

Leqembi® (Lecanemab-Irmb)

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[Instructions for Use](#)

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Related Community Plan Policy

- [Monoclonal Antibodies Directed Against Amyloid for the Treatment of Alzheimer's Disease](#)

Coverage Rationale

[See Benefit Considerations](#)

This policy refers to Leqembi (lecanemab-irmb) for administration by intravenous infusion by a healthcare professional. Leqembi IQLIK for self-administered subcutaneous injection is obtained under the pharmacy benefit, unless otherwise specified in the member's benefit plan documents. Exception: For members enrolled in UnitedHealthcare of California plans with a delegated provider group conducting the prior authorization review, the self-administered Leqembi IQLIK may be obtained under the medical benefit.

Leqembi is proven for the treatment of Alzheimer's disease (AD) when all of the following criteria are met:

- For **initial therapy**, all of the following:
 - Diagnosis of **one** of the following based on National Institute on Aging and the Alzheimer's Association (NIA-AA) criteria:
 - Mild cognitive impairment (MCI) due to Alzheimer's disease; **or**
 - Mild dementia due to Alzheimer's disease
 - and**
 - Presence of amyloid beta pathology has been confirmed; **and**
 - A baseline brain magnetic resonance imaging (MRI) has been completed within 12 months prior to initiating treatment; **and**
 - Not used in combination with other Aβ monoclonal antibodies (mAbs) for Alzheimer's Disease (e.g., Kisunla); **and**
 - Leqembi dosing is in accordance with the United States Food and Drug Administration approved labeling; **and**
 - Initial authorization will be for no more than 12 months
- For **continuation of therapy**, all of the following:
 - Diagnosis of Alzheimer's disease; **and**
 - Follow-up brain MRI has been completed after the initiation of therapy; **and**
 - Not used in combination with other Aβ monoclonal antibodies (mAbs) for Alzheimer's Disease (e.g., Kisunla); **and**
 - Leqembi dosing is in accordance with the United States Food and Drug Administration approved labeling; **and**
 - Reauthorization is for no more than 12 months

Leqembi (lecanemab-irmb) is medically necessary for the treatment of Alzheimer's disease (AD) when all of the following criteria are met:

- For **initial therapy**, all of the following:
 - Diagnosis of **one** of the following based on National Institute on Aging and the Alzheimer's Association (NIA-AA) criteria:

