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New Drug Evaluation: Ebglyss™ (lebrikizumab-lbkz) injection, for subcutaneous use

Date of Review: October 2025
Generic Name: lebrikizumab-lbkz

End date of Literature Search: 7/25/2025
Brand Name (Manufacturer): Ebglyss (Eli Lilly and Company)

Plain Language Summary:

- Atopic dermatitis, also known as eczema, is a common condition that causes dry, itchy, and red skin and can affect limbs, head, face, and other areas of body. While mild cases can be managed with topical moisturizers, moderate-to-severe cases often require other topical or systemic therapy.
- Lebrikizumab is a medicine that has been approved by the United States Food and Drug Administration for people aged 12 years and older who weigh at least 40 kg with moderate-to-severe atopic dermatitis whose disease is not adequately controlled with topical medicines. It can be used with or without topical corticosteroids (i.e. hydrocortisone).
- Lebrikizumab is a self-administered injection given every 2 weeks for 16 weeks and then every 4 weeks if symptoms have improved.
- In clinical studies, lebrikizumab improved skin symptoms, reduced itching, and enhanced quality of life better than placebo (or sugar pill).
- Lebrikizumab was relatively well tolerated. In studies, the most frequent side effect was eye inflammation (conjunctivitis).
- Providers must explain to the Oregon Health Authority (OHA) why someone needs lebrikizumab before OHA will pay for it. This process is called prior authorization.

Research Questions:

1. What are the benefits of lebrikizumab in patients with moderate-to-severe atopic dermatitis (AD)?
2. What are the harms of lebrikizumab in patients with moderate-to-severe AD?
3. Are there subpopulations for which lebrikizumab may be better tolerated or more effective?

Conclusions:

- Data from 4 randomized, double-blind clinical trials indicate that 16-week induction of lebrikizumab (administered every 2 weeks) is significantly more effective than placebo for improving skin clearance (Strength of Evidence: high), itching (Strength of Evidence: high), and quality of life (Strength of Evidence: high) for adults and adolescents with moderate-to-severe AD unresponsive to standard topical treatments.
- One trial, which re-randomized responders from two identical induction trials, indicated that continuing lebrikizumab (every 4 weeks) was associated with significantly better skin clearance, itching, and quality of life outcomes (Strength of Evidence: moderate) relative to placebo withdrawal.
- No head-to-head trials comparing lebrikizumab to other targeted immune modulators (TIMs) or other immunosuppressives have been conducted (Strength of Evidence: very low).

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- Lebrikizumab was generally well tolerated; the most commonly reported adverse effect was conjunctivitis (~5%).
- No evidence exists to indicate that treatment efficacy or safety differs by key demographic or disease-related characteristics.
- Safety of lebrikizumab in pregnant women has not been established. In animal studies, lebrikizumab was not associated with adverse fetal development.

Recommendations:

- Add lebrikizumab to the prior authorization criteria for targeted immune modulators (TIMs) for AD and asthma to ensure safe and appropriate use for adults and adolescents with moderate-to-severe AD. See Atopic Dermatitis Class Update for full criteria.

Background

Atopic dermatitis (AD), also known as eczema, is a chronic inflammatory skin disorder that presents as persistent or relapsing episodes of pruritus and eczematous lesions that can vary in morphology and distribution.[1,2] AD is common and affects both children (11%) and adults (6%) with peak incidence occurring in the first year of life.[3] Although most AD develops during childhood, active AD often persists into adolescence and adulthood. In the United States (US), the prevalence of AD is higher in Black children compared to White Children.[4] Although the cause is unknown, AD is believed to be influenced by a mix of genetic, immunologic, and environmental risk factors. In some individuals, AD is associated with increased immunoglobulin E (IgE) allergic reactivity and often presents with other allergic diseases (e.g. asthma, rhinitis).

AD is heterogenous with respect to its presentation and severity. Essential features of AD include eczematous lesions, intense pruritis, and a chronic or relapsing course of disease.[1] Other presenting characteristics (e.g. xerosis), can vary by age, race or ethnicity, and disease severity.[5] In particular, eczematous lesions manifest in distinct ways across age groups, with infants having higher rates of acute lesions that are widely distributed on head, face (especially the cheeks), and limbs, whereas adolescents are typically affected on flexural surfaces; adults typically have involvement limited to hands and feet. There are no definitive laboratory tests for AD, and diagnosis is based on clinical presentation. The American Academy of Dermatology mandates the presence of essential characteristics (pruritic, eczema) and important and supportive features (early age of onset, xerosis) as well as supportive, but non-specific signs for diagnosis of AD.[5]

AD has a substantial psychosocial impact on patients and their relatives. Intense itching is a hallmark of AD and is frequently reported as the most burdensome symptom, affecting sleep, daily activities, and social relationships.[6,7] The visible nature of AD can cause self-consciousness, social embarrassment, and isolation.[8] Patients with AD are at an increased risk of developing mental health disorders, including depression, anxiety, and suicidal ideation.[9] Caring for a child with moderate-to-severe eczema can significantly affect the mental wellbeing of caregivers. The economic burden of AD is also considerable, including direct costs of treatment and indirect costs such as loss of productivity.[10]

The management of AD is guided by disease severity, which is often categorized into mild, moderate, and severe categories, as well as age, co-occurring conditions and treatments.[11,12] Mild AD is characterized by erythema and xerosis with limited itching. Moderate AD can include areas of excoriation and lichenification impacting sleep and activities of daily living. Severe AD presents as widespread skin involvement that includes excoriation, extensive lichenification, bleeding, oozing, cracking, and changes in pigmentation with severe impact on sleep and quality of life. Treatment goals for AD include symptom alleviation and long-term disease control. Treatment is typically individualized based on clinical severity, skin area involved, and other patient factors (other medication or co-occurring disorders).

