Interleukin-4 (IL-4) Inhibitor Prior Authorization with Quantity Limit Program Summary

POLICY REVIEW CYCLE

Effective Date 03-17-2025

Date of Origin

FDA LABELED INDICATIONS AND DOSAGE

Agent(s)	FDA Indication(s)	Notes	Ref#
Dupixent® (dupilumab) Injection for subcutaneous use	Treatment of adult and pediatric patients aged 6 months and older with moderate-to-severe atopic dermatitis (AD) whose disease is not adequately controlled with topical prescription therapies or when those therapies are not advisable. Dupixent can be used with or without topical corticosteroids Add-on maintenance treatment of adult and pediatric patients aged 6 years and older with moderate-to-severe asthma characterized by an eosinophilic phenotype or with oral corticosteroid dependent asthma • Limitation of Use: Not indicated for the relief of acute bronchospasm or status asthmaticus Add-on maintenance treatment in adult and pediatric patients aged 12 years and older with inadequately controlled chronic rhinosinusitis with nasal polyps (CRSwNP) Treatment of adult and pediatric patients aged 1 year and older, weighing at least 15 kg, with eosinophilic esophagitis (EoE) Treatment of adult patients with prurigo nodularis (PN) Add-on maintenance treatment of adult patients with inadequately controlled chronic obstructive pulmonary disease (COPD) and an eosinophilic phenotype • Limitation of Use: Not indicated for the relief of acute bronchospasm		1

See package insert for FDA prescribing information: https://dailymed.nlm.nih.gov/dailymed/index.cfm

CLINICAL RATIONALE

Asthma	Asthma is a chronic inflammatory disorder of the airways. It is characterized by a history of respiratory symptoms along with variable expiratory airflow limitation, and is typically associated with bronchial hyperresponsiveness and underlying inflammation. Symptoms are variable and recurrent and include wheezing, coughing, shortness of breath, and chest tightness. Exercise, exposure to allergens and irritants, infections, and changes in the weather can be contributing factors to the variability in symptoms and airflow limitation.(11) Guidelines recommend evaluating respiratory symptoms, medical history, physical examination, and spirometry to determine a

	diagnosis of asthma.(9,11) Long-term goals for asthma management are to achieve control of symptoms, maintain normal activity level, and to minimize the future risk of exacerbations, decline in lung function, and medication side effects.(11) Different types of asthma and levels of severity exist. Moderate asthma is asthma that requires a low- or medium-dose inhaled corticosteroid (ICS) used in combination with a long-acting beta agonist (LABA) to be well controlled. Severe asthma is asthma that remains uncontrolled despite optimized treatment with high-dose ICS-LABA, or that requires high-dose ICS-LABA or biologic therapy to prevent it from becoming uncontrolled (e.g., asthma worsens when high-dose treatment is decreased). Severe asthma needs to be distinguished from difficult-to-treat asthma that remains symptomatic due to poor adherence, poor inhaler technique, comorbidities, and/or continued exposure to environmental agents since treatment and management differs between the two.(11) The European Respiratory Society (ERS)/American Thoracic Society (ATS) guidelines (2014; updated 2020) define uncontrolled asthma for adults and pediatric patients 6 years of age and older as a patient having at least one of the following:(10) • Frequent severe exacerbations (i.e., two or more bursts of systemic corticosteroids within the past 12 months) • Serious exacerbations (i.e., at least one hospitalization, intensive care unit stay, or mechanical ventilation in the past 12 months) • Airflow limitation (i.e., FEV1 less than 80% predicted) • Asthma that worsens upon tapering of high-dose ICS or systemic corticosteroids (or additional biologics) The Type 2 inflammatory asthma phenotype is found in the majority of people with severe asthma. Type 2 inflammation involves a systemic allergic response and elevated levels of Type 2 inflammatory cytokines such as interleukin (IL)-4, IL-5, and IL-13. Elevated eosinophils or an increased fractional exhaled nitric oxide (FeNO) are characteristics of the eosinophilic subtype of Type 2 inflammatory asthma, while the allergic asthma subtype is additionally characterized by elevated immunoglobulin E (IgE) levels and positive skin prick testing with common environmental allergens. Type 2 inflammation typically responds well to ICS treatment and rapidly improves, however, in severe asthma Type 2 inflammation may be relatively refractory to high-dose ICS. Maintenance oral corticosteroids (OCS) may elicit a response, but the risk of serious adverse effects with daily OCS use deters their usefulness and an alternative treatment should be sought.(11) Type 2 inflammation is considered refractory if any of the following are found while the patient is taking high dose ICS or daily OCS:(11) • Blood eosinophils greater than or equal to 150 cells/microliter • FeNO greater than or equal to 20 ppb • Sputum eosinophils greater than or equal to 2% • Asthma is clinically allergen-driven The Global Initiative for Asthma (GINA) guidelines recommend a stepwise approach for managing asthma. The 2024 GINA guidelines recommend all patients 6 years of age and older with asthma should receive ICS-containing controller medication to reduce the risk of serious exacerbation, even in patients with infrequent symptoms. It is recommended that patients with asthma symptoms most days should be started on low dose maintenance ICS-formoterol or an alternative ICS-LABA product. Patients' response to treatment should be reviewed after 2 to 3 months. If symptoms remain uncontrolled despite good adherence and correct inhaler technique, the next step up (Step 4) involves increasing controller therapy to medium or high dose ICS-formoterol (ICS-LABA). Other controller options that may be added on to ICS treatment at Step 4 include a long-acting muscarinic antagonist (LAMA), leukotriene receptor antagonist (LTRA), or theophylline. Both LTRA and theophylline are considered less efficacious than adding on a LABA or LAMA, and also come with safety concerns. Patients with uncontrolled symptoms and/or exacerbations despite being on Step 4 treatment for 3 to 6 months should be assessed for contributory factors, have their treatment optimized, and be referred for expert assessment, phenotyping, and potential add on

	biologic therapy. Maintenance oral corticosteroids (OCS) should be used only as last resort because short-term and long-term systemic side-effects are common and serious.(11) Biologic agents should be considered as add-on therapy for patients with refractory Type 2 inflammation with exacerbations and/or poor symptom control despite taking at least high dose ICS-LABA, and who have allergic or eosinophilic biomarkers or need maintenance OCS, and only after treatment has been optimized.(11) Tezepelumab is considered a broad-acting biologic and may be considered in patients without a Type 2 inflammatory phenotype due to it binding to circulating thymic stromal lymphopoietin (TSLP), which is upstream on the inflammatory cascade.(11,25) Based on efficacy results from clinical trials, the indication of use for tezepelumab is not restricted to a biomarker-defined phenotype.(25) 2024 GINA guidelines recommend the use of biologics based on the following patient eligibility factors:(11) • Anti-IgE (omalizumab) for moderate to severe allergic asthma o Sensitization to inhaled allergen(s) on skin prick testing for specific IgE o Total serum IgE and body weight within dosing range o Exacerbations within the last year • Anti-IL5 (mepolizumab, reslizumab) /Anti-IL5Ra (benralizumab) for severe eosinophilic asthma o Blood eosinophils greater than or equal to 150 cells/microliter or greater than or equal to 300 cells/microliter o Severe exacerbations within the last year • Anti-IL4Ra (dupilumab) for severe eosinophilic/Type 2 asthma or patients requiring maintenance OCS o Blood eosinophil greater than or equal to 150 cells/microliter but less than or equal to 1500 cells/microliter, or FeNO greater than or equal to 25 ppb, or taking maintenance OCS o Severe exacerbations within the last year • Anti-TSLP (tezepelumab) for severe asthma o Severe exacerbations within the last year Patient response to biologic therapy should be evaluated 4 months after initiating therapy, and the patient should be re-evaluated every 3 to 6 months. If response is unclear after 4 months, the trial should be extended to 6-12 months.(11) 2024 GINA guidelines recommend the following step-down therapy process in patients responding well to targeted biologic therapy:(11) • Re-evaluate the need for each asthma medication every 3 to 6 months, but inhaled therapy (e.g., ICS-containing therapy) should not be completely stopped • The order of reduction of treatments should be based on observed benefit, potential side-effects, cost, and patient preference. However, minimizing the use of OCS is a very high priority. • First, consider decreasing/stopping OCS due to their significant adverse effects. Then consider stopping other add-on asthma medications. • Then, if asthma is well controlled for 3-6 months, consider reducing maintenance ICS dose, but do not stop maintenance ICS-containing therapy (e.g., ICS-LABA) • Re-evaluate the need for ongoing biologic therapy, but a trial of withdrawal of the biologic should not be considered until after at least 12 months of treatment and only if asthma remains well controlled on medium-dose ICS-containing therapy o For allergic asthma, also confirm there is no further exposure to an allergic trigger
Atopic Dermatitis (AD)	Atopic dermatitis (AD), also known as atopic eczema, is a chronic, pruritic inflammatory dermatosis affecting up to 25% of children and 1-5% of adults. AD follows a relapsing course and is associated with elevated serum immunoglobulin (IgE)

