

Aduhelm™ (Aducanumab-Avwa)

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[Instructions for Use](#)

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Related Commercial Policy

- [Review at Launch for New to Market Medications](#)

Coverage Rationale

Aduhelm (aducanumab-avwa) been added to the Review at Launch program. Refer to the Medical Benefit Drug Policy titled [Review at Launch for New to Market Medications](#) for additional details.

Aduhelm is unproven and not medically necessary for the treatment of Alzheimer’s disease due to insufficient evidence of efficacy.

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 policy 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.

HCPCS Code	Description
J0172	Injection, aducanumab-avwa, 2 mg

Diagnosis Code	Description
G30.0	Alzheimer's disease with early onset
G30.1	Alzheimer's disease with late onset
G30.8	Other Alzheimer's disease
G30.9	Alzheimer's disease, unspecified

Background

Alzheimer’s disease (AD) is the most common cause of dementia and accounts for an estimated 60% to 80% of cases¹. After AD, the most common neurodegenerative dementias are Lewy body disease, characterized by chronic rapid eye movement

(REM) sleep behavior disorder, early visuospatial impairment, and parkinsonism; and Frontotemporal dementia, characterized by a behavioral variant or less often, a language impairment variant.²

AD is characterized by deposition of A β plaques and neurofibrillary tangles (comprised of abnormal tau protein) in the brain, accompanied by synaptic dysfunction and neurodegeneration.^{3,4} The deposition of A β (as amyloid plaques) generally begins decades before any symptoms of AD are observed. More specifically, A β deposition is followed sequentially by markers of neurodegeneration, accumulation of tau pathology, and brain volume loss. This pre-symptomatic phase of AD will precede the emergence of AD symptoms 10 to 20 years prior.⁵

Tau is the microtubule associated protein (MAP) of a normal mature neuron. Tau is a phosphoprotein that promotes the assembly of tubulin into microtubules and stabilization of their structure. In AD (and certain other related neurodegenerative diseases, called tauopathies), tau protein is abnormally hyperphosphorylated and aggregated into bundles of filaments. In AD, this tau pathology is seen as intraneuronal neurofibrillary tangles of paired helical filaments sometimes admixed with straight filaments. Aggregates of abnormally hyperphosphorylated filaments are also seen in dystrophic neurites surrounding the A β plaque core, and in the neuropil as neuropil threads.⁶

There are 2 ways to detect abnormal A β , either directly via PET imaging using tracers or indirectly by measuring the levels of the long form of A β in the CSF. P-tau and t-tau can also be detected using CSF and are used as biomarkers to detect the emergence of AD in patients with MCI.⁷

Age of AD onset:⁸

- Typical AD: AD is characteristically a disease of older age. The incidence and prevalence of AD increase exponentially with age, essentially doubling in prevalence every 5 years after the age of 65 years.
- Early-onset dementia: Although less common, early-onset dementia occurs in patients < 65 years of age. These patients often present with symptoms somewhat atypical for this disease, such as language, visual, or mood-behavioral changes rather than predominant memory loss. A study from the United Kingdom estimated that the incidence of dementia in individuals 30 to 65 years of age was approximately 54 per 100,000 person-years. The most common cause of dementia in these patients was AD (34%), followed by vascular dementia (18%), frontotemporal dementia (12%), dementia with Lewy bodies (7%), and alcohol-related dementia (10%).⁹
- Inherited forms of AD: These forms of AD are rare (< 1% of all AD cases) and routinely present before 65 years of age, frequently in the fifth decade or earlier. Inherited forms of AD typically exhibit an autosomal-dominant inheritance pattern related to mutations in genes that alter A β protein production or metabolism, including amyloid precursor protein (APP), presenilin-1 (PSEN1), and presenilin-2 (PSEN2).
- AD associated with Down syndrome: Patients with Down syndrome have an additional gene dose of APP due to trisomy of chromosome 21 and inevitably develop AD pathology. Symptoms tend to emerge at an earlier age, i.e., 10 to 20 years earlier than the general population with AD.

Risk factors for AD:²

- Aging is an important risk factor for dementia. AD affects 5% to 10% of people > 65 years of age, and 50% of those \geq 85 years of age.
- Nonmodifiable risk factors for AD include female gender, Black race, Hispanic ethnicity, and genetic factors such as presence of the APOE gene.
- Modifiable risk factors for all-cause dementia include hypertension, diabetes, diet, and limited cognitive, physical, and social activities.

