IL-13 ANTIBODIES FOR THE TREATMENT OF POST-INFLAMMATORY HYPERPIGMENTATION OR HYPOPIGMENTATION OF SKIN

SEQUENCE LISTING

[0001] The present application is being filed along with a Sequence Listing in ST.26 XML format. The Sequence Listing is provided as a file titled "31021_WO_000 Sequence Listing ST26 v2" created February 26, 2025, and is 20.1 kilobytes in size. The Sequence Listing information in the ST.26 XML format is incorporated herein by reference in its entirety.

FIELD

[0002] The present invention relates to methods, uses, and pharmaceutical compositions of antibodies that bind human IL-13 ("anti-IL-13 antibodies") for treating post-inflammatory hyperpigmentation or hypopigmentation of skin, e.g., post-inflammatory hyperpigmentation or hypopigmentation of skin associated with atopic dermatitis.

BACKGROUND

[0003] Interleukin (IL)-13 is a key mediator of T-helper type 2 (Th2) inflammation and signals through a heterodimeric receptor IL-4Rα/IL-13Rα1. Several lines of evidence suggest that IL-13 is a key pathogenetic component in atopic dermatitis (AD). Increased expression of IL-13 has consistently been reported in AD skin (Hamid Q, et al., J Allergy Clin Immunol 98:225-31 [1996]; Jeong CW, et al., Clin Exp Allergy 33:1717-24 [2003]; Tazawa T, et al., Arch Dermatol Res 295:459-64 [2004]; Neis MM, et al., J Allergy Clin Immunol 118:930-7 [2006]; Suárez-Fariñas M, et al., J Allergy Clin Immunol 132:361-70 [2013]; Choy DF, et al., J Allergy Clin Immunol.130:1335-43 [2012]) and some reports suggest a relationship between IL-13 expression and the severity of disease (La Grutta S, et al., Allergy 60:391-5 [2005]). Increased IL-13 has also been reported in the serum of AD patients (Novak N, et al., J Invest Dermatol 2002;119:870–5; WO2016149276), and several studies have reported an increase in IL-13-expressing T cells in the blood of AD patients (Akdis M, et al., J Immunol 1997;159:4611–9; Aleksza M, et al., Br J Dermatol 2002;147:1135–41; La Grutta S, et al., Allergy 2005;60:391–5).

[0004] Atopic dermatitis is a chronic relapsing and remitting inflammatory skin disorder affecting all age groups. Clinically, AD is characterized by xerosis, erythematous crusting rash, lichenification, an impaired skin barrier, and intense pruritus (Bieber T., N Engl J Med

2008;358:1483–94). There is a paucity of data to guide diagnosis and treatment in populations traditionally under-represented in clinical trials, including patients with skin of color (Chen et al., JAMA Dermatol. 2022;158(3):288-292). Compounding this issue is the complexity of diagnosing moderate-to-severe AD in this patient population, partially due to historical lack of representation in medical education resources and in some cases, limited exposure to patients with skin of color in medical training (Ebede et al., J Am Acad Dermatol. 2006;55(4):687-690; Adelekun et al., J Am Acad Dermatol. 2021;84(1):194-196; Ibraheim et al., *Clin Dermatol.* 2021; 25:S0738-081X(21)00257-1; Kurtti et al. 2021, Br J Dermatol. 2002;147(suppl 62):25 -54). Finally, the nuances of identifying erythema in patients with skin of color not only affect the accurate and timely diagnosis of AD but also confound the use of AD scales commonly used in clinical trials (Zhao et al., *Int J Womens Dermatol.* 2015;1(3):150-154).

[0005] There remains an unmet medical need for treating post-inflammatory hyperpigmentation or hypopigmentation of skin, e.g., post-inflammatory hyperpigmentation or hypopigmentation of skin associated with atopic dermatitis, especially for patients with skin of color.

SUMMARY OF INVENTION

[0006] Provided herein are methods, uses, and pharmaceutical compositions of anti-IL-13 antibodies (e.g., lebrikizumab) for treating post-inflammatory hyperpigmentation or hypopigmentation of skin, e.g., post-inflammatory hyperpigmentation or hypopigmentation of skin associated with atopic dermatitis.

[0007] In one aspect, provided herein are methods of treating post-inflammatory hyperpigmentation or hypopigmentation of skin in a patient in need thereof, the methods comprising administering to the patient a therapeutically effective amount of an anti-IL-13 antibody. In some embodiments, provided herein are methods of treating post-inflammatory hyperpigmentation or hypopigmentation of skin, the methods comprising: selecting a patient who has post-inflammatory hyperpigmentation or hypopigmentation of skin, and administering to the patient a therapeutically effective amount of an anti-IL-13 antibody. In some embodiments, the post-inflammatory hyperpigmentation or hypopigmentation of skin is associated with atopic dermatitis. In some embodiments, the patient's skin hyperpigmentation or hypopigmentation is measured by a post-inflammatory dyschromia scale. In some embodiments, the patient's skin hyperpigmentation or hypopigmentation is measured by the

Post-inflammatory Dyschromia Scale (PDS) or the Post-inflammatory Dyschromia Clinical Assessment (PDCA-DERM^{TM}).

[0008] In another aspect, provided herein are an anti-IL-13 antibody or pharmaceutical composition comprising an anti-IL-13 antibody for use in the treatment of post-inflammatory hyperpigmentation or hypopigmentation of skin. Also provided herein are uses of an anti-IL-13 antibody in the manufacture of a medicament for the treatment of post-inflammatory hyperpigmentation or hypopigmentation of skin. In some embodiments, the post-inflammatory hyperpigmentation or hypopigmentation of skin is associated with atopic dermatitis.

[0009] In some embodiments, the patient has moderate to severe AD for at least a year at the baseline. The moderate to severe AD can be determined by criteria known in the art, e.g., the American Academy of Dermatology Consensus Criteria for Chronic Atopic Dermatitis. In some embodiments, the patient has an EASI score of 16 or greater, an IGA score of 3 or greater, and more than 10% of body surface area (BSA) affected by AD at the baseline. In some embodiments, the patient has inadequate response to topical corticosteroids, topical calcineurin inhibitors, or crisaborole; or topical corticosteroids, topical calcineurin inhibitors, or crisaborole are medically inadvisable for the patient. In some embodiments, the patient is aged 12 years and older.

[0010] In some embodiments, the patient is Fitzpatrick Phototype of IV to VI. In some embodiments, the patient is not white. In some embodiments, the patient is Black or African American, American Indian, Alaska Native, Asian, Native Hawaiian, or Pacific Islander.

[0011] In some embodiments, the anti-IL-13 antibody comprises a heavy chain variable region (VH) and a light chain variable region (VL), wherein the VH comprises a HCDR1 comprising SEQ ID NO: 1, a HCDR2 comprising SEQ ID NO: 2, and a HCDR3 comprising SEQ ID NO: 3, and the VL comprises a LCDR1 comprising SEQ ID NO: 4, a LCDR2 comprising SEQ ID NO: 5, and a LCDR3 comprising SEQ ID NO: 6. In some embodiments, the anti-IL-13 antibody comprises a VH comprising SEQ ID NO: 7, and a VL comprising SEQ ID NO: 8. In some embodiments, the anti-IL-13 antibody comprises a VH comprising SEQ ID NO: 11, and a VL comprising SEQ ID NO: 12. In some embodiments, the anti-IL-13 antibody comprises a heavy chain comprising SEQ ID NO: 9, and a light chain comprising SEQ ID NO: 10. In some embodiments, the anti-IL-13 antibody comprises a heavy chain comprising SEQ ID NO: 13, and a light chain comprising SEQ ID NO: 14. In some embodiments, the anti-IL-13 antibody is lebrikizumab. In certain embodiments, the

anti-IL-13 antibody comprises a VH sequence set forth in SEQ ID NO: 11, a VL sequence set forth in SEQ ID NO: 12, and the human IgG sequence set forth in SEQ ID NO: 15. In certain embodiments, the anti-IL-13 antibody comprises a VH sequence set forth in SEQ ID NO: 11, a VL sequence set forth in SEQ ID NO: 12, a human Fc region comprising the human IgG sequence set forth in SEQ ID NO: 15, and a constant light chain sequence set forth by SEQ ID NO: 16.

[0012] In some embodiments, the anti-IL-13 antibody is administered subcutaneously to the patient. In some embodiments, the anti-IL-13 antibody is administered at a dose of 250 mg to 500 mg. In some embodiments, the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 250 mg once every two weeks. In some embodiments, the patient is further treated with a loading dose of 500 mg of the anti-IL-13 antibody. In some embodiments, the loading dose is administered once or twice to the patient. In some embodiments, the loading dose is administered to the patient at week 0 (baseline) and week 2. In some embodiments, the patient is treated with the anti-IL-13 antibody for a period of at least 16 weeks. In some embodiments, the patient is treated with the anti-IL-13 antibody at a loading dose of 500 mg at baseline (week 0) and week 2, followed by 250 mg once every two weeks for at least 14 weeks.

[0013] In some embodiments, the patient is further treated for a maintenance period of 8 to 36 weeks. In some embodiments, the patient is treated with a maintenance dose of 250 mg of the anti-IL-13 antibody once every two weeks during the maintenance period. In some embodiments, the patient is treated with a maintenance dose of 250 mg of the anti-IL-13 antibody once every four weeks during the maintenance period. In some embodiments, the patient is treated with a maintenance dose of 250 mg of anti-IL-13 antibody once every eight weeks during the maintenance period.

[0014] In some embodiments, the methods, uses, and pharmaceutical compositions described herein further comprise administrating one or more topical corticosteroids to the patient. In some embodiments, the anti-IL-13 antibody is used in simultaneous, separate, or sequential combination with one or more topical corticosteroids. In some embodiments, the topical corticosteroid is triamcinolone acetonide, hydrocortisone, or a combination of triamcinolone acetonide and hydrocortisone. In some embodiments, the topical corticosteroids are administered concomitantly with the anti-IL-13 antibody.