For all patients, AD management includes avoidance of individual trigger factors, skin barrier restoration using moisturizers, and a step-up and step-down

approach to reduce inflammation according to disease severity. Regardless of disease severity, all patients should regularly apply topical moisturizers, optimally fragrance-free emollients based on patient preference. In addition to moisturizers, most patients with AD will also require topical anti-inflammatory treatment, typically topical corticosteroids (TCS), which are the cornerstone to management of AD. For patients who wish to avoid TCS or have lesions in sensitive areas more adversely impacted by steroid side effects, topical calcineurin inhibitors (TCIs) can be used alone or in conjunction with TCS. Other topical anti-inflammatory therapies that are FDA-approved for management of mild-to-moderate AD include topical phosphodiesterase inhibitors (i.e. crisaborole, roflumilast) and topical Janus kinase inhibitors (JAK) (ruxolitinib). There is inadequate evidence to assess the relative efficacy and safety of topical crisaborole, roflumilast or ruxolitinib compared with TCI and TCS treatments.[13] For those with moderate-to-severe symptoms unresponsive to topical therapies, systemic immunomodulatory medications and/or phototherapy (narrowband ultraviolet B) can be added. Systemic immunomodulatory medications include oral JAK inhibitors (e.g. upadacitinib, abrocitinib), targeted immune modulators (TIMs) (e.g. dupilumab, tralokinumab, nemolizumab), or off-label immunosuppressants (e.g. methotrexate, azathioprine, cyclosporine, mycophenolate mofetil).

Several instruments and scales have been developed to assess severity of illness, disease impact, and quality of life for patients with AD.[14-17] Two of the most used scales include Eczema Area and Severity Index (EASI) and Investigators Global Assessment (IGA) severity score. The EASI assesses the severity of, and body surface area affected by, AD symptoms including erythema, induration/papulation/edema, excoriations, and lichenification.[15] Each symptom is graded systematically for specific anatomical regions (the head, trunk, arms and legs) and summarized in a composite score. EASI scores range from 0 to 72 points, with higher scores indicating greater severity and extent of AD.[15] EASI outcomes are measured as a percentage improvement in EASI score from baseline as EASI 50, 75, or 90.[15] The IGA is a clinician-reported outcome measure that has been used to evaluate severity of AD at a given point in time using a 5-point rating scale ranging from 0 (clear) to 4 (severe) symptoms.[16] In most clinical trials, scores less than or equal to 1 were generally classified as "treatment success," whereas scores greater than 1 were considered "treatment failure."[13]

Lebrikizumab-lbkz (Ebglyss™; Eli Lilly) is a monoclonal antibody that targets interleukin-13 (IL-13), which is a proinflammatory cytokine that is important in the pathogenesis of AD. With its approval in September 2024, lebrikizumab joined tralokinumab (IL-13) and dupilumab (IL-4) as FDA-approved monoclonal antibodies for the treatment of moderate to severe AD. Nemolizumab (Nemluvio™; Galderma) which targets IL-31 was approved for moderate to severe AD in December of 2024 and is reviewed in a separate new drug evaluation.

See **Appendix 1** for Highlights of Prescribing Information from the manufacturer, including Boxed Warnings and Risk Evaluation Mitigation Strategies (if applicable), indications, dosage and administration, formulations, contraindications, warnings and precautions, adverse reactions, drug interactions and use in specific populations.

Table 1: Pharmacology and Pharmacokinetic Properties[18]

Parameter	
Mechanism of Action	IL-13 antagonist that interrupts IL-13 mediated inflammatory signaling, important to the pathogenesis of AD
Distribution and Protein Binding	Volume of distribution = 5.14 L
Elimination	Lebrikizumab is enzymatically degraded into constituent peptides and amino acids similar to endogenous IgG.
Half-Life	24.5 days
Metabolism	No significant hepatic or renal elimination. Lebrikizumab is enzymatically degraded into constituent peptides and amino acids
Abbreviations: AD = atopic dermatitis; IgG = Immunoglobulin G; IL = interleukin; L = liter	

Clinical Efficacy:
Approval by the US FDA was granted on the basis of three phase 3 randomized controlled trials (RCTs) in adults and adolescents at least 12 of age (weighing ≥ 40 kg) with moderate-to-severe AD who remained uncontrolled with topical therapies.[19-21] All trials were placebo-controlled, double-blind, and enrolled subjects with moderate-to-severe AD affecting at least 10% of their body surface area for at least one year. Moderate-to-severe AD was based on an EASI score of at least 16 and an IGA score of at least 3. Patients with prior treatment with immunomodulating agents (systemic corticosteroids, JAK inhibitors) and phototherapy within 4 weeks and dupilumab or tralokinumab either entirely (ADvocate1, ADvocate2) or within 8-16 weeks (ADhere) were excluded.

For all 3 trials, enrolled patients were randomized (2:1) to lebrikizumab 250 mg subcutaneously (SC) every two weeks (Q2W) or placebo for 16 weeks with a 500 mg loading dose administered at baseline and at week 2. The 3 trials were differentiated in two important ways. ADvocate1 and ADvocate2 evaluated lebrikizumab monotherapy; the co-administration of topical (e.g. TCS, TCI) or systemic (e.g. oral steroids) therapies were prohibited. ADhere allowed co-administration of TCS (or TCI for sensitive areas). A second differentiating feature of ADvocate1 and ADvocate2, was that after 16 weeks (induction phase), subjects who responded (IGA score of 0 or 1 and $\geq$ 2 point reduction from baseline [IGA 2+] or a 75% improvement in EASI [EASI-75]), were re-randomized to either lebrikizumab dosed 250 mg Q2W, 250 mg every 4 weeks (Q4W), or placebo for 36 additional weeks (maintenance period).[21] Participants who did not respond received open-label lebrikizumab (250 mg Q2W); patients assigned to this "escape arm" who did not maintain an EASI-50 were terminated from the study.