	levels and a personal or family history of type I allergies, allergic rhinitis, and/or asthma. Onset is most common between 3 and 6 months of age, with approximately 60% of patients developing the eruption in the first year of life and 90% by age 5. While the majority of affected individuals have resolution of disease by adulthood, 10 to 30% do not, and a smaller percentage first develop symptoms as adults. AD has a complex pathogenesis involving genetic, immunologic, and environmental factors, which lead to a dysfunctional skin barrier and dysregulation of the immune system. Clinical findings include erythema, edema, xerosis, erosions/excoriations, oozing and crusting, and lichenification. These clinical findings vary by patient age and chronicity of lesions. Pruritus is a hallmark of the condition that is responsible for much of the disease burden borne by patients and their families. Typical patterns include facial, neck and extensor involvement in infants and children; flexure involvement in any age group, with sparing of groin and axillary regions.(2) Goals of treatment are to reduce symptoms (pruritus and dermatitis), prevent exacerbations, and minimize therapeutics risks.(13) Despite its relapsing and remitting nature, the majority of patients with AD can achieve clinical improvement and disease control with topical emollient/moisturizer use and conventional topical therapies (including corticosteroids and calcineurin inhibitors).(5,13) Moisturizers reduce signs, symptoms, and inflammation in AD, and can improve severity while also increasing time between flares. Moisturizers are considered generally safe and are strongly recommended to be used as part of a treatment regimen for AD, either as monotherapy or as concurrent use with pharmacologic treatments.(4) Topical therapies remain the mainstay of treatment due to their proven track record and generally favorable safety profile. They can be utilized individually or in combination with other topical, physical, and/or systemic treatments; as different classes of treatment have different mechanisms of action, combining therapies allows for the targeting of AD via multiple disease pathways. The American Academy of Dermatology (AAD) strongly recommends the following topical agents:(4) • Topical corticosteroids (TCS) • Calcineurin inhibitors (TCIs) (e.g., tacrolimus, pimecrolimus) • Topical PDE-4 inhibitors (e.g., crisaborole) [mild to moderate AD] • Topical JAK inhibitors (e.g., ruxolitinib) [mild to moderate AD] TCS are the most commonly utilized FDA-approved therapies in AD and are commonly used as first-line treatment for mild-to severe dermatitis in all skin regions. TCS target a variety of immune cells and suppress the release of proinflammatory cytokines. High to very high (super) potency TCS can be used to control flares and treat severe disease, while medium potency TCS are utilized for longer courses and as maintenance therapy. Lower potency TCS may be used, and it is important to consider the anatomical site (i.e., using lower potency agents on the face, neck, genitals, and body folds) and severity of the disease when choosing a steroid potency. Most studies of TCS in AD management involve twice daily application, but some studies (particularly for potent TCS) suggest once daily use may be sufficient. Traditionally, TCS were stopped once AD signs and symptoms of an AD flare were controlled. Maintenance in between AD flares with once to twice weekly use of TCS is another approach.(4) TCIs are a safe anti-inflammatory option for mild-to-severe AD, particularly when there is concern for adverse events secondary to corticosteroid use. Both tacrolimus and pimecrolimus have been shown to be effective in treating AD, but pimecrolimus may be more appropriate for patients who have milder disease or are sensitive to local reactions.(4) Prescribing information for pimecrolimus cream and tacrolimus ointment indicate evaluation after 6 weeks if symptoms of AD do not improve for adults and pediatrics.(6,12). When AD is more severe or refractory to topical treatment, advanced treatment with phototherapy or systemic medications can be considered. Phototherapy is conditionally

	recommended by the AAD as a treatment for AD based on low certainty evidence. The AAD strongly recommends the following systemic therapies:(5) • Monoclonal antibodies (biologics) (e.g., dupilumab, tralokinumab) • JAK inhibitors (e.g., upadacitinib, abrocitinib, baricitinib) In a change from the 2014 AAD AD guidelines, the use of systemic antimetabolites such as methotrexate, immunosuppressants such as systemic corticosteroids, mycophenolate mofetil, azathioprine, and cyclosporine are now conditionally recommended for AD only in a small number of select patients due to low or very low certainty of evidence and need for monitoring. The most favored first-line systemic is dupilumab.(5) There is no clear consensus on how to operationalize a definition of the FDA indication for treatment of patients with "moderate to severe" AD. The severity of AD can vary substantially over time and, from a patient's perspective, can include a complex combination of intensity of itch, location, body surface area (BSA) involvement, and degree of skin impairment. Given the variability of patient phenotype and lack of familiarity among clinicians with scoring systems used in clinical trials, it is advisable to create a broad clinically relevant definition inclusive of multiple specific measures of disease intensity for example:(26) One of the following: • Affected BSA greater than or equal to 10% • Investigator Global Assessment (IGA) greater than or equal to 3 • Eczema Area and Severity Index (EASI) greater than or equal to 16 OR One of the following: • Affected BSA greater than or equal to 10% • Involvement of body sites that are difficult to treat with prolonged topical corticosteroid therapy (e.g., hands, feet, face, neck, scalp, genitals/groin, skin folds) • Severe itch that has been unresponsive to topical therapies
Chronic Obstructive Pulmonary Disease (COPD)	Chronic obstructive pulmonary disease (COPD) is a common lung disease characterized by chronic respiratory symptoms caused by abnormalities of the airways and/or alveoli that cause persistent, and often progressive, airway obstruction. Symptoms include dyspnea, cough, sputum production, and/or exacerbations. COPD is one of the top causes of death in the United States, and prior to Covid 19, was the third leading cause of death in the world.(31) COPD develops due to a combination of environmental exposures and patient characteristics. Smoking and air pollution are the two leading environmental exposures leading to the development of COPD.(31) These toxins cause chronic inflammation, an increase in the number of goblet cells, mucus gland hyperplasia, fibrosis, and narrowing of the small airways.(32) Structural changes in the distal to terminal bronchiole lead to the development of emphysema. The structural changes also cause changes in normal ventilation-perfusion distributions.(31) Diagnosis of COPD is dependent on the presence of pulmonary symptoms (i.e., dyspnea, chronic cough, sputum production), patient's exposure history (e.g., current/previous smoker, history of recurrent lower respiratory tract infections), and evidence of airflow limitation. Diagnosis is confirmed by spirometry. A post-bronchodilator forced expiratory volume in 1 second (FEV1)/forced vital capacity (FVC) less than 0.7 is indicative of the diagnosis. Once diagnosis is confirmed it is important

to determine severity of airflow obstruction to appropriately guide treatment options.(31)

Exacerbations of COPD lead to increased dyspnea and/or cough and sputum that worsens over a less than 14 day period. They are often associated with increased airway inflammation, increased mucus production, and marked gas trapping. Symptoms of a COPD exacerbation are usually present for 7 to 10 days. They can be caused by infection, pollution, or other insult to the lungs. Moderate exacerbations are those that require treatment with a short acting bronchodilator (SABD) and oral corticosteroids, with or without an antibiotic. Severe exacerbations are associated with hospitalization or a visit to the emergency room, and may also be associated with acute respiratory failure requiring mechanical ventilation. Frequent exacerbations are defined as having two or more exacerbations per year and typically lead to a worse health status and morbidity for patients.(31)

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) ABE Assessment tool is recommended for determining initial pharmacotherapy for the management of COPD. The tool takes into account spirometry measures, disease severity, and frequency of exacerbations. The first portion of the assessment includes the GOLD grades, which determine airflow obstruction severity based on specific spirometry cut points.(31)

GOLD GRADES		
GOLD 1	Mild	FEV1 greater than or equal to 80% predicted
GOLD 2	Moderate	greater than or equal to 50% FEV1 to less than 80% predicted
GOLD 3	Severe	greater than or equal to 30% FEV1 to less than 50% predicted
GOLD 4	Very Severe	FEV1 less than 30% predicted

The tool also takes exacerbation history into account. Exacerbation history is broken into two sections. Patients then fall into one of three groups E, A, or B. For patients with 0 to 1 moderate exacerbations in the past year, the GOLD guideline recommends two symptom questionnaires to further establish initial therapy. The modified Medical Research Council (mMRC) dyspnea scale, assesses breathlessness, which is a key symptom for many patients with COPD. A more comprehensive questionnaire is the COPD Assessment Test (CAT).(31)

Initial Pharmacotherapy Treatment Algorithm		
greater than or equal to 2 exacerbations, or greater than or equal to 1 exacerbation leading to hospitalization within the past year	Group E	
0 to 1 moderate exacerbations (not leading to	Group A	Group B

hospitalization) within the past year		
	mMRC 0-1 CAT less than 10	mMRC 2 or greater CAT 10 or greater

Treatment recommendations based on GOLD ABE grouping are as follows(31):

- Group E:
    - Long-acting beta agonist (LABA) + long-acting muscarinic antagonist (LAMA)
    - Consider LABA + LAMA + inhaled corticosteroid (ICS) if blood eosinophil levels are 300 or greater
    - Use of LABA + ICS in COPD is not encouraged. If an ICS is indicated for use, ICS + LAMA + LABA has been shown to be superior to ICS + LABA
- Group A
    - A bronchodilator
- Group B
    - LABA + LAMA

The GOLD guideline has a separate algorithm for follow up therapy, based on persistence of dyspnea and occurrence of exacerbations. Patients not responding to initial therapy should have inhaler technique, adherence, and other interfering comorbidities addressed prior to initiating additional or changing therapies. Follow-up recommendations are as follows:(31)

- Dyspnea:
    - Initial therapy LABA or LAMA: switch to dual therapy (LABA/LAMA)
    - Initial therapy dual therapy: consider switching inhaler devices, implement or escalate non-pharmacologic therapies, or investigate other causes of dyspnea
- Exacerbations:
    - Initial therapy LABA or LAMA:
        - Blood eosinophils less than 300, switch to dual therapy (LABA/LAMA)
        - Blood eosinophils greater than or equal to 300, switch to triple therapy (LABA/LAMA/ICS)
    - Initial dual therapy:
        - Blood eosinophils less than 100, add roflumilast
        - Blood eosinophils greater than or equal to 100, switch to triple therapy (LABA/LAMA/ICS)
    - Exacerbations on triple therapy:
        - Add roflumilast (a phosphodiesterase-4 inhibitor)

