Overview

During the last two decades the interest in cardiovascular (CV) biomarkers as early screening tools has risen dramatically, largely fueled by the recognition that traditional CV risk factors (diabetes, smoking, hypertension and hyperlipidemia) do not fully explain individual variation in CV risk, and by advances in genetic and molecular research. Risk assessment for determining the 10-year risk for developing coronary heart disease (CHD) is traditionally carried out using the Framingham risk score.

Despite the Framingham risk-scoring tool, clinicians have sought non-traditional lipid and other biomarker measurements to predict CV events. The most promising biomarkers are the ones that closely correlate with the pathophysiological process of the disease. In general, there is evidence that some of these biomarkers may alter risk categorization (higher or lower) compared to traditional risk prediction, but it has not been established that changes in categorization provides clinically actionable information beyond that of traditional lipid measures. In addition, no study has provided high-quality evidence that measurement of non-traditional lipid and other biomarkers leads to changes in management that improve health outcomes.

To provide clinically useful knowledge, a biomarker should meet the following criteria:

- Adds clinical knowledge that improves patient outcomes;
- Provides risk information that is independent of established predictors;
- Is easy to measure and interpret in the clinical setting; and
- Is accurate, reproducible and standardized.
Guidelines

This policy denies coverage for all CV risk assessment panels, except the basic lipid panel, for symptomatic (with signs and symptoms) patients with suspected or documented CV disease because panel testing is not specific to a given patient’s lipid abnormality or disease. The policy indicates the medical indication(s) based on published scientific articles and consensus guidelines for individual lipid biomarkers that may be covered to characterize a given lipid abnormality or disease, to determine a treatment plan or to assist with intensification of therapy. Each individual lipid biomarker must be specifically ordered and the reason for the test order documented in the patient’s medical record. The policy denies coverage for all non-lipid biomarkers when used for CV risk assessment including but not limited to, biochemical, immunologic, hematologic, and genetic biomarkers for CV risk assessment regardless of whether ordered in a panel or individually.

The following biomarkers, when they are included in a CV risk assessment panel, are non-covered:

- Lipoprotein subclasses;
- LDL particles;
- Intermediate density lipoproteins;
- High density lipoprotein AI9LpAI and Al/AII;
- Lipoprotein(a);
- Apolipoprotein B (Apo B), apo A-I and apo E;
- Lipoprotein-associated phospholipase A2 (Lp-PLA2);
- BNP;
- Cystatin C;
- Thrombogenic/hematologic actors;
- Interleukin-6 (IL-6), tissue necrosis factor-a (TNF-a), plasminogen activator inhibitor-1 (PAI-1) and IL-6 promoter polymorphism;
- Free fatty acids;
- Visfatin, angiotensin-converting enzyme 1 (ACE2) and serum amyloid A;
- Microalbumin;
- Myeloperoxidase (MPO);
- Homocysteine and methylenetetrahydrofolate reductase (MTHFR) mutation testing;
- Uric acid;
- Vitamin D;
- White blood cell count;
- Long-chain omega-3 fatty acids in red blood cell membranes;
- Gamma-glutamyltransferase (GGT);
- Genomic profiling;
- Leptin, ghrelin, adiponectin and adipokines including retinol binding protein 4 (RBP4) and resistin;
- Inflammatory markers including VCAM-1, P-selectin (PSEL) and E-selectin (ESEL);
- Cardiovascular risk panels.

High-Sensitivity C-Reactive Protein (hs-CRP)

CRP is a protein produced in the liver during episodes of acute inflammation or infection. The hs-CRP test measures CRP that is in the normal range for healthy people, and is used to distinguish people with low normal levels from those with high normal levels. In recent years, prospective epidemiologic studies have demonstrated that inflammation is essential for CV disease pathogenesis and that high normal levels of hs-CRP correlate with an increased risk of CV events such as myocardial infarction (MI), stroke, sudden cardiac death and peripheral vascular disease (PVD) even when lipid levels are within acceptable ranges. The American Heart Association (AHA) and the US Centers for Disease Control and Prevent (CDC) recommend averaging two hs-CRP levels obtained two weeks apart. Based on hs-CRP test results, they recognize: low (3.0 mg/L) risk groups.

