methotrexate consist mostly of case reports, with few reported randomized controlled trials (Haeck IM, et al., J Am Acad Dermatol 2011;64:1074–84; Schram ME, et al., J Allergy Clin Immunol 2011;128:353–9). Prescribing practice demonstrates significant country-specific variation, highlighted by a recent survey of European dermatologists' which reported no clearly favored agent: 43% used cyclosporine, 31% oral corticosteroids, and 22% azathioprine (Proudfoot LE, et al., Br J Dermatol 2013;169:901–9).

[0017] Although the step-up therapies, including systemic immunosuppressants, used to treat patients with moderate to severe AD show evidence of modest to good efficacy, poor tolerability due to side effects limit their prolonged use. Even in cases where cyclosporine has demonstrated substantial efficacy, approximately 50% of patients relapse within 2 weeks, and 80% relapse within 6 weeks after cessation of therapy (Amor KT, et al., J Am Acad Dermatol 2010;63:925–46). The continued use of these agents despite harmful side effects and limitations indicates a high unmet medical need for safer and more effective therapies.

[0018] In attempts to find novel therapies for moderate to severe AD with an acceptable benefit-risk profile, a number of biologic agents that specifically target inflammatory cells and mediators have been tested (Taïeb A, et al., J Dtsch Dermatol Ges 2012;10:174–8; Guttman-Yassky E, et al., Expert Opin Biol Ther 2013;13:549–61). However, several of these studies and case reports were performed in only a small number of patients (as few as one or two) and have shown inconsistent efficacy and/or safety signals.

[0019] Accordingly, there is a need for new therapeutic treatments and treatment regimens that will reduce the severity of atopic dermatitis disease symptoms and maximize efficacy. In addition, there is a need for new therapeutic treatments and treatment regimens that provide an improved safety profile with limited toxicity compared to existing treatments or provide more tolerability or convenience for patients thereby improving patient compliance with usage of therapeutic agents and adherence to treatment regimens.

[0020] The invention described herein meets certain of the above-described needs and provides other benefits.

[0021] All references cited herein, including patent applications and publications, are incorporated by reference in their entirety for any purpose.

SUMMARY

[0022] The disclosure is based, at least in part, on the surprising and unexpected discovery that an anti-IL-13 antagonist monoclonal antibody, lebrikizumab, provides therapeutic benefit

when administered to atopic dermatitis patients using the dosing regimens provided herein, including patients using topical corticosteroids concomitantly, as assessed by several efficacy outcome measures. Accordingly, provided herein are uses of IL-13 antagonists, including anti-IL-13 antibodies such as lebrikizumab, in treating atopic dermatitis and methods of treating atopic dermatitis with IL-13 antagonists, including anti-IL-13 antibodies such as lebrikizumab.

[0023] Accordingly, in one aspect, there is provided use of a pharmaceutical composition comprising a therapeutically effective amount of an IL-13 antagonist, in the manufacture of a medicament for treating atopic dermatitis in a patient, wherein the pharmaceutical composition reduces disease severity in the patient and wherein disease severity is assessed by an Atopic Dermatitis Disease Severity Outcome Measure. Also provided are methods of treating atopic dermatitis in a patient comprising administering to the patient a pharmaceutical composition comprising a therapeutically effective amount of an IL-13 antagonist, wherein the pharmaceutical composition reduces disease severity in the patient and wherein disease severity is assessed by an Atopic Dermatitis Disease Severity Outcome Measure (ADDSOM). In some embodiments, the atopic dermatitis is moderate to severe as determined by Rajka/Langeland criteria score and wherein the Rajka/Langeland criteria score is determined to be between 4.5 and 9. In some embodiments, the use or method further comprises administration of one or more topical corticosteroids. In some embodiments, the one or more topical corticosteroids is administered before administration of the IL-13 antagonist, at the same time as administration of the IL-13 antagonist, or after administration of the IL-13 antagonist. In some embodiments, the one or more topical corticosteroids is selected from triamcinolone acetonide, hydrocortisone, and a combination of triamcinolone acetonide and hydrocortisone. In some embodiments, the patient is aged 12 years and over. In some embodiments, the patient is inadequately controlled on topical corticosteroids. In some embodiments, the IL-13 antagonist is a monoclonal anti-IL-13 antibody. In some embodiments, the anti-IL-13 antibody is an antibody comprising a VH comprising HVR-H1, HVR-H2, and HVR-H3, wherein the respective VH HVRs have the amino acid sequence of SEQ ID NO: 5, SEQ ID NO: 6, and SEQ ID NO: 7, and comprising a VL comprising HVR-L1, HVR-L2, and HVR-L3, wherein the respective VL HVRs have the amino acid sequence of SEQ ID NO: 8, SEQ ID NO: 9, and SEQ ID NO: 10. In certain embodiments, the anti-IL-13 antibody is an IgG1, IgG2, IgG3, or IgG4. In certain embodiments, the anti-IL-13 antibody is a human, humanized, or chimeric antibody. In some embodiments, the anti-IL-13 antibody is an antibody comprising a VH comprising a sequence selected from SEQ ID NO: 1 and SEQ ID NO: 3, and comprising a VL comprising a sequence selected from SEQ ID NO: 2 and SEQ ID NO: 4. In certain embodiments, the anti-IL-13 antibody is a full-length antibody or a fragment thereof that binds human IL-13. In some embodiments, the anti-IL-13 antibody is IgG4. In

