What is claimed is:

1. A method for reducing a type I interferon gene signature (IFNGS) in a subject in need thereof, the method comprising administering to the subject a pharmaceutically effective amount of an immunoglobulin-like transcript 7 (ILT7)-binding protein, wherein the ILT7 binding protein is administered to the subject when the type I IFNGS is elevated in the subject relative to the type I IFNGS in a normal subject.

2. The method of claim 1, wherein the type I IFNGS is measured in a test biological sample taken from the subject wherein the test sample is selected from the group consisting of blood, sputum, saliva, skin cells, skin biopsy samples, kidney cells, lung cells, liver cells, heart cells, brain cells, nervous tissue, thyroid cells, eye cells, skeletal muscle cells, cartilage, bone tissue, and cultured cells.

3. The method of claim 2, wherein the test biological sample is blood, skin cells or skin biopsy samples.

4. The method according to any one of the preceding claims, wherein the type I IFNGS is elevated by at least about 4-fold in the test biological sample relative to the normal biological sample.

5. The method according to any one of the preceding claims, wherein the type I IFNGS comprises the collective expression levels of two or more type I interferon (IFN)-inducible genes.

6. The method of claim 5, wherein the two or more type I interferon (IFN)-inducible genes are selected from the group consisting of SPATS2L, EPSTI1, HERC5, IFI27, IFI44, IFI44L, IFI6, IFIT1, IFIT3, ISG15, LAMP3, LY6E, MX1, OAS1, OAS2, OAS3, PLSCR1, RSAD2, RTP4, SIGLEC1, and USP18.

7. The method according to claim 5, wherein the type I IFNGS comprises the collective expression levels of all of SPATS2L, EPSTI1, HERC5, IFI27, IFI44, IFI44L, IFI6, IFIT1, IFIT3, ISG15, LAMP3, LY6E, MX1, OAS1, OAS2, OAS3, PLSCR1, RSAD2, RTP4, SIGLEC1, and USP18.

8. The method of claim 5, wherein the type I IFNGS is determined by assaying the mRNA levels of the two or more type I interferon (IFN)-inducible genes in the test biological sample.

9. The method of claim 7, wherein the type I IFNGS is determined by assaying the mRNA levels of the 21 type I interferon (IFN)-inducible genes in the test biological sample.

10. The method according to any one of the preceding claims, wherein administering the ILT7-binding protein causes a reduction in plasmacytoid dendritic cells (pDCs) in the subject.

11. The method of claim 10, wherein the pDCs are circulating pDCs.

12. The method of any one of claims 10 or 11, wherein the reduction in the pDCs is reversible.

13. The method according to any one of the preceding claims, wherein reducing the type I IFNGS treats an autoimmune disease in the subject.

14. The method of claim 13, wherein the autoimmune disease is selected from the group consisting of systemic lupus erythematosus (SLE), lupus nephritis, cutaneous lupus erythematosus (CLE), Sjögren's syndrome, inflammatory myositis, such as dermatomyositis, inclusion body myositis, juvenile myositis and polymyositis, systemic sclerosis, diabetes, Hashimoto's disease, autoimmune adrenal insufficiency, pure red cell anemia, multiple sclerosis, rheumatic carditis, psoriasis, psoriatic arthritis, rheumatoid arthritis, chronic inflammation, chronic rheumatism, vitiligo, alopecia areata, hidradenitis suppurativa, celiac disease, acute and chronic graft versus host disease (GVHD), vascular inflammation, myocardial infarction, and Type-1 interferonopathies.

15. The method of claim 14, wherein the autoimmune disease is SLE or CLE.

16. The method of claim 14, wherein the autoimmune disease is Sjögren's syndrome.

17. The method of claim 14, wherein the autoimmune disease is dermatomyositis.

18. The method of claim 14, wherein the autoimmune disease is polymyositis.

19. The method of claim 14, wherein the autoimmune disease is systemic sclerosis.

20. The method of claim 14, wherein the autoimmune disease is hidradenitis suppurativa.

21. The method of claim 14, wherein the autoimmune disease is vitiligo.

22. The method according to any one of the preceding claims, wherein the ILT7-binding protein is an antibody comprising heavy chain Complementarity-Determining Regions (HCDRs) HCDR1, HDR2, HCDR3, and light chain Complementarity Determining Regions (LCDRs) LCDR1, LCDR2, and LCDR3 comprising the amino acid sequences of SEQ ID NOs: 3, 4, 5, 6, 7, and 8, respectively.

