specimen-sparing perspective. While only TMB absolutely mandates this methodology, it may confer some benefit for MSI/dMMR and fusion detection over more traditional approaches. NGS CGP testing may especially benefit the 25% of patients with rare cancer types, those with no effective alternative treatment options, and underserved minority populations with less access to tumor molecular profiling or off-label therapies (7,35). Given the abundant (if conflicting) literature, and widespread societal support for NGS CGP testing in advanced cancer, National Government Services reservedly deems such testing appropriate for advanced somatic cancers, consistent with CMS NCD 90.2 (1). We echo calls for caution, given the evolving nature of these tests, and look forward to better standards along the lines of the Friends of Cancer Research TMB harmonization project.

General Information

Associated Information

N/A

Sources of Information

N/A

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Non-Small Cell Lung Cancer (NSCLC)

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Revision History Information

REVISION HISTORY DATE	REVISION HISTORY NUMBER	REVISION HISTORY EXPLANATION	REASONS FOR CHANGE
04/01/2022	R3	Based on receipt of Reconsideration Requests, limited coverage of Next-Generation Sequence (NGS) Comprehensive Genomic Profile (CGP)Testing language was added throughout the LCD.	• Provider Education/Guidance • Request for Coverage by a Practitioner (Part B)
10/03/2019	R2	This LCD was converted to the new "no-codes" format. There has been no change in coverage with this LCD revision.	• Revisions Due To Code Removal
08/15/2019	R1	Consistent with Change Request 10901, all coding	• Provider Education/Guidance

REVISION HISTORY DATE	REVISION HISTORY NUMBER	REVISION HISTORY EXPLANATION	REASONS FOR CHANGE
		information, National coverage provisions, and Associated Information (Documentation Requirements, Utilization Guidelines) have been removed from the LCD and placed in the related Billing and Coding Article, A56867. There has been no change in coverage with this LCD revision.	

Associated Documents

Attachments

N/A

Related Local Coverage Documents

Articles

A56867 - Billing and Coding: Genomic Sequence Analysis Panels in the Treatment of Solid Organ Neoplasms

A58984 - Response to Comments: Genomic Sequence Analysis Panels in the Treatment of Solid Organ Neoplasms

Related National Coverage Documents

N/A

Public Versions

UPDATED ON	EFFECTIVE DATES	STATUS
02/02/2022	04/01/2022 - N/A	Currently in Effect (This Version)
09/24/2019	10/03/2019 - 03/31/2022	Superseded
Some older versions have been archived. Please visit the MCD Archive Site to retrieve them.		

Keywords

N/A