one embodiment, the anti-IL-13 antibody is lebrikizumab. In one embodiment, the anti-IL-13 antibody comprises a heavy chain having the amino acid sequence of SEQ ID NO: 11 and a light chain having the amino acid sequence of SEQ ID NO: 12. In some embodiments, the IL-13 antagonist is administered to the patient using a subcutaneous administration device. In certain such embodiments, the subcutaneous administration device is selected from a prefilled syringe, disposable pen injection device, microneedle device, microinfuser device, needle-free injection device, and autoinjector device. In one embodiment, the IL-13 antagonist is lebrikizumab and lebrikizumab is administered to the patient using a prefilled syringe. In one embodiment, the IL-13 antagonist is lebrikizumab and lebrikizumab is administered to the patient using an autoinjector device.

[0024] In another embodiment, the pharmaceutical composition for use in treating atopic dermatitis comprises 125 mg or 250 mg or 500 mg or about 125 mg or about 250 mg or about 500 mg of an anti-IL-13 antibody. In some embodiments, the pharmaceutical composition comprises between 110 mg and 140 mg of an anti-IL-13 antibody or between 120 mg and 130 mg of an anti-IL-13 antibody. In some embodiments, the pharmaceutical composition comprises between 225 mg and 275 mg of an anti-IL-13 antibody or between 240 mg and 260 mg of an anti-IL-13 antibody. In some embodiments, the pharmaceutical composition comprises between 450 mg and 550 mg of an anti-IL-13 antibody or between 475 mg and 525 mg of an anti-IL-13 antibody or between 490 mg and 510 mg of an anti-IL-13 antibody. In some embodiments, the pharmaceutical composition comprises 125 mg, or about 125 mg, or between 110 mg and 140 mg, or between 120 mg and 130 mg, of the anti-IL-13 antibody and the composition is administered subcutaneously once every four weeks. In some embodiments, the pharmaceutical composition is administered for a period of 12 weeks or a period of 20 weeks or a period of 24 weeks. In some embodiments, the pharmaceutical composition comprises 250 mg, or about 250 mg, or between 225 mg and 275 mg, or between 240 mg and 260 mg of the anti-IL-13 antibody and the composition is administered subcutaneously once every four weeks or once every eight weeks. In some embodiments, the pharmaceutical composition is administered for a period of 24 weeks or more or for 24 weeks. In some embodiments, the atopic dermatitis is moderate to severe as determined by Rajka/Langeland criteria score and wherein the Rajka/Langeland criteria score is determined to be between 4.5 and 9. In some embodiments, the IL-13 antagonist is a monoclonal anti-IL-13 antibody. In some embodiments, the anti-IL-13 antibody is an antibody comprising a VH comprising HVR-H1, HVR-H2, and HVR-H3, wherein the respective VH HVRs have the

amino acid sequence of SEQ ID NO: 5, SEQ ID NO: 6, and SEQ ID NO: 7, and comprising a VL comprising HVR-L1, HVR-L2, and HVR-L3, wherein the respective VL HVRs have the amino acid sequence of SEQ ID NO: 8, SEQ ID NO: 9, and SEQ ID NO: 10. In certain embodiments, the anti-IL-13 antibody is an IgG1, IgG2, IgG3, or IgG4. In certain embodiments, the anti-IL-13 antibody is a human, humanized, or chimeric antibody. In some embodiments, the anti-IL-13 antibody is an antibody comprising a VH comprising a sequence selected from SEQ ID NO: 1 and SEQ ID NO: 3, and comprising a VL comprising a sequence selected from SEQ ID NO: 2 and SEQ ID NO: 4. In certain embodiments, the anti-IL-13 antibody is a full-length antibody or a fragment thereof that binds human IL-13. In some embodiments, the anti-IL-13 antibody is IgG4. In one embodiment, the anti-IL-13 antibody is lebrikizumab. In one embodiment, the anti-IL-13 antibody comprises a heavy chain having the amino acid sequence of SEQ ID NO: 11 and a light chain having the amino acid sequence of SEQ ID NO: 12. In some embodiments, the IL-13 antagonist is administered to the patient using a subcutaneous administration device. In certain such embodiments, the subcutaneous administration device is selected from a prefilled syringe, disposable pen injection device, microneedle device, microinfuser device, needle-free injection device, and autoinjector device. In one embodiment, the IL-13 antagonist is lebrikizumab and lebrikizumab is administered to the patient using a prefilled syringe. In one embodiment, the IL-13 antagonist is lebrikizumab and lebrikizumab is administered to the patient using an autoinjector device. In some embodiments, the use or method further comprises administration of one or more topical corticosteroids. In some embodiments, the one or more topical corticosteroids is administered before administration of the IL-13 antagonist, at the same time as administration of the IL-13 antagonist, or after administration of the IL-13 antagonist. In some embodiments, the one or more topical corticosteroids is selected from triamcinolone acetonide, hydrocortisone, and a combination of triamcinolone acetonide and hydrocortisone. In some embodiments, the patient is aged 12 years and over. In some embodiments, the patient is inadequately controlled on topical corticosteroids.