23. The method according to any one of the preceding claims, wherein the ILT7 binding protein is an antibody comprising a variable heavy chain (VH) that is at least 85%, 90%, 95%, 96%, 97%, 98% or 99% identical to SEQ ID NO:1 and/or a variable light chain (VL) that is at least 85%, 90%, 95%, 96%, 97%, 98% or 99% identical to SEQ ID NO:2.

24. The method according to any one of the preceding claims, wherein the ILT7-binding protein is an antibody comprising a heavy chain variable region (VH) of SEQ ID NO:1 and a light chain variable region (VL) of SEQ ID NO:2.

25. The method according to any one of claims 22-24, wherein the antibody is afucosylated.

26. The method according to any one of the preceding claims, wherein the pharmaceutically effective amount of the ILT7-binding protein ranges from about 0.1 mg to about 1000 mg.

27. The method of claim 26, wherein the pharmaceutically effective amount of the ILT7-binding protein is about 1 mg, about 5 mg, about 15 mg, about 50 mg, about 100 mg, or about 150 mg.

28. The method according to any one of the preceding claims, wherein the ILT7-binding protein is administered by subcutaneous injection.

29. The method according to any one of the preceding claims, wherein administration of the ILT7-binding protein leads to at least about 50% reduction in the type I IFNGS in the subject, compared to the type I IFNGS prior to administration of the ILT7-binding protein.

30. The method according to any one of the preceding claims, wherein the ILT7-binding protein induces antibody-dependent cell-mediated cytotoxicity (ADCC) activity against pDCs.

31. The method according to any one of claims 1-29, wherein the ILT7-binding protein suppresses release of type I interferon (IFN) from pDCs.

32. The method of claim 31, wherein the type I IFN is IFNα.

33. The method according to any one of the preceding claims, wherein the ILT7-binding protein specifically binds to ILT7.

34. The method of claim 33, wherein the ILT7 is located on pDCs.

35. A method of monitoring effectiveness of treatment of a condition marked by activated plasmacytoid dendritic cells (pDCs) in a subject, comprising the steps of:

(a) measuring a type I interferon gene signature (IFNGS) in a biological sample taken from the subject to obtain a baseline value of the type I IFNGS; and

(b) measuring the type I IFNGS in a biological sample taken from the subject after administering a treatment, wherein the treatment comprises administering an immunoglobulin-like transcript 7 (ILT7)-binding protein to the subject.

36. A method of reducing plasmacytoid dendritic cells (pDCs) in a tissue of a subject in need thereof, the method comprising administering to the subject a pharmaceutically effective amount of an immunoglobulin-like transcript 7 (ILT7)-binding protein.

37. The method of claim 36, wherein the tissue is selected from the group consisting of skin cells, skin biopsy samples, kidney cells, lung cells, liver cells, heart cells, brain cells, nervous tissue, thyroid cells, eye cells, skeletal muscle cells, cartilage, bone tissue, and cells from airway passages.

38. The method of claim 37, wherein the tissue is a skin cell.

39. The method of claim 37, wherein the tissue is a skin biopsy sample.

40. The method of any one of claims 36-39, wherein the method results in a decrease in pDCs in the tissue compared to a baseline value.

41. The method of claim 40, wherein the decrease in pDCs in the tissue compared to the baseline value ranges from about 1% to about 99%.

42. The method of claim 40 or 41, wherein the decrease in pDCs in the tissue compared to the baseline value is at least about 50%.

43. The method of any one of claims 36-42, wherein the ILT7-binding protein is an antibody comprising heavy chain Complementarity-Determining Regions (HCDRs) HCDR1, HDR2, HCDR3, and light chain Complementarity Determining Regions (LCDRs) LCDR1, LCDR2, and LCDR3 comprising the amino acid sequences of SEQ ID NOs: 3, 4, 5, 6, 7, and 8, respectively.

44. A method of treating an autoimmune disorder in a subject in need thereof, the method comprising administering to the subject a pharmaceutically effective amount of an immunoglobulin-like transcript 7 (ILT7)-binding protein, wherein the pharmaceutically effective amount of the ILT7-binding protein is about 1 mg, about 5 mg, about 15 mg, about 50 mg, about 100 mg, or about 150 mg.

45. The method of any one of claims 1-34, or 44, wherein the pharmaceutically effective amount of the ILT7-binding protein is about 50 mg.

46. The method of any one of claims 1-34, or 44, wherein the pharmaceutically effective amount of the ILT7-binding protein is about 150 mg.