	• Add a macrolide (i.e., azithromycin) • Dupilumab (an anti-interleukin[IL]-4 receptor alpha biologic) has been shown to reduce exacerbations and improve FEV1, symptoms and quality of life in patients with a baseline blood eosinophil count greater than or equal to 300 cells/microliter
Chronic Rhinosinusitis with Nasal Polyps (CRSwNP)	Chronic rhinosinusitis with nasal polyps (CRSwNP) is an inflammatory condition affecting the paranasal sinuses.(16) The exact cause of CRSwNP is unknown, but biopsies of nasal polyps have shown elevated levels of eosinophils. Sinus computed tomography (CT) and/or nasal endoscopy are needed to determine the presence of sinonasal inflammation and nasal polyps.(15) The International Consensus Statement on Allergy and Rhinology: Rhinosinusitis (ICAR-RS) indicates that the diagnostic criteria for CRSwNP consist of ALL the following:(24) • Symptoms greater than or equal to 12 weeks • Two of the following symptoms: o Nasal discharge (rhinorrhea or post-nasal drainage) o Nasal obstruction or congestion o Hyposmia (loss or decreased sense of smell) o Facial pressure or pain • One or more of the following findings: o Evidence of inflammation on nasal endoscopy or computed tomography o Evidence of purulence coming from paranasal sinuses or ostiomeatal complex • Presence of nasal polyps Topical saline irrigation and intranasal corticosteroids (INCS) are recommended in the guidelines as initial treatment for CRSwNP.(16,18,24) Nasal saline irrigation used as adjunct treatment with other therapies improves symptoms and quality of life (QoL) outcomes and is considered an important aspect of management of CRSwNP. Saline irrigation can improve nasal mucosa function through the mechanical clearance of thick mucus and inflammatory mediators, including eosinophilic mucin.(18,24) INCS can have a positive impact on the disease and improve symptoms, reduce nasal polyp size, and improve sense of smell.(18,24) The ICAR-RS strongly recommends INCS before or after sinus surgery.(24) INCS are well tolerated and long term treatment is effective and safe. Many different INCS have been used in the treatment of CRSwNP, including triamcinolone, mometasone, fluticasone, and budesonide, but no differences were shown to recommend a specific formulation.(18) For patients using INCS for at least 4 weeks and who continue to have high disease burden, biologics may be considered and preferred over other medical treatment choices.(16) Oral systemic corticosteroids (OCS), used as a short course, can result in a significant reduction in symptoms and nasal polyps for up to three months after the start of treatment. Up to 2 courses per year, taken in addition to INCS, can be useful for patients with partially or uncontrolled disease.(18) The ICAR-RS strongly recommends the use of OCS in the short term management of CRSwNP, but does not recommend longer term use due to the increased risk of adverse effects.(24) Endoscopic sinus surgery (ESS) is aimed at improving symptoms and creating better conditions for local treatment. Sinus surgery should be considered when disease is refractory and remains symptomatic despite trial of primary medical therapy (e.g., nasal sinus irrigation, INCS, oral corticosteroids). Based on current evidence, delaying surgical intervention can be detrimental to symptom improvement and outcomes.(18,24) After surgery, patients need to continue other treatments due to the

	chronic nature of the disease and nasal polyps potentially reoccurring despite surgery.(15,18) INCS can help to prevent nasal polyp recurrence.(18,24) Biologics can be considered in patients where their disease remains uncontrolled despite appropriate medical treatment and sinus surgery.(16,27) Biologics vary in their magnitude of benefits and harms and certainty of evidence across outcomes. Dupilumab and omalizumab are the most beneficial for most patient important outcomes when comparing with other biologics, followed by mepolizumab.(16)
Eosinophilic Esophagitis (EoE)	Eosinophilic esophagitis (EoE) is an allergen/immune-mediated disease characterized by symptoms of esophageal dysfunction and marked eosinophilic inflammation of the esophageal mucosa in the absence of secondary causes. EoE has dramatically increased in prevalence over the years. EoE is characterized by symptoms related to esophageal dysfunction and histologically with eosinophil-predominant inflammation (a peak count of greater than or equal to 15 eosinophils per high-power field on esophageal biopsy). Atopic and allergic inflammatory conditions commonly occur concomitantly with EoE.(19) The symptoms of EoE are age dependent. Young children may refuse to eat, have decreased appetite, recurring abdominal pain, trouble swallowing, and vomiting. Young adults and adults have the same symptoms, but often struggle to swallow dry or dense, solid foods due to inflammation. Food impaction is a common cause for emergency room visits in patients with EoE. Patients may also have concurrent gastroesophageal reflux disease (GERD). EoE is a progressive disease if left untreated. The chronic inflammation can lead to tissue fibrosis and strictures in the esophagus that require esophageal dilation.(20) The diagnosis of EoE is suspected on the basis of chronic symptoms such as dysphagia, food impaction, food refusal, failure to progress with food introduction, heartburn, regurgitation, vomiting, chest pain, odynophagia, abdominal pain, and malnutrition. Due to the wide range of chronic symptoms, the diagnosis should be highly considered in the presence of concomitant atopic conditions and if there are endoscopic findings. Endoscopic findings associated with EoE include esophageal rings, longitudinal furrows, exudates, edema, strictures, or narrow caliber esophagus. Assessment of non-EoE disorders and esophageal biopsy are required to confirm the diagnosis of EoE, with at least 15 eosinophils (eos)/ high-power field (hpf) present on esophageal biopsy.(21) Nonpharmacological treatment of EoE includes dilation and diet. Dilation is only conditionally recommended for patients with dysphagia associated with strictures due to EoE, noting that the dilation does not address the underlying inflammation.(22) Both elemental and elimination diets have been shown to be effective, however, barriers of adherence and cost make this treatment modality feasible only for select patients.(3,22) Proton pump inhibitors (PPIs) are a first line treatment option for patients with EoE, and PPI monotherapy is widely used in practice. PPIs have a longstanding safety profile and have shown to be effective based on symptom response and histological remission. The 2020 American Gastroenterological Association (AGA) and the Joint Task Force on Allergy-Immunology Practice Parameters (JTF) guidelines conditionally recommend their use while the 2022 British Society of Gastroenterology (BSG) and British Society of Pediatric Gastroenterology, Hepatology and Nutrition (BSPGHAN) guidelines strongly recommend their use.(3,22) The AGA/JTF and BSG/BSPGHAN both strongly recommend the use of topical glucocorticoids for the treatment of EoE. Studies showed that topical (swallowed) budesonide or topical fluticasone induced histological remission significantly better than placebo and had similar adverse events to placebo. Due to the chronic nature of the disease and the risk of progression, topical corticosteroids may be continued as maintenance therapy after remission with short term use. A clinical review of the patient should guide this decision based on preference to avoid long term adverse

	effects of topical steroids, or to prevent undesirable outcomes of the disease itself.(3,22)
Prurigo Nodularis (PN)	Prurigo nodularis (PN) is a skin disorder that is defined by the presence of chronic pruritus and multiple elevated, firm, and nodular lesions. PN is more common in older adults but can occur in children. The underlying cause of PN is unknown, but it appears neural and immunologic processes both play a role in its development. The nodules form in a subset of patients that have chronic pruritus, with the nodules forming in areas with continuous scratching over prolonged periods of time. There is significant disease burden associated with PN including sleep disruption, anxiety, and depression. The nodules are typically firm, dome-shaped, and itchy and range in size from millimeters to several centimeters. The nodules can range in color from flesh tones to brown/black and can range in number from a few to hundreds. The pruritis associated with PN can range from sporadic to continuous, and generally the underlying cause is unknown. There are a number of conditions, both dermatologic and other diseases, that are associated with PN, such as atopic dermatitis, kidney disease, diabetes, and HIV.(23) The diagnosis of PN is generally one of exclusion. The American Academy of Dermatology (AAD) indicates that the diagnostic workup should include a clinical examination with a complete review of systems and assessment of PN severity, which should include both disease burden (e.g., quality of life, sleep disturbances) and pruritis intensity. The AAD notes three core features associated with PN:(23) • Presence of firm, nodular lesions • Pruritus that lasts for at least 6 weeks • History and/or signs of repeated scratching, picking, or rubbing Management requires a multifaceted approach with a focus on reducing pruritis, interrupting the itch-scratch cycle, and healing lesions.(23) General measures that should be used at baseline include gentle skin care, moisturizers, and antipruritic emollients.(17,23) Treatment may need to address both the neural and immunologic components of pruritis based on patient signs and symptoms, and often involves the use of topical and systemic therapies.(30) Most therapies for PN have not been adequately studied, and their evidence for use is based on small clinical trials, observational studies, and case reports.(23) Topical therapies are the initial treatment for limited disease. Topical corticosteroids (TCS) target the immunologic component of PN.(23) The International Forum for the Study of Itch (IFSI) 2020 guideline on chronic prurigo including prurigo nodularis strongly recommends moderate to very potent topical corticosteroids on lesional skin.(17) Intralesional corticosteroids may be directly injected into thicker lesions where required, but use should be limited to patients with less than 10 lesions. Topical calcineurin inhibitors and topical calcipotriol have also been used in patients who failed TCS therapy and a prolonged course of a topical immunomodulator is desired. Topical capsaicin, which targets the neural component of PN, has limited clinical evidence and tends to have short term efficacy.(23) Systemic therapies are used for widespread disease or disease refractory to topical therapy.(23,30) Phototherapy is reasonably tolerated and addresses both the immunologic and neural components of PN.(23) However, phototherapy combined with topical therapy will not be adequate for most patients, and the majority will require supplemental systemic therapy.(30) Oral immunosuppressants, such as methotrexate and cyclosporine, have shown to reduce pruritis and heal lesions per limited data available. Methotrexate is generally preferred due to its more favorable side effect profile in comparison to cyclosporine, and cyclosporine should only be considered in more severe cases.(23,30) Other systemic therapies that have shown to be less efficacious and treat the neural component of PN include thalidomide, gabapentin, pregabalin, antidepressants, aprepitant, and naltrexone.(23) Since PN is a nonhistaminergic condition, antihistamines are unlikely to be effective and are not recommended.(23,30)

