What is claimed:

1. A method for determining the efficacy of a human TNFα antibody, or an antigen-binding portion thereof, for treating ankylosing spondylitis (AS), said method comprising comparing a pre-determined level of a collagen degradation biomarker and/or a synovitis biomarker from a patient having AS following treatment with the human TNFα antibody, with a known standard level of the collagen degradation biomarker and/or the synovitis biomarker associated with the disease state; and

assessing whether the patient's post-treatment collagen degradation biomarker and/or synovitis biomarker level is lower than the known standard level of the collagen degradation biomarker and/or synovitis biomarker, wherein a lower collagen degradation biomarker and/or synovitis biomarker level from the patient following treatment with the human TNFα antibody relative to the known standard level indicates efficacy of the human TNFα antibody for the treatment of AS.

2. The method of claim 1, wherein the collagen degradation biomarker is type II collagen C-telopeptide (CTX-II).

3. The method of claim 2, wherein the collagen degradation biomarker is urinary type II collagen C-telopeptide (CTX-II).

4. The method of claim 1, wherein the synovitis biomarker is matrix metalloprotease 3 (MMP3).

5. The method of claim 4, wherein the synovitis biomarker is serum metalloprotease 3 (MMP3).

6. The method of claim 1, wherein the efficacy of the human TNFα antibody, or an antigen-binding portion thereof, for improving structural damage associated with AS is determined.

7. The method of any one of claims 1-6, further comprising comparing the patient's C-reactive protein (CRP) level with a known standard CRP level associated with the disease state; and

assessing whether the patient's CRP level is higher than the known standard CRP level, wherein a lower C-reactive protein level relative to the known standard indicates efficacy of treatment.

8. The method of any one of claims 1-6, wherein the human TNFα antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less.

9. The method of any one of claims 1-6, wherein the human TNFα antibody, or antigen-binding portion thereof, has the following characteristics:

a) dissociates from human TNFα with a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, as determined by surface plasmon resonance;

b) has a light chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8 or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8 and/or 9;

c) has a heavy chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11 or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11 and/or 12.

10. The method any one of claims 1-6, wherein the human TNFα antibody, or antigen-binding portion thereof, comprises a light chain variable region (LCVR) having a CDR3 domain comprising the amino acid sequence of SEQ ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8, and comprises a heavy chain variable region (HCVR) having a CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11.

11. The method of any one of claims 1-6, wherein the human TNFα antibody, or antigen-binding portion thereof, comprises a light chain variable region (LCVR) comprising the amino acid sequence of SEQ ID NO: 1 and a heavy chain variable region (HCVR) comprising the amino acid sequence of SEQ ID NO: 2.

12. The method of any one of claims 1-6, wherein the human TNFα antibody, or antigen-binding portion thereof, is adalimumab.

13. A method for determining the efficacy of a human TNFα antibody, or an antigen-binding portion thereof, for ankylosing spondylitis (AS) comprising comparing a pre-treatment level of a collagen degradation biomarker and/or a synovitis biomarker

obtained from a patient having AS with a post-treatment level of the collagen degradation biomarker and/or the synovitis biomarker obtained from said patient, wherein a lower post-treatment biomarker level indicates efficacy of the human TNFα antibody, or an antigen-binding portion thereof.

14. The method of claim 13, wherein the collagen degradation biomarker is type II collagen C-telopeptide.

15. The method of claim 14, wherein the collagen degradation biomarker is urinary type II collagen C-telopeptide.

16. The method of claim 13, wherein the synovitis biomarker is matrix metalloprotease 3 (MMP3).

17. The method of claim 16, wherein the synovitis biomarker is serum matrix metalloprotease 3 (MMP3).

18. A method of monitoring the efficacy of a human TNFα antibody, or an antigen-binding portion thereof, for decreasing the progression of structural damage associated with ankylosing spondylitis (AS) in a patient, the method comprising determining the level of a collagen degradation biomarker and/or a synovitis biomarker in a patient and comparing the level of the collagen degradation biomarker and/or a synovitis biomarker with a known standard level of the collagen degradation biomarker and/or a synovitis biomarker associated with AS, wherein a decrease in the level of the biomarker indicates that the human TNFα antibody, or an antigen-binding portion thereof, is efficacious for decreasing the rate of progression of structural damage associated with AS in the patient.