- Mild cognitive impairment (MCI) due to Alzheimer's disease; **or**
 - Mild dementia due to Alzheimer's disease
- and**
- Submission of medical records (e.g., chart notes, laboratory values) documenting **one** of the following:
 - Mini-Mental State Examination (MMSE) score of 20 to 30
 - Montreal Cognitive Assessment (MoCA) score of 17 to 30
 - Saint Louis University Mental Status (SLUMS) score of 17 to 30
- and**
- Submission of medical records (e.g., chart notes, laboratory values) documenting the presence of amyloid beta pathology, as evidenced by **one** of the following:
 - Positive amyloid positron emission tomography (PET) brain imaging; **or**
 - Cerebrospinal fluid (CSF) biomarker testing documents abnormalities suggestive of beta-amyloid accumulation in the brain (e.g., A β 42/40 ratio, p-tau 181/A β 42 ratio, t-tau/A β 42 ratio)
- and**
- Other differential diagnoses [e.g., dementia with Lewy bodies (DLB), frontotemporal dementia (FTD), vascular dementia, pseudodementia due to mood disorder, vitamin B12 deficiency, encephalopathy, etc.] have been ruled out; **and**
 - **One** of the following:
 - Patient is not currently taking an anticoagulant (e.g., warfarin, dabigatran); **or**
 - **Both** of the following:
 - Patient is currently taking an anticoagulant (e.g., warfarin, dabigatran); **and**
 - Counseling has been provided that the combined use of Leqembi with anti-coagulant drugs may increase the risk of cerebral macrohemorrhage and prescriber attests that the patient has shared in decision-making to initiate Leqembi therapy
- and**
- Patient has no history of intracerebral hemorrhage within the previous year prior to initiating treatment; **and**
 - Counseling has been provided on the risk of amyloid-related imaging abnormalities [ARIA characterized as ARIA with edema (ARIA-E) and ARIA with hemosiderin (ARIA-H)] and patient is aware to monitor for headache, dizziness, visual disturbances, nausea, and vomiting; **and**
 - **All** of the following:
 - Counseling has been provided on how testing for apolipoprotein E (ApoE) epsilon 4 (ϵ 4) status informs the risk of developing ARIA when deciding to initiate treatment with Leqembi; **and**
 - Testing for ApoE ϵ 4 status has been offered to the patient and prescriber attests that the patient has shared in decision-making to initiate Leqembi therapy
- and**
- A baseline brain magnetic resonance imaging (MRI) has been completed within 12 months prior to initiating treatment; **and**
 - Not used in combination with other A β monoclonal antibodies (mAbs) for Alzheimer's Disease (e.g., Kisunla); **and**
 - Prescribed by a neurologist, geriatric psychiatrist, or geriatrician who specializes in treating dementia; **and**
 - Leqembi dosing is in accordance with the United States Food and Drug Administration approved labeling; **and**
 - Initial authorization will be for no more than 12 months
- For **continuation of therapy**, **all** of the following:
 - **Both** of the following:
 - Submission of medical records (e.g., chart notes) confirming follow-up brain magnetic resonance imaging (MRI) has been completed after the initiation of therapy; **and**
 - **One** of the following:
 - ARIA has not been observed on MRI; **or**
 - All of the following:
 - ARIA has been observed on MRI; **and**
 - Prescriber attests that continuation of therapy with Leqembi is appropriate based on the severity of the patient's clinical symptoms; **and**
 - **One** of the following:
 - Follow-up MRI demonstrates radiographic resolution and/or stabilization; **or**
 - Prescriber attests that continuation of therapy with Leqembi is appropriate based on the radiographic severity of ARIA
- and**
- Not used in combination with other A β monoclonal antibodies (mAbs) for Alzheimer's Disease (e.g., Kisunla); **and**
 - Prescribed by a neurologist, geriatric psychiatrist, or geriatrician who specializes in treating dementia; **and**
 - Leqembi dosing is in accordance with the United States Food and Drug Administration approved labeling; **and**
 - Reauthorization is for no more than 12 months

Leqembi (lecanemab-irmb) is unproven and not medically necessary for any indication other than Alzheimer's disease.

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
J0174	Lecanemab-irmb, for intravenous injection, 1 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 cases. AD is characterized by deposition of amyloid-beta A β plaques and neurofibrillary tangles (comprised of abnormal tau protein) in the brain, accompanied by synaptic dysfunction and neurodegeneration. 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.

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.

Abnormal A β may be detected 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.

Age of AD onset:

- 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 AD: Early-onset AD is less common, and 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.
- 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:

- 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:

- The APOE gene is located on chromosome 19 and exists in 3 alleles: epsilon 2, 3, and 4. The APOE epsilon 4 (ε4) allele has been confirmed to be an important risk factor for AD in many clinical trials.
- Factors that may influence the impact of APOE ε4 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. A CDR-SB score ranging from 0.5 - 4.0 has been reported to correspond to a Global CDR (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.

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.

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.

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.

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 National Institute on Aging and the Alzheimer’s Association (NIA-AA) research framework committee created a numeric clinical staging scheme applicable for diagnosing those in the Alzheimer’s continuum. The six-stage numeric clinical staging scheme was brought forward largely unchanged (table below) into an Alzheimer’s Association 2024 revision of the 2018 framework.

Stage	Numeric Clinical Staging–Applicable Only to Individuals in the Alzheimer’s Disease Continuum
Stage 0 Asymptomatic, deterministic gene	<ul style="list-style-type: none"> No evidence of clinical change. Biomarkers in normal range.
Stage 1 Asymptomatic, biomarker evidence only	<ul style="list-style-type: none"> Performance within expected range on objective cognitive tests. No evidence of recent cognitive decline or new symptoms.
Stage 2 Transitional decline: mild detectable change, but minimal impact on daily function	<ul style="list-style-type: none"> Normal performance within expected range on objective cognitive tests. Decline from previous level of cognitive or neurobehavioral function that represents a change from individual baseline within the past 1 to 3 years, and has been persistent for at least 6 months. May be documented by evidence of subtle decline on longitudinal cognitive testing, which may involve memory or other cognitive domains but performance still within normal range. May be documented through subjective report of cognitive decline. May be documented with recent onset change in mood, anxiety, motivation not explained by life events. Remains fully independent with no or minimal functional impact on activities of daily living (ADLs).
Stage 3 Cognitive impairment with early functional impact	<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 assessments. Performs daily life activities independently, but cognitive difficulty may result in detectable functional impact on complex ADLs (i.e., may take more time or be less efficient but still can complete—either self-reported or corroborated by an observer).