	Biologic agents are the first therapies to gain approval from the US Food and Drug Administration (FDA) for the treatment of PN. These immunomodulating drugs are believed to target molecules expressed by specific cell types that release a variety of itching mediators that directly or indirectly stimulate receptors on nerve endings in the skin. Biologic agents disrupt this cycle and have been proven to alleviate both pruritus and PN lesions.(30)
Efficacy	**Asthma(1)** The asthma development program included three randomized, double-blind, placebo controlled, parallel-group, multi-center trials (AS Trials 1, 2, and 3) of 24 to 52 weeks in treatment duration which enrolled a total of 2888 subjects (12 years of age and older). Subjects enrolled in AS Trials 1 and 2 were required to have a history of 1 or more asthma exacerbations that required treatment with systemic corticosteroids or emergency department visit or hospitalization for the treatment of asthma in the year prior to trial entry. Subjects enrolled in AS Trial 3 required dependence on daily oral corticosteroids in addition to regular use of high-dose inhaled corticosteroids plus an additional controller(s). In all 3 trials, subjects were enrolled without requiring a minimum baseline blood eosinophil count. In AS Trials 2 and 3 subjects with screening blood eosinophil level of greater than 1500 cells/mcL (less than 1.3%) were excluded. Dupixent was administered as add-on to background asthma treatment. Subjects continued background asthma therapy throughout the duration of the studies, except in AS Trial 3 in which OCS dose was tapered as described below. AS Trial 1 was a 24-week dose-ranging study which included 776 subjects (18 years of age and older). Dupixent compared with placebo was evaluated in adult subjects with moderate to severe asthma on a medium or high-dose inhaled corticosteroid and a long acting beta agonist. Subjects were randomized to receive either 200 mg (N equal to 150) or 300 mg (N equal to 157) Dupixent every other week (Q2W) or 200 mg (N equal to 154) or 300 mg (N equal to 157) Dupixent every 4 weeks following an initial dose of 400 mg, 600 mg or placebo (N equal to 158), respectively. The primary endpoint was mean change from baseline to Week 12 in FEV1 (L) in subjects with baseline blood eosinophils greater than or equal to 300 cells/mcL. Other endpoints included percent change from baseline in FEV1 and annualized rate of severe asthma exacerbation events during the 24-week placebo controlled treatment period. Results were evaluated in the overall population and subgroups based on baseline blood eosinophil count (greater than or equal to 300 cells/mcL and less than 300 cells/mcL. Additional secondary endpoints included responder rates in the patient reported Asthma Control Questionnaire (ACQ-5) and Asthma Quality of Life Questionnaire, Standardized Version (AQLQ(S)) scores. AS Trial 2 was a 52-week study which included 1902 subjects (12 years of age and older). Dupixent compared with placebo was evaluated in 107 adolescents and 1795 adult subjects with moderate-to-severe asthma on a medium or high-dose inhaled corticosteroid (ICS) and a minimum of one and up to two additional controller medications. Subjects were randomized to receive either 200 mg (N=631) or 300 mg (N=633) Dupixent Q2W (or matching placebo for either 200 mg [N equal to 317] or 300 mg [N equal to 321] Q2W) following an initial dose of 400 mg, 600 mg or placebo respectively. The primary endpoints were the annualized rate of severe exacerbation events during the 52-week placebo controlled period and change from baseline in pre-bronchodilator FEV1 at Week 12 in the overall population (unrestricted by minimum baseline blood eosinophils count). Additional secondary endpoints included annualized severe exacerbation rates and FEV1 in patients with different baseline levels of blood eosinophils as well as responder rates in the ACQ-5 and AQLQ(S) scores. AS Trial 3 was a 24-week oral corticosteroid-reduction study in 210 subjects with asthma who required daily oral corticosteroids in addition to regular use of high dose inhaled corticosteroids plus an additional controller. After optimizing the OCS dose during the screening period, subjects received 300 mg Dupixent (N=103) or placebo (N=107) once Q2W for 24 weeks following an initial dose of 600 mg or placebo. Subjects continued to receive their existing asthma medicine during the study; however, their OCS dose was reduced every 4 weeks during the OCS reduction phase (Week 4-20), as long as asthma control was maintained. The primary endpoint was

	the percent reduction of oral corticosteroid dose at Weeks 20 to 24 compared with the baseline dose, while maintaining asthma control in the overall population (unrestricted by minimum baseline blood eosinophils count). Additional secondary endpoints included the annualized rate of severe exacerbation events during treatment period and responder rate in the ACQ-5 and AQLQ(S) scores. AS Trials 1 and 2 evaluated the frequency of severe asthma exacerbations defined as deterioration of asthma requiring the use of systemic corticosteroids for at least 3 days or hospitalization or emergency room visit due to asthma that required systemic corticosteroids. In the primary analysis population (subjects with baseline blood eosinophil count of greater than or equal to 300 cells/mcL in AS Trial 1 and the overall population in AS Trial 2), subjects receiving either Dupixent 200 mg or 300 mg Q2W had significant reductions in the rate of asthma exacerbations compared to placebo. In the overall population in AS Trial 2, the rate of severe exacerbations was 0.46 and 0.52 for Dupixent 200 mg Q2W and 300 mg Q2W, respectively, compared to matched placebo rates of 0.87 and 0.97. The rate ratio of severe exacerbations compared to placebo was 0.52 (95% CI: 0.41, 0.66) and 0.54 (95% CI: 0.43, 0.68) for Dupixent 200 mg Q2W and 300 mg Q2W, respectively. Prespecified subgroup analyses of AS Trials 1 and 2 demonstrated that there were greater reductions in severe exacerbations in subjects with higher baseline blood eosinophil levels. In AS Trial 2, reductions in exacerbations were significant in the subgroup of subjects with baseline blood eosinophils greater than or equal to 150 cells/mcL. In subjects with baseline blood eosinophil count less than 150 cells/mcL, similar severe exacerbation rates were observed between Dupixent and placebo. Significant increases in pre-bronchodilator FEV1 were observed at Week 12 for AS Trials 1 and 2 in the primary analysis populations (subjects with baseline blood eosinophil count of greater than or equal to 300 cells/mcL in AS Trial 1 and the overall population in AS Trial 2). In the overall population in AS Trial 2, the FEV1 LS mean change from baseline was 0.32 L (21%) and 0.34 L (23%) for Dupixent 200 mg Q2W and 300 mg Q2W, respectively, compared to matched placebo means of 0.18 L (12%) and 0.21 L (14%). The mean treatment difference versus placebo was 0.14 L (95% CI: 0.08, 0.19) and 0.13 L (95% CI: 0.08, 0.18) for Dupixent 200 mg Q2W and 300 mg Q2W, respectively. Subgroup analysis of AS Trials 1 and 2 demonstrated greater improvement in subjects with higher baseline blood eosinophils. **Atopic Dermatitis(1,7,8)** Dupilumab was FDA approved through two randomized, double-blind, placebo-controlled phase 3 trials (SOLO 1 and SOLO 2). All patients in both trials were at least 18 years old, had chronic AD (according to American Academy of Dermatology Consensus Criteria Eichenfield 2014) that had been present for at least 3 years, and had greater than or equal to 10% body surface area (BSA) involvement at the screening and baseline visits. Additionally, all patients had a documented recent history (within 6 months before the screening visit) of inadequate response to treatment with topical medications (defined as failure to achieve and maintain remission or a low disease activity state despite treatment with a daily regimen of topical corticosteroids of medium to higher potency applied for greater than or equal to 28 days or for the maximum duration recommended by the product prescribing information [e.g., 14 days for super-potent topical corticosteroids], whichever is shorter), or whom topical treatments are otherwise medically inadvisable. The primary outcome measure in both trials was proportion of patients with both IGA (Investigator Global Assessment) 0 to 1 (on a 5-point scale) and a reduction from baseline of greater than or equal to 2 points at week 16. There were several secondary endpoints included. Some examples include: proportion of patients with Eczema Area and Severity Index (EASI) -75 (greater than or equal to 75% improvement from baseline) at week 16, percent change from baseline to week 16 in pruritus numerical rating scale (NRS), change from baseline to week 16 in % BSA, and changes in quality of life, anxiety, and depression.