19. The method of claim 18, wherein the collagen degradation biomarker is type II collagen C-telopeptide.

20. The method of claim 19, wherein the collagen degradation biomarker is urinary type II collagen C-telopeptide.

21. The method of claim 18, wherein the synovitis biomarker is matrix metalloprotease 3 (MMP3).

22. The method of claim 21, wherein the synovitis biomarker is serum matrix metalloprotease 3 (MMP3).

23. The method of any one of claims 18-22, wherein the human TNFα antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less.

24. The method of any one of claims 18-22, wherein the human TNFα antibody, or antigen-binding portion thereof, has the following characteristics:

a) dissociates from human TNFα with a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, as determined by surface plasmon resonance;

b) has a light chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8 or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8 and/or 9;

c) has a heavy chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11 or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11 and/or 12.

25. The method of any one of claims 18-22, wherein the human TNFα antibody, or antigen-binding portion thereof, comprises a light chain variable region (LCVR) having a CDR3 domain comprising the amino acid sequence of SEQ ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8, and comprises a heavy chain variable region (HCVR) having a CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11.

26. The method of any one of claims 18-22, wherein the human TNFα antibody, or antigen-binding portion thereof, comprises a light chain variable region (LCVR) comprising the amino acid sequence of SEQ ID NO: 1 and a heavy chain variable region (HCVR) comprising the amino acid sequence of SEQ ID NO: 2.

27. The method of any one of claims 18-22, wherein the TNFα antibody, or antigen-binding portion thereof, is adalimumab.

28. A method for predicting the efficacy of a human TNFα antibody, or an antigen-binding portion thereof, for the treatment of AS in a patient, said method comprising

comparing a pre-determined level of a collagen degradation biomarker and/or a synovitis biomarker from the patient following treatment with the human TNFα antibody, or an antigen-binding portion thereof, with a known standard level of the collagen degradation biomarker and/or a synovitis biomarker associated with AS; and

assessing whether the patient's post-treatment collagen degradation biomarker and/or a synovitis biomarker level is lower than the known standard level of the collagen degradation biomarker and/or a synovitis biomarker, wherein a lower collagen degradation biomarker and/or a synovitis biomarker level from the patient relative to the known standard level indicates that the human TNFα antibody, or an antigen-binding portion thereof, is predicted to be effective for the treatment of AS in the patient.

29. The method of claim 28, wherein the collagen degradation biomarker is type II collagen C-telopeptide.

30. The method of claim 29, wherein the collagen degradation biomarker is urinary type II collagen C-telopeptide.

31. The method of claim 28, wherein the synovitis biomarker is matrix metalloprotease 3 (MMP3).

32. The method of claim 31, wherein the synovitis biomarker is serum matrix metalloprotease 3 (MMP3).

33. The method of any one of claims 28-32, wherein the human TNFα antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less.

34. The method of any one of claims 28-32, wherein the human TNFα antibody, or antigen-binding portion thereof, has the following characteristics:

a) dissociates from human TNFα with a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, as determined by surface plasmon resonance;

b) has a light chain CDR3 domain comprising the amino acid sequence of SEQ

ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8 or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8 and/or 9;

c) has a heavy chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11 or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11 and/or 12.

35. The method of any one of claims 28-32, wherein the human TNFα antibody, or antigen-binding portion thereof, comprises a light chain variable region (LCVR) having a CDR3 domain comprising the amino acid sequence of SEQ ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8, and comprises a heavy chain variable region (HCVR) having a CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11.

36. The method of any one of claims 28-32, wherein the human TNFα antibody, or antigen-binding portion thereof, comprises a light chain variable region (LCVR) comprising the amino acid sequence of SEQ ID NO: 1 and a heavy chain variable region (HCVR) comprising the amino acid sequence of SEQ ID NO: 2.

37. The method of any one of claims 28-32, wherein the TNFα antibody, or antigen-binding portion thereof, is adalimumab.

38. The method of any one of claims 1-37, wherein the level of the biomarker is determined using ELISA.

39. A kit for performing the method of any one of claims 1-37, comprising

a) a detectable agent that specifically recognizes the collagen degradation biomarker and/or a synovitis biomarker;

b) instructions for use; and

c) optionally, reagents for isolating a sample from the patient.