BRIEF DESCRIPTION OF THE DRAWINGS

[0015] **Figure 1** is a schematic diagram of the study design described in Example 1.

[0016] **Figure 2** shows the PDCA-DERM™ scale.

[0017] **Figure 3A** shows the baseline demographics of enrolled patients. **Figure 3B** shows the baseline disease characteristics of enrolled patients.

[0018] **Figure 4** shows the percentage of patients treated with lebrikizumab achieved IGA (0,1) with ≥2-point improvement.

[0019] **Figure 5** shows the percentage of patients treated with lebrikizumab achieved EASI-75.

[0020] **Figure 6** shows the mean pruritus NRS percentage change from baseline.

[0021] **Figure 7A** shows the baseline PDCA-DERM™ scores. **Figure 7B** shows PDCA-DERM™ changes from baseline to Week 16 for hyperpigmented lesions. Footnotes: [a] For patients with multiple hypopigmented or hyperpigmented lesions at baseline, only the lesion with the most severe score was included in the analysis for each lesion type. In the event of a tie, the change from baseline representing a smaller improvement or worsening in condition was included; [b] The reduction from 27 to 21 patients with hyperpigmented lesions between Week 0 and Week 16 can be attributed to the study discontinuation of 3 patients and missing data for another 3 patients; [c] Improved = +3 to +2/+1/0, +2 to +1/0 or +1 to 0; [d] Normal skin tone = +3/+2/+1 to 0. [e] No change/Not improved = +3 to +3/-1/-2/-3, +2 to +3/+2/-1/-2/-3, +1 to +3/+2/+1/-1/-2/-3. **Figure 7C** shows PDCA-DERM™ changes from baseline to Week 16 for hypopigmented lesions. Footnotes: [a] For patients with multiple hypopigmented or hyperpigmented lesions at baseline, only the lesion with the most severe score was included in the analysis for each lesion type. In the event of a tie, the change from baseline representing a smaller improvement or worsening in condition was included; [b] The reduction from 9 to 5 patients with hypopigmented lesions between baseline and Week 0 can be attributed to the study discontinuation of 2 patients and missing data for another 2 patients; [c] Improved = -3 to -2/-1/0, -2 to -1/0 or -1 to 0; [d] Normal skin tone = -3/-2/-1 to 0. [e] No change/Not improved = -3 to -3/+1/+2/+3, -2 to -3/-2/+1/+2/+3, -1 to -3/-2/-1/+1/+2/+3.

DETAILED DESCRIPTION

[0022] Provided herein are methods, uses, and pharmaceutical compositions of anti-IL-13 antibodies for treating post-inflammatory hyperpigmentation or hypopigmentation of skin, e.g., post-inflammatory hyperpigmentation or hypopigmentation of skin associated with AD.

[0023] In one aspect, provided herein are methods of treating post-inflammatory hyperpigmentation or hypopigmentation of skin in a patient in need thereof, the methods comprising administering to the patient a therapeutically effective amount of an anti-IL-13 antibody. In some embodiments, provided herein are methods of treating post-inflammatory hyperpigmentation or hypopigmentation of skin, the methods comprising: selecting a patient who has post-inflammatory hyperpigmentation or hypopigmentation of skin, and administering to the patient a therapeutically effective amount of an anti-IL-13 antibody. In some embodiments, the post-inflammatory hyperpigmentation or hypopigmentation of skin is associated with AD. In some embodiments, the patient's skin hyperpigmentation or hypopigmentation is measured by a post-inflammatory dyschromia scale such as the Post-inflammatory Dyschromia Scale (PDS) or the Post-inflammatory Dyschromia Clinical Assessment (PDCA-DERM^{TM}).

[0024] In another aspect, provided herein are an anti-IL-13 antibody or pharmaceutical composition comprising an anti-IL-13 antibody for use in the treatment of post-inflammatory hyperpigmentation or hypopigmentation of skin. Also provided herein are uses of an anti-IL-13 antibody in the manufacture of a medicament for the treatment of post-inflammatory hyperpigmentation or hypopigmentation of skin. In some embodiments, the post-inflammatory hyperpigmentation or hypopigmentation of skin is associated with AD.

[0025] In some embodiments, the patient has moderate to severe AD for at least a year at the baseline. In some embodiments, the patient has an EASI score of 16 or greater, an IGA score of 3 or greater, and more than 10% of body surface area (BSA) affected by AD at the baseline. In some embodiments, the patient has inadequate response to topical corticosteroids, topical calcineurin inhibitors, or crisaborole; or topical corticosteroids, topical calcineurin inhibitors, or crisaborole are medically inadvisable for the patient. In some embodiments, the patient is aged 12 years and older.

[0026] In some embodiments, the patient is Fitzpatrick Phototype of IV to VI. In some embodiments, the patient is not white. In some embodiments, the patient is Black or African American, American Indian, Alaska Native, Asian, Native Hawaiian, or Pacific Islander.

[0027] In some embodiments, the AD severity can be determined by criteria known in the art, e.g., Hanifin and Rajka criteria (*Acta Derm Venereol (Stockh)* 1980; Suppl 92:44-7); Rajka and Langeland criteria (Rajka G and Langeland T, *Acta Derm Venereol (Stockh)* 1989; 144(Suppl):13–4); or the American Academy of Dermatology Consensus Criteria for Chronic Atopic Dermatitis (Eichenfield LF, et al. *J Am Acad Dermatol.* 2014;70(2):338-351). In some embodiments, the AD severity is determined by the American Academy of Dermatology Consensus Criteria for Chronic Atopic Dermatitis. Under the criteria, the essential features of AD include pruritus; eczema (acute, subacute, chronic); typical morphology and age specific patterns; chronic or relapsing history. The typical morphology and age specific patterns include facial, neck, and extensor involvement in infants and children; current or previous flexural lesions in any age group; sparing of the groin and axillary regions. Other important features that add support to the diagnosis include early age of onset; atopy; personal and/or family history; immunoglobulin E reactivity; xerosis. The associated features that could help to suggest the diagnosis of atopic dermatitis but are nonspecific to be used for defining or detecting AD for research and epidemiologic studies: atypical vascular responses (e.g. facial pallor, white dermographism, delayed blanch response); keratosis pilaris/ pityriasis alba/ hyperlinear palms/ icthyosis; ocular/periorbital changes; perifollicular accentuation/ lichenification/ prurigo lesions. On occasion, skin biopsy specimens or other tests (such as serum immunoglobulin E, potassium hydroxide preparation, patch testing, and/or genetic testing) may be helpful to rule out other or associated skin conditions. The exclusionary conditions include scabies; seborrheic dermatitis; contact dermatitis (irritant or allergic); icthyoses; cutaneous T-cell lymphoma; psoriasis; photosensitivity dermatoses; immune deficiency diseases; erythroderma of other causes.

[0028] The anti-IL-13 antibodies suitable for use in the methods and uses provided herein have been described previously, e.g., WO2005062967. In some embodiments, the anti-IL-13 antibody binds IL-13 with high affinity and blocks signaling through the active IL-4Ralpha/IL-13Ralpha1 heterodimer. In some embodiments, the anti-IL-13 antibody comprises a heavy chain variable region (VH) and a light chain variable region (VL), wherein the VH comprises a HCDR1 comprising SEQ ID NO: 1, a HCDR2 comprising SEQ ID NO: 2, and a HCDR3 comprising SEQ ID NO: 3, and the VL comprises a LCDR1 comprising SEQ ID NO: 4, a LCDR2 comprising SEQ ID NO: 5, and a LCDR3 comprising SEQ ID NO: 6. In some embodiments, the anti-IL-13 antibody comprises a VH comprising SEQ ID NO:

7, and a VL comprising SEQ ID NO: 8. In some embodiments, the anti-IL-13 antibody comprises a VH comprising SEQ ID NO: 11, and a VL comprising SEQ ID NO: 12. In some embodiments, the anti-IL-13 antibody comprises a heavy chain comprising SEQ ID NO: 9, and a light chain comprising SEQ ID NO: 10. In some embodiments, the anti-IL-13 antibody comprises a heavy chain comprising SEQ ID NO: 13, and a light chain comprising SEQ ID NO: 14. In certain embodiments, the anti-IL-13 antibody comprises a VH sequence set forth in SEQ ID NO: 11, a VL sequence set forth in SEQ ID NO: 12, and the human IgG sequence set forth in SEQ ID NO: 15. In certain embodiments, the anti-IL-13 antibody comprises a VH sequence set forth in SEQ ID NO: 11, a VL sequence set forth in SEQ ID NO: 12, a human Fc region comprising the human IgG sequence set forth in SEQ ID NO: 15, and a constant light chain sequence set forth by SEQ ID NO: 16. In some embodiments, the anti-IL-13 antibody is lebrikizumab (CAS No. 953400-68-5). Lebrikizumab is a humanized monoclonal IgG4 antibody that specifically binds IL-13 with high affinity and blocks signaling through the active IL-4Ralpha/IL-13Ralpha1 heterodimer. The amino acid sequences of lebrikizumab are provided in Table 1. C-terminal clipping of IgG antibodies could occur when one or two C-terminal amino acids are removed from the heavy chain of the IgG antibodies. For example, if a C-terminal lysine (K) is present, it may be truncated or clipped off from the heavy chain. A penultimate glycine (G) may also be truncated or clipped off from the heavy chain as well. Modification of N-terminal amino acid of IgG could also occur. For example, the N-terminal glutamine (Q) or glutamic acid (E) can cyclize into pyro-glutamate (pE) spontaneously. SEQ ID NO: 9 reflects these potential modifications of lebrikizumab heavy chain. Similarly, SEQ ID NO: 11, 13, and 15 reflect these potential modifications of lebrikizumab variant VH, lebrikizumab variant HC, and human IgG1 Fc region, respectively.

