neuromuscular blocker; an antimicrobial; an antipsoriatic; a corticosteroid; an anabolic steroid; an erythropoietin; an immunization; an immunoglobulin; an immunosuppressive; a growth hormone; a hormone replacement drug; a radiopharmaceutical; an antidepressant; an antipsychotic; a stimulant; an asthma medication; a beta agonist; an inhaled steroid; an oral steroid; an epinephrine or analog thereof; a cytokine; or a cytokine antagonist.

42. ~~A method for treating a mammal comprising the step of administering to the mammal~~ Use of an effective amount of the composition of claim 39, in the manufacture of a medicament for treating a mammal.

43. A method for reducing human TNF-α activity comprising contacting human TNF-α with the binding protein of claim 1 such that human TNF-α activity is reduced.

44. ~~A method for reducing human TNF-α activity in a human subject suffering from a disorder in which TNF-α activity is detrimental, comprising administering to the human subject~~ Use of the binding protein of claim 1 in the manufacture of a medicament for reducing human TNF-α activity in a human subject suffering from a disorder in which TNF-α activity is detrimental~~such that human TNF-α activity in the human subject is reduced~~.

45. ~~A method for treating a subject for a disease or a disorder in which TNF-α activity is detrimental by administering to the subject~~ Use of the binding protein of claim 1 in the manufacture of a medicament for treating a subject for a disease or a disorder in which TNF-α activity is detrimental~~that treatment is achieved~~.

46. The ~~method~~ use of claim 45, wherein the disorder is an autoimmune and/or inflammatory disorder.

46. The ~~method~~ use of claims 46, wherein the disorder is Crohn's disease, plaque psoriasis, rheumatoid arthritis, psoratic arthritis, osteoarthritis, juvenile idiopathic arthritis, multiple sclerosis, systemic lupus erythematosus, ankylosing spondylitis, insulin-dependent diabetes mellitus, autoimmune diabetes, allergy, and autoimmune uveitis.

47. The ~~method~~ use of claim 46, wherein the disorder is a respiratory disorder; asthma; allergic and nonallergic asthma; asthma due to infection; asthma due to infection with respiratory syncytial virus (RSV); chronic obstructive pulmonary disease (COPD); a condition involving airway inflammation; eosinophilia; fibrosis and excess mucus production; cystic fibrosis; pulmonary fibrosis; an atopic disorder; atopic dermatitis; urticaria; eczema; allergic rhinitis; allergic enterogastritis; an inflammatory and/or autoimmune condition of the skin; an inflammatory and/or autoimmune condition of gastrointestinal organs; inflammatory bowel diseases (IBD); ulcerative colitis; Crohn's disease; an inflammatory and/or autoimmune condition of the liver; liver cirrhosis; liver fibrosis; liver fibrosis caused by hepatitis B and/or C virus; scleroderma; tumors or cancers;

hepatocellular carcinoma; glioblastoma; lymphoma; Hodgkin's lymphoma; a viral infection; a bacterial infection; a parasitic infection; HTLV-1 infection; suppression of expression of protective type 1 immune responses, and suppression of expression of a protective type 1 immune response during vaccination.

48. ~~A method of treating a patient suffering from a disorder in which TNF-α is detrimental comprising administering~~ Use of the binding protein of claim 1 before, concurrent, or after the administration of a second agent, wherein the second agent is an antibody, or fragment thereof, capable of binding human IL-12; PGE2; LPA; NGF; CGRP; SubP; RAGE; histamine; a histamine receptor blocker; bradykinin; IL-1alpha; IL-1beta; VEGF; PLGF; methotrexate; a corticosteroid, a glucocorticoid receptor modulator; cyclosporin, rapamycin, FK506, or a non-steroidal anti-inflammatory agent in the manufacture of a medicament for treating a patient suffering from a disorder in which TNF-α is detrimental.