While the genetic basis for early-onset AD is much better understood, the genetic basis of late-onset AD is considered far more complex, with susceptibility conferred by a variety of more common but less penetrant genetic factors likely interacting with environmental and epigenetic influences. To date, the most firmly established genetic risk factor for late-onset disease is APOE:¹⁰

- The APOE gene is located on chromosome 19 and exists in 3 alleles: epsilon 2, 3, and 4. The APOE epsilon 4 (ϵ 4) allele has been confirmed to be an important as a risk factor for AD in many clinical trials.
- Factors that may influence the impact of APOE ϵ 4 on AD risk include female gender, African/African-American race (although there are conflicting data), vascular risk factors (e.g., smoking, diabetes, hypertension, and hypercholesterolemia), and modifier genes/environment.

- Genetic testing is available for the known causative genes in early-onset AD but has not been widely adopted, likely in part because of the current lack of highly effective preventive or therapeutic strategies.

The symptoms at early stage AD are less pronounced than in later stages of AD, and therefore require measures that are different from those used in later stages.

The Clinical Dementia Rating-Sum of Boxes (CDR-SB) is an integrated scale that assesses both daily function and cognitive effects and was shown to be sufficiently sensitive and specific to detect change over time in early symptomatic AD patients. The scale integrates assessments from 3 domains of cognition (memory, orientation, judgment/problem-solving) and 3 domains of function (community affairs, home/hobbies, personal care). CDR-SB scores range from 0-18, with higher scores indicating greater disease severity. A minimal clinically important difference in CDR-SB has not been clearly defined but has been estimated to be 1-2 points.^{5,11,41} A CDR-SB score ranging from 0.5 - 4.0 has been reported to correspond to a CDR-G score of 0.5. A CDR-SB score ranging from 4.5-9.0 has been reported to correspond to a CDR-G score of 1.²⁶

CDR-SB Score	Disease Severity
0	Normal
0.5 - 4.0	Suggests questionable cognitive impairment to very mild dementia
0.5 - 2.5	Suggests questionable cognitive impairment
3.0 - 4.0	Suggests very mild dementia
4.5 - 9.0	Suggests mild dementia
9.5 - 15.5	Suggests moderate dementia
16.0 - 18.0	Suggests severe dementia

The Mini-Mental State Exam (MMSE) is a widely used performance-based test of global cognitive status. The MMSE is a measure of cognition that includes 11 tasks relating to topics of word recall, attention and calculation, language ability, and visuospatial function. The scale ranges from 0 to 30 with a lower score reflecting greater cognitive impairment. It has several known limitations impacting sensitivity to change, particularly in earlier disease stages: substantial ceiling effects, sensitivity to practice effects, scores are impacted by patients' educational achievement, and learning effects are observed. The minimal clinically important difference of the MMSE in AD is estimated to be 1-3 points, and in early AD to be 1-2 points.^{5,11,12,27,41}

MMSE Score	Disease Severity
25 - 30	Normal to questionable cognitive impairment
19 - 24	Suggests mild dementia
10 - 18	Suggests moderate dementia
0 - 9	Suggests severe dementia

The Alzheimer's Disease Assessment Scale - Cognitive Subscale (13-Item version) (ADAS-Cog13) comprises both cognitive tasks and clinical ratings of cognitive performance. The scale items capture word recall, ability to follow commands, the ability to correctly copy or draw an image, naming, the ability to interact with everyday objects, orientation, word recognition, memory, comprehension of spoken language, word-finding, and language ability, with a measure for delayed word recall and concentration/distractibility. The total score ranges from 0 to 85 with an increase in score over time indicates increasing cognitive impairment. The minimal clinically important difference of the ADAS-COG 13 in early AD is estimated to be 3 points.^{5,11,42}

The Montreal Cognitive Assessment (MoCA) is a widely used screening test specifically designed to detect more subtle cognitive deficits that characterize mild cognitive impairment. Like the MMSE, the MoCA is scored on a 30-point scale, with items that assess delayed word recall, visuospatial/executive function, language, attention/concentration, and orientation. Studies examining head-to-head performance of patients on the MMSE and MoCA have shown that the MoCA is more difficult; MoCA scores are consistently lower than those obtained on the MMSE. The MoCA appears to be more sensitive than the MMSE for detecting MCI, though perhaps slightly less specific. A minimum clinically important difference of the MoCA in AD has not been described.⁴³