Table 1. Lebrikizumab Sequences

SEQ ID NO:	Description	Sequence
1	Lebrikizumab HCDR1	AYSVN
2	Lebrikizumab HCDR2	MIWGDGKIVYNSALKS
3	Lebrikizumab HCDR3	DGYYPYAMDN
4	Lebrikizumab LCDR1	RASKSVDSYGNSFMH
5	lebrikizumab LCDR2	LASNLES
6	Lebrikizumab LCDR3	QQNNEDPRT
7	Lebrikizumab heavy chain variable region (VH)	VTLRESGPALVKPTQTLTLTCTVSGFSLSAYSVN WIRQPPGKALEWLAMIWGDGKIVYNSALKSRLTI SKDTSKNQVVLTMTNMDPVDTATYYCAGDGYY PYAMDNWGQGSLVTVSS
8	Lebrikizumab light chain variable region (VL)	DIVMTQSPDSLSVSLGERATINCRASKSVDSYGN SFMHWYQQKPGQPPKLLIYLASNLESGVPDRFSG SGSGTDFTLTISSLQAEDVAVYYCQQNNEDPRTF GGGTKVEIK
9	Lebrikizumab heavy chain (HC)	Xaa1VTLRESGPALVKPTQTLTLTCTVSGFSLSAYS VNWIRQPPGKALEWLAMIWGDGKIVYNSALKSR LTISKDTSKNQVVLTMTNMDPVDTATYYCAGDG YYPYAMDNWGQGSLVTVSSASTKGPSVFPLAPC SRSTSESTAALGCLVKDYFPEPVTVSWNSGALTS GVHTFPAVLQSSGLYSLSSVVTVPSSSLGTKTYTC NVDHKPSNTKVDKRVESKYGPPCPPCPAPEFLGG PSVFLFPPKPKDTLMISRTPEVTCVVVDVSQEDPE VQFNWYVDGVEVHNAKTKPREEQFNSTYRVVS VLTVLHQDWLNGKEYKCKVSNKGLPSSIEKTISK AKGQPREPQVYTLPPSQEEMTKNQVSLTCLVKG FYPSDIAVEWESNGQPENNYKTTPPVLDSDGSFF LYSRLTVDKSRWQEGNVFSCSVMHEALHNHYTQ KSLSLSLXaa2Xaa3 wherein Xaa1 is Q, pE, or absent; Xaa2 is G or absent; Xaa3 is K or absent.

10	Lebrikizumab light chain (LC)	DIVMTQSPDSLSVSLGERATINCRASKSVDSYGN SFMHWYQQKPGQPPKLLIYLASNLESGVPDRFSG SGSGTDFTLTISSLQAEDVAVYYCQQNNEDPRTF GGGTKVEIKRTVAAPSVFIFPPSDEQLKSGTASVV CLLNNFYPREAKVQWKVDNALQSGNSQESVTEQ DSKDSTYSLSSTLTLSKADYEKHKVYACEVTHQ GLSSPVTKSFNRGEC
11	Lebrikizumab variant heavy chain variable region (VH)	Xaa1VQLQESGPGLVKPSETLSLTCTVSGFSLNAY SVNWIRQPPGKGLEWLGMIWGDGKIVYNSALKS RLTISKDSSKNQVSLKLSSVTAADTAVYYCAGDG YYPYAMDNWGQGTTVTVSS wherein Xaa1 is E, pE, or absent
12	Lebrikizumab variant light chain variable region (VL)	DIQLTQSPSSLSASVGDRVTITCRASKSVDSYGNS FMHWYQQKPGKAPKLLIYLASNLESGVPSRFSGS GSGTDFTLTISSLQPEDFATYYCQQNNEDPRTFG GGTKVEIK
13	Lebrikizumab variant heavy chain (HC)	Xaa1VQLQESGPGLVKPSETLSLTCTVSGFSLNAY SVNWIRQPPGKGLEWLGMIWGDGKIVYNSALKS RLTISKDSSKNQVSLKLSSVTAADTAVYYCAGDG YYPYAMDNWGQGTTVTVSSASTKGPSVFPLAPS SKSTSGGTAALGCLVKDYFPEPVTUSWNSGALTS GVHTFPAVLQSSGLYSLSSVVTVPSSSLGTQTYIC NVNHKPSNTKVDKKVEPKSCDKTHTCPPCPAPE AAGGPSVFLFPPKPKDTLYITREPEVTCVVVDVS HEDPEVKFNWYVDGVEVHNAKTKPREEQYNST YRVVSVLTVLHQDWLNGKEYKCKVSNKALPAPI EKTISKAKGQPREPQVYTLPPSRDELTKNQVSLTC LVKGFYPSDIAVEWESNGQPENNYKTTPPVLDSD GSFFLYSKLTVDKSRWQQGNVFSC SVMHEALHNHYTQKSLSLSXaa2Xaa3 wherein Xaa1 is E, pE, or absent; Xaa2 is P or absent; Xaa3 is G or absent
14	Lebrikizumab variant light chain (LC)	DIQLTQSPSSLSASVGDRVTITCRASKSVDSYGNS FMHWYQQKPGKAPKLLIYLASNLESGVPSRFSGS GSGTDFTLTISSLQPEDFATYYCQQNNEDPRTFG GGTKVEIKRTVAAPSVFIFPPSDEQLKSGTASVVC LLNNFYPREAKVQWKVDNALQSGNSQESVTEQD SKDSTYSLSSTLTLSKADYEKHKVYACEVTHQGL SSPVTKSFNRGEC
15	hlgGI-LALA-YTE HC	ASTKGPSVFPLAPSSKSTSGGTAALGCLVKDYFP EPVTUSWNSGALTSGVHTFPAVLQSSGLYSLSSV

	constant domain	VTVPSSSLGTQTYICNVNHKPSNTKVDKKVEPKS CDKTHTCPPCPAPEAAGGPSVFLFPPKPKDTLYIT REPEVTCVVVDVSHEDPEVKFNWYVDGVEVHN AKTKPREEQYNSTYRVVSVLTVLHQDWLNGKEY KCKVSNKALPAPIEKTISKAKGQPREPQVYTLPPS RDELTKNQVSLTCLVKGFYPSDIAVEWESNGQPE NNYKTTPPVLDSDGSFFLYSKLTVDKSRWQQGN VFSC SVMHEALHNHYTQKSLSLSXaa1Xaa2 wherein Xaa1 is P or absent; Xaa2 is G or absent
16	LC human kappa LC	RTVAAPSVFIFPPSDEQLKSGTASVVCLLNNFYPR EAKVQWKVDNALQSGNSQESVTEQDSKDSTYSL SSTLTLSKADYEKHKVYACEVTHQGLSSPVTKSF NRGEC

[0029] In some embodiments, the anti-IL-13 antibody is a lebrikizumab variant that comprises the same HCDR and LCDR sequences as lebrikizumab. In some embodiments, the anti-IL-13 antibody is a lebrikizumab variant described in WO2023245187, e.g., construct 133, 134, 136, 141. In some embodiments, the anti-IL-13 antibody is APG777. The amino acid sequences of a lebrikizumab variant are also provided in Table 1.

[0030] Other exemplary anti-IL-13 antibodies include, but not limited to, IMA-026, IMA-638 (also referred to as, anrukinzumab, QAX-576, CAS No. 910649-32-0), tralokinumab (also referred to as CAT-354, CAS No. 1044515-88-9); cendakimab (also referred to as CC-93538, RPC4046, ABT-308, CAS No. 2151032-62-9), AER-001, ABT-308 (also referred to as humanized 13C5.5 antibody). Examples of such anti-IL-13 antibodies and other inhibitors of IL-13 are disclosed, for example, in WO2008/086395, WO2006/085938, US 7,615,213, US 7,501,121, US 7,935,343, US 7,829,090, US7,947,273, WO2007/036745, WO2010/073119, WO2007/045477, and WO 2014/165771. In some embodiments, the anti-IL-13 antibody is tralokinumab. In some embodiments, the anti-IL-13 antibody is cendakimab.

[0031] The anti-IL-13 antibodies, e.g., lebrikizumab, can be formulated with suitable carriers or excipients into a pharmaceutical composition that is suitable for administration to patients. For example, the anti-IL-13 antibodies, e.g., lebrikizumab, can be formulated in a pharmaceutical composition as described in WO 2013/066866. The pharmaceutical composition can comprise 100 mg, 150 mg, 200 mg, 250 mg, 300 mg, 350 mg, 400 mg, 450 mg, or 500 mg of the anti-IL-13 antibody. In some embodiments, the pharmaceutical composition comprises 250 mg or 500 mg of the anti-IL-13 antibody. In some embodiments,

the anti-IL-13 antibody concentration in the pharmaceutical composition is between 100 mg/mL and 150 mg/mL, e.g., 125 mg/mL. The pharmaceutical composition can also comprise 5 mM - 40 mM histidine acetate buffer, pH 5.4 to 6.0. In some embodiments, the pharmaceutical composition further comprises a polyol (e.g., sugar) that has a concentration between 100 mM and 200 mM, and/or a surfactant (e.g., polysorbate 20) that has a concentration of 0.01% - 0.1%. In one embodiment, the pharmaceutical composition comprises 125 mg/mL of anti-IL-13 antibody (e.g., lebrikizumab), 20 mM histidine acetate buffer, pH 5.7, 175 mM sucrose and 0.03% polysorbate 20. In some embodiments, provided herein are pharmaceutical compositions comprising a means for inhibiting IL-13 and a pharmaceutically acceptable carrier or excipient.