40. The kit of claim 39, wherein the detectable agent recognizes either urinary CTX-II or serum MMP3.

41. A method of determining the efficacy of a TNFα inhibitor for the treatment of AS in a patient, said method comprising

comparing a pre-determined level of CTX-II from the patient following treatment with the TNFα inhibitor with a known standard level of CTX-II associated with the disease state; and

assessing whether the patient's post-treatment CTX-II level is lower than the known standard level of CTX-II, wherein a lower CTX-II level from the patient relative to the known standard level indicates that the TNFα inhibitor is effective for the treatment of AS in the patient.

42. A method for determining the efficacy of a TNFα inhibitor for decreasing structural damage associated with ankylosing spondylitis (AS) in a patient, said method comprising comparing a pre-determined level of CTX-II from the patient having AS following treatment with the TNFα inhibitor with a known standard level of CTX-II associated with the disease state; and

assessing whether the patient's post-treatment CTX-II level is lower than the known standard level of CTX-II, wherein a lower CTX-II level from the patient following treatment with the TNFα inhibitor relative to the known standard level indicates that the TNFα inhibitor is effective at decreasing structural damage associated with AS in the patient.

43. A method of determining the efficacy of a TNFα inhibitor for the treatment of AS in a patient, said method comprising

comparing a pre-determined, post-treatment level of CTX-II obtained from the patient with a pre-determined, pre-treatment level of CTX-II obtained from the patient; and

assessing whether the post-treatment CTX-II level is lower than the pre-treatment CTX-II level, wherein a lower post-treatment CTX-II level from the patient relative to the pre-treatment CTX-II level indicates that the TNFα inhibitor is effective for the treatment of AS in the patient.

44. The method of any one of claims 41-43, wherein the post-treatment CTX-II level is at least about a 9% decrease relative to the pre-treatment CTX-II level.

45. The method of any one of claims 41-43, wherein the CTX-II is urinary CTX-II.

46. The method of any one of claims 41-43, wherein the CTX-II level is determined using ELISA.

47. The method of any one of claims 41-43, wherein the TNFα inhibitor is selected from the group consisting of a TNFα antibody, or an antigen-binding portion thereof, a TNF fusion protein, or a recombinant TNF binding protein.

48. The method of claim 47, wherein the TNF fusion protein is etanercept.

49. The method of claim 47, wherein the TNFα antibody, or antigen-binding portion thereof, is selected from the group consisting of a chimeric antibody, a humanized antibody, a human antibody, and a multivalent antibody.

50. The method of claim 47, wherein the anti-TNFα antibody, or antigen-binding portion thereof, is selected from the group consisting of infliximab, golimumab, and adalimumab.

51. The method of claim 49, wherein the human antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less.

52. The method of claim 49, wherein the human antibody, or antigen-binding portion thereof, has the following characteristics:

a) dissociates from human TNFα with a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, as determined by surface plasmon resonance;

b) has a light chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8 or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8 and/or 9;

c) has a heavy chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11 or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11 and/or 12.

53. The method of claim 49, wherein the human antibody, or antigen-binding portion thereof, comprises a light chain variable region (LCVR) comprising the amino acid sequence of SEQ ID NO: 1 and a heavy chain variable region (HCVR) comprising the amino acid sequence of SEQ ID NO: 2

54. The method of any one of claims 41-43, further comprising comparing a pre-determined, post-treatment level of a synovitis biomarker obtained from the patient with a known standard level of the synovitis biomarker associated with AS; and

assessing whether the post-treatment synovitis biomarker level is lower than the known standard synovitis biomarker level, wherein a lower post-treatment synovitis biomarker relative to the known standard synovitis biomarker level indicates that the TNFα inhibitor is effective for the treatment of AS in the patient.

55. The method of claim 54, wherein the synovitis biomarker is MMP-3.

56. The method of any one of claims 41-54, wherein the level of the biomarker is determined using ELISA.

57. A kit for performing the method of any one of claims 41-54, comprising

a) a detectable agent that specifically recognizes CTX-II;

b) instructions for use; and

c) optionally, reagents for isolating a sample from the patient.