Stage	Numeric Clinical Staging—Applicable Only to Individuals in the Alzheimer’s Disease Continuum
Stage 4 Dementia with mild functional impairment	<ul style="list-style-type: none"> Progressive cognitive and mild functional impairment on instrumental ADLs, with independence in basic ADLs.
Stage 5 Dementia with moderate functional impairment	<ul style="list-style-type: none"> Progressive cognitive and moderate functional impairment on basic ADLs requiring assistance.
Stage 6 Dementia with severe functional impairment	<ul style="list-style-type: none"> Progressive cognitive and functional impairment, and complete dependence for basic ADLs.

Leqembi (lecanemab-irmb) is a humanized immunoglobulin gamma 1 (IgG1) monoclonal antibody directed against aggregated soluble and insoluble forms of amyloid beta. The accumulation of amyloid beta plaques in the brain is a defining pathophysiological feature of Alzheimer’s disease.

Benefit Considerations

Some Certificates of Coverage allow for coverage of experimental/investigational/unproven treatments for life-threatening illnesses when certain conditions are met. The member specific benefit plan document must be consulted to make coverage decisions for this service. Some states mandate benefit coverage for off-label use of medications for some diagnoses or under some circumstances when certain conditions are met. Where such mandates apply, they supersede language in the benefit document or in the medical or drug policy.

Clinical Evidence

Multiple investigational anti-Aβ antibodies have been developed with the goal of either reducing production of Aβ or lowering levels of aggregated Aβ 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-Aβ antibodies include the following:

- Inclusion of patients in clinical trials without evidence of Aβ pathology
- Unknown or no target engagement prior to initiation of Phase 3 study (i.e., poor selectivity of drug for neurotoxic Aβ)
- Lack of robust and sustained inhibition of soluble Aβ 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

FDA approval for lecanemab was based on Study 201, an 18-month, Phase 2b, double-blind, placebo controlled, multicenter, randomized control trial that evaluated the safety and efficacy of lecanemab. The study aimed to establish the effective dose 90% (ED90), defined as the simplest dose that achieves ≥ 90% of the maximum treatment effect. The primary endpoint was Bayesian analysis of 12-month clinical change on the Alzheimer’s Disease Composite Score (ADCOMS) for the ED90 dose, which required an 80% probability of ≥ 25% clinical reduction in decline versus placebo. Study 201 enrolled 854 patients, who were treated with lecanemab (609/854) or placebo (245/854). Of the total number of patients randomized, 71.4% were ApoE ε4 carriers and 28.6% were ApoE ε4 non-carriers. During the study, the protocol was amended to no longer randomize ApoE ε4 carriers to the 10 mg/kg every two weeks dose arm. ApoE ε4 carriers who had been receiving lecanemab 10 mg/kg every two weeks for 6 months or less were discontinued from study drug. The primary analysis conducted at Month 12 of treatment indicated that the 10 mg/kg IV biweekly dose (the effective dose) had a 64% probability to be better than placebo by 25% on ADCOMS at 12 months, missing the prespecified 80% probability threshold for the primary outcome.

The results for the Bayesian analysis for reduction of clinical decline at 18 months vs placebo for 10 mg/kg biweekly on ADCOMS (-27%, with 97.7% probability to be superior to placebo), CDR-SB (33%, with 96.4% probability to be superior to placebo), and ADASCog14 (56%, with a 98.8% probability to be superior to placebo) were similar to the results from the corresponding conventional analyses for clinical measures when comparing mean change from baseline and least squares (LS) mean data.