For all 3 trials, the primary outcome was an IGA score of 0 or 1 with a reduction of 2 or more points from baseline (IGA 2+) which was assessed following the induction phase (week 16). Other secondary outcomes included:

1) a 75% improvement in EASI score (EASI-75),
2) a 90% improvement in EASI score (EASI-90),
3) a 4-point reduction in the Pruritus Numeric Ratings Scale (NRS-4),
4) a 2-points reduction on the Sleep-Loss Scale (SLS), and
5) a 4-point reduction in the Dermatology Life Quality Index (DLQI).

For the maintenance phase (week 52) of ADvocate1 and ADvocate2, outcomes included maintenance of EASI-75, IGA 2+, NRS-4, and percentage change in EASI from baseline. ADvocate1 and ADvocate2 study populations were combined in a pooled analyses of both efficacy and safety.

In addition to the trials used as the basis for US approval, a phase 3 induction trial conducted in Japan was identified (ADhere-J; NCT04760314). Similar to ADhere and ADvocate1& 2, Adhere-J was a 16-week randomized, double-blind trial in adults and adolescent with moderate-to-severe AD. In contrast to the US trial program, Adhere-J evaluated two different induction doses of lebrikizumab: 250 mg SC Q2W and 250 mg SC Q4W relative to placebo. Similar to ADhere, co-administration with TCS was permitted. Co-primary outcomes were IGA 2+ and EASI-75 measured at week 16. The secondary outcomes were similar to the US trial program (ADvocate1&2, ADhere).

Results
Induction Trials[19,20,22]
Across all 3 induction trials evaluated by the FDA[19,20], a total of 717 participants were randomized 2:1 to lebrikizumab 250 mg Q2W (Adhere n=153; ADvocate1 n=283; ADvocate2 n=281) and 362 participants were randomized to placebo (Adhere n=75; ADvocate1 n=141; ADvocate2 n=146). The mean age of participants ranged from 34 to 37 years, and 12% to 22% of participants were under 18 years of age. About 50% of participants were female and 58% to 62% were White,

with the next most prevent race being Asian (12% to 30%) and Black (7% to 14%). The average percentage of body surface area affected ranged from 38% to 48%, and average EASI scores at baseline were 26 to 31 points. About half of participants (46% to 60%) had prior systemic treatment. Patient characteristics across randomized groups at baseline were similar.

Lebrikizumab was statistically significantly superior to placebo for the primary and secondary outcomes (**Table 2**). For the primary outcome of IGA 2+, treatment effects varied from 18.3 to 29.7% difference compared to placebo after 16 weeks (number needed to treat [NNT] 4-6). The treatment effect was smallest within the ADhere trial (difference 18.3%; NNT=6) where co-administration of TCS was permitted. Lebrikizumab induction was also associated with significant improvements in secondary endpoints including EASI-75 (difference 26.4% to 42%; NNT 3-4), pruritis (P-NRS 4+ difference 19.2% to 32.9%; NNT 4-6), and quality of life (DLQI 4+ difference 19.2% to 32.9%; NNT 4-6) compared to placebo. Similar to the primary outcome, the treatment effects were lowest in the ADhere trial for secondary outcomes.

The treatment effect for the primary outcome (IGA 2+; 22.3%; NNT=4) and key secondary outcomes were similar within the Japanese trial ADhere-J. For the primary outcome and most secondary outcomes, symptom improvement was qualitatively similar for both dosing arms of lebrikizumab, although statistical tests were not reported and these data were not evaluated by FDA.

Only one trial (ADhere) reported conducting analyses by key demographic characteristics (subgroup analyses). In this study, the authors report that lebrikizumab efficacy, as measured by EASI-75 and EASI-90, differed significantly by sex with male participants exhibiting "a greater risk difference." However, a detailed summary of this analysis was not provided.

Although all trials were sponsored by, and had significant input from, the manufacturer (Eli Lilly and Company), they generally had low risk of selection, performance, attrition, and detection bias. Except for ADhere-J, which was conducted exclusively in Japan, the 3 trials conducted for US approval were demographically diverse and broadly applicable to a US population. Consistency of treatment effects across trials, precision of estimates, clinical endpoint relevance and strong internal validity of trials indicates lebrikizumab improve skin clearance (IGA 2+, EASI-75), pruritis (P-NRS 4+), and quality of life (DLQI 4+) compared to placebo after 16 weeks (Strength of Evidence: high). The comparative efficacy of lebrikizumab relative to other targeted immune modulators (i.e. dupilumab, tralokinumab) or other immunosuppressive has not been established (Strength of Evidence: very low). Additionally, no studies have been conducted to evaluate the efficacy of lebrikizumab following non-response or intolerance to TIMs or other immunosuppressives (Strength of Evidence: very low).

Table 2: Summary of treatment effect across 16-week lebrikizumab induction trials

Outcomes at 16 weeks	**Trials**				**Strength of Evidence (SoE)**
	ADhere (n=228)	ADvocate1 (n=424)	ADvocate2 (n=427)	ADhere-J (n=286)	
IGA 2+	LEB: 41.2% PLB: 22.1% Difference 18.3% (95% CI 5.1% to 31.5%) NNT= 6	LEB: 43.1% PLB: 12.7% Difference 29.7% (95% CI 21.6% to 37.8%) NNT = 4	LEB: 32.2% PLB: 10.8% Difference 21.9% (95% CI 14.2% to 29.6%) NNT = 5	LEB (Q2W)[‡]: 33.4% LEB (Q4W): 29.1% PLB: 6.1% Difference Q2W[‡]: 27.3% (95% CI 17.%5 to 37.0%) Difference Q4W: 22.6% (95% CI 11.6% to 33.6%)	RoB: low risk Imprecision: sufficiently precise Inconsistency: consistent Indirectness: none Publication bias: likely none **SoE Conclusion: High**