49. ~~A method of treating a patient suffering from a disorder in which TNF-α is detrimental, the method comprising the step of administering~~ Use of the binding protein of claim 1 before, concurrently, or after the administration of a second agent, wherein the second agent is chosen from TNF antagonists; a soluble fragment of a TNF receptor; ENBREL®; TNF enzyme antagonists; TNF converting enzyme (TACE) inhibitors; muscarinic receptor antagonists; TGF-beta antagonists; interferon gamma; perfenidone; chemotherapeutic agents, methotrexate; leflunomide; sirolimus (rapamycin) or an analog thereof, CCI-779; COX2 or cPLA2 inhibitors; NSAIDs; immunomodulators; p38 inhibitors; TPL-2, MK-2 and NFkB inhibitors; budenoside; epidermal growth factor; corticosteroids; cyclosporine; sulfasalazine; aminosalicylates; 6-mercaptopurine; azathioprine; metronidazole; lipoxygenase inhibitors; mesalamine; olsalazine; balsalazide; antioxidants; thromboxane inhibitors; IL-1 receptor antagonists; anti-IL-1b antibodies; anti-IL-6 antibodies; growth factors; elastase inhibitors; pyridinyl-imidazole compounds; antibodies or agonists of TNF, LT, IL-1β, IL-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-8, IL-9, IL-10, IL-11, IL-12, IL-14, IL-15, IL-16, IL-17, IL-18, IL-19, IL-20, IL-21, IL-22, IL-23, IL-24, IL-25, IL-26, IL-27, IL-28, IL-29, IL-30, IL-31, IL-32, IL-33, EMAP-II, GM-CSF, FGF, or PDGF; antibodies of CD2, CD3, CD4, CD8, CD25, CD28, CD30, CD40, CD45, CD69, CD90 or their ligands; FK506; rapamycin; mycophenolate mofetil; ibuprofen; prednisolone; phosphodiesterase inhibitors; adensosine agonists; antithrombotic agents; complement inhibitors; adrenergic agents; IRAK, NIK, IKK, p38, or MAP kinase inhibitors; IL-1β converting enzyme inhibitors; TNFa converting enzyme inhibitors; T-cell signaling inhibitors; metalloproteinase inhibitors; 6-mercaptopurines; angiotensin converting enzyme inhibitors; soluble cytokine receptors; soluble p55 TNF receptor; soluble p75 TNF receptor; sIL-1RI; sIL-1RII; sIL-6R; anti-inflammatory cytokines; and TGFb in the manufacture of a medicament for treating a patient suffering from a disorder in which TNF-α is detrimental.

50. The ~~method~~ use of claims 40-48, wherein said administering to the subject is by at least parenteral, subcutaneous, intramuscular, intravenous, intra-articular, intrabronchial, intraabdominal, intracapsular, intracartilaginous, intracavitary, intracelial, intracerebellar, intracerebroventricular, intracolic, intracervical, intragastric, intrahepatic, intramyocardial, intraosteal, intrapelvic, intrapericardiac, intraperitoneal, intrapleural, intraprostatic, intrapulmonary, intrarectal, intrarenal, intraretinal, intraspinal, intrasynovial, intrathoracic, intrauterine, intravesical, bolus, vaginal, rectal, buccal, sublingual, intranasal, or transdermal.

51. A method of detecting human TNF-α in a sample comprising:
(i) contacting the sample with an TNF-α binding protein as described in claim 1 or an TNF-α binding portion thereof;

and

(ii) detecting formation of a complex between the TNF-α binding protein or binding portion thereof and TNF-α in the sample, wherein a statistically significant change in the formation of the complex in the sample relative to that in a control sample or relative TNF-α in the sample.

52. The method according to claim 51, wherein the sample is whole blood, plasma, serum, urine, saliva, or tissue biopsy.

53. A method of detecting human TNF-α in a human subject comprising:
(i) administering an TNF-α binding protein as described in claim 1, or an TNF-α binding portion thereof, to a test subject or a control subject under conditions that allow binding of the TNF-α binding protein, or TNF-α binding portion thereof, to human TNF-α;

and

(ii) detecting formation of a complex between the binding protein or binding portion thereof and TNF-α, wherein a statistically significant change in the formation of the complex in the test subject relative to the control subject or relative to formation of the complex in the test subject at an earlier time point is indicative of the presence of TNF-α.