[0025] In yet another embodiment, methods of treating atopic dermatitis in a patient comprising administering to the patient a pharmaceutical composition comprising a therapeutically effective amount of an IL-13 antagonist, wherein the pharmaceutical composition reduces disease severity in the patient and wherein disease severity is assessed by an Atopic Dermatitis Disease Severity Outcome Measure (ADDSOM) are provided, and wherein the Atopic Dermatitis Disease Severity Outcome Measure is Eczema Area and Severity Index

the IL-13 antagonist is a monoclonal anti-IL-13 antibody. In some embodiments, the anti-IL-13 antibody is an antibody comprising a VH comprising HVR-H1, HVR-H2, and HVR-H3, wherein the respective VH HVRs have the amino acid sequence of SEQ ID NO: 5, SEQ ID NO: 6, and SEQ ID NO: 7, and comprising a VL comprising HVR-L1, HVR-L2, and HVR-L3, wherein the respective VL HVRs have the amino acid sequence of SEQ ID NO: 8, SEQ ID NO: 9, and SEQ ID NO: 10. In certain embodiments, the anti-IL-13 antibody is an IgG1, IgG2, IgG3, or IgG4. In certain embodiments, the anti-IL-13 antibody is a human, humanized, or chimeric antibody. In some embodiments, the anti-IL-13 antibody is an antibody comprising a VH comprising a sequence selected from SEQ ID NO: 1 and SEQ ID NO: 3, and comprising a VL comprising a sequence selected from SEQ ID NO: 2 and SEQ ID NO: 4. In certain embodiments, the anti-IL-13 antibody is a full-length antibody or a fragment thereof that binds human IL-13. In some embodiments, the anti-IL-13 antibody is IgG4. In one embodiment, the anti-IL-13 antibody is lebrikizumab. In one embodiment, the anti-IL-13 antibody comprises a heavy chain having the amino acid sequence of SEQ ID NO: 11 and a light chain having the amino acid sequence of SEQ ID NO: 12. In some embodiments, the IL-13 antagonist is administered to the patient using a subcutaneous administration device. In certain such embodiments, the subcutaneous administration device is selected from a prefilled syringe, disposable pen injection device, microneedle device, microinfuser device, needle-free injection device, and autoinjector device. In one embodiment, the IL-13 antagonist is lebrikizumab and lebrikizumab is administered to the patient using a prefilled syringe. In one embodiment, the IL-13 antagonist is lebrikizumab and lebrikizumab is administered to the patient using an autoinjector device.

[0026] In another aspect, there is provided use of a therapeutically effective amount of an IL-13 antagonist, in the manufacture of a medicament for treating atopic dermatitis in a patient, wherein the therapeutically effective amount is selected from 125 mg and 250mg and wherein the IL-13 antagonist is to be administered subcutaneously once every four weeks or once every eight weeks. Also provided are methods comprising administering to a patient a therapeutically effective amount of an IL-13 antagonist, wherein the therapeutically effective amount is selected from 125 mg and 250 mg and wherein the IL-13 antagonist is administered subcutaneously once every four weeks. In some embodiments, the therapeutically effective amount is about 125 mg or between 110 mg and 140 mg of an anti-IL-13 antibody or between 120 mg and 130 mg of an anti-IL-13 antibody. In some embodiments, the therapeutically effective amount is about 250 mg or between 225 mg and 275 mg of an anti-IL-13 antibody or between 240 mg and 260 mg of an anti-IL-13 antibody. In some embodiments, the IL-13 antagonist is a monoclonal anti-IL-13 antibody. In some embodiments, the anti-IL-13 antibody is an antibody comprising a VH comprising HVR-H1, HVR-H2, and HVR-H3, wherein the respective VH HVRs have the amino acid sequence of