58. The kit of claim 57, further comprising a detectable agent that specifically recognizes MMP-3.

Claims

1. A method for determining the efficacy of a human anti-TNFα antibody, or an antigen binding portion thereof, for the treatment of ankylosing spondylitis (AS) in a subject having AS, said method comprising

determining a post-treatment level of type II collagen C-telopeptide (CTX-II) and a post-treatment level of matrix metalloproteinase-3 (MMP-3) in a sample(s) obtained from the subject having AS,

wherein a lower post-treatment level of CTX-II in the sample(s) relative to a known standard level of CTX-II based on a subject(s) having AS and a lower post-treatment level of MMP-3 in the sample(s) relative to a known standard level of MMP-3 based on a subject(s) having AS indicates that the human anti-TNFα antibody, or antigen-binding portion thereof, is efficacious for the treatment of AS in the subject; and

wherein the human anti-TNFα antibody or antigen binding portion thereof comprises a light chain variable region comprising

a complementary determining region (CDR) 1 domain comprising the amino acid sequence set forth in SEQ ID NO:7;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:5; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7, or 8, or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8, and/or 9; and

comprises a heavy chain variable region comprising

a CDR 1 domain comprising the amino acid sequence set forth in SEQ ID NO:8;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:6; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10, or 11, or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11, and/or 12.

2. A method for determining the efficacy of a human anti-TNFα antibody, or an antigen-binding portion thereof, for the treatment of ankylosing spondylitis (AS) in a subject having AS, said method comprising

determining a pre-treatment level of type II collagen C-telopeptide (CTX-II) and a pre-treatment level of matrix metalloproteinase-3 (MMP-3) in a sample(s) obtained from the subject having AS,

wherein a lower post-treatment level of CTX-II relative to the pre-treatment level of CTX-II and a lower post-treatment level of MMP-3 relative to the pre-treatment level of MMP-3 indicates that the human anti-TNFα antibody, or antigen-binding portion thereof, is efficacious for the treatment of AS in the subject, and

wherein the human anti-TNFα antibody or antigen binding portion thereof comprises a light chain variable region comprising

a complementary determining region (CDR) 1 domain comprising the amino acid sequence set forth in SEQ ID NO:7;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:5; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7, or 8, or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8, and/or 9; and

comprises a heavy chain variable region comprising

a CDR 1 domain comprising the amino acid sequence set forth in SEQ ID NO:8;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:6; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10, or 11, or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11, and/or 12.

3. A method for determining the efficacy of a human anti-TNFα antibody, or an antigen-binding portion thereof, for the treatment of ankylosing spondylitis (AS) in a subject having AS, said method comprising

determining a post-treatment level of type II collagen C-telopeptide (CTX-II) and a post-treatment level of matrix metalloproteinase-3 (MMP-3) in a sample(s) obtained from the subject having AS,

wherein a lower post-treatment level of CTX-II relative to a pre-treatment level of CTX-II and a lower post-treatment level of MMP-3 relative to a pre-treatment level of MMP-3 indicates that the human anti-TNFα antibody, or antigen-binding portion thereof, is efficacious for the treatment of AS in the subject, and

wherein the human anti-TNFα antibody or antigen binding portion thereof comprises a light chain variable region comprising

a complementary determining region (CDR) 1 domain comprising the amino acid sequence set forth in SEQ ID NO:7;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:5; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7, or 8, or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8, and/or 9; and

comprises a heavy chain variable region comprising

a CDR 1 domain comprising the amino acid sequence set forth in SEQ ID NO:8;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:6; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10, or 11, or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11, and/or 12.