In 2010, The American College of Cardiology Foundation and the American Heart Association (ACCF/AHA) published guidance as to when and in whom to measure blood levels of hs-CRP. The guidance states that hs-CRP levels may assist in the selection of patients for statin therapy according to the following criteria (Class Ila; Level of evidence (LOE): B):

- Men > 50 years of age, or women > 60 years of age or older,
- LDL-C < 130 mg/dL,
- Patients not on lipid-lowering, hormone replacement, or immunosuppressant therapy,
Patients without clinical CHD, diabetes, chronic kidney disease, severe inflammatory conditions, or contraindications to statins.

For example, a patient may appear to have a low or low-moderate elevated risk of CV events based on traditional risk factor scoring with cholesterol levels, weight, level of exercise, smoking history, diabetes and hypertension. However, an elevated hs-CRP level would indicate that the cardiac risk may be substantially greater than traditional risk factors suggest, and that treatment might be considered. For patients who are already known to have high risk, according to current recommendations, hs-CRP levels will not add any substantially new information, since the patient should already be receiving all available therapy including statins to reduce the risk.

The ACCF/AHA recommended measurement of hs-CRP for CV risk assessment in asymptomatic intermediate-risk men 50 years of age or younger, or women 60 years of age or younger (Class IIb; LOE B). Since screening (asymptomatic patient) is statutorily excluded from coverage, hs-CRP testing for these individuals is not a Medicare benefit. They found no benefit for hs-CRP testing in asymptomatic high-risk adults or men and women below the ages stated above. (Class III; LOE B).

In summary, testing is expected to be limited to the following criteria:

- Patient has intermediate CV risk (10-20% risk of CVD per 10 years using the Framingham point score); and
- Patient has LDL-C between 100-130 mg/dL; and
- Patient has two or more CHD major risk factors, including:
  - Age (Men > 50 years; Women > 60 years).
  - Current cigarette smoking.
  - Family history of premature CHD (CHD in male first degree relative < 55; CHD in female first degree relative).
  - Hypertension (Systolic > 140 mm Hg, or on anti-hypertensive medication).
  - Low HDL-C.

The use of hs-CRP testing to evaluate the effects of treatment or to motivate patients to improve lifestyle behaviors are not considered medically reasonable and necessary, and therefore not covered by Medicare.

**Lipoprotein Subclasses**

Lipoprotein subclass determination based on density, electric charge and other physical chemistry aspect of particles such as nuclear magnetic resonance allow more specific characterization of the major subclasses (VLDL, LDL, IDL and HDL). Studies showed that small, dense LDL particles were highly associated with the occurrence of CVD and diabetes.

**LDL Particles (LDL-P) (aka LDL or Lipoprotein Particles or Particle Number, LDL or Lipid Subfractionation, Lipid Phenotyping, Nuclear Magnetic Resonance, or NMR Profile)**

Small dense LDL with elevated triglyceride levels and low HDL-cholesterol levels constitute the “atherogenic lipoprotein phenotype” form of dyslipidemia that is a feature of type II diabetes and the metabolic syndrome. Measurement of LDL particle density has been proposed as a technique to further risk stratification in patients with elevated LDL levels or for patients with normal LDL levels who have other high risk factors for Coronary Artery Disease (CAD), or to predict response to a particular therapy.

Although great progress has been made in the development of refined lipoprotein assessment and such measurements have helped in understanding the atherosclerotic process, it is not known whether measurements beyond traditional lipids can identify CV risk subgroups and how treatment would differ based on subgroup classification. Furthermore, it is not known whether this additional information helps the health care provider to identify with greater precision and accuracy the person who will develop clinical or subclinical CVD.

The National Academy of Clinical Biochemistry (NACB) does not recommend testing as there is insufficient data that measurement of lipoprotein subclasses can identify CV risk subgroups, how treatment would differ based on subgroup classification and whether, over time, measurement is useful to evaluate the effects of treatments. In addition, the 2010 ACCF/AHA guidelines for assessment of lipoprotein, other lipoprotein parameters and modified lipids state that “measurement of lipid parameters, including lipoproteins, apolipoproteins, particle size, and density, beyond standard fasting lipid profile is not recommended for cardiovascular disease risk assessment in asymptomatic adults.”
Unlike lipoprotein size or subclass measures, which seek to improve CV risk assessment beyond conventional lipid testing, LDL particle number tests (NMR LDL-P) and apoB are simply alternate measures of LDL quantity. Current data supports the ability of LDL particle number to provide clinically actionable information beyond traditional lipid measures to adjudicate individual response to treatment and guide adjustment in therapy.