SEQ ID NO: 5, SEQ ID NO: 6, and SEQ ID NO: 7, and comprising a VL comprising HVR-L1, HVR-L2, and HVR-L3, wherein the respective VL HVRs have the amino acid sequence of SEQ ID NO: 8, SEQ ID NO: 9, and SEQ ID NO: 10. In certain embodiments, the anti-IL-13 antibody is an IgG1, IgG2, IgG3, or IgG4. In certain embodiments, the anti-IL-13 antibody is a human, humanized, or chimeric antibody. In some embodiments, the anti-IL-13 antibody is an antibody comprising a VH comprising a sequence selected from SEQ ID NO: 1 and SEQ ID NO: 3, and comprising a VL comprising a sequence selected from SEQ ID NO: 2 and SEQ ID NO: 4. In certain embodiments, the anti-IL-13 antibody is a full-length antibody or a fragment thereof that binds human IL-13. In some embodiments, the anti-IL-13 antibody is IgG4. In one embodiment, the anti-IL-13 antibody is lebrikizumab. In one embodiment, the anti-IL-13 antibody comprises a heavy chain having the amino acid sequence of SEQ ID NO: 11 and a light chain having the amino acid sequence of SEQ ID NO: 12. In some embodiments, the IL-13 antagonist is administered to the patient using a subcutaneous administration device. In certain such embodiments, the subcutaneous administration device is selected from a prefilled syringe, disposable pen injection device, microneedle device, microinfuser device, needle-free injection device, and autoinjector device. In one embodiment, the IL-13 antagonist is lebrikizumab and lebrikizumab is administered to the patient using a prefilled syringe. In one embodiment, the IL-13 antagonist is lebrikizumab and lebrikizumab is administered to the patient using an autoinjector device. In some embodiments, the atopic dermatitis is moderate to severe as determined by Rajka/Langeland criteria score and wherein the Rajka/Langeland criteria score is determined to be between 4.5 and 9. In some embodiments, the use or method further comprises administration of one or more topical corticosteroids. In some embodiments, the one or more topical corticosteroids is administered before administration of the IL-13 antagonist, at the same time as administration of the IL-13 antagonist, or after administration of the IL-13 antagonist. In some embodiments, the one or more topical corticosteroids is selected from triamcinolone acetonide, hydrocortisone, and a combination of triamcinolone acetonide and hydrocortisone. In some embodiments, the patient is aged 12 years and over. In some embodiments, the patient is inadequately controlled on topical corticosteroids. In some embodiments, the therapeutically effective amount reduces disease severity in the patient and disease severity is assessed by an Atopic Dermatitis Disease Severity Outcome Measure (ADDSOM). In some embodiments, the ADDSOM is Eczema Area and Severity Index (EASI), or Severity Scoring of Atopic Dermatitis (SCORAD), or Investigator Global Assessment (IGA), or a Patient Reported

Outcome (PRO). In some embodiments, the ADDSOM is EASI and the therapeutically effective amount reduces the EASI by 50% or 75% or 90% compared to the EASI determined prior to administration of a first dose of the IL-13 antagonist. In some embodiments, the EASI is determined 12 weeks after administration of the first dose or 20 weeks after administration of the first dose or 24 weeks after administration of the first dose. In some embodiments, the ADDSOM is SCORAD and the therapeutically effective amount reduces the SCORAD by 50% compared to the SCORAD determined prior to administration of a first dose of the IL-13 antagonist. In some embodiments, the ADDSOM is SCORAD and the therapeutically effective amount reduces the SCORAD by 75% compared to the SCORAD determined prior to administration of a first dose of the IL-13 antagonist. In some embodiments, the SCORAD is determined 12 weeks after administration of the first dose. In some embodiments, the ADDSOM is IGA and the therapeutically effective amount reduces the IGA to zero or one. In some embodiments, the IGA is determined 12 weeks after administration of a first dose of the IL-13 antagonist. In some embodiments, the ADDSOM is PRO and the PRO is pruritus visual analogue scale (VAS), sleep loss VAS, or Atopic Dermatitis Impact Questionnaire (ADIQ) score. In some embodiments, the PRO is determined 12 weeks after administration of a first dose of the IL-13 antagonist. In some embodiments, the PRO is pruritus VAS and the therapeutically effective amount reduces the pruritus VAS by 40% to 55%. In some embodiments, the PRO is sleep loss VAS and the therapeutically effective amount reduces the sleep loss VAS by 53% to 61%. In some embodiments, the PRO is ADIQ and the therapeutically effective amount reduces the ADIQ score by 54% to 65%.