4. A method of monitoring the efficacy of a human anti-TNFα antibody, or an antigen-binding portion thereof, for decreasing the progression of structural damage associated with ankylosing spondylitis (AS) in a subject having AS, the method comprising

determining a post-treatment level of type II collagen C-telopeptide (CTX-II) and a post-treatment level of matrix metalloproteinase-3 (MMP-3) in a sample(s) obtained from the subject having AS,

wherein a lower post-treatment level of CTX-II in the sample(s) relative to a known standard level of CTX-II based on a subject(s) having AS and a lower post-treatment level of MMP-3 in the sample(s) relative to a known standard level of MMP-3 based on a subject(s) having AS indicates that the human anti-TNFα antibody, or antigen-binding portion thereof, is efficacious for decreasing the rate of progression of structural damage associated with AS in the subject, and

wherein the human anti-TNFα antibody or antigen binding portion thereof comprises a light chain variable region comprising

a complementary determining region (CDR) 1 domain comprising the amino acid sequence set forth in SEQ ID NO:7;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:5; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7, or 8, or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8, and/or 9; and

comprises a heavy chain variable region comprising

a CDR 1 domain comprising the amino acid sequence set forth in SEQ ID NO:8;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:6; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10, or 11, or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11, and/or 12.

5. A method for monitoring the efficacy of a human anti-TNFα antibody, or an antigen-binding portion thereof, for decreasing the progression of structural damage associated with ankylosing spondylitis (AS) in a subject having AS, said method comprising

determining a pre-treatment level of type II collagen C-telopeptide (CTX-II) and a pre-treatment level of matrix metalloproteinase-3 (MMP-3) in a sample(s) obtained from the subject having AS,

wherein a lower post-treatment level of CTX-II relative to the pre-treatment level of CTX-II and a lower post-treatment level of MMP-3 relative to the pre-treatment level of MMP-3 indicates that the human anti-TNFα antibody, or antigen-binding portion thereof, is efficacious for decreasing the rate of progression of structural damage associated with AS in the subject, and

wherein the human anti-TNFα antibody or antigen binding portion thereof comprises a light chain variable region comprising

a complementary determining region (CDR) 1 domain comprising the amino acid sequence set forth in SEQ ID NO:7;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:5; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7, or 8, or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8, and/or 9; and

comprises a heavy chain variable region comprising

a CDR 1 domain comprising the amino acid sequence set forth in SEQ ID NO:8;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:6; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10, or 11, or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11, and/or 12.

6. A method for monitoring the efficacy of a human anti-TNFα antibody, or an antigen-binding portion thereof, for decreasing the progression of structural damage associated with ankylosing spondylitis (AS) in a subject having AS, said method comprising

determining a post-treatment level of type II collagen C-telopeptide (CTX-II) and a post-treatment level of matrix metalloproteinase-3 (MMP-3) in a sample(s) obtained from the subject having AS,

wherein a lower post-treatment level of CTX-II relative to a pre-treatment level of CTX-II and a lower post-treatment level of MMP-3 relative to a pre-treatment level of MMP-3 indicates that the human anti-TNFα antibody, or antigen-binding portion thereof, is efficacious for decreasing the rate of progression of structural damage associated with AS in the subject, and

wherein the human anti-TNFα antibody or antigen binding portion thereof comprises a light chain variable region comprising

a complementary determining region (CDR) 1 domain comprising the amino acid sequence set forth in SEQ ID NO:7;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:5; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7, or 8, or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8, and/or 9; and

comprises a heavy chain variable region comprising

a CDR 1 domain comprising the amino acid sequence set forth in SEQ ID NO:8;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:6; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10, or 11, or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11, and/or 12.

7. A method for determining the efficacy of a human anti-TNFα antibody, or an antigen-binding portion thereof, for the treatment of ankylosing spondylitis (AS) in a subject having AS, said method comprising

determining a post-treatment level of type II collagen C-telopeptide (CTX-II) in a sample(s) obtained from the subject having AS,

wherein a decrease in the post-treatment level of CTX-II of at least 9% relative to a known standard level of CTX-II based on a subject(s) having AS indicates that the human anti-TNFα antibody, or antigen-binding portion thereof, is efficacious for the treatment of AS in the subject, and

wherein the human anti-TNFα antibody or antigen binding portion thereof comprises a light chain variable region comprising

a complementary determining region (CDR) 1 domain comprising the amino acid sequence set forth in SEQ ID NO:7;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:5; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7, or 8, or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8, and/or 9; and

comprises a heavy chain variable region comprising

a CDR 1 domain comprising the amino acid sequence set forth in SEQ ID NO:8;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:6; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10, or 11, or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11, and/or 12.