Assessment Scale	Minimal Clinical Important Difference
Clinical Dementia Rating-Sum of Boxes (CDR-SB)	1-2 points
Mini-Mental State Exam (MMSE)	1-3 points
Alzheimer’s Disease Assessment Scale – Cognitive Subscale (13-Item version) (ADAS-Cog13)	3 points

The stages of AD dementia can be defined by the MMSE and MoCA scores below:¹²

- Mild dementia (MMSE 19 to 26; MoCA 12 to 16)
- Moderate dementia (MMSE 10 to 18; MoCA 4 to 11)
- Severe dementia (MMSE < 10; MoCA < 4)

The National Institute on Aging and the Alzheimer’s Association (NIA-AA) research framework committee created a numeric clinical staging scheme (table below) applicable for diagnosing those in the Alzheimer’s continuum. This staging scheme reflects the sequential evolution of AD from an initial stage characterized by the appearance of abnormal AD biomarkers in asymptomatic individuals. As biomarker abnormalities progress, the earliest subtle symptoms become detectable.⁴⁰

Stage	Numeric Clinical Staging—Applicable Only to Individuals in the Alzheimer’s Continuum
Stage 1	<ul style="list-style-type: none"> • Performance within expected range on objective cognitive tests. Cognitive test performance may be compared to normative data of the investigator’s choice, with or without adjustment (the choice of the investigators) for age, sex, education, etc.* • Does not report recent decline in cognition or new onset of neurobehavioral symptoms of concern. • No evidence of recent cognitive decline or new neurobehavioral symptoms by report of an observer (e.g., study partner) or by longitudinal cognitive testing if available.
Stage 2	<ul style="list-style-type: none"> • Normal performance within expected range on objective cognitive tests. • Transitional cognitive decline: Decline in previous level of cognitive function, which may involve any cognitive domain(s) (i.e., not exclusively memory). • May be documented through subjective report of cognitive decline that is of concern to the participant. • Represents a change from individual baseline within past 1–3 years, and persistent for at least 6 months. • May be corroborated by informant but not required. • Or may be documented by evidence of subtle decline on longitudinal cognitive testing but not required. • Or may be documented by both subjective report of decline and objective evidence on longitudinal testing. • Although cognition is the core feature, mild neurobehavioral changes—for example, changes in mood, anxiety, or motivation—may coexist. In some individuals, the primary complaint may be neurobehavioral rather than cognitive. Neurobehavioral symptoms should have a clearly defined recent onset, which persists and cannot be explained by life events† • No functional impact on daily life activities
Stage 3	<ul style="list-style-type: none"> • Performance in the impaired/abnormal range on objective cognitive tests. • Evidence of decline from baseline, documented by the individual’s report or by observer (e.g., study partner) report or by change on longitudinal cognitive testing or neurobehavioral behavioral assessments. • May be characterized by cognitive presentations that are not primarily amnesic‡ • Performs daily life activities independently, but cognitive difficulty may result in detectable but mild functional impact on the more complex activities of daily life, that is, may take more time or be less efficient but still can complete, either self-reported or corroborated by a study partner.

Stage	Numeric Clinical Staging—Applicable Only to Individuals in the Alzheimer’s Continuum
Stage 4	<ul style="list-style-type: none"> Mild dementia Substantial progressive cognitive impairment affecting several domains, and/or neurobehavioral disturbance. Documented by the individual’s report or by observer (e.g., study partner) report or by change on longitudinal cognitive testing. Clearly evident functional impact on daily life, affecting mainly instrumental activities. No longer fully independent/requires occasional assistance with daily life activities.
Stage 5	<ul style="list-style-type: none"> Moderate dementia Progressive cognitive impairment or neurobehavioral changes. Extensive functional impact on daily life with impairment in basic activities. No longer independent and requires frequent assistance with daily life activities.
Stage 6	<ul style="list-style-type: none"> Severe dementia Progressive cognitive impairment or neurobehavioral changes. Clinical interview may not be possible. Complete dependency due to severe functional impact on daily life with impairment in basic activities, including basic self-care.
Notes	<p>* For stages 1-6: Cognitive test performance may be compared to normative data of the investigator’s choice, with or without adjustment (choice of the investigators) for age, sex, education, etc.</p> <p>† For stages 2-6: Although cognition is the core feature, neurobehavioral changes – for example, changes in mood, anxiety, or motivation – may coexist.</p> <p>‡ For stages 3-6: Cognitive impairment may be characterized by presentations that are not primarily amnesic.</p>