[0027] In another aspect, there is provided use of a therapeutically effective amount of an IL-13 antagonist, in the manufacture of a medicament for treating atopic dermatitis in a patient, wherein the administration comprises administration of at least one loading dose and administration of at least one subsequent maintenance dose, wherein each of the at least one loading dose and each of the at least one maintenance dose is to be administered subcutaneously at a flat dose. Also provided are methods of treating atopic dermatitis in a patient by administering to the patient a therapeutically effective amount of an IL-13 antagonist, where the administration comprises administration of at least one loading dose and administration of at least one subsequent maintenance dose, and where each of the at least one loading dose and each of the at least one maintenance dose is administered subcutaneously at a flat dose. In some embodiments, the atopic dermatitis is moderate to severe as determined by Rajka/Langeland criteria score and wherein the Rajka/Langeland criteria score is determined to be between 4.5 and 9. In some embodiments, the use or method further comprises administration of one or more topical corticosteroids. In some embodiments, the one or more topical corticosteroids is administered before administration of the IL-13 antagonist, at the same time as administration of the IL-13 antagonist, or after administration of the IL-13 antagonist. In some embodiments, the one or more topical corticosteroids is selected from triamcinolone

CLAIMS:

1. Use of a pharmaceutical composition comprising a therapeutically effective amount of an IL-13 antagonist, in the manufacture of a medicament for treating atopic dermatitis in a patient, wherein the pharmaceutical composition reduces disease severity in the patient and wherein disease severity is assessed by an Atopic Dermatitis Disease Severity Outcome Measure.

2. The use of claim 1, wherein the IL-13 antagonist is a monoclonal anti-IL-13 antibody.

3. The use of claim 2, wherein the anti-IL-13 antibody is an antibody comprising a VH comprising HVR-H1, HVR-H2, and HVR-H3, wherein the respective VH HVRs have the amino acid sequence of SEQ ID NO: 5, SEQ ID NO: 6, and SEQ ID NO: 7, and comprising a VL comprising HVR-L1, HVR-L2, and HVR-L3, wherein the respective VL HVRs have the amino acid sequence of SEQ ID NO: 8, SEQ ID NO: 9, and SEQ ID NO: 10.

4. The use of claim 3, wherein the anti-IL-13 antibody is an antibody comprising a VH comprising a sequence selected from SEQ ID NO: 1 and SEQ ID NO: 3, and comprising a VL comprising a sequence selected from SEQ ID NO: 2 and SEQ ID NO: 4.

5. The use of claim 3, wherein the anti-IL-13 antibody is an IgG1, IgG2, IgG3, or IgG4.

6. The use of claim 5, wherein the anti-IL-13 antibody is a human, humanized, or chimeric antibody.

7. The use of claim 6, wherein the anti-IL-13 antibody is a full-length antibody or a fragment thereof that binds human IL-13.

8. The use of claim 5, wherein the anti-IL-13 antibody is IgG4.

9. The use of claim 8, wherein the anti-IL-13 antibody is lebrikizumab.

10. The use of claim 4, wherein the anti-IL-13 antibody comprises a heavy chain having the amino acid sequence of SEQ ID NO: 11 and a light chain having the amino acid sequence of SEQ ID NO: 12.

11. The use of claim 3, wherein the pharmaceutical composition comprises 125 mg or 250 mg or 500 mg of the anti-IL-13 antibody.

12. The use of claim 11, wherein the pharmaceutical composition comprises 125 mg of the anti-IL-13 antibody and wherein the composition is to be administered subcutaneously once every four weeks.

13. The use of claim 12, wherein the pharmaceutical composition is to be administered for a period of 12 weeks or a period of 20 weeks or a period of 24 weeks.

14. The use of claim 11, wherein the pharmaceutical composition comprises 250 mg of the anti-IL-13 antibody and wherein the composition is to be administered subcutaneously once every four weeks.
15. The use of claim 11, wherein the pharmaceutical composition comprises 250 mg of the anti-IL-13 antibody and wherein the composition is to be administered subcutaneously once every eight weeks.
16. The use of claim 14 or claim 15, wherein the pharmaceutical composition is to be administered for a period of 24 weeks or more.
17. The use of claim 16, wherein the pharmaceutical composition is to be administered for a period of 24 weeks.
18. The use of claim 1, wherein the Atopic Dermatitis Disease Severity Outcome Measure is Eczema Area and Severity Index (EASI).
19. The use of claim 18, wherein the pharmaceutical composition reduces the EASI by 50% or 75% or 90% compared to the EASI determined prior to administration of a first dose of the pharmaceutical composition.
20. The use of claim 19, wherein the EASI is determined 12 weeks after administration of the first dose or 20 weeks after administration of the first dose or 24 weeks after administration of the first dose.
21. The use of claim 1, wherein the Atopic Dermatitis Disease Severity Outcome Measure is Severity Scoring of Atopic Dermatitis (SCORAD).
22. The use of claim 21, wherein the pharmaceutical composition reduces the SCORAD by 50% or 75% compared to the SCORAD determined prior to administration of a first dose of the pharmaceutical composition.
23. The use of claim 22, wherein the SCORAD is determined 12 weeks after administration of the first dose.
24. The use of claim 1, wherein the Atopic Dermatitis Disease Severity Outcome Measure is Investigator Global Assessment (IGA).
25. The use of claim 24, wherein the pharmaceutical composition reduces the IGA to zero or one.
26. The use of claim 25, wherein the IGA is determined 12 weeks after administration of a first dose of the pharmaceutical composition.