				NNT = 4	NNT = 4	
EASI-75	LEB: 69.5% PLB: 42.2% Difference 26.4% (95% CI 12.1% to 40.8%) NNT = 4	LEB: 58.8% PLB: 16.2% Difference 42% (95% CI 33.3% to 50.6%) NNT = 3	LEB: 52.1% PLB: 18.1% Difference 33.3% (95% CI 24.4% to 42.2%) NNT = 3	LEB (Q2W) ‡: 51.2% LEB (Q4W): 47.2% PLB: 13.4% Difference Q2W‡: 37.6% (95% CI 26.2% to 49.0%) NNT = 3	 Difference Q4W: 33.2% (95% CI 20.6% to 45.8%) NNT = 3	RoB: low risk Imprecision: sufficiently precise Inconsistency: consistent Indirectness: none Publication bias: likely none **SoE Conclusion: High**
P-NRS 4+	LEB: 50.6% PLB: 31.9% Difference 19.2% (95% CI 4.3% to 34.1%) NNT = 6	LEB: 45.9% PLB: 13.0% Difference 32.9% (95% CI 24.6% to 41.3%) NNT = 4	LEB: 39.8% PLB: 11.5% Difference 28.3% (95% CI 20.0% to 36.5%) NNT=4	LEB (Q2W) ‡: 32.7% LEB (Q4W): 23.8% PLB: 3.3% Difference Q2W‡: 29.2% (95% CI 17.9% to 40.4%) NNT = 3	 Difference Q4W: 20.6% (95% CI 8.7% to 32.4%) NNT = 5	RoB: low risk (1) Imprecision: sufficiently precise Inconsistency: consistent Indirectness: none Publication bias: likely none **SoE Conclusion: High**
DLQI 4+	LEB: 77.4% PLB: 58.7% Difference 17.2% (95% CI 0.1% to 34.3%) NNT = 6	LEB: 71.2% PLB: 29.3% Difference 41.9% (95% CI 31.8% to 52.0%) NNT = 3	LEB: 62.3% PLB: 31.3% Difference 31.4% (95% CI 20.7% to 42.1%) NNT=4	LEB (Q2W): 68.8% LEB (Q4W): 53.3% PLB: 20.6% Difference Q2W‡: 48.1% (95% CI 34.5% to 61.7%) NNT = 2	 Difference Q4W: 32.8% (95% CI 16.6% to 48.9%) NNT = 3	RoB: low risk (1) Imprecision: sufficiently precise Inconsistency: consistent Indirectness: none Publication bias: likely none **SoE Conclusion: High**

Abbreviations: LEB = lebrikizumab, NNT = number needed to treat; PLB = placebo; SoE = Strength of Evidence; Q2W = every 2 weeks dosing; Q4W = every 4 weeks dosing; 95% CI = 95% confidence interval; ‡ FDA approved regimen

Maintenance Trial[21]

Participants enrolled in ADvocate1 and ADvocate2 who achieved an IGA 2+ or EASI-75 response by week 16 were re-randomized to a maintenance phase where they received lebrikizumab 250 mg SC Q2W, lebrikizumab 250 mg SC Q4W, or placebo (randomization ratio 2:2:1) from week 16 through week 52. Higher proportions of patients who were randomized to lebrikizumab compared to the placebo withdrawal group maintained their response as measured by EASI-75 (Q2W 78%, Q4W 82% vs. 66% for placebo; p-values not reported) or IGA 2+ (Q2W 71%, Q4W 77% vs. 48% for placebo; p-values not reported). A dose response was not evident and those receiving the less frequently dosed regimen (Q4W) had qualitatively similar rates of response to the more frequent administration schedule (Q2W). Although a substantial proportion of patients randomized to placebo after initial response maintained their IGA 2+ (48%) and EASI-75 (66%) response, the FDA approved maintenance dose for responders at week 16 is 250 mg Q4W. Although two trials with high internal validity provide evidence that

maintenance with lebrikizumab improves relevant outcomes relative to placebo withdrawal, precision of estimates is uncertain (Strength of Evidence: moderate).

Clinical Safety:
Lebrikizumab was generally well tolerated during the 16-week induction and 52-week maintenance trials reviewed by the FDA. As summarized in **Table 3,** rates of serious adverse events (AEs) were uncommon and occurred at similar rates between groups for both the induction and maintenance phase. The most frequently reported treatment emergent AE was conjunctivitis and herpes among those receiving lebrikizumab and atopic dermatitis for those receiving placebo. The significance of the higher incidence of COVID-19 among lebrikizumab patients (Q2W) is unclear and may be due to delta variant emergence during the pandemic. Injection site reactions, which were slightly more common with lebrikizumab, were uncommon (<5%).

Table 3: Adverse events occurring during the lebrikizumab trial program (ADvocate1, ADvocate2, ADhere)[23]

Adverse events occurring during the 16-week lebrikizumab induction trials (ADvocate1, ADvocate2, ADhere)			
Subjects with AE	**Lebrikizumab (n=805)**		**Placebo (n=417)**
Serious AEs	1.4%		1.7%
AEs leading to discontinuation	2.3%		1.4%
Treatment emergent AEs >=5%			
Any AE	48.9%		53.0%
Conjunctivitis	6.3%		1.7%
Atopic dermatitis	5.8%		17.7%
Adverse events occurring during week 16 through week 52 of maintenance trial (ADvocate1, ADvocate2)[21]			
	Lebrikizumab Q4W (n=118)	**Lebrikizumab Q2W (n=113)**	**Placebo (n=60)**
Serious AEs	1.7%	1.8%	1.7%
AEs leading to discontinuation	1.7%	0.9%	0%
Treatment emergent AEs >=5%			
COVID-19	9.3%	2.7%	3.3%
Conjunctivitis	5.9%	1.8%	3.3%
Atopic dermatitis	5.9%	4.4%	11.7%
Nasopharyngitis	7.6%	3.5%	5.0%
Herpes infections	5.9%	2.7%	3.3%

Other warnings and precautions
Patients with parasitic infections were excluded from trial participation. It is recommended patients be treated for these infections prior to treatment with lebrikizumab. Lebrikizumab may affect an individual's response following administration of live vaccines. It is recommended that all age-appropriate vaccinations be administered prior to treatment with lebrikizumab and to avoid live vaccines immediately prior to, or during, treatment.