[0032] In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered subcutaneously to the patient. In some embodiments, the anti-IL-13 antibody is administered at a dose of 250 mg to 500 mg. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered to the patient once every two weeks, once every four weeks, or once every eight weeks. In some embodiments, the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 250 mg once every two weeks. In some embodiments, the patient is further treated with a loading dose of 500 mg of the anti-IL-13 antibody. In some embodiments, the loading dose is administered once or twice to the patient. In some embodiments, the loading dose is administered to the patient at week 0 (baseline) and week 2. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered to the patient once every eight weeks, once every twelve weeks or once every twenty-four weeks, once every 36 weeks, or once every 52 weeks. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered to the patient once about every eight weeks, once about every twelve weeks, once about every twenty-four weeks, once about every 36 weeks, or once about every 52 weeks. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 360 mg once every eight weeks. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 360 mg once every twelve weeks. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 360 mg once every twenty-four weeks. In some embodiments, the anti-IL-13

antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 360 mg once every thirty-six weeks. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 360 mg once every fifty-two weeks. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 360 mg once about every eight weeks. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 360 mg once about every twelve weeks. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 360 mg once about every twenty-four weeks. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 360 mg once about every thirty-six weeks. In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 360 mg once about every fifty-two weeks. In some embodiments, the patient is further treated with a loading dose of 720 mg of the anti-IL-13 antibody. In some embodiments, the loading dose is administered once or twice to the patient. In some embodiments, the loading dose is administered to the patient at week 0 (baseline) and week 2.

[0033] In some embodiments, the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody is administered to the patient using a subcutaneous administration device. The subcutaneous administration device can be selected from a prefilled syringe, disposable pen injection device, microneedle device, microinfuser device, needle-free injection device, or autoinjector device. Various subcutaneous administration devices, including autoinjector devices, are known in the art and are commercially available. Exemplary devices include, but are not limited to, prefilled syringes (such as BD HYPAK SCF®, READYFILL™, and STERIFILL SCF™ from Becton Dickinson; CLEARSHOT™ copolymer prefilled syringes from Baxter; and Daikyo Seiko CRYSTAL ZENITH® prefilled syringes available from West Pharmaceutical Services); disposable pen injection devices such as BD Pen from Becton Dickinson; ultra-sharp and microneedle devices (such as INJECT-EASE™ and microinfuser devices from Becton Dickinson; and H-PATCH™ available from Valeritas) as well as needle-free injection devices (such as BIOJECTOR® and IJECT® available from Bioject; and SOF-SERTER® and patch devices available from Medtronic). In

some embodiments, the subcutaneous administration device is an autoinjector device described in WO 2008/112472, WO 2011/109205, WO 2014/062488, and/or WO 2016/089864.

[0034] In some embodiments, the patient can be treated with the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody for a period of 16 to 52 weeks, e.g., 16 weeks, 18 weeks, 20 weeks, 22 weeks, 24 weeks, 26 weeks, 28 weeks, 30 weeks, 32 weeks, 34 weeks, 36 weeks, 38 weeks, 40 weeks, 42 weeks, 44 weeks, 46 weeks, 48 weeks, 50 weeks, 52 weeks.

[0035] In some embodiments, the patient is treated with the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody for a treatment period (e.g., an induction period or the first period) of at least 16 weeks. In such embodiments, the anti-IL-13 antibody is administered at a loading dose of 500 mg at baseline (week 0) and week 2, followed by a subsequent dose of 250 mg once every two weeks for at least 14 weeks. In some embodiments, the patient is treated with the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody for a treatment period (e.g., an induction period or the first period) of 16 weeks. In such embodiments, the anti-IL-13 antibody is administered at a loading dose of 500 mg at baseline (week 0) and week 2, followed by a subsequent dose of 250 mg once every two weeks for 14 weeks.

[0036] Before, during and after the treatment of the anti-IL-13 antibody, the patient can be assessed for one or more characteristics, which determine certain signs, symptoms, features, or parameters that have been associated with AD and that can be quantitatively or qualitatively assessed. Exemplary characteristics include, but are not limited to, Eczema Area and Severity Index (EASI), Investigator Global Assessment (IGA), body surface area (BSA), Face-Investigator's Global Assessment (F-IGA), Post-inflammatory Dyschromia Scale (PDS) or Post-inflammatory Dyschromia Clinical Assessment (PDCA-DERM^{TM}), Patient-Oriented SCORing Atopic Dermatitis (PO-SCORAD), Pruritus Numerical Rating Scale (NRS), Skin pain NRS score, Sleep-Loss Scale, Patient-Oriented Eczema Measure (POEM), Dermatology Life Quality Index (DLQI) or Children Dermatology Life Quality Index (cDLQI), Atopic Dermatitis Control Tool (ADCT), Work Productivity and Activity Impairment Questionnaire–Atopic Dermatitis (WPAI-AD), Participant-Reported Satisfaction Question, Fitzpatrick Skin Phototype Assessment or Fitzpatrick Scale. The characteristics can be measured at baseline and at one or more time points after administration of the anti-IL-13 antibody or pharmaceutical composition comprising the anti-IL-13 antibody. The difference between the value of the characteristics at a particular time point following

initiation of treatment and the value of the characteristics at baseline is used to establish whether there has been an improvement (e.g., a reduction) in the characteristics.

[0037] In some embodiments, the methods and therapeutic uses described herein further comprise determining one or more of the following characteristics of the patient at baseline and during and after the induction period (or the first period): the EASI score; the IGA score; the F-IGA score; the percentage of BSA affected by AD; PDS or PDCA-DERM^{TM}; PO-SCORAD; pruritus NRS score; Sleep-Loss score; Skin Pain NRS; POEM; DLQI or cDLQI score; ADCT; WPAI-AD; Fitzpatrick score.

[0038] In some embodiments, the patient's EASI score is determined after the treatment period (e.g., the induction period or the first period). In some embodiments, the patient's EASI score determined after the treatment period (e.g., the induction period or the first period) is reduced by 50% or greater compared to the patient's EASI score at the baseline, which means the patient has achieved "EASI-50". In some embodiments, the patient's EASI score determined after the treatment period (e.g., the induction period or the first period) is reduced by 75% or greater compared to the patient's EASI score at the baseline, which means the patient has achieved "EASI-75". In some embodiments, the patient's EASI score determined after the treatment period (e.g., the induction period or the first period) is reduced by 90% or greater compared to the patient's EASI score at the baseline, which means the patient has achieved "EASI-90". A patient is considered a responder to the anti-IL13 antibody when the patient reached EASI-75 after the induction period (or the first period).

[0039] In some embodiments, the patient's IGA score is determined after the treatment period (e.g., the induction period or the first period). A patient is considered a responder to the anti-IL13 antibody when the patient's IGA score is 0 or 1 after the treatment period (e.g., the induction period or the first period). In some embodiments, a patient is considered a responder to the anti-IL13 antibody when the patient's IGA score is 0 or 1 after the treatment period (e.g., the induction period or the first period), and the patient's IGA score after the treatment period (e.g., the induction period or the first period) is reduced by 2 points or greater compared to the patient's IGA score determined at the baseline.

[0040] After completion of the induction period (or the first period), the patient can enter a maintenance period (or a second period). During the maintenance period (or the second period), the patient is further treated with the anti-IL13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody. The dosing regimen for the maintenance period (or the second period) can be selected based on the patient's assessment and response to the IL-13 antibody after the induction period (or the first period), e.g., the patient's IGA or

EASI score after the induction period (or the first period), and/or the patient's own characteristics, e.g., weight, age, race.

[0041] The maintenance period (or the second period) can be 8 to 36 weeks (e.g., 8 weeks, 12 weeks, 16 weeks, 20 weeks, 24 weeks, 28 weeks, 32 weeks, 36 weeks). In some embodiments, the maintenance period (or the second period) is 8 weeks. In some embodiments, the maintenance period (or the second period) is 36 weeks.

[0042] In some embodiments, the methods and therapeutic uses described herein further comprise determining one or more of the following characteristics of the patient during and after the maintenance period (or the second period): the EASI score; the IGA score; the F-IGA score; the percentage of BSA affected by AD; PDS or PDCA-DERM™; PO-SCORAD; pruritus NRS score; Sleep-Loss score; Skin Pain NRS; POEM; DLQI or cDLQI score; ADCT; WPAI-AD; Fitzpatrick score.

[0043] The "Investigator Global Assessment" or "IGA" is an investigator-reported, single-item scale that rates the severity of the participant's AD (Simpson E, et al. *J Am Acad Dermatol.* 2020;83(3):839-846). The IGA is composed of a 5-point scale ranging from 0 (clear) to 4 (severe), and a score is selected using descriptors that best describe the overall appearance of the lesions at a given time point (see Table 2).

Table 2. Investigator Global Assessment (IGA)

Score	Grade	Definition
0	Clear	Minor, residual discoloration; no erythema or induration/papulation; no oozing/crusting; no edema.
1	Almost clear	Trace, faint pink erythema with barely perceptible induration/papulation and no oozing/crusting; no edema.
2	Mild	Faint-pink erythema with papulation and edema perceptible upon palpation and no oozing/crusting; minimal induration.
3	Moderate	Pink-red erythema with definite edema of skin papules and plaques; there may be some oozing/crusting; palpable induration.
4	Severe	Deep/bright red erythema with significant swelling and obvious raised borders of papules and plaques with oozing/crusting; significant induration.

[0044] The "Eczema Area and Severity Index" or "EASI" is a measure used in clinical settings to assess the severity and extent of AD (Hanifin et al., *Exp Dermatol.* 2001; 10:11-18). The EASI is an investigator-reported, 20-item scale that evaluates two dimensions of AD: extent of disease at four body regions (head or neck, trunk, upper and lower extremities)

and four clinical signs (erythema, induration or papulation, excoriation, and lichenification). The clinical signs are assessed for severity on a scale of 0 (absent) to 3 (severe). The scores are added up for each of the four body regions. The assigned percentages of BSA for each section of the body are 10% for head or neck, 20% for upper extremities, 30% for trunk, and 40% for lower extremities, respectively. Each subtotal score is multiplied by the BSA represented by that region. In addition, an area score of 0 to 6 is assigned for each body region, depending on the percentage of AD-affected skin in that area: 0 (none), 1 (1% to 9%), 2 (10% to 29%), 3 (30% to 49%), 4 (50% to 69%), 5 (70% to 89%), or 6 (90% to 100%). Each of the body area scores is multiplied by the area affected. The resulting EASI ranges from 0 to 72 points, with the highest score indicating worse severity of AD (Hanifin et al. 2001). The recall period of this scale is present time.