27. The use of claim 1, wherein the Atopic Dermatitis Disease Severity Outcome Measure is a Patient Reported Outcome (PRO).
28. The use of claim 27, wherein the PRO is pruritus visual analogue scale (VAS), sleep loss VAS, or Atopic Dermatitis Impact Questionnaire (ADIQ) score.
29. The use of claim 28, wherein the PRO is determined 12 weeks after administration of a first dose of the pharmaceutical composition.
30. The use of claim 28 or claim 29, wherein the pharmaceutical composition reduces the pruritus VAS by 40% to 55%.
31. The use of claim 28 or claim 29, wherein the pharmaceutical composition reduces the sleep loss VAS by 53% to 61%.
32. The use of claim 28 or claim 29, wherein the pharmaceutical composition reduces the ADIQ score by 54% to 65%.
33. The use of claim 1, wherein the atopic dermatitis is moderate to severe as determined by Rajka/Langeland criteria score and wherein the Rajka/Langeland criteria score is determined to be between 4.5 and 9.
34. Use of a therapeutically effective amount of an IL-13 antagonist, in the manufacture of a medicament for treating atopic dermatitis in a patient, wherein the therapeutically effective amount is selected from 125 mg and 250mg and wherein the IL-13 antagonist is to be administered subcutaneously once every four weeks or once every eight weeks.
35. The use of claim 34, wherein the therapeutically effective amount is 125 mg and wherein the IL-13 antagonist is to be administered once every four weeks.
36. The use of claim 34, wherein the therapeutically effective amount is 250 mg and wherein the IL-13 antagonist is to be administered once every four weeks.
37. The use of claim 34, wherein the therapeutically effective amount is 250 mg and wherein the IL-13 antagonist is to be administered once every eight weeks.
38. The use of claim 34, wherein the IL-13 antagonist is a monoclonal anti-IL-13 antibody.
39. The use of claim 38, wherein the anti-IL-13 antibody is an antibody comprising a VH comprising HVR-H1, HVR-H2, and HVR-H3, wherein the respective VH HVRs have the amino acid sequence of SEQ ID NO: 5, SEQ ID NO: 6, and SEQ ID NO: 7, and comprising a VL comprising HVR-L1, HVR-L2, and HVR-L3, wherein the respective VL HVRs have the amino acid sequence of SEQ ID NO: 8, SEQ ID NO: 9, and SEQ ID NO: 10.

40. The use of claim 39, wherein the anti-IL-13 antibody is an antibody comprising a VH comprising a sequence selected from SEQ ID NO: 1 and SEQ ID NO: 3, and comprising a VL comprising a sequence selected from SEQ ID NO: 2 and SEQ ID NO: 4.

41. The use of claim 39, wherein the anti-IL-13 antibody is an IgG1, IgG2, IgG3, or IgG4.

42. The use of claim 41, wherein the anti-IL-13 antibody is a human, humanized, or chimeric antibody.

43. The use of claim 42, wherein the anti-IL-13 antibody is a full-length antibody or a fragment thereof that binds human IL-13.

44. The use of claim 41, wherein the anti-IL-13 antibody is IgG4.

45. The use of claim 44, wherein the anti-IL-13 antibody is lebrikizumab.

46. The use of claim 40, wherein the anti-IL-13 antibody comprises a heavy chain having the amino acid sequence of SEQ ID NO: 11 and a light chain having the amino acid sequence of SEQ ID NO: 12.

47. The use of any one of claims 34-46, wherein the therapeutically effective amount reduces disease severity in the patient and wherein disease severity is assessed by an Atopic Dermatitis Disease Severity Outcome Measure.

48. The use of claim 47, wherein the Atopic Dermatitis Disease Severity Outcome Measure is Eczema Area and Severity Index (EASI).