Conclusions and Policy Recommendations

Lebrikizumab is a monoclonal antibody approved for the treatment of moderate to severe atopic dermatitis in patients ≥ 12 years old who weigh ≥ 40 kg and are not adequately controlled with topical prescription therapies. It can be used with or without other topical therapies (TCS or TCI). During the 16-week induction period, patients treated with lebrikizumab experienced improved skin clearance (High Strength of Evidence; IGA 2+, EASI-75), reduced pruritus (High Strength of Evidence; NRS 4+), and enhanced quality of life (High Strength of Evidence; DLQI 4+). Among responders continuing on maintenance therapy, lebrikizumab was also superior to placebo at improving these outcome in trials up to 52 weeks (Moderate Strength of Evidence). Lebrikizumab has not been directly compared to other monoclonal antibodies or other systemic immunomodulators for AD (Very low Strength of Evidence). Lebrikizumab was generally well tolerated with low rates of AEs during the induction and maintenance phases with conjunctivitis and herpes most frequently observed (~5%).

We recommend prior authorization criteria for lebrikizumab consistent with other monoclonal antibodies for AD to ensure safe and appropriate use for adults and adolescents with moderate-to-severe AD. For individuals approved for lebrikizumab, we recommend approving for 16 weeks at induction doses (250 mg Q2W including initial loading dose) with renewal criteria assessed at 16 weeks to ensure patients have responded and are prescribed the maintenance dose (250 mg Q4W).

Comparative Endpoints:

Clinically Meaningful Endpoints:
1) IGA 2+
2) EASI-75 / EASI-90
3) P-NRS 4+
4) DLQI 4+
5) Serious adverse events
6) Study withdrawal due to an adverse event

Primary Study Endpoint(s):
1) IGA 2+

Table 4: Evidence Tables: Lebrikizumab induction and maintenance clinical trials

Study Summary	Treatment Descriptions	Patient Population	Sample Size	Efficacy Endpoints	NNT	Safety Endpoints	Quality Rating (Risk of Bias)
Simpson EL, et al.[19] ADhere NCT04250337 DB, PC, RCT, MC 16-week induction	A. LEB Q2W; 500 mg SC injection for first 2 doses, then 250 mg Q2W B. PLB SC injection Q2W Low- to mid-potency TCS/ TCI allowed Randomized 2:1 `	Demographics: -Mean age: 37 years -age <18: 22% -age ≥18: 78% -Female: 49% -White: 62% -Black: 13% -Asian: 15% -Prior systemic treatment: 47% -IGA-4: 31% -Mean EASI: 26-27 -Mean P-NRS: 6.8-7.3 -Mean BSA affected: 38%-40% Inclusion: -Age: ≥12 years -ADCC moderate-to severe AD >=1 year -EASI >=16 -IGA >=3 -BSA >=10% -History of inadequate response to topical medications Exclusions: -TCS, TCI, PDE-4, rx moisturizers within 1 week -TIMs, Immunosuppressives, phototherapy within 4 weeks – 6 months -Ongoing chronic disease requiring oral steroids -chronic or acute infection	ITT (mITT) A.153 (145) B. 75 (66) Attrition A. 8 (5%) B. 9 (12%) mITT was used as 17 subjects from a study site were excluded because of eligibility could not be confirmed.	Primary Endpoints: (at 16 weeks) % with 2+ IGA (0,1) A. 41.2% B. 22.1% RD: 18.3% (95% CI 5.1% to 31.5%) P<0.05 Secondary Endpoints: (at 16 weeks) % with EASI-75 A. 69.5% B. 42.2% RD: 26.4 % (95% CI 12.1% to 40.8 %) P<0.01 % with 4+ P-NRS A. 50.6% B. 31.9% RD: 19.2% (95% CI 4.3% to 34.1 %) P<0.05 % with 4+ DLQI A. 77.4% B. 58.7% RD: 17.2% (95% CI 0.1 to 34.3%) P<0.05	6 4 6 6	Important AEs Serious AE: A. 1.4% B. 1.5% DC due to AE: A. 2.1% B. 0% Deaths: None Common AEs: Infections A. 17% B. 14%	**Risk of Bias (low/high/unclear)**: Selection Bias: Low. Randomized via electronic randomization stratified by region, age, IGA severity. Performance Bias: Low. Patients, investigators, personnel blinded. Placebo identical in appearance to LEB. Detection Bias: Low. Patients, investigators, personnel blinded. Placebo identical in appearance to LEB. Attrition Bias: High. Post randomization exclusion may have introduced systematic differences between groups. Other potential biases: Eli Lilly involved in funding, design, data collection, analysis, and preparation of manuscript. **External validity** Patient: Patients were typical with those with AD. Exclusions for prior treatment not overly restrictive. Intervention: Intervention dosed appropriately Comparator: placebo control appropriate to determine efficacy Outcomes: use of multiple symptom scales appropriate Setting: 45 outpatient sites in US, Europe, and Canada