The CLARITY AD Phase 3 study was conducted to evaluate the efficacy of lecanemab in participants with early Alzheimer’s disease (EAD) by determining the superiority of lecanemab compared with placebo on the change from baseline in the Clinical Dementia Rating-Sum of Boxes (CDR-SB) at 18 months of treatment in the Core Study. This study will also evaluate the long-term safety and tolerability of lecanemab in participants with EAD in the Extension Phase and

whether the long-term effects of lecanemab as measured by the CDR-SB at the end of the Core Study is maintained over time in the Extension Phase. CLARITY AD was an 18-month, multicenter, double-blind, phase 3 trial involving persons 50 to 90 years of age with early Alzheimer's disease (mild cognitive impairment or mild dementia due to Alzheimer's disease) with evidence of amyloid on positron-emission tomography (PET) or by cerebrospinal fluid testing. Participants were randomly assigned in a 1:1 ratio to receive intravenous lecanemab (10 mg per kilogram of body weight every 2 weeks) or placebo. The primary end point was the change from baseline at 18 months in the score on the Clinical Dementia Rating–Sum of Boxes (CDR-SB; range, 0 to 18, with higher scores indicating greater impairment). Key secondary end points were the change in amyloid burden on PET, the score on the 14-item cognitive subscale of the Alzheimer's Disease Assessment Scale (ADAS-cog14; range, 0 to 90; higher scores indicate greater impairment), the Alzheimer's Disease Composite Score (ADCOMS; range, 0 to 1.97; higher scores indicate greater impairment), and the score on the Alzheimer's Disease Cooperative Study–Activities of Daily Living Scale for Mild Cognitive Impairment (ADCSMCI-ADL; range, 0 to 53; lower scores indicate greater impairment). A total of 1795 participants were enrolled, with 898 assigned to receive lecanemab and 897 to receive placebo. The mean CDR-SB score at baseline was approximately 3.2 in both groups. The adjusted least-squares mean change from baseline at 18 months was 1.21 with lecanemab and 1.66 with placebo [difference, -0.45; 95% confidence interval (CI), -0.67 to -0.23; $p < 0.001$]. Furthermore, a slope analysis demonstrated that lecanemab took 5.5 to 6 months more time to achieve the same CDR-SB as placebo at 18 months, indicating a 5.5 to 6 month slowing of progression. A β plaque reduction was a secondary endpoint and was studied in a subset of patients ($n = 698$). The adjusted mean change from baseline at 18 months was -55.48 centiloids in the lecanemab group vs 3.64 centiloids in the placebo group (adjusted mean difference, -59.12 centiloids; 95% CI, -62.64 to -55.60; $p < 0.001$). Lecanemab resulted in infusion-related reactions in 26.4% of the participants and amyloid-related imaging abnormalities with edema or effusions in 12.6%. The incidence of ARIA-E with lecanemab was 12.5% vs 1.7% with placebo (symptomatic ARIA-E: 2.8% vs 0% with placebo). The incidence of ARIA-H was 17.0% vs 8.7% with placebo (symptomatic ARIA-H: 0.7% vs 0.2% in placebo group). In a sub study involving 698 participants, there were greater reductions in brain amyloid burden with lecanemab than with placebo (difference, -59.1 centiloids; 95% CI, -62.6 to -55.6). Other mean differences between the two groups in the change from baseline favoring lecanemab were as follows: for the ADAS-cog14 score, -1.44 (95% CI, -2.27 to -0.61; $p < 0.001$); for the ADCOMS, -0.050 [(95% CI, -0.074 to -0.027; $p < 0.001$); and for the ADCS-MCIADL score, 2.0 (95% CI, 1.2 to 2.8; $p < 0.001$).

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.

Leqembi (lecanemab-irmb) is indicated for the treatment of Alzheimer's disease. Treatment with Leqembi 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.

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Policy History/Revision Information

Date	Summary of Changes
04/01/2026	<p>Applicable Codes</p> <ul style="list-style-type: none"> Updated list of applicable HCPCS codes to reflect quarterly edits; revised description for J0174 <p>Supporting Information</p> <ul style="list-style-type: none"> Archived previous policy version 2026D0125D

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, 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.