Despite the existence of several FDA-approved therapies for AD, there is an unmet medical need for treatments that are intended to address the biological basis of AD. Currently approved treatments do not target the underlying pathology of AD.⁵ Cholinesterase inhibitors (donepezil, galantamine, and rivastigmine) and the NMDA-antagonist, memantine, are the only FDA-approved and guideline-recommended treatments for AD dementia.¹³ The majority of patients with newly diagnosed AD should be offered a trial of a cholinesterase inhibitor for symptomatic treatment of cognition and global functioning. However, the degree of expected benefit is modest, and therapy should only be continued in patients who appear to be benefiting.¹²

Aducanumab is a human immunoglobulin gamma 1 (IgG1) monoclonal antibody that selectively targets aggregated forms of A β , including soluble oligomers and insoluble fibrils. Aducanumab crosses the blood-brain barrier and targets aggregated forms of A β in brain tissue, ultimately leading to clearance of A β plaques through a microglia-mediated phagocytosis process.^{4,7}

Clinical Evidence

Multiple investigational anti-A β antibodies have been developed with the goal of either reducing production of A β or lowering levels of aggregated A β present in the brain, the latter of which has been the most pursued approach. Many of these investigational drugs have failed to demonstrate efficacy and/or safety. Some explanations for the failures of previous anti-A β antibodies include the following:^{5,14}

- Inclusion of patients in clinical trials without evidence of A β pathology
- Unknown or no target engagement prior to initiation of Phase 3 study (i.e., poor selectivity of drug for neurotoxic A β)
- Lack of robust and sustained inhibition of soluble A β oligomers
- Use of subtherapeutic doses (possibly due to decreased brain penetration)
- Inclusion of patients at later stages of AD dementia, when significant irreversible neurodegeneration has already occurred

Aducanumab is the first anti-A β antibody to achieve proof of concept prior to Phase 3 studies.⁵ After the completion of PRIME (Study 103; N = 165), which demonstrated a dose-dependent effect with aducanumab, 2 identically-designed studies (ENGAGE [Study 301] and EMERGE [Study 302]) were initiated. Both studies were Phase 3, double-blind, placebo-controlled, multicenter (global), randomized controlled trials (ENGAGE, N = 1647; EMERGE, N = 1638) and were designed to have a titration period of 6 months (to minimize risk of AEs), followed by administration of 14 doses of aducanumab over 12 months. Key inclusion criteria included patients age 50 to 85 years, a positive amyloid PET scan, a Clinical Dementia Rating Global (CDR-G) score of

0.5, a Repeatable Battery for the Assessment of Neuropsychological Status (RBANS) score of ≤ 85 , a Mini-Mental State Exam (MMSE) score of ≥ 24 . Key exclusion criteria included a transient ischemic attack or stroke or any unexplained loss of consciousness within 1 year prior to screening, baseline MRI showing signs of previous hemorrhage, the history of a bleeding disorder, or the use of an antiplatelet or anticoagulant (aspirin at a dose of ≤ 325 mg was allowed).

A total of 6757 patients were screened for entry into the EMERGE study and 1638 (24%) patients were randomized. The most common reasons reported for screening failure were having a specific AD functional score (CDR, MMSE or RBANS) outside the allowed range (62%) or not having a positive amyloid PET scan (16%). A total of 6173 patients were screened for entry into the ENGAGE study and 1647 (27%) patients were randomized. The most common reasons reported for screening failure were having a specific AD functional score (CDR, MMSE, or RBANS) outside the allowed range (67%) or not having a positive amyloid PET scan (12%). Patients were randomized 1:1:1 to the low-dose aducanumab, high-dose aducanumab, or placebo group, and stratified based on apolipoprotein E (APOE) $\epsilon 4$ carrier status. A protocol amendment (protocol 4) eventually allowed for all patients in the high-dose aducanumab group to receive the 10 mg/kg target dose (previously only non-carriers of APOE $\epsilon 4$ received this target dose). At baseline, 80% of patient were diagnosed with mild cognitive impairment (MCI) with 20% having mild AD, patients had a mean age of 70 and a mean score on the CDR-SB of 2.4.^{5,7}