Study Summary	Treatment Descriptions	Patient Population	Sample Size	Efficacy Endpoints	NNT	Safety Endpoints	Quality Rating (Risk of Bias)
Silverberg JI, et al. [20] Advocate1 NCT04146363 DB, PC, RCT, MC 16-week induction	A. LEB Q2W; 500 mg SC injection for first 2 doses, then 250 mg Q2W B. PLB SC injection Q2W Randomized 2:1 for 16 weeks	Demographics: -Mean age: 34-36 years -age <18: 13% -age ≥18: 87% -Female: 51% -White: 68% -Black: 12% -Asian: 17% -Prior systemic treatment: 54% -IGA-4: 40% -Mean EASI: 29-31 -Mean P-NRS: 7.3 -Mean BSA affected: 45%-47% Inclusion: -Age: ≥12 years -ADCC moderate-to severe AD ≥1 year -EASI ≥16 -IGA ≥3 -BSA ≥10% -History of inadequate response to topical medications Exclusions: -Treatment with LEB, dupilumab, tralokinumab -TCS, TCI, PDE-4, rx moisturizers within 1 week -Immunosuppressives, phototherapy within 4 weeks – 6 months -Ongoing chronic disease requiring oral steroids -Chronic or acute infection	ITT A. 283 B. 141 Attrition A. 4 (1.4%) B. 1 (0.7%)	Primary Endpoints: (at 16 weeks) % with 2+ IGA (0,1) A. 43.1% B. 12.7% RD: 29.7% (95% CI 21.6% to 37.8%) P<0.001 Secondary Endpoints: (at 16 weeks) % with EASI-75 A. 58.8% B. 16.2% RD: 42% (95% CI 33.3% to 50.6%) P<0.001 % with 4+ P-NRS A. 45.9% B. 13.0% RD: 32.9% (95% CI 24.6% to 41.3%) P<0.001 % with 4+ DLQI A. 71.2% B. 29.3% RD: 41.9% (95% CI 31.8% to 52.0%) p-value not reported	4 3 4 3	Important AEs Serious AE: A. 2.1% B. 0.7% DC due to AE: A. 1.1% B. 0.7% Deaths: None Common AEs Infections A. 21.6% B. 19.9% AD exacerbation A. 6.0% B. 21.3% Conjunctivitis A. 7.4% B. 2.8% Skin infection A. 2.8% B. 5.7%	**Risk of Bias (low/high/unclear)**: Selection Bias: Low. Randomized via interactive web response system stratified by region, age, and IGA severity. Performance Bias: Low. Patients, investigators, personnel blinded. Placebo identical in appearance to LEB. Detection Bias: Low. Patients, investigators, personnel blinded. Placebo identical in appearance to LEB. Attrition Bias: Low. Attrition similar between groups and around 1% overall Other potential biases: Eli Lilly involved in funding, design, data collection, analysis, and preparation of manuscript **External validity** Patient: Patients were typical with those with moderate to severe AD. Exclusions reflect a sample with less prior TIMs treatment experience. Intervention: Intervention dosed appropriately Comparator: Placebo control appropriate Outcomes: Use of multiple symptom scales appropriate with consistent direction of effect. Setting: 94 outpatient sites in US, Europe, Korea, Australia, and Canada

Study Summary	Treatment Descriptions	Patient Population	Sample Size	Efficacy Endpoints	NNT	Safety Endpoints	Quality Rating (Risk of Bias)
Silverberg JI, et al.[20] Advocate2 NCT04178967 DB, PC, RCT, MC 16-week induction	A. LEB 250 mg SC Q2W* B. PLB SC Q2W Randomized 2:1 for 16 weeks *500 mg loading dose at baseline and week 2	Demographics: -Mean age: 35-37 years -age <18: 11% -age ≥18: 89% -Female: 49% -White: 59% -Black: 8% -Asian: 29% -Prior systemic treatment: 56% -IGA-4: 37% -Mean EASI: 30 -Mean P-NRS: 7 -Mean BSA affected: 46% Inclusion: -Age: ≥12 years -ADCC moderate-to severe AD ≥1 year -EASI ≥16 -IGA ≥3 -BSA ≥10% -History of inadequate response to topical medications Exclusions: -Tx with LEB, dupilumab, tralokinumab -TCS, TCI, PDE-4, rx moisturizers within 1 week -immunosuppressives, phototherapy within 4 weeks – 6 months -Ongoing chronic disease requiring oral steroids -Chronic or acute infection	ITT A. 281 B. 146 Attrition A. 1 (0.4%) B. 2 (1.4%)	Primary Endpoints: (at 16 weeks) % with 2+ IGA (0,1) A. 33.2% B. 10.8% RD: 21.9% (95% CI 14.2 to 29.6%) P<.001 Secondary Endpoints: (at 16 weeks) % with EASI-75 A. 52.1% B. 18.1% RD: 33.3% (95% CI 24.4% to 42.2%) P<.001 % with 4+ P-NRS A. 39.8% B. 11.5% RD: 28.3% (95% CI 20.0% to 36.5%) P<.001 % with 4+ DLQI A. 62.3% B. 31.3% RD: 31.4% (95% CI 20.7% to 42.1%) p-value not reported	5 3 4 4	Important AEs Serious AE: A. 0.7% B. 2.8% DC due to AE: A. 3.2% B. 2.8% Deaths: A. 0 B. 1 Common AEs Infections A. 23.1% B. 20.7% AD exacerbation A. 10.3% B. 26.9% Conjunctivitis A. 7.5% B. 2.1% Skin infection A. 1.4% B. 6.2%	**Risk of Bias (low/high/unclear)**: Selection Bias: Low. Randomized via interactive web response system stratified by region, age, IGA severity. Slight imbalance in racial demographics (fewer Asian patients received LEB) Performance Bias: Low. Patients, investigators, personnel blinded. Placebo identical in appearance to LEB. Detection Bias: Low. Patients, investigators, personnel blinded. Placebo identical in appearance to LEB. Attrition Bias: Low. Attrition similar between groups and around 1% overall Other potential biases: Eli Lilly involved in funding, design, data collection, analysis, and preparation of manuscript **External validity** Patient: Patients were typical with those with moderate to severe AD. Exclusions reflect a sample with less prior TIMs treatment experience. Intervention: Intervention dosed appropriately Comparator: Placebo control appropriate Outcomes: Use of multiple symptom scales appropriate Setting: 94 outpatient sites in US, Europe, Mexico, Taiwan, Singapore, and Canada