49. The use of claim 48, wherein the therapeutically effective amount reduces the EASI by 50% or 75% or 90% compared to the EASI determined prior to administration of a first dose of the IL-13 antagonist.

50. The use of claim 49, wherein the EASI is determined 12 weeks after administration of the first dose or 20 weeks after administration of the first dose or 24 weeks after administration of the first dose.

51. The use of claim 47, wherein the Atopic Dermatitis Disease Severity Outcome Measure is Severity Scoring of Atopic Dermatitis (SCORAD).

52. The use of claim 51, wherein the therapeutically effective amount reduces the SCORAD by 50% or 75 % compared to the SCORAD determined prior to administration of a first dose of the IL-13 antagonist.

53. The use of claim 52, wherein the SCORAD is determined 12 weeks after administration of the first dose.

54. The use of claim 47, wherein the Atopic Dermatitis Disease Severity Outcome Measure is Investigator Global Assessment (IGA).
55. The use of claim 54, wherein the therapeutically effective amount reduces the IGA to zero or one.
56. The use of claim 55, wherein the IGA is determined 12 weeks after administration of a first dose of the IL-13 antagonist.
57. The use of claim 47, wherein the Atopic Dermatitis Disease Severity Outcome Measure is a Patient Reported Outcome (PRO).
58. The use of claim 57, wherein the PRO is pruritus visual analogue scale (VAS), sleep loss VAS, or Atopic Dermatitis Impact Questionnaire (ADIQ) score.
59. The use of claim 58, wherein the PRO is determined 12 weeks after administration of a first dose of the IL-13 antagonist.
60. The use of claim 58 or claim 59, wherein the therapeutically effective amount reduces the pruritus VAS by 40% to 55%.
61. The use of claim 58 or claim 59, wherein the therapeutically effective amount reduces the sleep loss VAS by 53% to 61%.
62. The use of claim 58 or claim 59, wherein the therapeutically effective amount reduces the ADIQ score by 54% to 65%.
63. The use of claim 34, wherein the atopic dermatitis is moderate to severe as determined by Rajka/Langeland criteria score and wherein the Rajka/Langeland criteria score is determined to be between 4.5 and 9.
64. Use of a therapeutically effective amount of an IL-13 antagonist, in the manufacture of a medicament for treating atopic dermatitis in a patient, wherein the administration comprises administration of at least one loading dose and administration of at least one subsequent maintenance dose, wherein each of the at least one loading dose and each of the at least one maintenance dose is to be administered subcutaneously at a flat dose.
65. The use of claim 64, wherein the IL-13 antagonist is a monoclonal anti-IL-13 antibody.
66. The use of claim 65, wherein the anti-IL-13 antibody is an antibody comprising a VH comprising HVR-H1, HVR-H2, and HVR-H3, wherein the respective VH HVRs have the amino acid sequence of SEQ ID NO: 5, SEQ ID NO: 6, and SEQ ID NO: 7, and comprising a

VL comprising HVR-L1, HVR-L2, and HVR-L3, wherein the respective VL HVRs have the amino acid sequence of SEQ ID NO: 8, SEQ ID NO: 9, and SEQ ID NO: 10.

67. The use of claim 66, wherein the anti-IL-13 antibody is an antibody comprising a VH comprising a sequence selected from SEQ ID NO: 1 and SEQ ID NO: 3, and comprising a VL comprising a sequence selected from SEQ ID NO: 2 and SEQ ID NO: 4.

68. The use of claim 66, wherein the anti-IL-13 antibody is an IgG1, IgG2, IgG3, or IgG4.

69. The use of claim 68, wherein the anti-IL-13 antibody is a human, humanized, or chimeric antibody.

70. The use of claim 69, wherein the anti-IL-13 antibody is a full-length antibody or a fragment thereof that binds human IL-13.

71. The use of claim 68, wherein the anti-IL-13 antibody is IgG4.

72. The use of claim 71, wherein the anti-IL-13 antibody is lebrikizumab.

73. The use of claim 67, wherein the anti-IL-13 antibody comprises a heavy chain having the amino acid sequence of SEQ ID NO: 11 and a light chain having the amino acid sequence of SEQ ID NO: 12.

74. The use of claim 64, wherein the loading dose is 250 mg or 500 mg and the maintenance dose is 125 mg.

75. The use of claim 74, wherein the maintenance dose is to be administered four weeks after administration of the loading dose and wherein the maintenance dose is to be administered once every four weeks thereafter for the duration of treatment.

76. The use of claim 75, wherein the loading dose is 250 mg and the maintenance dose is 125 mg.

77. The use of claim 64, wherein the loading dose is 250 mg and wherein the loading dose is to be administered followed by a second loading dose administration 15 days later and wherein the maintenance dose is 125 mg.