In March 2019, Biogen conducted a prespecified interim futility analysis on pooled data from ENGAGE and EMERGE studies. Results of this futility analysis demonstrated that aducanumab failed to meet its objectives, which resulted in Biogen terminating Phase 3 clinical trials. Subsequent examination of individual study results that included additional data that accrued during the time the futility analysis revealed findings that differed from the results of the prespecified futility analysis. Most notably, statistically significant results were observed from EMERGE.^{5,15}

In the EMERGE study, high-dose aducanumab demonstrated statistically significant treatment effect on change from baseline in CDR-SB compared to placebo (-0.39, [95%CI -0.69 to -0.09], $p=0.012$). Statistically significant differences from placebo were also demonstrated in secondary endpoints of Mini Mental State Examination (MMSE), Alzheimer's Disease Cooperative Study Group - Activities of Daily Living (ADCS-ADL-MCI), and Alzheimer's Disease Assessment Scale - Cognitive Subscale (ADAS-Cog13). In an exploratory analysis, statistically significant reductions in amyloid beta examined by PET were demonstrated in the subset of evaluated patients treated with aducanumab at week 26 and 28. However, additional studies are needed to establish a clinical benefit.

The ENGAGE study, in contrast, failed to meet its primary and secondary endpoints at both low- and high-dose aducanumab. Consistent with the EMERGE study finding, statistically significant reductions in amyloid beta examined by PET were demonstrated in the subset of evaluated patients treated with aducanumab at week 26 and 28. A post-hoc analyses was conducted to understand why EMERGE demonstrated statistically significant outcomes while ENGAGE did not demonstrate benefit. Biogen concluded the following:

- A smaller subset of patients received the target dose of 10 mg/kg (due to the protocol amendment that occurred later in the study) in ENGAGE.
- Based on their post-hoc analysis (using an extra 3 months of data [December 2018 to March 2019]), patients who did receive a sufficient number of aducanumab 10 mg/kg doses did demonstrate statistically significant results in this study.
- There were a greater number of rapidly progressing AD patients in the high-dose group of the ENGAGE study, which could have contributed to the lack of benefit.

Both the ENGAGE and EMERGE studies demonstrated safety concerns with the development of amyloid related imaging abnormalities (ARIA) in patients treated with aducanumab. ARIA can be classified as ARIA-E, ARIA with associated brain edema or sulcal effusions, or ARIA-H, ARIA which includes microhemorrhage and superficial siderosis. Of the patients who received high dose aducanumab, 41% experienced ARIA in ENGAGE and EMERGE. ARIA-E occurred in 35.0% and ARIA-H was observed in 28.3% in the high-dose arm across the two trials, compared with only 2.7% and 8.7% in the placebo arms, respectively. The risk of ARIA was greater in patients receiving aducanumab who were APOE $\epsilon 4$ carriers compared to noncarriers. ARIA led to discontinuation of study therapy in 7% of participants receiving the high dose of aducanumab compared with only 0.6% of participants in the placebo arm. Due to these findings, aducanumab has a labeled warning for ARIA. A baseline MRI is required prior to initiating aducanumab and follow-up MRIs are also required prior to the 7th and 12th infusions of aducanumab to monitor for ARIA.

On November 6, 2020, the FDA PCNS advisory committee convened to examine data supporting the approval of aducanumab for AD. The advisory committee voted to not approve aducanumab. Key questions and corresponding responses are listed below:¹⁶

- “Does Study 302 (EMERGE), viewed independently and without regard for Study 301 (ENGAGE), provide strong evidence that supports the effectiveness of aducanumab for the treatment of AD?” Responses: 1 voted yes, 8 voted no, and 2 were uncertain.
- “Has the Applicant presented strong evidence of a pharmacodynamic effect of aducanumab on AD pathophysiology?” Responses: 0 voted yes, 7 voted no, and 4 were uncertain.
- “Does Study 103 (PRIME) provide supportive evidence of the effectiveness of aducanumab for the treatment of AD?” Responses: 5 voted yes, 0 voted no, and 6 were uncertain.
- “In light of the understanding provided by the exploratory analyses of Study 301 and Study 302, along with the results of Study 103 and evidence of a pharmacodynamic effect on AD pathophysiology, it is reasonable to consider Study 302 as primary evidence of effectiveness of aducanumab for the treatment of AD?” Responses: 0 voted yes, 10 voted no, and 1 was uncertain.