Katoh N, et al.[22] Adhere-J NCT04760314 DB, PC, RCT, MC 16-week induction	A. LEB 250 mg SC Q2W* (FDA approved induction dose) B. LEB 250 mg SC Q4W** C. PLB SC Q2W Plus, low or mid-potency TCS/TCI allowed Randomized 3:2:2 for 16 weeks *500 mg loading dose at baseline and week 2 **500 mg loading dose at baseline	<u>Demographics</u>: -Mean age: 35-38 years -age <18: 6% -age ≥18: 94% -Female: 31% -Asian: 100% -History biologic treatment: 2% History systemic steroids: 34%-42% -IGA-4: 32% -Mean EASI: 32-34 -Mean P-NRS: 5.1-5.4 -Mean BSA affected: 58%-61% <u>Inclusion:</u> -Age: 12-17 (≥40 kg), ≥ 18 years -ADCC moderate-to severe AD ≥1 year -EASI ≥16 -IGA ≥3 -BSA ≥10% -History of inadequate response to topical medications (TCS/TCI) <u>Exclusions:</u> - Recent treatment with LEB, dupilumab, JAK inhibitors, PDE-4, B-cell depleting agents (time periods differed for each therapy) -High potency TCS, topical JAK, PDE-4, within 1 week -immunosuppressives, phototherapy within 4 weeks -Ongoing chronic disease requiring oral steroids -Chronic or acute infection	<u>ITT</u> A. 123 B. 81 C. 82 <u>Attrition</u> A. 3 (2.4%) B. 1 (1.2%) C. 0 (0%)	<u>Primary Endpoints</u>: (at 16 weeks) % with 2+ IGA (0,1) A. 33.4% B. 29.1% C. 6.1% RD A vs C: 27.3% (95% CI 17.5% to 37.0%)* RD B vs C: 22.6% (95% CI 11.6% to 33.6%)* % with EASI-75 A. 51.2% B. 47.2% C. 13.4% RD A vs C: 37.6% (95% CI 26.2% to 49.0%)* RD B vs C: 33.2% (95% CI 20.6% to 45.8%)* <u>Secondary Endpoints</u>: (at 16 weeks) % with EASI-90 A. 34.3% B. 28.4% C. 9.8% RD A vs C: 24.2% (95% CI 13.9% to 34.5%) * RD B vs C: 18.4% (95% CI 6.8% to 29.9%)** % with 4+ P-NRS A. 32.7% B. 23.8% C. 3.3% RD A vs C: 29.2% (95% CI 17.9% to 40.4%) * RD B vs C: 20.6% (95% CI 8.7% to 32.4%)** % with 4+ DLQI A. 68.8% B. 53.3% C. 20.6% RD A vs C: 48.1% (95% CI 34.5% to 61.7%)* RD B vs C: 32.8% (95% CI 16.6% to 48.9%) * P<0.001 **P<0.01 (relative to placebo)	4 4 3 3 5 6 4 5 3 4	Important AEs <u>Serious AE</u>: A. 0.8% B. 0% C. 2.4% <u>DC due to AE:</u> A. 1.6% B. 0% C. 0% <u>Deaths:</u> A. 0% B. 0% C. 0% Common AEs <u>Pyrexia:</u> A. 20.3% B. 18.5% C. 15.9% <u>Nasopharyngitis:</u> A. 5.7% B. 6.2% C. 2.4% <u>Allergic conjunctivitis:</u> A. 17.1% B. 12.3% C. 4.9% <u>Conjunctivitis:</u> A. 9.8% B. 6.2% C. 2.4% <u>Headache:</u> A. 3.3% B. 3.7% C. 11.1% <u>Skin infection:</u> A. 7.3% B. 8.6% C. 17.1%	**Risk of Bias (low/high/unclear)**: <u>Selection Bias</u>: Low. Randomized via interactive web response system stratified by age, IGA severity. Baseline characteristics balanced. <u>Performance Bias</u>: Low. Patients, investigators, personnel blinded. Placebo identical in appearance to LEB. <u>Detection Bias</u>: Low. Patients, investigators, personnel blinded. Placebo identical in appearance to LEB. <u>Attrition Bias</u>: Low. Attrition similar between groups and around 1% overall <u>Other potential biases:</u> Eli Lilly involved in funding, design, data collection, analysis, and preparation of manuscript **External validity** <u>Patient:</u> None, patients typical with those with AD. Exclusions for prior treatment not overly restrictive. Demographically homogenous. <u>Intervention:</u> Intervention dosed appropriately <u>Comparator:</u> Placebo control appropriate <u>Outcomes:</u> Use of multiple symptom scales appropriate <u>Setting:</u> Japanese population