[0045] The body surface area (BSA) is an investigator-reported assessment, which estimates the extent of disease or skin involvement of a participant's AD. BSA is expressed as a percentage of total body surface and will be reported by body location. The recall period of this assessment is present time.

[0046] The Face-Investigator's Global Assessment (F-IGA) is an investigator-administered, single-item scale that rates the severity of the participant's AD on the face. The F-IGA is composed of a 5-point scale ranging from 0 (clear) to 4 (severe), and a score is selected using descriptors that best describe the overall appearance of the facial lesions at a given time point.

[0047] The Post-inflammatory Dyschromia Scale (PDS) or the Post-inflammatory Dyschromia Clinical Assessment (PDCA-DERM^{TM}) is an investigator assessment of cutaneous post-inflammatory hyperpigmentation and/or hypopigmentation. This is a measure of post-inflammatory cutaneous change; as such, pigmented actively inflamed AD lesions should not be evaluated with this scale. Areas of normal pigmentation, with no post-inflammatory hyper- or hypo- pigmentation, will receive a score of 0. Areas of post-inflammatory hyperpigmentation, as compared to adjacent normal skin tone (0), should receive a score of 1 (mild/barely perceptible hyperpigmentation), 2 (moderate/clearly perceptible hyperpigmentation) or 3 (severe/prominent hyperpigmentation). Areas of post-inflammatory hypopigmentation, as compared to adjacent normal skin tone (0), should receive a score of -1 (mild/barely perceptible hypopigmentation), -2 (moderate/clearly perceptible hypopigmentation) or -3 (severe/prominent hypopigmentation). See Figure 2.

[0048] The Patient-Oriented SCORing Atopic Dermatitis (PO-SCORAD) is a patient-reported scale used to evaluate the condition of a patient's eczema over the last 3 days in both

adults and adolescent participants. Questions cover the spread of the eczema over the body's surface, the severity of dry skin outside of eczema-affected areas, the symptom intensity on eczema-affected areas (including redness/darkness, swelling, oozing/scabs, scratch marks, and thickening of the skin), the intensity of eczema-related itching, and trouble sleeping. There are 9 items, and response options vary across each item. For the questions about symptom intensity, an accompanying user guide shows visual and descriptive anchors for each response option. The PO-SCORAD total score ranges from 0 to 104, with higher scores indicating more severe AD (Stalder et al., *Allergy*. 2011;66(8):1114-21; Faye et al., *J Eur Acad Dermatol Venereol*. 2020;34(4):795-799). The recall period of this scale is over the last 3 days.

[0049] The Pruritus Numeric Rating Scale (NRS) is a participant-reported, single-item, daily, 11-point scale. The Pruritus NRS is used by participants to rate their worst itch severity over the past 24 hours with 0 indicating "No itch" and 10 indicating "Worst itch imaginable." Assessments will be recorded daily by the participant. The minimal clinically important change is 3 points. (Yosipovitch et al., *J Dermatol*. 2019; 181(4):761-769; Yosipovitch et al., *Revolutionizing Atopic Dermatitis* – December 2021).

[0050] The Sleep-Loss Scale is a participant-reported, single-item, daily scale that measures the extent of sleep-loss due to interference of itch over the last night. The Sleep-Loss Scale is rated based on a 5-point Likert scale (0 [not at all] to 4 [unable to sleep at all]). Assessment will be recorded daily by the participant. The minimal clinically important change is 3 points. (Yosipovitch et al., 2021).

[0051] The Skin Pain NRS is a participant-reported, 11-point horizontal scale anchored at 0 and 10, with 0 representing "no pain" and 10 representing "worst pain imaginable." Overall severity of a participant's skin pain is indicated by selecting the number that best describes the worst level of skin pain in the past 24 hours. Assessment will be recorded daily by the participant (Newton L, et al. *J Patient Rep Outcomes*. 2019 Jul 16; 3:42; Silverberg et al. 2021).

[0052] The Patient-Oriented Eczema Measure (POEM) is a simple, participant-reported, 7-item scale that assesses disease severity in children and adults. Participants respond to questions about the frequency of 7 symptoms (itching, sleep disturbance, bleeding, weeping/oozing, cracking, flaking, and dryness/roughness) over the past week. Response categories include "No days," "1-2 days," "3-4 days," "5-6 days," and "Every day" with corresponding scores of 0, 1, 2, 3, and 4, respectively. Scores range from 0 to 28, with higher

total scores indicating greater disease severity (Charman et al., *Arch Dermatol.* 2004;140(12):1513-1519).

[0053] The Dermatology Life Quality Index (DLQI) is a simple, participant-reported, 10-item, QoL questionnaire in adults that covers 6 domains (symptoms and feelings, daily activities, leisure, work and school, personal relationships, and treatment). The recall period of this scale is over the "last week." Response categories include "not at all,", "a little", "a lot," and "very much," with corresponding scores of 0, 1, 2, and 3, respectively, and unanswered (or "not relevant") responses scored as 0. Scores range from 0 to 30 with higher scores indicating greater impairment of QoL. A DLQI total score of 0 to 1 is considered as having no effect on a participant's health-related QoL (Hongbo et al., *J Invest Dermatol.* 2005;125(4):659-664), and a 4-point change from baseline is considered as the minimal clinically important difference threshold (Khilji et al., *Br J Dermatol.* 2002;147(suppl 62):25-54; Basra et al., *Dermatology.* 2015;230(1):27 33).

[0054] Participants <16 years at baseline will complete the children's DLQI (cDLQI) and should continue to complete the cDLQI for the duration of the study (Lewis-Jones MS, Finlay AY. *British Journal of Dermatology*, 1995; 132:942-949).

[0055] The Atopic Dermatitis Control Tool (ADCT) is a patient-reported, simple, brief tool for adults and adolescents that evaluates 6 symptoms and effects associated with AD over the past week: overall severity of symptoms, days with intense episodes of itching, intensity of bother, problem with sleep, impact on daily activities, and impact on mood or emotions. Each of the 6 ADCT items has a score range from 0 (no problem) to 4 (worst), rating the severity of each concept; the total score ranges from 0 to 24, which is the summation of the responses to all the items. A score of ≥7 points was derived as the threshold to identify patients "not in control". The threshold for meaningful within-person change was estimated to be 5 points (Simpson et al., *BMC Dermatol.* 2019;19(1):15; Pariser et al., *Curr Med Res Opin.* 2020;36(3):367-376).

[0056] The Work Productivity and Activity Impairment Questionnaire–Atopic Dermatitis (WPAI-AD) is a participant-reported, 6-item questionnaire that records impairment due to AD over the past 7 days in adolescents and adults. The WPAI-AD consists of 6 items grouped into 4 domains: absenteeism (work time missed), presenteeism (impairment at work/reduced on the job effectiveness), work productivity loss (overall work impairment/absenteeism plus presenteeism), and activity impairment. Absenteeism, presentism, and work productivity will only be reported by those who are currently employed (working for pay) at the time of the scale completion. The recall period for the scale is over

the past 7 days. Scores are calculated as impairment percentages (Reilly et al., *Pharmacoeconomics*. 1993;4(5):353 365), with higher scores indicating greater impairment and less productivity.

[0057] For Participant-Reported Satisfaction Question, the question asks "How satisfied are you with this treatment's ability to treat your skin condition?" The response options range from 1 (not satisfied) to 5 (completely satisfied).

[0058] The Fitzpatrick Skin Phototype Assessment uses Fitzpatrick Scale, which is a clinician-rated scale and is based on a participant's cutaneous reaction to sun exposure and baseline skin pigmentation. Fitzpatrick skin phototypes range from I to VI, with a score of I indicating "always burns, does not tan, white skin tone", and a score of VI indicating "never burns, tans very easily, skin color black" (High et al., *Dermatology (3rd ed.)*. China: Elsevier;2012:8). Investigators will follow the descriptive terms included in this protocol when recording the Fitzpatrick skin phototype. The sun sensitivity measure will represent the Fitzpatrick Scale score for each participant. The recall period for this scale is the present time.

[0059] In some embodiments, the methods and uses described herein further comprise administrating one or more topical corticosteroids to the patient. In some embodiments, the anti-IL-13 antibody is used in simultaneous, separate, or sequential combination with one or more topical corticosteroids. Exemplary topical corticosteroids include, but are not limited to, triamcinolone acetonide, hydrocortisone, and a combination of triamcinolone acetonide and hydrocortisone. Triamcinolone acetonide is typically formulated at a concentration of 0.1% in a cream, and hydrocortisone is typically formulated at a concentration of 1% or 2.5% in a cream. Certain topical corticosteroids are considered very high potency such as, for example, betamethasone dipropionate, clobetasol propionate, diflorasone diacetate, fluocinonide, and halobetasol propionate. Certain topical corticosteroids are considered high potency such as, for example, amcinonide, desoximetasone, halcinonide, and triamcinolone acetonide. Certain topical corticosteroids are considered medium potency, such as, for example, betamethasone valerate, clocortolone pivalate, fluocinolone acetonide, flurandrenolide, fluocinonide, fluticasone propionate, hydrocortisone butyrate, hydrocortisone valerate, mometasone furoate, and prednicarbate. Certain topical corticosteroids are considered low potency, such as, for example, alclometasone dipropionate, desonide, and hydrocortisone. TCS may be applied to affected areas once daily, twice daily, three times per day, or as needed. In some embodiments, the patient is inadequately controlled on topical corticosteroids. In some embodiments, the topical corticosteroid is triamcinolone acetonide, hydrocortisone, or a

combination of triamcinolone acetonide and hydrocortisone. In some embodiments, the topical corticosteroids are administered concomitantly or sequentially with the anti-IL-13 antibody. In some embodiments, the topical corticosteroids are administered concomitantly with the anti-IL-13 antibody.