LDL particle number (NMR LDL-P), rather than LDL size or subclass, has been shown to be significantly associated with CV risk independent of traditional lipid and established risk factors. The American Association of Clinical Endocrinologists (AACE), the National Lipid Association (NLA), the American Diabetes Association (ADA) in conjunction with the American College of Cardiology (ACC), and the American Association of Clinical Chemistry (AACC) have developed consensus position statement on lipoprotein particle management in individuals at risk for CVD. The 2013 AACE Comprehensive Diabetes Management Algorithm, as well as the 2015 joint AACE/American College of Endocrinology Clinical Practice Guidelines for Comprehensive Diabetes Mellitus Care, advocates specific LDL particle number goals for statin treated diabetic patients at high CV risk.

**Intermediate Density Lipoproteins (Remnant Proteins)**

Intermediate density lipoproteins (IDLs) have a density that falls between LDLS and VLDLs, and may be referred to as remnant lipoproteins because they vary in size and contain varying proportion of triglycerides and cholesterol. Although there is abundant evidence the remnant lipoproteins are atherogenic, and a risk factor for CAD, there is no evidence how testing improves patient outcomes.

**High Density Lipoprotein (HDL) Subclass (Lipoprotein AI 9LpAI) and Lipoprotein AI/All (LpAI/All) and/or HDL3 and HDL2**

HDL cholesterol (HDL-C) is the risk indicator most often used in associated with CHD risk. HDL subfractions have been used for risk prediction. However, data is lacking how the subfractions aid in the diagnosis and management of CHD. Neither the NCEP nor ACCF/AHA guidelines recommend the routine measurement of HDL subspecies in CHD risk assessment.

**Lipoprotein(a)(Lp(a))**

Lp(a) is a modified form of LDL in which a large glycoprotein, apolipoprotein(a) is bound to apolipoprotein B. It promotes foam cell formation and the deposition of cholesterol in atherosclerotic plaques, and, because it is structurally similar to plasminogen, Lp(a) may contribute to clot formation. However, the complete role of lipoprotein(a) is not fully understood.

There is no standardized scale for measuring Lp(a) because there is no level that is considered “normal”. Because Lp(a) levels are controlled predominantly by genes, cholesterol-lowering drugs have little effect on lowering Lp(a) levels.

The NACB specifies that Lp(a) screening is not warranted for primary prevention and assessment of cardiovascular risk. They comment that Lp(a) measurement may be done at the physician’s discretion if the risk is intermediate (10%-20%) and uncertainty remains as to the use of preventive therapies such as statins or aspirin (Recommendation-IIb; LOE-C). They further note there is insufficient evidence to support therapeutic monitoring of Lp(a) concentrations for evaluating the effects of treatment.

**Apolipoprotein B (Apo B), Apolipoprotein A-I (Apo AI), and Apolipoprotein E (Apo E)**

Apo B is a constituent of LDL particles, and serves as an indirect measurement of the number of LDL particles. Consequently, elevated levels of Apo B suggest increased levels of small dense LDL particles that are thought to be atherogenic.

Apo AI is the major protein constituent of HDL-C. However, its measurement has not been established as a clinically useful test in determining clinical therapy for patients with CAD or dyslipidemia at the current time.

While Apo B and Apo A-I are thought to be the main structural proteins of atherogenic and anti-atherogenic lipoproteins and particles, testing for these compounds has not been validated as a tool for risk assessment. As such, the 2010 ACCF/AHA guidelines indicate that apolipoproteins testing is not recommended for CV risk assessment in asymptomatic adults. However, AACE recommends apoB testing to assess residual risk in patients for CAD (even when LDL-C levels are controlled) in patient when the triglyceride concentration is > 150 mg/dL or the HDL-C concentration is < 40 mg/dL.
Medicare expects testing to be limited to assessment of residual risk in patients with CAD with triglyceride concentrations of > 150 mg/dL or HDL-C of < 40 mg/dL.

Apo E, the major constituent of VLDL and chylomicrons, acts as the primary binding protein for LDL receptors in the liver and is thought to play a role in lipid metabolism. Although some individuals hypothesize that Apo E genotypes may be useful in the selection of drug therapy, the value of Apo E testing in the diagnosis and management of CHD is insufficient and needs further evaluation.