	The manufacturer reports the following results from SOLO 1 and SOLO 2. In SOLO 1, the primary outcome (an IGA of 0-1 and a reduction of greater than or equal to 2 points from baseline at week 16) occurred in 85 patients (38%) who received dupilumab every other week and in 83 (37%) who received dupilumab weekly, as compared with 23 (10%) who received placebo (P less than 0.001 for both comparisons with placebo). The results were similar in SOLO 2, with the primary outcome occurring in 84 patients (36%) who received dupilumab every other week and in 87 (36%) who received dupilumab weekly, as compared with 20 (8%) who received placebo (P less than 0.001 for both comparisons). In addition, in the two trials, an improvement from baseline to week 16 of at least 75% on the Eczema Area and Severity Index was reported in significantly more patients who received each regimen of dupilumab than in patients who received placebo (P less than 0.001 for all comparisons). Dupilumab was also associated with improvement in other clinical end points, including reduction in pruritus and symptoms of anxiety or depression and improvement in quality of life. The efficacy and safety of Dupixent monotherapy in adolescent subjects was evaluated in a multicenter, randomized, double-blind, placebo-controlled trial in 251 adolescent subjects 12 to 17 years of age, with moderate-to-severe AD and a minimum BSA involvement of greater than or equal to 10%. Eligible subjects enrolled into this trial had previous inadequate response to topical medication. Subjects in the Dupixent group with baseline weight of less than 60 kg received an initial dose of 400 mg at Week 0, followed by 200 mg every 2 weeks for 16 weeks. Subjects with baseline weight of greater than or equal to 60 kg received an initial dose of 600 mg at Week 0, followed by 300 mg every 2 weeks for 16 weeks. Subjects were permitted to receive rescue treatment at the discretion of the investigator. Subjects who received rescue treatment were considered non-responders. The primary endpoint was the proportion of subjects with an IGA 0 (clear) or 1 (almost clear) and at least a 2-point improvement from baseline to Week 16. Other evaluated outcomes included the proportion of subjects with EASI-75 or EASI-90 (improvement of at least 75% or 90% in EASI from baseline, respectively), and reduction in itch as measured by the Peak Pruritus NRS (greater than or equal to 4-point improvement). The efficacy results at Week 16 were as follows: • IGA 0 or 1: 24% for Dupixent and 2% for placebo • EASI-75: 42% for Dupixent and 8% for placebo • EASI-90: 23% for Dupixent and 2% for placebo • Peak Pruritus NRS (greater than or equal to 4-point improvement): 37% for Dupixent and 5% for placebo **COPD(1,28,29)** The efficacy of Dupixent as add-on maintenance treatment of adult patients with inadequately controlled COPD and an eosinophilic phenotype was evaluated in two randomized, double-blind, multicenter, parallel-group, placebo-controlled trials (BOREAS [NCT03930732] and NOTUS [NCT04456673]) of 52 weeks duration. The two trials enrolled a total of 1874 adult subjects with COPD. Both trials enrolled subjects with a diagnosis of COPD with moderate to severe airflow limitation (post-bronchodilator FEV1/FVC ratio less than 0.7 and post-bronchodilator FEV1 of 30% to 70% predicted) and a minimum blood eosinophil count of 300 cells/mcL at screening. Patients had to have been receiving maintenance triple therapy consisting of a long-acting muscarinic antagonist (LAMA), long-acting beta agonist (LABA), and inhaled corticosteroid (ICS) for at least 3 months before randomization; LAMA/LABA dual therapy was allowed if ICS use was contraindicated. Trial enrollment required an exacerbation history of at least 2 moderate or 1 severe exacerbation(s) in the previous year despite receiving maintenance therapy, and symptoms of chronic productive cough for at least 3 months in the past year. Exacerbations of COPD were defined as clinically significant worsening of COPD symptoms including increases in

	dyspnea, wheezing, cough, sputum volume, and/or increase in sputum purulence. Exacerbation severity was further defined as moderate if treatment with systemic corticosteroids and/or antibiotics was required, or severe if they resulted in hospitalization or observation for over 24 hours in an emergency department or urgent care facility. One of the two required moderate exacerbations had to require the use of systemic corticosteroids. Greater than or equal to 95% of subjects in each trial had chronic bronchitis. Subjects also had a Medical Research Council (MRC) dyspnea score greater than or equal to 2 (range 0-4). In both trials, subjects were randomized to receive Dupixent 300 mg subcutaneously every two weeks (Q2W) or placebo in addition to their background maintenance therapy for 52 weeks. The primary endpoint for BOREAS and NOTUS trials was the annualized rate of moderate or severe COPD exacerbations during the 52-week treatment period. In both trials, Dupixent demonstrated a significant reduction in the annualized rate of moderate or severe COPD exacerbations compared to placebo when added to background maintenance therapy. In the BOREAS trial, the annualized rate of moderate or severe exacerbations of COPD was 0.78 (95% confidence interval [CI], 0.64 to 0.93) in the dupilumab group and 1.10 (95% CI, 0.93 to 1.30) in the placebo group (rate ratio, 0.70; 95% CI, 0.58 to 0.86; P value less than 0.001). In the NOTUS trial, the annualized rate of moderate or severe exacerbations of COPD was lower in the dupilumab group (0.86; 95% CI, 0.70 to 1.06) than in the placebo group (1.30; 95% CI, 1.05 to 1.60), resulting in a rate ratio of 0.66 (95% CI, 0.54 to 0.82; P value less than 0.001). Treatment with Dupixent decreased the risk of a moderate to severe COPD exacerbation as measured by time to first exacerbation when compared with placebo in BOREAS (HR: 0.80; 95% CI: 0.66, 0.98) and NOTUS (HR: 0.71; 95% CI: 0.57, 0.89). In both trials (BOREAS and NOTUS), Dupixent demonstrated numerical improvement in post-bronchodilator FEV1 at Weeks 12 and 52 compared to placebo when added to background maintenance therapy. Significant improvements of similar magnitude were observed in change from baseline in pre-bronchodilator FEV1 at Weeks 12 and 52 in subjects treated with Dupixent compared to placebo across both trials. In both trials (BOREAS and NOTUS), the St. George's Respiratory Questionnaire (SGRQ) total score responder rate (defined as the proportion of subjects with SGRQ improvement from baseline of at least 4 points) at Week 52 was evaluated. SGRQ is a 50-item questionnaire designed to measure and quantify health status in adult patients with chronic airflow limitation and scores range from 0 to 100, with lower scores indicating a better quality of life. In BOREAS, the responder rate was 51% for subjects treated with Dupixent versus 43% for placebo (N=939, odds ratio: 1.44; 95% CI: 1.10, 1.89). In NOTUS, the responder rate was 51% for subjects treated with Dupixent versus 47% for placebo (N=721, odds ratio: 1.16; 95% CI: 0.86, 1.58). **CRSwNP(1)** Two randomized, double-blind, parallel-group, multicenter, placebo-controlled studies (CSNP Trial 1 and CSNP Trial 2) evaluated Dupixent in CRSwNP. There were 724 subjects aged 18 years and older on background intranasal corticosteroids (INCS) included in the trials. These studies included subjects with CRSwNP despite prior sinonasal surgery or treatment with, or who were ineligible to receive or were intolerant to, systemic corticosteroids in the past 2 years. Patients with chronic rhinosinusitis without nasal polyposis were not included in these trials. Rescue with systemic corticosteroids or surgery was allowed during the studies at the investigator's discretion. In CSNP Trial 1, a total of 276 subjects were randomized to receive either 300 mg Dupixent (N=143) or placebo (N=133) every other week for 24 weeks. In CSNP Trial 2, 448 subjects were randomized to receive either 300 mg Dupixent (N=150) every other week for 52 weeks, 300 mg Dupixent (N=145) every other week until week 24 followed by 300 mg Dupixent every 4 weeks until week 52, or placebo (N=153). All subjects had evidence of sinus opacification on the Lund Mackay (LMK) sinus CT scan and 73% to 90% of subjects had opacification of all sinuses. Subjects were stratified based on their histories of prior surgery and co-morbid

	asthma/nonsteroidal anti-inflammatory drug exacerbated respiratory disease (NSAID-ERD). A total of 63% of subjects reported previous sinus surgery, with a mean number of 2.0 prior surgeries, 74% used systemic corticosteroids in the previous 2 years with a mean number of 1.6 systemic corticosteroid courses in the previous 2 years, 59% had co-morbid asthma, and 28% had NSAID-ERD. The co-primary efficacy endpoints were change from baseline to Week 24 in bilateral endoscopic nasal polyps score (NPS; 0-8 scale) as graded by central blinded readers and change from baseline to Week 24 in nasal congestion/obstruction score averaged over 28 days (NC; 0-3 scale), as determined by subjects using a daily diary. In both studies, key secondary end-points at Week 24 included change from baseline in: LMK sinus CT scan score, daily loss of smell, and 22-item sinonasal outcome test (SNOT-22). In the pooled efficacy results, the reduction in the proportion of subjects rescued with systemic corticosteroids and/or sinonasal surgery (up to Week 52) were evaluated. Statistically significant efficacy was observed in CSNP Trial 2 with regard to improvement in bilateral endoscopic NPS score at week 24 and week 52. Similar results were seen in CSNP Trial 1 at Week 24. In the post-treatment period when subjects were off Dupixent, the treatment effect diminished over time. In both studies, significant improvements in nasal congestion were observed as early as the first assessment at Week 4. A significant decrease in the LMK sinus CT scan score was observed. Dupilumab significantly improved the loss of smell compared to placebo. In both studies, significant improvements in daily loss of smell severity were observed as early as the first assessment at Week 4. Dupilumab significantly decreased sinonasal symptoms as measured by SNOT-22 compared to placebo. In the pre-specified multiplicity-adjusted pooled analysis of two studies, treatment with Dupixent resulted in significant reduction of systemic corticosteroid use and need for sinonasal surgery versus placebo (HR of 0.24; 95% CI: 0.17, 0.35). The proportion of subjects who required systemic corticosteroids was reduced by 74% (HR of 0.26; 95% CI: 0.18, 0.38). The total number of systemic corticosteroid courses per year was reduced by 75% (RR of 0.25; 95% CI: 0.17, 0.37). The proportion of subjects who required surgery was reduced by 83% (HR of 0.17; 95% CI: 0.07, 0.46). The effects of Dupixent on the primary endpoints of NPS and nasal congestion and the key secondary endpoint of LMK sinus CT scan score were consistent in patients with prior surgery and without prior surgery. **EoE(1)** A single randomized, double-blind, parallel-group, multicenter, placebo-controlled trial, including two 24-week treatment periods (Parts A and B), was conducted in adult and pediatric subjects 12 to 17 years of age, weighing at least 40 kg, with EoE (NCT03633617). In both parts, subjects were randomized to receive 300 mg Dupixent every week or placebo. Eligible subjects had greater than or equal to 15 intraepithelial eosinophils per high-power field (eos/hpf) following a treatment course of a proton pump inhibitor (PPI) either prior to or during the screening period and symptoms of dysphagia as measured by the Dysphagia Symptom Questionnaire (DSQ). At baseline, 43% of subjects in Part A and 37% of subjects in Part B had a history of prior esophageal dilations. The coprimary efficacy endpoints in Parts A and B were the (1) proportion of subjects achieving histological remission defined as peak esophageal intraepithelial eosinophil count of less than or equal to 6 eos/hpf at week 24; and (2) the absolute change in the subject reported DSQ score from baseline to week 24. In Parts A and B, a greater proportion of subjects randomized to Dupixent achieved histological remission (peak esophageal intraepithelial eosinophil count less than or equal to 6 eos/hpf) compared to placebo (Part A: 25% vs 2%; Part B: 47% vs 5%). Treatment with Dupixent also resulted in a significant improvement in LS mean change