[0060] As used herein, the term "a," "an," "the" and similar terms used in the context of the present disclosure (especially in the context of the claims) are to be construed to cover both the singular and plural unless otherwise indicated herein or clearly contradicted by the context.

[0061] The term "about" as used herein, means in reasonable vicinity of the stated numerical value, such as plus or minus 10% of the stated numerical value.

[0062] The term "antibody," as used herein, refers to an immunoglobulin molecule that binds an antigen. Embodiments of an antibody include a monoclonal antibody, polyclonal antibody, human antibody, humanized antibody, chimeric antibody, or conjugated antibody. The antibodies can be of any class (e.g., IgG, IgE, IgM, IgD, IgA) and any subclass (e.g., IgG1, IgG2, IgG3, IgG4).

[0063] An exemplary antibody is an immunoglobulin G (IgG) type antibody comprised of four polypeptide chains: two heavy chains (HC) and two light chains (LC) that are cross-linked via inter-chain disulfide bonds. The amino-terminal portion of each of the four polypeptide chains includes a variable region of about 100-125 or more amino acids primarily responsible for antigen recognition. The carboxyl-terminal portion of each of the four polypeptide chains contains a constant region primarily responsible for effector function. Each heavy chain is comprised of a heavy chain variable region (VH) and a heavy chain constant region. Each light chain is comprised of a light chain variable region (VL) and a light chain constant region. The IgG isotype may be further divided into subclasses (e.g., IgG1, IgG2, IgG3, and IgG4).

[0064] The VH and VL regions can be further subdivided into regions of hyper-variability, termed complementarity determining regions (CDRs), interspersed with regions that are more conserved, termed framework regions (FR). The CDRs are exposed on the surface of the protein and are important regions of the antibody for antigen binding specificity. Each VH and VL is composed of three CDRs and four FRs, arranged from amino-terminus to carboxyl-terminus in the following order: FR1, CDR1, FR2, CDR2, FR3, CDR3, FR4. Herein, the three CDRs of the heavy chain are referred to as "HCDR1, HCDR2, and HCDR3" and the three CDRs of the light chain are referred to as "LCDR1, LCDR2 and LCDR3". The CDRs contain most of the residues that form specific interactions with the

antigen. Assignment of amino acid residues to the CDRs may be done according to the well-known schemes, including those described in Kabat (Kabat et al., "Sequences of Proteins of Immunological Interest," National Institutes of Health, Bethesda, Md. (1991)), Chothia (Chothia et al., "Canonical structures for the hypervariable regions of immunoglobulins", Journal of Molecular Biology, 196, 901-917 (1987); Al-Lazikani et al., "Standard conformations for the canonical structures of immunoglobulins", Journal of Molecular Biology, 273, 927-948 (1997)), North (North et al., "A New Clustering of Antibody CDR Loop Conformations", Journal of Molecular Biology, 406, 228-256 (2011)), or IMGT (the international ImMunoGeneTics database available on at www.imgt.org; see Lefranc et al., Nucleic Acids Res. 1999; 27:209-212).

[0065] Exemplary embodiments of antibodies of the present disclosure also include antibody fragments or antigen-binding fragments, which comprise at least a portion of an antibody retaining the ability to specifically interact with an antigen such as Fab, Fab', $F(ab')_2$, Fv fragments, scFv, scFab, disulfide-linked Fvs (sdFv), a Fd fragment and linear antibodies.

[0066] The term "baseline", as used herein, means prior to or at the time of administration of the first dose (week 0) of the anti-IL-13 antibody or a pharmaceutical composition comprising the anti-IL-13 antibody.

[0067] The terms "bind" and "binds", as used herein are intended to mean, unless indicated otherwise, the ability of a protein or molecule to form a chemical bond or attractive interaction with another protein or molecule, which results in proximity of the two proteins or molecules as determined by common methods known in the art.

[0068] The term "high affinity" as used herein refers to the strength of binding of an antibody to human IL-13 with an equilibrium dissociation constant (K_D) of less than about 10^{-8} M, e.g., from 10^{-15} M to 10^{-8} M, or from 10^{-12} M to 10^{-9} M.

[0069] The term "human IL-13" refers to human interleukin 13 (also known as P600), an immunoregulatory cytokine produced primarily by activated Th2 cells. There are two known human IL-13 isoforms: isoform a and isoform b. The term "human IL-13" as used herein refers collectively to all human IL-13 isoforms. The amino acid sequence for human IL-13 isoform a can be found at NCBI Accession No. NP_002179.2. The amino acid sequence for human IL-13 isoform b can be found at NCBI Accession No. NP_001341922.1.

[0070] The term "inadequate response" as used herein refers to inability to achieve good disease control (e.g., not able to achieve IGA $\leq$2 or EASI 75 for AD) after use of the

treatment for the duration recommended by the product prescribing information, or flare of AD occurs while on the treatment.

[0071] The term "intolerance" or "intolerant" as used herein refers to unacceptable toxicity (e.g., elevated creatinine, elevated liver function tests, uncontrolled hypertension, paranesthesia, headache, nausea, hypertrichosis), or requirement for a drug at doses or duration beyond those specified in the prescribing information.

[0072] The term "patient", as used herein, refers to a human patient.

[0073] The term "topical corticosteroid" or "TCS", as used herein includes Group I, Group II, Group III and Group IV topical corticosteroids. According to the Anatomical Therapeutic Chemical (ATC) Classification System of World Health Organization, the corticosteroids are classified as weak (Group I), moderately potent (Group II) and potent (Group III) and very potent (Group IV), based on their activity as compared to hydrocortisone. Group IV TCS (very potent) are up to 600 times as potent as hydrocortisone and include clobetasol and halcinonide. Group III TCS (potent) are 50 to 100 times as potent as hydrocortisone and include, but are not limited to, betamethasone valerate, betamethasone dipropionate, diflucortolone valerate, hydrocortisone-17-butyrate, mometasone furoate, and methylprednisolone aceponate. Group II TCS (moderately potent) are 2 to 25 times as potent as hydrocortisone and include, but are not limited to, clobetasone butyrate, and triamcinolone acetonide. Group I TCS (weak or mild) includes hydrocortisone, prednisolone, and methylprednisolone.

[0074] The term "topical calcineurin inhibitor" or "TCI", as used herein, includes pimecrolimus, tacrolimus, and other inhibitors that suppress calcineurin activities and can be topically applied to a patient's skin.

[0075] As used herein, "treat", "treatment" or "treating" refers to all processes wherein there may be a slowing, controlling, delaying, or stopping of the progression of the disorders or disease disclosed herein, or ameliorating disorder or disease symptoms, but does not necessarily indicate a total elimination of all disorder or disease symptoms. Treatment includes administration of a protein or nucleic acid or vector or composition for treatment of a disease or condition in a patient, particularly in a human.

EXAMPLES

Example 1. A Study to Assess the Safety and Efficacy of Lebrikizumab in Adult and Adolescent Participants with Moderate-to-Severe Atopic Dermatitis and Skin of Color.

Patient Population

[0076] **Inclusion Criteria**: Participants are eligible to be included in the study only if all of the following criteria apply:

1. Participants must be ≥12 years of age inclusive, at the time of signing the informed consent or assent.
2. Participants who are Fitzpatrick phototype IV-VI.
3. Participants who are self-reported race other than White, including but not limited to persons who self-identify as Black or African American, American Indian or Alaska Native, Asian, Native Hawaiian, or Other Pacific Islander.
4. Have chronic AD (according to American Academy of Dermatology Consensus Criteria for Chronic Atopic Dermatitis) that has been present for ≥1 year before screening visit.
5. Have Eczema Area and Severity Index (EASI) score ≥16 at the baseline visit.
6. Have Investigator's Global Assessment (IGA) score ≥3 (scale of 0 to 4) at the baseline visit.
7. Have ≥10% body surface area (BSA) of AD involvement at the baseline visit.
8. Have a history of inadequate response to treatment with topical medications; or determination that topical treatments are otherwise medically inadvisable.
9. For adolescent participants, body weight ≥40 kg at baseline.
10. Male and/or female
    a. No male contraception is required except in compliance with specific local government study requirements.
    b. Female participants of child-bearing potential must agree to remain abstinent (refrain from heterosexual intercourse) or use a highly effective contraceptive method during the treatment period and for at least 18 weeks after the last dose of study drug. Women not of child-bearing potential (WNOCBP) may participate without any contraception requirements.
11. Informed consent/assent
    a. For adult participants: capable of giving signed informed consent.

b. For adolescent participants: a parent or legal guardian must be able to read, understand, and give documented informed consent for a child to participate in this study

12. Are willing and able to comply with all clinic visits and study-related procedures an questionnaires.

[0077] **Exclusion Criteria**: Participants are excluded from the study if any of the following criteria apply:

1. History of human immunodeficiency virus (HIV) infection or positive HIV serology at screening.
2. Have a current infection or chronic infection with hepatitis B virus (HBV) at screening, that is, positive for hepatitis B surface antigen (HBsAg) and/or polymerase chain reaction positive for HBV DNA.
3. Have a current infection with hepatitis C virus (HCV) at screening, that is, positive for HCV RNA.
4. Have an uncontrolled chronic disease that might require multiple intermittent uses of oral corticosteroids at screening.
5. Have uncontrolled asthma that
    a. might require bursts of oral or systemic corticosteroids, or
    b. required the following due to ≥1 exacerbations within 12 months before baseline
        i. systemic (oral and/or parenteral) corticosteroid treatment, or
        ii. hospitalization for >24 hours.
6. Have known liver cirrhosis and/or chronic hepatitis of any etiology.
7. History of malignancy, including mycosis fungoides or cutaneous T-cell lymphoma, within 5 years before the screening, except completely treated in situ carcinoma of the cervix of completely treated and resolved non-metastatic squamous or basal cell carcinoma of the skin with no evidence of recurrence in the past 12 weeks.
8. Are diagnosed with active endoparasitic infections or at high risk of these infections.
9. Have a known or suspected history of immunosuppression, including history of invasive opportunistic infections (e.g., tuberculosis, histoplasmosis, listeriosis, coccidioidomycosis, pneumocystosis, and aspergillosis) despite infection resolution; or unusually frequent, recurrent, or prolonged infections, per the investigator's judgment.
10. Have presence of skin comorbidities that may interfere with study assessments.