78. The use of claim 77, wherein the maintenance dose is to be administered two weeks after administration of the second loading dose and wherein the maintenance dose is to be administered once every four weeks thereafter for the duration of treatment.

79. The use of claim 64, wherein the loading dose is 250 mg and wherein the loading dose is to be administered followed by a second loading dose administration 29 days later and wherein the maintenance dose is 125 mg.

80. The use of claim 79, wherein the maintenance dose is to be administered four weeks after administration of the second loading dose and wherein the maintenance dose is to be administered once every four weeks thereafter for the duration of treatment.
81. The use of claim 64, wherein the loading dose is 500 mg and the maintenance dose is 250 mg.
82. The use of claim 81, wherein the maintenance dose is to be administered four weeks after administration of the loading dose and wherein the maintenance dose is to be administered once every four weeks thereafter for the duration of treatment.
83. The use of claim 81, wherein the maintenance dose is to be administered four weeks after administration of the loading dose and wherein the maintenance dose is to be administered once every eight weeks thereafter for the duration of treatment.
84. The use of any one of claims 75, 76, 78, 80, 82, or 83, wherein the duration of treatment is 24 weeks or more.
85. The use of claim 84, wherein the duration of treatment is 24 weeks.
86. The use of any one of claims 64-85, wherein the therapeutically effective amount reduces disease severity in the patient and wherein disease severity is assessed by an Atopic Dermatitis Disease Severity Outcome Measure.
87. The use of claim 86, wherein the Atopic Dermatitis Disease Severity Outcome Measure is Eczema Area and Severity Index (EASI).
88. The use of claim 87, wherein the therapeutically effective amount reduces the EASI by 50% or 75% or 90% compared to the EASI determined prior to administration of a first dose of the IL-13 antagonist.
89. The use of claim 88, wherein the EASI is determined 12 weeks after administration of the first dose or 20 weeks after administration of the first dose or 24 weeks after administration of the first dose.
90. The use of claim 86, wherein the Atopic Dermatitis Disease Severity Outcome Measure is Severity Scoring of Atopic Dermatitis (SCORAD).
91. The use of claim 90, wherein the therapeutically effective amount reduces the SCORAD by 50% or 75% compared to the SCORAD determined prior to administration of a first dose of the IL-13 antagonist.
92. The use of claim 91, wherein the SCORAD is determined 12 weeks after administration of the first dose.

93. The use of claim 86, wherein the Atopic Dermatitis Disease Severity Outcome Measure is Investigator Global Assessment (IGA).

94. The use of claim 93, wherein the therapeutically effective amount reduces the IGA to zero or one.

95. The use of claim 94, wherein the IGA is determined 12 weeks after administration of a first dose of the IL-13 antagonist.

96. The use of claim 86, wherein the Atopic Dermatitis Disease Severity Outcome Measure is a Patient Reported Outcome (PRO).

97. The use of claim 96, wherein the PRO is pruritus visual analogue scale (VAS), sleep loss VAS, or Atopic Dermatitis Impact Questionnaire (ADIQ) score.

98. The use of claim 97, wherein the PRO is determined 12 weeks after administration of a first dose of the IL-13 antagonist.

99. The use of claim 97 or claim 98, wherein the therapeutically effective amount reduces the pruritus VAS by 40% to 55%.

100. The use of claim 97 or claim 98, wherein the therapeutically effective amount reduces the sleep loss VAS by 53% to 61%.

101. The use of claim 97 or claim 98, wherein the therapeutically effective amount reduces the ADIQ score by 54% to 65%.

102. The use of claim 64, wherein the atopic dermatitis is moderate to severe as determined by Rajka/Langeland criteria score and wherein the Rajka/Langeland criteria score is determined to be between 4.5 and 9.

103. The use of any one of claims 1-102, wherein the IL-13 antagonist is to be administered to the patient using a subcutaneous administration device.

104. The use of claim 103, wherein the subcutaneous administration device is selected from a prefilled syringe, disposable pen injection device, microneedle device, microinfuser device, needle-free injection device, and autoinjector device.

105. The use of any one of claims 1-104, wherein the method further comprises administration of one or more topical corticosteroids.

106. The use of claim 105, wherein the one or more topical corticosteroids is to be administered before administration of the IL-13 antagonist, at the same time as administration of the IL-13 antagonist, or after administration of the IL-13 antagonist.

107. The use of claim 106, wherein the one or more topical corticosteroids is selected from triamcinolone acetonide, hydrocortisone, and a combination of triamcinolone acetonide and hydrocortisone.

108. The use of any one of claims 1-107, wherein the patient is aged 12 years and over.

109. The use of any one of claims 1-108, wherein the patient is inadequately controlled on topical corticosteroids.