	in DSQ score compared to placebo at week 24 (Part A: -21.9 vs -9.6; Part B -23.8 vs -13.9). The results of the anchor-based analyses that incorporated the subjects' perspectives indicated that the observed improvement in dysphagia from Parts A and B is representative of a clinically meaningful within-subject improvement. **PN(1,14)** The prurigo nodularis (PN) development program included two 24-week randomized, double-blind, placebo-controlled, multicenter, parallel-group trials (PRIME [NCT04183335] and PRIME 2 [NCT04202679]) in 311 adult subjects 18 years of age and older with pruritus (WINRS greater than or equal to 7 on a scale of 0 to 10) and greater than or equal to 20 nodular lesions. PRIME and PRIME 2 assessed the effect of Dupixent on pruritus improvement as well as its effect on PN lesions. In these two trials, subjects received either subcutaneous Dupixent 600 mg (two 300 mg injections) on day 1, followed by 300 mg once every other week (Q2W) for 24 weeks, or matching placebo. At baseline, the mean Worst Itch-Numeric Rating Scale (WI-NRS) was 8.5, 66% had 20 to 100 nodules (moderate), and 34% had greater than 100 nodules (severe). Patients were required to have failed at least a 2-week trial of a medium to super potent topical corticosteroid or topical corticosteroids were not medically advised. The WI-NRS is comprised of a single item, rated on a scale from 0 (no itch) to 10 (worst imaginable itch). Subjects were asked to rate the intensity of their worst pruritus (itch) over the past 24 hours using this scale. The Investigator's Global Assessment for Prurigo Nodularis-Stage (IGA PN-S) is a scale that measures the approximate number of nodules using a 5-point scale from 0 (clear) to 4 (severe). Efficacy was assessed with the proportion of subjects with improvement (reduction) in WI-NRS by greater than or equal to 4 points, the proportion of subjects with IGA PN-S 0 or 1 (the equivalent of 0-5 nodules), and the proportion of subjects who achieved a response in both WI-NRS and IGA PN-S per the criteria described above. Overall, patients treated with Dupixent saw improvement in all endpoints over placebo.
Safety	Dupixent (dupilumab) is contraindicated in patients who have a known hypersensitivity to dupilumab or any excipients of the product.(1)

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30	Yook HJ, Lee JH. Prurigo nodularis: Pathogenesis and the horizon of potential therapeutics. *International Journal of Molecular Sciences*. 2024;25(10):1-26. doi:10.3390/ijms25105164
31	Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease (2024 Report). Available from: https://goldcopd.org/2024-gold-report/
32	McDonough JE, Yuan R, Suzuki M, et al. Small-Airway obstruction and emphysema in chronic obstructive pulmonary disease. *New England Journal of Medicine*. 2011;365(17):1567-1575. doi:10.1056/nejmoa1106955

POLICY AGENT SUMMARY PRIOR AUTHORIZATION

Target Brand Agent(s)	Target Generic Agent(s)	Strength	Targeted MSC	Available MSC	Final Age Limit	Preferred Status
Dupixent	dupilumab subcutaneous soln auto-injector ; dupilumab subcutaneous soln prefilled syringe	100 MG/0.67ML ; 200 MG/1.14ML ; 300 MG/2ML	M ; N ; O ; Y	N		

POLICY AGENT SUMMARY QUANTITY LIMIT

Target Brand Agent Name(s)	Target Generic Agent Name(s)	Strength	QL Amount	Dose Form	Day Supply	Duration	Addtl QL Info	Allowed Exceptions	Targeted NDCs When Exclusions Exist
Dupixent	Dupilumab Subcutaneous Soln Pen-injector 200 MG/1.14ML	200 MG/1.14 ML	2	Pens	28	DAYS			
Dupixent	Dupilumab Subcutaneous Soln Pen-injector 300 MG/2ML	300 MG/2ML	4	Pens	28	DAYS			
Dupixent	Dupilumab Subcutaneous Soln Prefilled Syringe	100 MG/0.67 ML	2	Syringes	28	DAYS			
Dupixent	Dupilumab Subcutaneous Soln Prefilled Syringe 200 MG/1.14ML	200 MG/1.14 ML	2	Syringes	28	DAYS			
Dupixent	Dupilumab Subcutaneous Soln Prefilled Syringe 300 MG/2ML	300 MG/2ML	4	Syringes	28	DAYS			

CLIENT SUMMARY – PRIOR AUTHORIZATION

Target Brand Agent Name(s)	Target Generic Agent Name(s)	Strength	Client Formulary
Dupixent	dupilumab subcutaneous soln auto-injector ; dupilumab subcutaneous soln prefilled syringe	100 MG/0.67ML ; 200 MG/1.14ML ; 300 MG/2ML	Commercial ; HIM ; ResultsRx

CLIENT SUMMARY – QUANTITY LIMITS

Target Brand Agent Name(s)	Target Generic Agent Name(s)	Strength	Client Formulary
Dupixent	Dupilumab Subcutaneous Soln Pen-injector 200 MG/1.14ML	200 MG/1.14ML	Commercial ; HIM ; ResultsRx
Dupixent	Dupilumab Subcutaneous Soln Pen-injector 300 MG/2ML	300 MG/2ML	Commercial ; HIM ; ResultsRx
Dupixent	Dupilumab Subcutaneous Soln Prefilled Syringe	100 MG/0.67ML	Commercial ; HIM ; ResultsRx
Dupixent	Dupilumab Subcutaneous Soln Prefilled Syringe 200 MG/1.14ML	200 MG/1.14ML	Commercial ; HIM ; ResultsRx
Dupixent	Dupilumab Subcutaneous Soln Prefilled Syringe 300 MG/2ML	300 MG/2ML	Commercial ; HIM ; ResultsRx

PRIOR AUTHORIZATION CLINICAL CRITERIA FOR APPROVAL

Module	Clinical Criteria for Approval
PA	**Initial Evaluation** **Target Agent(s)** will be approved when ALL of the following are met: 1. ONE of the following: A. The requested agent is eligible for continuation of therapy AND ONE of the following: **Agents Eligible for Continuation of Therapy** All target agents are eligible for continuation of therapy 1. The patient has been treated with the requested agent (starting on samples is not approvable) within the past 90 days **OR** 2. The prescriber states the patient has been treated with the requested agent (starting on samples is not approvable) within the past 90 days AND is at risk if therapy is changed **OR** B. BOTH of the following: 1. ONE of the following: A. The patient has a diagnosis of moderate-to-severe atopic dermatitis (AD) AND ALL of the following: 1. ONE of the following: A. The patient has at least 10% body surface area involvement **OR** B. The patient has involvement of body sites that are difficult to treat with prolonged topical corticosteroid therapy (e.g., hands, feet, face, neck, scalp, genitals/groin, skin folds) **OR** C. The patient has an Eczema Area and Severity Index (EASI) score greater than or equal to 16 **OR** D. The patient has an Investigator Global Assessment (IGA) score greater than or equal to 3 **AND** 2. ONE of the following: A. BOTH of the following: 1. ONE of the following:

Module	Clinical Criteria for Approval
	A. The patient has tried and had an inadequate response to at least a medium-potency topical corticosteroid used in the treatment of AD after at least a 4-week duration of therapy **OR** B. The patient has an intolerance or hypersensitivity to at least a medium-potency topical corticosteroid used in the treatment of AD **OR** C. The patient has an FDA labeled contraindication to ALL medium-, high-, and super-potency topical corticosteroids used in the treatment of AD **AND** 2. ONE of the following: A. The patient has tried and had an inadequate response to a topical calcineurin inhibitor (e.g., Elidel/pimecrolimus, Protopic/tacrolimus) used in the treatment of AD after at least a 6-week duration of therapy **OR** B. The patient has an intolerance or hypersensitivity to a topical calcineurin inhibitor used in the treatment of AD **OR** C. The patient has an FDA labeled contraindication to ALL topical calcineurin inhibitors used in the treatment of AD **OR** B. The patient's medication history indicates use of another biologic immunomodulator agent that is FDA labeled or supported in compendia for the treatment of AD **AND** 3. The prescriber has documented the patient's baseline (prior to therapy with the requested agent) pruritus and other symptom severity (e.g., erythema, edema, xerosis, erosions/excoriations, oozing and crusting, and/or lichenification) **OR** B. The patient has a diagnosis of moderate to severe asthma AND BOTH of the following: 1. ONE of the following: A. The patient has eosinophilic type asthma AND ONE of the following: 1. The patient has a baseline (prior to therapy with the requested agent) blood eosinophil count of 150 cells/microliter or higher while on high-dose inhaled corticosteroids or daily oral corticosteroids **OR** 2. The patient has sputum eosinophils 2% or higher while on high-dose inhaled corticosteroids or daily oral corticosteroids **OR** B. The patient has oral corticosteroid dependent type asthma **AND** 2. The patient has a history of uncontrolled asthma while on asthma control therapy as demonstrated by ONE of the following:

Module	Clinical Criteria for Approval
	A. Frequent severe asthma exacerbations requiring two or more courses of systemic corticosteroids (steroid burst) within the past 12 months **OR** B. Serious asthma exacerbations requiring hospitalization, mechanical ventilation, or visit to the emergency room or urgent care within the past 12 months **OR** C. Controlled asthma that worsens when the doses of inhaled and/or systemic corticosteroids are tapered **OR** D. The patient has baseline (prior to therapy with the requested agent) Forced Expiratory Volume (FEV1) that is less than 80% of predicted **OR** C. The patient has a diagnosis of chronic obstructive pulmonary disease (COPD) AND ALL of the following: 1. The patient's diagnosis was confirmed by spirometry with a post-bronchodilator FEV1/FVC ratio less than 0.7 **AND** 2. The patient has a post-bronchodilator FEV1 between 30% to 70% predicted **AND** 3. ONE of the following: A. The patient has a modified Medical Research Council (mMRC) dyspnea score of 2 or greater **OR** B. The patient has a COPD Assessment Test (CAT) score greater than or equal to 10 **AND** 4. The patient has a baseline (prior to therapy with the requested agent) blood eosinophil count of 300 cells/microliter or higher **AND** 5. The patient has a history of inadequately controlled COPD while on COPD inhaled maintenance therapy as demonstrated by ONE of the following: A. Frequent COPD exacerbations requiring one or more courses of systemic corticosteroids within the past 12 months **OR** B. A severe COPD exacerbation requiring hospitalization, mechanical ventilation, or visit to the emergency room or urgent care within the past 12 months **OR** D. The patient has a diagnosis of chronic rhinosinusitis with nasal polyps (CRSwNP) AND ALL of the following: 1. The patient has at least TWO of the following symptoms consistent with chronic rhinosinusitis (CRS): A. Nasal discharge (rhinorrhea or post-nasal drainage) B. Nasal obstruction or congestion C. Loss or decreased sense of smell (hyposmia) D. Facial pressure or pain **AND** 2. The patient has had symptoms consistent with chronic rhinosinusitis (CRS) for at least 12 consecutive weeks **AND** 3. The patient's diagnosis was confirmed by ONE of the following: A. Anterior rhinoscopy or endoscopy **OR** B. Computed tomography (CT) of the sinuses **AND** 4. ONE of the following: A. The patient has tried and had an inadequate response to ONE intranasal corticosteroid therapy (e.g., fluticasone nasal spray, mometasone nasal spray, Sinuva) after at least a 4-week duration of therapy **OR** B. The patient has an intolerance or hypersensitivity to ONE intranasal corticosteroid therapy (e.g.,

Module	Clinical Criteria for Approval
	fluticasone nasal spray, mometasone nasal spray, Sinuva) **OR** C. The patient has an FDA labeled contraindication to ALL intranasal corticosteroids **OR** E. The patient has a diagnosis of eosinophilic esophagitis (EoE) AND BOTH of the following: 1. The patient's diagnosis was confirmed by ALL of the following: A. Chronic symptoms of esophageal dysfunction **AND** B. Greater than or equal to 15 eosinophils per high-power field on esophageal biopsy **AND** C. Other causes that may be responsible for or contributing to symptoms and esophageal eosinophilia have been ruled out **AND** 2. ONE of the following: A. The patient has tried and had an inadequate response to ONE standard corticosteroid therapy used in the treatment of EoE (i.e., budesonide oral suspension, swallowed budesonide nebulizer suspension, swallowed fluticasone MDI) **OR** B. The patient has an intolerance or hypersensitivity to ONE standard corticosteroid therapy used in the treatment of EoE **OR** C. The patient has an FDA labeled contraindication to ALL standard corticosteroid therapies used in the treatment of EoE **OR** D. The patient has tried and had an inadequate response to ONE proton pump inhibitor (PPI) used in the treatment of EoE **OR** E. The patient has an intolerance or hypersensitivity to ONE PPI used in the treatment of EoE **OR** F. The patient has an FDA labeled contraindication to ALL PPI therapies used in the treatment of EoE **OR** F. The patient has a diagnosis of prurigo nodularis (PN) and BOTH of the following: 1. The patient has ALL of the following features associated with PN: A. Presence of greater than or equal to 20 firm, nodular lesions **AND** B. Pruritus that has lasted for at least 6 weeks **AND** C. History and/or signs of repeated scratching, picking, or rubbing **AND** 2. ONE of the following: A. The patient has tried and had an inadequate response to at least a medium-potency topical corticosteroid used in the treatment of PN after at least a 2-week duration of therapy **OR** B. The patient has an intolerance or hypersensitivity to therapy with at least a medium-potency topical corticosteroid used in the treatment of PN **OR** C. The patient has an FDA labeled contraindication to ALL medium-, high-, and super-potency topical corticosteroids used in the treatment of PN **OR** G. The patient has another FDA labeled indication for the requested agent and route of administration **AND** 2. If the patient has an FDA labeled indication, then ONE of the following: A. The patient's age is within FDA labeling for the requested indication for the requested agent **OR** B. There is support for using the requested agent for the patient's age for the requested indication **OR**

Module	Clinical Criteria for Approval
	C. The patient has another indication that is supported in compendia for the requested agent and route of administration **AND** 2. If the patient has a diagnosis of moderate-to-severe atopic dermatitis (AD), then BOTH of the following: A. The patient is currently treated with topical emollients and practicing good skin care **AND** B. The patient will continue the use of topical emollients and good skin care practices in combination with the requested agent **AND** 3. If the patient has a diagnosis of chronic obstructive pulmonary disease (COPD), then ALL of the following: A. ONE of the following: 1. The patient is currently treated with an inhaled corticosteroid for at least 3 months AND has been adherent for 90 days within the past 120 days **OR** 2. The patient has an intolerance or hypersensitivity to therapy with an inhaled corticosteroid **OR** 3. The patient has an FDA labeled contraindication to ALL inhaled corticosteroids **AND** B. ONE of the following: 1. The patient is currently treated with a long-acting muscarinic antagonist (LAMA) AND a long-acting beta-2 agonist (LABA) used in combination for at least 3 months AND has been adherent for 90 days within the past 120 days **OR** 2. The patient has an intolerance or hypersensitivity to therapy with a LAMA AND a LABA used in combination **OR** 3. The patient has an FDA labeled contraindication to ALL long-acting muscarinic antagonists (LAMA) AND long-acting beta-2 agonists (LABA) **AND** C. The patient will continue COPD inhaled maintenance therapy (e.g., ICS/LAMA/LABA triple therapy, LAMA/LABA) in combination with the requested agent **AND** 4. If the patient has a diagnosis of chronic rhinosinusitis with nasal polyps (CRSwNP), then BOTH of the following: A. The patient is currently treated with standard nasal polyp maintenance therapy (e.g., nasal saline irrigation, intranasal corticosteroids [e.g., fluticasone nasal spray, mometasone nasal spray, Sinuva]) **AND** B. The patient will continue standard nasal polyp maintenance therapy (e.g., nasal saline irrigation, intranasal corticosteroids [e.g., fluticasone nasal spray, mometasone nasal spray, Sinuva]) in combination with the requested agent **AND** 5. If the patient has a diagnosis of moderate to severe asthma, then ALL of the following: A. ONE of the following: 1. The patient is NOT currently treated with the requested agent AND is currently treated with a maximally tolerated inhaled corticosteroid for at least 3 months AND has been adherent for 90 days within the past 120 days **OR** 2. The patient is currently treated with the requested agent AND ONE of the following: A. The patient is currently treated with an inhaled corticosteroid for at least 3 months that is adequately dosed to control symptoms AND has been adherent for 90 days within the past 120 days **OR** B. The patient is currently treated with a maximally tolerated inhaled corticosteroid for at least 3 months AND has been adherent for 90 days within the past 120 days **OR** 3. The patient has an intolerance or hypersensitivity to therapy with an inhaled corticosteroid **OR** 4. The patient has an FDA labeled contraindication to ALL inhaled corticosteroids **AND** B. ONE of the following: 1. The patient is currently treated for at least 3 months AND has been adherent for 90 days within the past 120 days with ONE of the following: A. A long-acting beta-2 agonist (LABA) **OR**