8. A method for determining the efficacy of a human anti-TNFα antibody, or an antigen-binding portion thereof, for the treatment of ankylosing spondylitis (AS) in a subject having AS, said method comprising

determining a pre-treatment level of type II collagen C-telopeptide (CTX-II) in a sample(s) obtained from the subject having AS,

wherein a decrease in a post-treatment level of CTX-II of at least 9% relative the pre-treatment level of CTX-II indicates that the human anti-TNFα antibody, or antigen-binding portion thereof, is efficacious for the treatment of AS in the subject, and

wherein the human anti-TNFα antibody or antigen binding portion thereof comprises a light chain variable region comprising

a complementary determining region (CDR) 1 domain comprising the amino acid sequence set forth in SEQ ID NO:7;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:5; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7, or 8, or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8, and/or 9; and

comprises a heavy chain variable region comprising

a CDR 1 domain comprising the amino acid sequence set forth in SEQ ID NO:8;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:6; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10, or 11, or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11, and/or 12.

9. A method for determining the efficacy of a human anti-TNFα antibody, or an antigen-binding portion thereof, for the treatment of ankylosing spondylitis (AS) in a subject having AS, said method comprising

determining a post-treatment level of type II collagen C-telopeptide (CTX-II) in a sample(s) obtained from the subject,

wherein a decrease in the post-treatment level of CTX-II of at least 9% relative a pre-treatment level of CTX-II indicates that the human anti-TNFα antibody, or antigen-binding portion thereof, is efficacious for the treatment of AS in the subject, and

wherein the human anti-TNFα antibody or antigen binding portion thereof comprises a light chain variable region comprising

a complementary determining region (CDR) 1 domain comprising the amino acid sequence set forth in SEQ ID NO:7;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:5; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7, or 8, or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8, and/or 9; and

comprises a heavy chain variable region comprising

a CDR 1 domain comprising the amino acid sequence set forth in SEQ ID NO:8;

a CDR2 domain comprising the amino acid sequence set forth in SEQ ID NO:6; and

a CDR3 domain comprising the amino acid sequence set forth in SEQ ID NO. 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10, or 11, or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11, and/or 12.

10. The method of any one of claims 1-9, wherein the CTX-II is urinary CTX-II.

11. The method of any one of claims 1-6, wherein the MMP-3 is serum MMP-3.

12. The method of any one of claims 1-3 and 7-9, wherein the efficacy of the human anti-TNFα antibody, or antigen-binding portion thereof, for improving structural damage associated with AS is determined.

13. The method of any one of claims 1-9, further comprising determining the subject's C-reactive protein (CRP) level,

wherein a lower C-reactive protein level relative to a known standard level of CRP based on a subject(s) having AS indicates efficacy of treatment.

14. The method of any one of claims 1-9, wherein the human anti-TNFα antibody, or antigen-binding portion thereof, comprises a light chain variable region (LCVR) comprising the amino acid sequence of SEQ ID NO: 1 and a heavy

chain variable region (HCVR) comprising the amino acid sequence of SEQ ID NO: 2.

15. The method of any one of claims 1-9, wherein the human anti-TNFα antibody, or antigen-binding portion thereof, is adalimumab.

16. The method of claim 1 or 4, wherein the post-treatment level of the CTX-II in the sample(s) obtained from the subject is at least about a 9% decrease relative to the known standard level of the CTX-II based on a subject(s) having AS.

17. The method of claim 1 or 4, wherein the post-treatment level of the MMP-3 in the sample(s) obtained from the subject is at least about a 8% decrease relative to the known standard level of the MMP-3 based on a subject(s) having AS.

18. The method of any one of claims 1-9, wherein the level of CTX-II is determined using ELISA.

19. The method of any one of claims 1-6, wherein the level of the MMP-3 is determined using ELISA.

20. The method of any one of claims 7-9 , further comprising determining a post-treatment level of a matrix metalloproteinase-3 (MMP-3) in a sample(s) obtained from the subject
wherein a lower post-treatment level of the MMP-3 in the sample(s) relative to the known standard level of the MMP-3 level based on a subject(s) having AS indicates that the human anti-TNFα antibody, or antigen-binding fragment thereof, is efficacious for the treatment of AS in the subject.