The National Cholesterol Education Program (NCEP) expert panel concluded that Apo AI is carried in HDL and it is usually low when HDL is reduced. A low Apo AI thus is associated with increased risk of CHD, but not independently of low HDL. Whether it has independent predictive power beyond HDL-C is uncertain and its measurement is not recommended for routine risk assessment in Adult Treatment Panel (ATP III) Guidelines.

**Testing for Lipoproteins**

**Apolipoproteins**

Apolipoproteins are measured in routine clinical laboratories with the use of immunonephelometric or immunoturbidimetric assays. ApoB reflects the number of potentially atherogenic lipoprotein particles because each particle of VLDL, IDL, LDL and lipoprotein(a) particle carries on its surface 1 Apo B100 protein. Most of plasma Apo B is found in LDL particles. HDL particles do not carry Apo B. Instead they carry Apo AI, which does not correspond directly to the concentration of HDL particles in a 1-to-1 fashion.

**LDL Gradient Gel Electrophoresis (GGE) (used by Berkeley Heart Lab, Berkeley, CA)**

GGE is the most commonly used lab technique to measure LDL particle density. It has been promoted as an important criteria of CHD risk, and as a guide to drug and diet therapy in patients with CAD. While the measurement of LDL subclass patterns may be useful in elucidating possible atherogenic dyslipidemia in patients without abnormal total cholesterol, HDL, LDL and triglycerides, there is inadequate evidence that LDL sub-classification by GGE improves outcomes in patients with CV disease.

**Density Gradient Ultracentrifugation (DGU) (used by Atherotech Inc, Birmingham, AL)**

The Vertical Auto Profile (VAP) test measures the relative distribution of cholesterol within various lipoprotein subfractions, quantifying the cholesterol content in the VLDL, IDL, LDL, lipoprotein(a) and HDL subclasses. It includes components (e.g., total cholesterol, direct measured LDL-C, HDL-C and triglycerides), LDL density (i.e., pattern A versus pattern B), IDL, LDL sub types, VLDL density and Lp(a), and non-lipid CV risk assessment biomarkers including hs-CRP, homocysteine, Lp-PLA2, apo-E genotype, vitamin D, cystatin and NT-proBNP.

**Nuclear Magnetic Resonance Spectroscopy**

In this method (NMR LipoProfile® is FDA cleared and available from LipoScience Inc, Raleigh, NC) particle concentrations of lipoprotein subfractions of different size are obtained from the measured amplitudes of their lipid methyl group NMR signals. Lipoprotein particle sizes are then derived from the sum of the diameter of each subclass multiplied by its relative mass percentage based on the amplitude of its methyl NMR signal. Note: FDA clearance does not mean the test has clinical utility.

**Ion-Mobility Analysis**

This method (available from Quest Diagnostics Inc, Madison, NJ) measures both the size and concentration of lipoprotein particle subclasses on the basis of gas-phase differential electric mobility.

**Summary of Lipoprotein Testing**

At the current time, none of the above tests for lipoproteins have better predictive strength than total/HDL-C ratio and there has been no clear benefit for measuring particle number in most studies to date. Additional research is needed to establish the utility of following changes in lipoproteins as a therapeutic target and determine if any subgroups of patients benefit.

**Lipoprotein-Associated Phospholipase A2 (Lp-PLA2)**

Lp-PLA2 is also known as platelet activating factor acetylhydrolase. This enzyme hydrolyzes phospholipids and is primarily associated with LDLs. It has been suggested that this enzyme has a proinflammatory role in the development of
atherosclerosis. Studies show that Lp-PLA2 is an independent predictor of CV risk but fail to demonstrate improved health outcomes. To improve outcomes, studies must demonstrate how risk factors improve risk classification and change in physician practice to improve patient outcomes.

The NCEP ATP III panel concluded that routine measurement of inflammatory markers (including Lp-PLA2) for the purpose of modifying LDL-cholesterol goals in primary prevention is not warranted. In the 2010 ACCF/AHA guidelines for assessment of CV risk, the experts concluded “lipoprotein-associated phospholipase (Lp-PLA2) might be reasonable for cardiovascular risk assessment in intermediate risk asymptomatic adults”. However, at the current time, it is not known whether Lp-PLA2 concentrations are clinically effective for motivating patients, guiding treatment, or improving outcomes.