11. Have a severe concomitant illness(es) that in the investigator's judgment would adversely affect the patient's participation in the study.
12. Have any other medical or psychological condition that in the opinion of the investigator may suggest a new and/or insufficiently understood disease, may present an unreasonable risk to the study participant because of his/her participation in this clinical trial, may make patient's participation unreliable, or may interfere with study assessments.
13. Have had any of the following types of infection within 3 months of screening or develop any of these infections during screening:
    a. Serious (requiring hospitalization, and/or intravenous or equivalent oral antibiotic treatment)
    b. Opportunistic
    c. Chronic (duration of symptoms, signs, and/or treatment of 6 weeks or longer)
    d. Recurring (including, but not limited to recurring cellulitis, chronic osteomyelitis).
14. Have an active or acute infection requiring treatment with systemic antibiotics, antivirals, antiparasitics, antiprotozoals, or antifungals within 2 weeks before the baseline visit (baseline; Visit 2), or superficial skin infections within 1 week before the baseline visit.
15. Prior treatment with tralokinumab.
16. Prior treatment with dupilumab.
17. Treatment with topical agents: corticosteroids, calcineurin inhibitors, JAK inhibitors, or phosphodiesterase-4 inhibitors, such as crisaborole within 2 weeks prior to baseline visit.
18. Treatment with any of the following agents within 4 weeks prior to the baseline visit:
    a. systemic immunosuppressive or immunomodulating drugs (e.g., systemic corticosteroids, cyclosporine, mycophenolate mofetil, IFN-gamma, azathioprine, methotrexate, and other immunosuppressants); small molecules (JAK inhibitors)
    b. small molecules (e.g. JAK inhibitors)
    c. phototherapy and photochemotherapy for AD.
19. Have regularly used (more than 2 visits per week) a tanning booth or parlor within 4 weeks of the screening visit.
20. Are currently receiving build-up dosing of allergen immunotherapy (allergy shots).

21. Treatment with B-cell-depleting biologics, including rituximab, within 6 months prior to the baseline visit.
22. Use of prescription moisturizers within 7 days of the baseline visit.
23. Have received any live attenuated vaccine within less than 4 weeks of the baseline visit, or intend to receive a live attenuated vaccine during the study, or within 4 weeks after receiving the last dose of study intervention.
24. Have received a Bacillus Calmette-Guerin vaccination or treatment within less than 4 weeks before the baseline visit, or intend to receive Bacillus Calmette-Guerin vaccination or treatment during the study.
25. Use of cannabis or cannabinoids for the treatment of pruritus, pain, and/or AD.
26. In the opinion of the investigator, have any clinically significant laboratory results from the chemistry or hematology tests obtained at the screening visit (Visit 1).
27. Are currently enrolled in any other clinical study involving an investigational product or any other type of medical research judged not to be scientifically or medically compatible with this study.
28. Treatment with an investigational drug within 8 weeks or within 5 half-lives (if known), whichever is longer prior to the baseline visit.
29. Have received a dose of lebrikizumab in any prior lebrikizumab clinical study.
30. Are Lilly employees, or are employees of any third-party involved in study who require exclusion of their employees.
31. Are investigator site personnel directly affiliated with this study and/or their immediate families. Immediate family is defined as a spouse, parent, child, or sibling, whether biological or legally adopted.
32. Are pregnant or breastfeeding or are planning to become pregnant or to breastfeed during the study.
33. Have a known hypersensitivity to any component of lebrikizumab.
34. Participant or caregiver who is unwilling to administer SC injections of study medication.

Study Drug:

[0078] Pharmaceutical compositions containing 125 mg/mL lebrikizumab or placebo are supplied as sterile pre-filled syringes with a pre-assembled needle safety device (PFS-NSD) for subcutaneous administration to the patients. Lebrikizumab sequences are provided in

Table 1. The placebo solution is identical in appearance and volume to the active solution except that it does not contain lebrikizumab.

Study Design:

[0079] The study design is shown in Figure 1.

[0080] This trial has three study periods:

- Screening (≤30 days prior to baseline);
- Treatment (24 weeks);
- Safety follow-up (at Week 34 or approximately 12 weeks after last treatment).

[0081] During the 24-week Treatment Period, approximately 95 participants will receive treatment:

- 500 mg subcutaneous (SC) once every 2 weeks (Q2W) loading dose at baseline and Week 2, followed by 250 mg SC Q2W until Week 16.
- Responders, defined as achieving IGA (0,1) or EASI-75 at Week 16, will receive 250 mg SC once every 4 weeks (Q4W).
- Inadequate responders at Week 16 will continue to receive 250 mg SC Q2W.

Objectives and Endpoints

[0082] The objectives and endpoints of this study are shown in Table 3.

Table 3. Objectives and Endpoints

Objectives	Endpoints
Primary	
• To assess the effect of lebrikizumab 250 mg Q2W in reducing the signs and symptoms of AD in participants with moderate-to-severe AD and skin of color	• Percentage of participants achieving EASI-75 (≥75% reduction from baseline in EASI) at Week 16
Secondary	
• To evaluate the effect of treatment with lebrikizumab in the signs and symptoms of AD and health outcome measures, in participants with moderate-to-severe AD and skin of color	• Percentage of participants achieving EASI-75 at Week 24 • Percentage of participants with an IGA score of 0 or 1 and a reduction ≥2 points from baseline to Weeks 16 and 24 • Percentage change in EASI total from baseline to Weeks 16 and 24 • Change in EASI total from baseline to Weeks 16 and 24 • Percentage of participants achieving EASI 90 at Weeks 16 and 24

	• Percentage of participants with a Pruritus NRS of ≥4 points at baseline who achieve a 4-point reduction at Weeks 16 and 24 • Percentage of participants with a Pruritus NRS ≥3 points at baseline who achieve at least 3-point reduction at Weeks 16 and 24 • Percentage change in Pruritus NRS score from baseline to Weeks 16 and 24 • Percentage of participants with a Sleep-Loss Scale score of ≥2 points at baseline who achieve a 2-point reduction at Weeks 16 and 24 • Percentage change in Sleep-Loss Scale score from baseline to Weeks 16 and 24 • Percentage of participants with a Skin Pain NRS of ≥4 points at baseline who achieve a 4-point reduction from baseline to Weeks 16 and 24 • Change in POEM from baseline to Weeks 16 and 24 • Change in DLQI from baseline to Weeks 16 and 24 for participants ≥16 years of age at baseline • Change in cDLQI from baseline to Weeks 16 and 24 for participants <16 years of age at baseline • Percentage of participants with a DLQI of ≥4 points at baseline who achieve a ≥4-point improvement in DLQI at Weeks 16 and 24 for participants ≥16 years of age at baseline
Exploratory	

• To evaluate the efficacy and health outcome measures of lebrikizumab from baseline by visit in participants with skin of color and moderate-to-severe AD	• Percentage change in PO-SCORAD from baseline by visit
	• Change in Post-inflammatory Dyschromia Scale or PDCA-DERM™ from baseline by visit
	• Percentage change in ADCT from baseline by visit
	• Using WPAI-AD by visit, change in baseline in
	  ○ absenteeism in those who are currently employed (working for pay)
	  ○ presenteeism in those who are currently employed (working for pay)

	○ overall work impairment in those who are currently employed (working for pay), and ○ impairment in activities (all participants) • Percentage of participants with an F-IGA score of 0 or 1 and a reduction ≥2 points from baseline by visit • Lichenification change from baseline (calculated from EASI) • Participant satisfaction change from baseline (1 question) • Percentage of participants achieving EASI-75 by visit • Percentage of participants with an IGA score of 0 or 1 and a reduction ≥2 points from baseline by visit • Percentage change in EASI total by visit • Change in EASI total by visit • Percentage of participants achieving EASI-90 by visit • Percentage of participants with a Pruritus NRS of ≥4 points at baseline who achieve a 4-point reduction by visit • Percentage of participants with a Pruritus NRS of ≥3 points at baseline who achieve a 3-point reduction by visit • Percentage change in Pruritus NRS score by visit • Percentage of participants with a Sleep-Loss Scale score of ≥2 points at baseline who achieve a 2-point reduction by visit • Percentage change in Sleep-Loss Scale score from baseline by visit • Percentage of participants with a Skin Pain NRS of ≥4 points at baseline who achieve a 4-point reduction from baseline by visit • Change from baseline in POEM by visit
•	•

[0083] Statistical analyses are performed for the primary, secondary and exploratory endpoints.

Results

[0084] Results are shown in Figures 3-6. Figure 3A shows baseline demographics of enrolled patients. Figure 3B shows baseline disease characteristics of enrolled patients. Figure 4 shows 39% of patients treated with lebrikizumab achieved IGA (0,1) with ≥2-point improvement at Week 16. Figure 5 shows 68% of patients treated with lebrikizumab achieved EASI-75 at Week 16. Figure 6 shows the mean pruritus NRS percentage change from baseline was -54% at Week 16.