47. A method of treating an autoimmune disorder in a subject in need thereof, the method comprising administering to the subject a pharmaceutically effective amount of an immunoglobulin-like transcript 7 (ILT7)-binding protein, wherein the pharmaceutically effective amount of the ILT7-binding protein is about 50 mg.

48. A method of treating an autoimmune disorder in a subject in need thereof, the method comprising administering to the subject a pharmaceutically effective amount of an immunoglobulin-like transcript 7 (ILT7)-binding protein, wherein the pharmaceutically effective amount of the ILT7-binding protein is about 150 mg.

49. A method of reducing plasmacytoid dendritic cells (pDCs) in a tissue of a subject in need thereof, the method comprising administering to the subject a pharmaceutically effective

amount of an immunoglobulin-like transcript 7 (ILT7)-binding protein, wherein the pharmaceutically effective amount of the ILT7-binding protein is about 50 mg.

50. A method of reducing plasmacytoid dendritic cells (pDCs) in a tissue of a subject in need thereof, the method comprising administering to the subject a pharmaceutically effective amount of an immunoglobulin-like transcript 7 (ILT7)-binding protein, wherein the pharmaceutically effective amount of the ILT7-binding protein is about 150 mg.

51. The method of claim 49 or claim 50, wherein the decrease in pDCs in the tissue compared to the baseline value ranges from about 1% to about 99%.

52. The method of claim 49 or claim 50, wherein the decrease in pDCs in the tissue compared to the baseline value is at least about 50%.

53. The method of any one of claims 1-52, wherein the subject has a high blood type I IFNGS level prior to administration of the ILT7-binding protein.

54. The method of any one of claims 1-53, wherein the subject has a high pDC level in a tissue biopsy prior to administration of the ILT7-binding protein.

55. The method of any one of claims 44-54, where the autoimmune disease is systemic lupus erythematosus (SLE), lupus nephritis, cutaneous lupus erythematosus (CLE), Sjögren's syndrome, inflammatory myositis, such as dermatomyositis, inclusion body myositis, juvenile myositis and polymyositis, systemic sclerosis, diabetes, Hashimoto's disease, autoimmune adrenal insufficiency, pure red cell anemia, multiple sclerosis, rheumatic carditis, psoriasis, psoriatic arthritis, rheumatoid arthritis, chronic inflammation, chronic rheumatism, vitiligo, alopecia areata, hidradenitis suppurativa, celiac disease, acute and chronic graft versus host disease (GVHD), vascular inflammation, myocardial infarction, and Type-1 interferonopathies.

56. The method of claim 55, wherein the autoimmune disease is SLE.

57. The method of claim 55, wherein the autoimmune disease is CLE.

58. The method of any one of claims 44-54, wherein the autoimmune disease is lupus.

59. The method of any one of claims 56-58, wherein the subject does not have discoid lupus erythematosus (DLE).

60. A method for selecting a patient for treatment with an ILT7-binding protein, the method comprising:

(i) determining the baseline blood type I IFNGS level of the patient, and

(ii) selecting those patients with high baseline blood type I IFNGS levels for treatment with the ILT7-binding protein.

61. A method of treating an autoimmune disorder in a subject in need thereof, the method comprising administering to the subject a pharmaceutically effective amount of an immunoglobulin-like transcript 7 (ILT7)-binding protein, wherein the subject is determined to have a high blood type I IFNGS level prior to administration of the ILT7-binding protein.

62. The method according to any one of claims 44-61, wherein the ILT7-binding protein is an antibody comprising heavy chain Complementarity-Determining Regions (HCDRs) HCDR1, HDR2, HCDR3, and light chain Complementarity Determining Regions (LCDRs) LCDR1, LCDR2, and LCDR3 comprising the amino acid sequences of SEQ ID NOs: 3, 4, 5, 6, 7, and 8, respectively.

63. The method according to any one of the claims 44-62, wherein the ILT7 binding protein is an antibody comprising a variable heavy chain (VH) that is at least 85%, 90%, 95%, 96%, 97%, 98% or 99% identical to SEQ ID NO:1 and/or a variable light chain (VL) that is at least 85%, 90%, 95%, 96%, 97%, 98% or 99% identical to SEQ ID NO:2.

64. The method according to any one of the claims 44-63, wherein the ILT7-binding protein is an antibody comprising a heavy chain variable region (VH) of SEQ ID NO:1 and a light chain variable region (VL) of SEQ ID NO:2.

65. The method according to any one of claims 60-64, wherein the antibody is afucosylated.