Cystatin C

Cystatin C, encoded by the CST3 gene, is a small serine protease inhibitor protein secreted by all functional cells in the body. It is used as a biomarker for renal function, and in CV risk assessment although there is no evidence that this marker improves outcomes when used in clinical care. The NACB guidelines on Biomarkers of Renal Function and Cardiovascular Disease Risk do not recommend testing. The NCEP advocates clinical studies to characterize the utility of these markers in the global assessment of CV disease risk.

Homocysteine and Methylenetetrahydrofolate Reductase (MTHFR) Mutation Testing

Homocysteine is an amino acid found in the blood. Observational evidence generally supports the association of homocysteine levels with CV risk, particularly observational data that patients with hereditary homocystinuria, an inborn error of metabolism associated with high plasma levels of homocysteine, have markedly increased risk of CV disease. Folic acid and the B vitamins are involved in the metabolism of homocysteine. Several studies found the higher levels of B vitamins are associated with lower homocysteine levels, while other evidence shows that low levels of folic acid are linked to a higher risk of CHD and stroke. However, large randomized controlled trials do not support a protective effect of folic acid supplementation (rectifying homocysteine levels) in cardiovascular disease.

MTHFR is a key enzyme in folate metabolism. Two variants of the MTHFR polymorphisms result in reduced enzyme activity, impaired methylation and increased risk of CVD, stroke, and hypertension. MTHFR mutation testing has been advocated to evaluate the cause of elevated homocysteine levels. However, in 2009, the US Preventive Services Task Force (USPSTF) concluded that the evidence was insufficient to assess the benefits and harms of using non-traditional risk factors to screen asymptomatic adults with no history of CHD to prevent CHD events.

Long-chain Omega-3 Fatty Acids in Red Blood Cell (RBC) Membranes

It has been proposed that the fatty acid composition of RBCs is an index of long-term intake of eicosapentaenoic (EPA) plus docosahexaenoic (DPA) acids. The omega-3 fatty acids are considered a new modifiable and clinically relevant risk factor for death from CHD. Most studies to date have focused on the association between fish consumption and risk of CHD. Not only is there no association between fish intake and EPA + DHA levels regarding prevention of HF, there is no scientific evidence regarding how measurements of RBC omega-3 fatty acids composition would affect management of individuals at risk for or patients with CHD.

Documentation Requirements

The patient's medical record must contain documentation that fully supports the medical necessity for services included within this policy guideline. This documentation includes, but is not limited to, relevant medical history, physical examination, and results of pertinent diagnostic tests or procedures. Documentation supporting the medical necessity should be legible, maintained in the patient's medical record, and must be made available upon request.

Nationally Non-Covered Indications

Compliance with the provisions in this policy is subject to monitoring by post payment data analysis and subsequent medical review. Title XVIII of the Social Security Act, Section 1862(a)(1)(A) states "...no Medicare payment shall be made for items or services which are not reasonable and necessary for the diagnosis and treatment of illness or injury...". Furthermore, it has been longstanding CMS policy that "tests that are performed in the absence of signs, symptoms, complaints, or personal history of disease or injury are not covered unless explicitly authorized by statute".
Applicable Codes

The following list(s) of procedure and/or diagnosis codes is provided for reference purposes only and may not be all inclusive. Listing of a code in this guideline does not imply that the service described by the code is a covered or non-covered health service. Benefit coverage for health services is determined by the member specific benefit plan document and applicable laws that may require coverage for a specific service. The inclusion of a code does not imply any right to reimbursement or guarantee claim payment. Other Policies and Guidelines may apply.

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<th>CPT Code</th>
<th>Description</th>
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<td>82172</td>
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<td>Cystatin C</td>
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<td>83090</td>
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<td>83695</td>
<td>Lipoprotein (a)</td>
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<td>86141</td>
<td>C-reactive protein; high sensitivity (hsCRP)</td>
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*CPT® is a registered trademark of the American Medical Association*

Coding Clarifications: For ICD-10 diagnosis codes, refer to the related National and Local Coverage Determinations References.

Non-Covered Diagnosis Code

Non-Covered Diagnosis Codes List

This list contains diagnosis codes that are never covered when given as the primary reason for the test. If a code from this section is given as the reason for the test and you know or have reason to believe the service may not be covered, call UnitedHealthcare to issue an Integrated Denial Notice (IDN) to the member and you. The IDN informs the member of their liability for the non-covered service or item and appeal rights. You must make sure the member has received the IDN prior to rendering or referring for non-covered services or items in order to collect payment.