Module	Clinical Criteria for Approval
	B. A long-acting muscarinic antagonist (LAMA) **OR** C. A leukotriene receptor antagonist (LTRA) **OR** D. Theophylline **OR** 2. The patient has an intolerance or hypersensitivity to therapy with a long-acting beta-2 agonist (LABA), a long-acting muscarinic antagonist (LAMA), a leukotriene receptor antagonist (LTRA), or theophylline **OR** 3. The patient has an FDA labeled contraindication to ALL long-acting beta-2 agonists (LABA) AND long-acting muscarinic antagonists (LAMA) **AND** C. The patient will continue asthma control therapy (e.g., ICS, ICS/LABA, LTRA, LAMA, theophylline) in combination with the requested agent **AND** 6. The prescriber is a specialist in the area of the patient's diagnosis (e.g., atopic dermatitis or PN-dermatologist, allergist, immunologist; asthma or COPD-allergist, immunologist, pulmonologist; CRSwNP-otolaryngologist, allergist, pulmonologist; EoE-allergist, gastroenterologist) or the prescriber has consulted with a specialist in the area of the patient's diagnosis **AND** 7. ONE of the following (please refer to "Agents NOT to be used Concomitantly" table): A. The patient will NOT be using the requested agent in combination with another immunomodulatory agent (e.g., TNF inhibitors, JAK inhibitors, IL-4 inhibitors) **OR** B. The patient will be using the requested agent in combination with another immunomodulatory agent AND BOTH of the following: 1. The prescribing information for the requested agent does NOT limit the use with another immunomodulatory agent **AND** 2. There is support for the use of combination therapy (submitted copy of clinical trials, phase III studies, or guidelines required) **AND** 8. The patient does NOT have any FDA labeled contraindications to the requested agent **Compendia Allowed:** AHFS, DrugDex 1 or 2a level of evidence, or NCCN 1 or 2a recommended use **Length of Approval:** 6 months for moderate-to-severe AD, moderate-to-severe asthma, CRSwNP, EoE, and PN; 12 months for COPD and all other indications NOTE: Quantity Limit applies, please refer to Quantity Limit Criteria. **Renewal Evaluation** **Target Agent(s)** will be approved when ALL of the following are met: 1. The patient has been previously approved for the requested agent through the plan's Prior Authorization process [Note: patients not previously approved for the requested agent will require initial evaluation review] **AND** 2. ONE of the following: A. The patient has a diagnosis of moderate-to-severe atopic dermatitis (AD) AND BOTH of the following: 1. The patient has had a reduction or stabilization from baseline (prior to therapy with the requested agent) of ONE of the following: A. Affected body surface area **OR** B. Flares **OR** C. Pruritus, erythema, edema, xerosis, erosions/excoriations, oozing and crusting, and/or lichenification **OR** D. A decrease in the Eczema Area and Severity Index (EASI) score **OR** E. A decrease in the Investigator Global Assessment (IGA) score **AND** 2. The patient will continue standard maintenance therapies (e.g., topical emollients, good skin care practices) in combination with the requested agent **OR**

Module	Clinical Criteria for Approval
	B. The patient has a diagnosis of moderate to severe asthma AND BOTH of the following: 1. The patient has had improvements or stabilization with the requested agent from baseline (prior to therapy with the requested agent) as indicated by ONE of the following: A. The patient has had an increase in percent predicted Forced Expiratory Volume (FEV1) **OR** B. The patient has had a decrease in the dose of inhaled corticosteroids required to control the patient's asthma **OR** C. The patient has had a decrease in need for treatment with systemic corticosteroids due to exacerbations of asthma **OR** D. The patient has had a decrease in number of hospitalizations, need for mechanical ventilation, or visits to urgent care or emergency room due to exacerbations of asthma **AND** 2. The patient is currently treated within the past 90 days and is compliant with asthma control therapy (e.g., inhaled corticosteroids [ICS], ICS/long-acting beta-2 agonist [LABA], leukotriene receptor antagonist [LTRA], long-acting muscarinic antagonist [LAMA], theophylline) **OR** C. The patient has a diagnosis of chronic obstructive pulmonary disease (COPD) AND BOTH of the following: 1. The patient has had clinical benefit with the requested agent **AND** 2. The patient is currently treated within the past 90 days and is compliant with COPD inhaled maintenance therapy (e.g., inhaled corticosteroid [ICS]/long-acting muscarinic antagonist [LAMA]/long-acting beta-2 agonist [LABA] triple therapy, LAMA/LABA) **OR** D. The patient has a diagnosis of chronic rhinosinusitis with nasal polyps (CRSwNP) AND BOTH of the following: 1. The patient has had clinical benefit with the requested agent **AND** 2. The patient will continue standard nasal polyp maintenance therapy (e.g., nasal saline irrigation, intranasal corticosteroids [e.g., fluticasone nasal spray, mometasone nasal spray, Sinuva]) in combination with the requested agent **OR** E. The patient has a diagnosis other than moderate-to-severe atopic dermatitis (AD), moderate to severe asthma, COPD, or CRSwNP AND has had clinical benefit with the requested agent **AND** 3. The prescriber is a specialist in the area of the patient's diagnosis (e.g., atopic dermatitis or PN-dermatologist, allergist, immunologist; asthma or COPD-allergist, immunologist, pulmonologist; CRSwNP-otolaryngologist, allergist, pulmonologist; EoE-allergist, gastroenterologist) or the prescriber has consulted with a specialist in the area of the patient's diagnosis **AND** 4. ONE of the following (please refer to "Agents NOT to be used Concomitantly" table): A. The patient will NOT be using the requested agent in combination with another immunomodulatory agent (e.g., TNF inhibitors, JAK inhibitors, IL-4 inhibitors) **OR** B. The patient will be using the requested agent in combination with another immunomodulatory agent AND BOTH of the following: 1. The prescribing information for the requested agent does NOT limit the use with another immunomodulatory agent **AND** 2. There is support for use of combination therapy (submitted copy of clinical trials, phase III studies, or guidelines required) **AND** 5. The patient does NOT have any FDA labeled contraindications to the requested agent **Compendia Allowed:** AHFS, DrugDex 1 or 2a level of evidence, or NCCN 1 or 2a recommended use **Length of Approval:** 12 months NOTE: Quantity Limit applies, please refer to Quantity Limit Criteria.

QUANTITY LIMIT CLINICAL CRITERIA FOR APPROVAL

Module	Clinical Criteria for Approval
Universal QL	**Quantity limit for the Target Agent(s)** will be approved when ONE of the following is met: 1. The requested quantity (dose) does NOT exceed the program quantity limit **OR** 2. The requested quantity (dose) exceeds the program quantity limit AND ONE of the following: A. BOTH of the following: 1. The requested agent does NOT have a maximum FDA labeled dose for the requested indication **AND** 2. There is support for therapy with a higher dose for the requested indication **OR** B. BOTH of the following: 1. The requested quantity (dose) does NOT exceed the maximum FDA labeled dose for the requested indication **AND** 2. There is support for why the requested quantity (dose) cannot be achieved with a lower quantity of a higher strength that does NOT exceed the program quantity limit **Length of Approval:** up to 12 months Note: If approving initial loading dose, please approve initial loading dose for asthma, atopic dermatitis, or prurigo nodularis only. The loading dose plus maintenance dose may be approved for 1 month per FDA labeling, followed by maintenance dosing for the remainder of the length of approval.

CONTRAINDICATION AGENTS

Contraindicated as Concomitant Therapy
Agents NOT to be used Concomitantly Abrilada (adalimumab-afzb) Actemra (tocilizumab) Adalimumab Adbry (tralokinumab-ldrm) Amjevita (adalimumab-atto) Arcalyst (rilonacept) Avsola (infliximab-axxq) Benlysta (belimumab) Bimzelx (bimekizumab-bkzx) Cibinqo (abrocitinib) Cimzia (certolizumab) Cinqair (reslizumab) Cosentyx (secukinumab) Cyltezo (adalimumab-adbm) Dupixent (dupilumab) Ebglyss (lebrikizumab-lbkz) Enbrel (etanercept) Entyvio (vedolizumab) Fasenra (benralizumab) Hadlima (adalimumab-bwwd) Hulio (adalimumab-fkjp) Humira (adalimumab) Hyrimoz (adalimumab-adaz) Idacio (adalimumab-aacf) Ilaris (canakinumab) Ilumya (tildrakizumab-asmn) Imuldosa (ustekinumab-srlf) Inflectra (infliximab-dyyb) Infliximab

Contraindicated as Concomitant Therapy
Kevzara (sarilumab)
Kineret (anakinra)
Leqselvi (deuruxolitinib)
Litfulo (ritlecitinib)
Nemluvio (nemolizumab-ilto)
Nucala (mepolizumab)
Olumiant (baricitinib)
Omvoh (mirikizumab-mrkz)
Opzelura (ruxolitinib)
Orencia (abatacept)
Otezla (apremilast)
Otulfi (ustekinumab-aauz)
Pyzchiva (ustekinumab-ttwe)
Remicade (infliximab)
Renflexis (infliximab-abda)
Riabni (rituximab-arrx)
Rinvoq (upadacitinib)
Rituxan (rituximab)
Rituxan Hycela (rituximab/hyaluronidase human)
Ruxience (rituximab-pvvr)
Saphnelo (anifrolumab-fnia)
Selarsdi (ustekinumab-aekn)
Siliq (brodalumab)
Simlandi (adalimumab-ryvk)
Simponi (golimumab)
Simponi ARIA (golimumab)
Skyrizi (risankizumab-rzaa)
Sotyktu (deucravacitinib)
Spevigo (spesolimab-sbzo) subcutaneous injection
Stelara (ustekinumab)
Steqeyma (ustekinumab-stba)
Taltz (ixekizumab)
Tezspire (tezepelumab-ekko)
Tofidence (tocilizumab-bavi)
Tremfya (guselkumab)
Truxima (rituximab-abbs)
Tyenne (tocilizumab-aazg)
Tysabri (natalizumab)
Ustekinumab
Velsipity (etrasimod)
Wezlana (ustekinumab-auub)
Xeljanz (tofacitinib)
Xeljanz XR (tofacitinib extended release)
Xolair (omalizumab)
Yesintek (ustekinumab-kfce)
Yuflyma (adalimumab-aaty)
Yusimry (adalimumab-aqvh)
Zeposia (ozanimod)
Zymfentra (infliximab-dyyb)