In March 2020, a global, OL, single-arm, Phase 3b clinical trial (EMBARK [NCT04241068]) in approximately 2400 patients was initiated with the primary objective of assessing the long-term safety of aducanumab 10 mg/kg in AD patients who were actively participating in aducanumab clinical trials (PRIME [Phase 1b], EVOLVE [Phase 2], ENGAGE [Phase 3], and EMERGE [Phase 3]) at the time of their early termination (March 2019).^{17,18} Secondary study objectives include long-term efficacy, and long-term effect of aducanumab on biomarkers and pharmacokinetic endpoints. Another goal of EMBARK is to provide answers regarding the effect of prolonged treatment interruption and improve understanding of the durability of aducanumab’s treatment effects.

U.S. Food and Drug Administration (FDA)

This section is to be used for informational purposes only. FDA approval alone is not a basis for coverage.

Aduhelm (aducanumab-avwa) is an A β -targeting antibody indicated for the treatment of Alzheimer’s disease. Treatment with Aduhelm should be initiated in patients with mild cognitive impairment or mild dementia stage of disease, the population in which treatment was initiated in clinical trials. There are no safety or effectiveness data on initiating treatment at earlier or later stages of the disease than were studied. This indication is approved under accelerated approval based on reduction in amyloid beta plaques observed in patients treated with Aduhelm. Continued approval for this indication may be contingent upon verification of clinical benefit in confirmatory trial(s).

To monitor for Amyloid Related Imaging Abnormalities (ARIA), a recent (within one year) brain magnetic resonance imaging (MRI) prior to initiating treatment is required. MRIs are required prior to the 7th infusion (first dose of 10 mg/kg) and 12th infusion (sixth dose of 10 mg/kg). The safety of Aduhelm in patients with any pre-treatment localized superficial siderosis, 10 or more brain microhemorrhages, and/or with a brain hemorrhage greater than 1 cm within one year of treatment initiation has not been established. If 10 or more new incident microhemorrhages or > 2 focal areas of superficial siderosis (radiographic severe ARIA-H) is observed while receiving Aduhelm, treatment may be continued with caution only after a clinical evaluation and a follow-up MRI demonstrates radiographic stabilization (i.e., no increase in size or number of ARIA-H).

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Policy History/Revision Information

Date	Summary of Changes
06/01/2022	<ul style="list-style-type: none"> • New Medical Benefit Drug Policy

Instructions for Use

This Medical Benefit Drug Policy provides assistance in interpreting UnitedHealthcare standard benefit plans. When deciding coverage, the member specific benefit plan document must be referenced as the terms of the member specific benefit plan may differ from the standard plan. In the event of a conflict, the member specific benefit plan document governs. Before using this policy, please check the member specific benefit plan document and any applicable federal or state mandates. UnitedHealthcare reserves the right to modify its Policies and Guidelines as necessary. This Medical Benefit Drug Policy is provided for informational purposes. It does not constitute medical advice.

This Medical Benefit Drug Policy may also be applied to Medicare Advantage plans in certain instances. In the absence of a Medicare National Coverage Determination (NCD), Local Coverage Determination (LCD), or other Medicare coverage guidance, CMS allows a Medicare Advantage Organization (MAO) to create its own coverage determinations, using objective evidence-based rationale relying on authoritative evidence ([Medicare IOM Pub. No. 100-16, Ch. 4, §90.5](#)).

UnitedHealthcare may also use tools developed by third parties, such as the InterQual® criteria, to assist us in administering health benefits. UnitedHealthcare Medical Benefit Drug Policies are intended to be used in connection with the independent professional medical judgment of a qualified health care provider and do not constitute the practice of medicine or medical advice.