[0085] Figure 7A shows the baseline PDCA-DERM™ scores. For patients with multiple hypopigmented or hyperpigmented lesions at baseline, only the lesion with the most severe score was included in the analysis for each lesion type. In the event of a tie, the change from baseline representing a smaller improvement or worsening in condition was included. Figure 7B shows PDCA-DERM™ changes from baseline to Week 16 for hyperpigmented lesions. 12 out of 21 (57.1%) patients showed an improvement in hyperpigmentation of skin, and 6 out of 21 (28.6%) patients improved to normal skin tone. Figure 7C shows PDCA-DERM™ changes from baseline to Week 16 for hypopigmented lesions. 1 out of 5 (20%) patients showed an improvement in hypopigmentation of skin.

[0086] These data show lebrikizumab treatment improved AD symptoms in patients with skin of color after 16 weeks of treatment. There were no serious adverse events. PDCA-DERM™ scale showed lebrikizumab treatment resulted in improvement in hyperpigmentation or hypopigmentation of skin in some patients.

CLAIMS

1. A method of treating post-inflammatory hyperpigmentation or hypopigmentation of skin in a patient in need thereof, the method comprising administering to the patient a therapeutically effective amount of an anti-IL-13 antibody, wherein the anti-IL-13 antibody comprises a heavy chain variable region (VH) and a light chain variable region (VL), wherein the VH comprises a HCDR1 comprising SEQ ID NO: 1, a HCDR2 comprising SEQ ID NO: 2, and a HCDR3 comprising SEQ ID NO: 3, and the VL comprises a LCDR1 comprising SEQ ID NO: 4, a LCDR2 comprising SEQ ID NO: 5, and a LCDR3 comprising SEQ ID NO: 6.

2. A method of treating post-inflammatory hyperpigmentation or hypopigmentation of skin, the method comprising:
selecting a patient who has post-inflammatory hyperpigmentation or hypopigmentation of skin, and
administering to the patient a therapeutically effective amount of an anti-IL-13 antibody, wherein the anti-IL-13 antibody comprises a heavy chain variable region (VH) and a light chain variable region (VL), wherein the VH comprises a HCDR1 comprising SEQ ID NO: 1, a HCDR2 comprising SEQ ID NO: 2, and a HCDR3 comprising SEQ ID NO: 3, and the VL comprises a LCDR1 comprising SEQ ID NO: 4, a LCDR2 comprising SEQ ID NO: 5, and a LCDR3 comprising SEQ ID NO: 6.

3. The method of claim 1 or 2, wherein the post-inflammatory hyperpigmentation or hypopigmentation of skin is associated with atopic dermatitis (AD).

4. The method of any one of claims 1-3, wherein the anti-IL-13 antibody comprises a VH comprising SEQ ID NO: 7, and a VL comprising SEQ ID NO: 8.

5. The method of any one of claims 1-4, wherein the anti-IL-13 antibody comprises a heavy chain comprising SEQ ID NO: 9, and a light chain comprising SEQ ID NO: 10.

6. The method of any one of claims 1-5, wherein the anti-IL-13 antibody is lebrikizumab.

7. The method of any one of claims 1-6, wherein the anti-IL-13 antibody is administered subcutaneously to the patient.

8. The method of any one of claims 1-7, wherein the anti-IL-13 antibody is administered at a dose of 250 mg to 500 mg.

9. The method of any one of claims 1-8, wherein the anti-IL-13 antibody is administered subcutaneously to the patient at a dose of 250 mg once every two weeks.

10. The method of claim 9, wherein the patient is further treated with a loading dose of 500 mg of the anti-IL-13 antibody.

11. The method of claim 10, wherein the loading dose is administered to the patient at week 0 and week 2.

12. The method of any one of claims 1-11, wherein the patient is treated with the anti-IL-13 antibody for a period of at least 16 weeks.

13. The method of any one of claims 1-12, wherein the anti-IL-13 antibody is administered at 500 mg at baseline (week 0) and week 2, followed by 250 mg once every two weeks for at least 14 weeks.

14. The method of any one of claims 1-13, further comprising determining if the patient is a responder to the anti-IL-13 antibody after the treatment.

15. The method of claim 14, wherein the patient is a responder when the patient's EASI score determined after the treatment is reduced by 75% or greater compared to the patient's EASI score at the baseline.

16. The method of claim 14, wherein the patient is a responder when the patient's IGA score is 0 or 1 after the treatment, and the patient's IGA score after the treatment is reduced by 2 points or greater compared to the patient's IGA score at the baseline.

17. The method of any one of claims 1-16, wherein the patient is further treated for a maintenance period of 8 to 36 weeks.

18. The method of any one of claims 1-16, wherein the patient is further treated for a maintenance period of 8 weeks.

19. The method of claim 17 or 18, wherein the patient is treated with a maintenance dose of 250 mg of anti-IL-13 antibody once every two weeks during the maintenance period.

20. The method of claim 17 or 18, wherein the patient is treated with a maintenance dose of 250 mg of the anti-IL-13 antibody once every four weeks during the maintenance period.

21. The method of claim 17 or 18, wherein the patient is treated with a maintenance dose of 250 mg of the anti-IL-13 antibody once every eight weeks during the maintenance period.

22. The method of any one of claims 1-21, wherein the anti-IL-13 antibody is administered to the patient using a subcutaneous administration device.

23. The method of claim 22, wherein the subcutaneous administration device is selected from a prefilled syringe, disposable pen injection device, microneedle device, microinfuser device, needle-free injection device, or autoinjector device.

24. The method of any one of claims 1-23, wherein the patient has moderate to severe AD for at least a year at the baseline.

25. The method of claim 24, wherein the moderate to severe AD is determined by the American Academy of Dermatology Consensus Criteria for Chronic Atopic Dermatitis.

26. The method of any one of claims 1-25, wherein, at the baseline, the patient has an EASI score of 16 or greater, an IGA score of 3 or greater, and more than 10% of body surface area (BSA) affected by atopic dermatitis.

27. The method of any one of claims 1-26, wherein the patient has inadequate response to topical corticosteroids, topical calcineurin inhibitors, or crisaborole; or topical corticosteroids, topical calcineurin inhibitors, or crisaborole are medically inadvisable for the patient.

28. The method of any one of claims 1-27, wherein the patient is aged 12 years and older.

29. The method of any one of claims 1-28, wherein the patient is Fitzpatrick Phototype of IV to VI.

30. The method of any one of claims 1-29, wherein the patient is not white.

31. The method of any one of claims 1-30, wherein the patient is Black or African American, American Indian or Alaska Native, Asian, Native Hawaiian, or Pacific Islander.

32. The method of any one of claims 1-31, wherein the patient's skin hyperpigmentation or hypopigmentation is measured by a post-inflammatory dyschromia scale .

33. The method of any one of claims 1-32, wherein the method further comprises administrating one or more topical corticosteroids to the patient.

34. The method of claim 33, wherein the one or more topical corticosteroids is triamcinolone acetonide, hydrocortisone, or a combination of triamcinolone acetonide and hydrocortisone.

35. An anti-IL-13 antibody, or a pharmaceutical composition comprising an anti-IL-13 antibody, for use in the treatment of post-inflammatory hyperpigmentation or hypopigmentation of skin, wherein the anti-IL-13 antibody comprises a heavy chain variable region (VH) and a light chain variable region (VL), wherein the VH comprises a HCDR1 comprising SEQ ID NO: 1, a HCDR2 comprising SEQ ID NO: 2, and a HCDR3 comprising SEQ ID NO: 3, and the VL comprises a LCDR1 comprising SEQ ID NO: 4, a LCDR2 comprising SEQ ID NO: 5, and a LCDR3 comprising SEQ ID NO: 6.

36. Use of an anti-IL-13 antibody in the manufacture of a medicament for the treatment of post-inflammatory hyperpigmentation or hypopigmentation of skin, wherein the anti-IL-13 antibody comprises a heavy chain variable region (VH) and a light chain variable region (VL), wherein the VH comprises a HCDR1 comprising SEQ ID NO: 1, a HCDR2 comprising SEQ ID NO: 2, and a HCDR3 comprising SEQ ID NO: 3, and the VL comprises a LCDR1 comprising SEQ ID NO: 4, a LCDR2 comprising SEQ ID NO: 5, and a LCDR3 comprising SEQ ID NO: 6.

37. The anti-IL-13 antibody for use of claim 35 or use of claim 36, wherein the post-inflammatory hyperpigmentation or hypopigmentation of skin is associated with AD.

38. The anti-IL-13 antibody for use of claim 35 or 37, or use of claim 36 or 37, wherein the patient has moderate to severe AD for at least a year at the baseline.

39. The anti-IL-13 antibody for use of any one of claims 35, 37-38, or use of any one of claims 36-38, wherein, at the baseline, the patient has an EASI score of 16 or greater, an IGA score of 3 or greater, and more than 10% of body surface area (BSA) affected by AD.

40. The anti-IL-13 antibody for use of any one of claims 35, 37-39, or use of any one of claims 36-40, wherein the patient has inadequate response to topical corticosteroids, topical calcineurin inhibitors, or crisaborole; or topical corticosteroids, topical calcineurin inhibitors, or crisaborole are medically inadvisable for the patient.

41. The anti-IL-13 antibody for use of any one of claims 35, 37-40, or use of any one of claims 36-40, wherein the patient is aged 12 years and older.

42. The anti-IL-13 antibody for use of any one of claims 35, 37-41, or use of any one of claims 36-41, wherein the patient is Fitzpatrick Phototype of IV to VI.

43. The anti-IL-13 antibody for use of any one of claims 35, 37-42, or use of any one of claims 36-42, wherein the patient is not white.

44. The anti-IL-13 antibody for use of any one of claims 35, 37-43, or use of any one of claims 36-43, wherein the patient is Black or African American, American Indian or Alaska Native, Asian, Native Hawaiian, or Pacific Islander.

45. The anti-IL-13 antibody for use of any one of claims 35, 37-44, or use of any one of claims 36-44, wherein the anti-IL-13 antibody is used in simultaneous, separate, or sequential combination with one or more topical corticosteroids.