Study Summary	Treatment Descriptions	Patient Population	Sample Size	Efficacy Endpoints	NNT	Safety Endpoints	Quality Rating (Risk of Bias)
Blauvelt A, et al.[21] Advocate1 NCT04146363 Advocate2 NCT04178967 DB, PC, RCT, MC 52-week maintenance to evaluate durability of response	A. LEB 250 mg SC Q2W B. LEB 250 mg SC Q4W (FDA approved dose) C. PLB SC Q2W (LEB withdrawal arm) Randomized 2:2:1 for 36 weeks; 52 weeks after initial induction) Intermittent rescue with TCS therapy allowed Rescue medication use: A. 12.4% B. 16.1% C. 18.3%	<u>Demographics</u>: -Mean age: 34 to 36 years -age <18: 13% -age ≥18: 87% -Female: 54% -White: 68% -Black: 10% -Asian: 18% <u>Inclusion / Exclusion:</u> see ADvocate1 & ADvocate2: -LEB treated and response at week 16 (75% reduction in EASI-75 or 2+ IGA (0,1))	<u>ITT</u> A. 113 B. 118 C. 60 <u>Attrition</u> A. 12 (11%) B. 8 (7%) C. 6 (10%)	<u>Endpoints:</u> (at 52 weeks) % who maintained EASI-75 A. 78.4% B. 81.7% C. 66.4% RD A vs C: 12.0%* RD B vs C: 15.3%* % who maintained IGA 2+ A. 71.2% B. 76.9% C. 47.9% RD A vs C: 23.3%* RD B vs C: 29.0%* % who maintained 4+ P-NRS A. 84.6% B. 84.7% C. 66.3% RD A vs C: 18.3%* RD B vs C: 18.4%* *p-values / 95% CI not reported	A. 9 B. 7 A. 5 B. 4 A. 6 B. 6	Important AEs <u>Serious AE</u>: A. 1.8% B. 1.7% C. 1.7% <u>DC due to AE:</u> A. 0.9% B. 1.7% C. 0% <u>Deaths:</u> A. 0% B. 0% C. 0% Common AEs <u>COVID-19:</u> A. 2.7% B. 9.3% C. 3.3% <u>Nasopharyngitis:</u> A. 3.5% B. 7.6% C. 5.0% <u>Headache:</u> A. 0.9% B. 5.2% C. 1.7% <u>AD exacerbation:</u> A. 4.4% B. 5.9% C. 11.7% <u>Conjunctivitis:</u> A. 0% B. 5.1% C. 5.0% <u>Herpes:</u> A. 2.7% B. 5.9% C. 3.3%	**Risk of Bias (low/high/unclear)**: <u>Selection Bias</u>: Low. Randomized via electronic data capture system. Imbalance in racial demographics (PLB had fewer White patients) <u>Performance Bias</u>: Low. Patients, investigators, personnel blinded. Placebo identical in appearance to LEB. <u>Detection Bias</u>: Low. Patients, investigators, personnel blinded. Placebo identical in appearance to LEB. <u>Attrition Bias</u>: Low. Attrition similar between groups and around 10% overall <u>Other potential biases:</u> Eli Lilly involved in funding, design, data collection, analysis, and preparation of manuscript **External validity** <u>Patient:</u> None, patients typical with those with AD. Exclusions for prior treatment not overly restrictive. <u>Intervention:</u> Intervention dosed appropriately <u>Comparator:</u> Placebo control appropriate <u>Outcomes:</u> Use of multiple symptom scales appropriate <u>Setting:</u> 94 outpatient sites in US, Europe, Mexico, Taiwan, Singapore, and Canada

Study Summary	Treatment Descriptions	Patient Population	Sample Size	Efficacy Endpoints	NNT	Safety Endpoints	Quality Rating (Risk of Bias)

Key: Abbreviations: AD = atopic dermatitis; ADCC = American Academy of Dermatology Consensus Criteria; AE = adverse event; BSA = body surface area; CI = confidence interval; DB = double-blind; DC = discontinue; DLQI = Dermatology Life Quality Index score; EASI = Eczema Area and Severity Index score; IGA = Investigators Global Assessment score; JAK = Janus kinase; ITT = intention to treat; LEB = lebrikizumab; MC = multicenter; mITT = modified intention to treat; NNT = number needed to treat; PDE = phosphodiesterase inhibitor; PC = placebo- controlled; PLB = placebo; P-NRS = Pruritus Numeric Rating Scale; Q2W = every 2 weeks; Q4W = every 4 weeks; RCT= randomized controlled trial; RD = risk difference; RX = prescription; SC = subcutaneous; TCI = topical calcineurin inhibitors; TCS = topical corticosteroids

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Appendix 1: Prescribing Information Highlights

HIGHLIGHTS OF PRESCRIBING INFORMATION

These highlights do not include all the information needed to use EBGLYSS safely and effectively. See full prescribing information for EBGLYSS.

EBGLYSS (lebrikizumab-lbkz), injection, for subcutaneous use
Initial U.S. Approval: 2024

---------- INDICATIONS AND USAGE ----------

EBGLYSS™ is an interleukin-13 antagonist indicated for the treatment of adults and pediatric patients 12 years of age and older who weigh at least 40 kg with moderate-to-severe atopic dermatitis whose disease is not adequately controlled with topical prescription therapies or when those therapies are not advisable. EBGLYSS can be used with or without topical corticosteroids. (1)

---------- DOSAGE AND ADMINISTRATION ----------

- Prior to EBGLYSS treatment, complete all age-appropriate vaccinations according to current immunization guidelines. (2.1)
- The recommended dosage of EBGLYSS is 500 mg (two 250 mg injections) at Week 0 and Week 2, followed by 250 mg (one injection) every 2 weeks until Week 16 or later, when adequate clinical response is achieved. The maintenance dose is EBGLYSS 250 mg every 4 weeks. (2.2)
- Administer by subcutaneous injection. (2.4)

---------- DOSAGE FORMS AND STRENGTHS ----------

Injection:

- 250 mg/2 mL in a single-dose prefilled pen (3)
- 250 mg/2 mL (125 mg/mL) in a single-dose prefilled syringe with needle shield (3)

---------- CONTRAINDICATIONS ----------

Prior serious hypersensitivity to lebrikizumab-lbkz or any excipients in EBGLYSS. (4)

---------- WARNINGS AND PRECAUTIONS ----------

- Hypersensitivity: Hypersensitivity reactions including angioedema and urticaria, have occurred after administration of EBGLYSS. Discontinue EBGLYSS in the event of a serious hypersensitivity reaction. (5.1)
- Conjunctivitis and Keratitis: Report new onset or worsening eye symptoms to a healthcare provider. (5.2)
- Parasitic (Helminth) Infections: Treat patients with pre-existing helminth infections before initiating EBGLYSS. If patients become infected while receiving EBGLYSS and do not respond to anti-helminth treatment, discontinue treatment with EBGLYSS until the infection resolves. (5.3)
- Vaccinations: Avoid use of live vaccines during treatment with EBGLYSS. (5.4)

---------- ADVERSE REACTIONS ----------

Most common (≥1%) adverse reactions are conjunctivitis, injection site reactions, and herpes zoster. (6.1)

To report SUSPECTED ADVERSE REACTIONS, contact Eli Lilly and Company at 1-800-LillyRx (1-800-545-5979) or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

See 17 for PATIENT COUNSELING INFORMATION and FDA-approved patient labeling.

Revised: 05/2025