References

CMS National Coverage Determinations (NCDs)

NCD 190.23 Lipid Testing

CMS Local Coverage Determinations (LCDs) and Articles

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### Cystatin C Measurement

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### Homocysteine Level, Serum

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### Frequency of Laboratory Testing

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### CMS Benefit Policy Manual

Chapter 15; § 80.1 - 80.1.3 Clinical Laboratory Services

### CMS Claims Processing Manual

Chapter 16, § 10.2 General Explanation of Payment; § 20 Calculation of Payment Rates - Clinical Laboratory Test Fee Schedules; § 40 Billing for Clinical Laboratory Tests

### Other(s)

CMS Clinical Laboratory Fee Schedule, CMS Website
Palmetto GBA MolDx Website
Palmetto GBA MolDx Manual

### Guideline History/Revision Information

Revisions to this summary document do not in any way modify the requirement that services be provided and documented in accordance with the Medicare guidelines in effect on the date of service in question.

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<td>10/11/2023</td>
<td><strong>Applicable Codes</strong>&lt;br&gt;Non-Covered Diagnosis Codes&lt;br&gt;● Removed Z59.0, Z59.4, and Z59.8&lt;br&gt;Supporting Information&lt;br&gt;● Updated References section to reflect the most current information</td>
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Purpose

The Medicare Advantage Policy Guideline documents are generally used to support UnitedHealthcare Medicare Advantage claims processing activities and facilitate providers’ submission of accurate claims for the specified services. The document can be used as a guide to help determine applicable:

- Medicare coding or billing requirements, and/or
- Medical necessity coverage guidelines; including documentation requirements.

UnitedHealthcare follows Medicare guidelines such as NCDs, LCDs, LCAs, and other Medicare manuals for the purposes of determining coverage. It is expected providers retain or have access to appropriate documentation when requested to support coverage. Please utilize the links in the References section above to view the Medicare source materials used to develop this resource document. This document is not a replacement for the Medicare source materials that outline Medicare coverage requirements. Where there is a conflict between this document and Medicare source materials, the Medicare source materials will apply.

Terms and Conditions

The Medicare Advantage Policy Guidelines are applicable to UnitedHealthcare Medicare Advantage Plans offered by UnitedHealthcare and its affiliates.

These Policy Guidelines are provided for informational purposes, and do not constitute medical advice. Treating physicians and healthcare providers are solely responsible for determining what care to provide to their patients. Members should always consult their physician before making any decisions about medical care.

Benefit coverage for health services is determined by the member specific benefit plan document* and applicable laws that may require coverage for a specific service. The member specific benefit plan document identifies which services are covered, which are excluded, and which are subject to limitations. In the event of a conflict, the member specific benefit plan document supersedes the Medicare Advantage Policy Guidelines.

Medicare Advantage Policy Guidelines are developed as needed, are regularly reviewed and updated, and are subject to change. They represent a portion of the resources used to support UnitedHealthcare coverage decision making. UnitedHealthcare may modify these Policy Guidelines at any time by publishing a new version of the policy on this website. Medicare source materials used to develop these guidelines include, but are not limited to, CMS National Coverage Determinations (NCDs), Local Coverage Determinations (LCDs), Medicare Benefit Policy Manual, Medicare Claims Processing Manual, Medicare Program Integrity Manual, Medicare Managed Care Manual, etc. The information presented in the Medicare Advantage Policy Guidelines is believed to be accurate and current as of the date of publication and is provided on an “AS IS” basis. Where there is a conflict between this document and Medicare source materials, the Medicare source materials will apply.

You are responsible for submission of accurate claims. Medicare Advantage Policy Guidelines are intended to ensure that coverage decisions are made accurately based on the code or codes that correctly describe the health care services provided. UnitedHealthcare Medicare Advantage Policy Guidelines use Current Procedural Terminology (CPT®), Centers for Medicare and Medicaid Services (CMS), or other coding guidelines. References to CPT® or other sources are for definitional purposes only and do not imply any right to reimbursement or guarantee claims payment.

Medicare Advantage Policy Guidelines are the property of UnitedHealthcare. Unauthorized copying, use, and distribution of this information are strictly prohibited.

*For more information on a specific member’s benefit coverage, please call the customer service number on the back of the member ID card or refer to the Administrative Guide.