38. The isolated nucleic acid of claim 37, wherein the IL-2 mutein comprises a T3 substitution.

39. The isolated nucleic acid of claim 38, wherein the IL-2 mutein comprises a T3N or T3A substitution.

40. The isolated nucleic acid of claim 39, wherein the IL-2 mutein comprises a T3N substitution.

41. The isolated nucleic acid of claim 40, wherein the IL-2 mutein further comprises an S5 mutation.

42. The isolated nucleic acid of claim 41, wherein the IL-2 mutein further comprises an S5T mutation.

43. An expression vector comprising the isolated nucleic acid of any of claims 25-42 operably linked to a promoter.

44. A host cell comprising the isolated nucleic acid of any of claims 25-42.

45. The host cell of claim 44, wherein the isolated nucleic acid is operably linked to a promoter.

46. The host cell of claim 44 or 45, wherein said host cell is a prokaryotic cell.

47. The host cell of claim 46, wherein the host cell is *E. coli*.

48. The host cell of claim 44 or 45, wherein said host cell is a eukaryotic cell.

49. The host cell of claim 48, wherein the host cell is a mammalian cell.

50. The host cell of claim 49, wherein the host cell is a Chinese hamster ovary (CHO) cell line.

51. A method of making a human IL-2 mutein, comprising culturing a host cell of any of claims 45 to 50 under conditions in which said promoter is expressed and harvesting the human IL-2 mutein from said culture.

52. A method of making a Fc-fusion protein, comprising culturing a host cell of any of claims 45 to 50 under conditions in which said promoter is expressed and harvesting the Fc-fusion protein from said culture.

53. An *in vitro* method of increasing the ratio of regulatory T cells (Tregs) to non-regulatory T cells within a population of T cells, comprising contacting the population of T cells with an effective amount of a human IL-2 mutein of any of claims 1-5.

54. The method of claim 53, wherein the ratio of CD3+FoxP3+ cells to CD3+FoxP3- increases.

55. The method of claim 54, wherein the ratio of CD3+FoxP3+ cells to CD3+FoxP3- increases at least 50%.

56. An *in vitro* method of increasing the ratio of regulatory T cells (Tregs) to non-regulatory T cells within a population of T cells, comprising contacting the population of T cells with an effective amount of an Fc-fusion protein of any of claims 6-24.

57. The method of claim 56, wherein the ratio of CD3+FoxP3+ cells to CD3+FoxP3- increases.

58. The method of claim 57, wherein the ratio of CD3+FoxP3+ cells to CD3+FoxP3- increases at least 50%.

59. Use of a human IL-2 mutein of any of claims 1-5 in the manufacture of a medicament for ~~A method of~~ increasing the ratio of regulatory T cells (Tregs) to non-regulatory T cells within peripheral blood of a subject~~, comprising administering an effective amount of a human IL-2 mutein of any of claims 1-5~~.

60. The ~~method~~ use of claim 59, wherein the ratio of CD3+FoxP3+ cells to CD3+FoxP3- increases.

61. The ~~method~~ use of claim 60, wherein the ratio of CD3+FoxP3+ cells to CD3+FoxP3- increases at least 50%.

62. Use of an Fc-fusion protein of any of claims 6-24 in the manufacture of a medicament for ~~A method of~~ increasing the ratio of regulatory T cells (Tregs) to non-regulatory T cells within the peripheral blood of a subject~~, comprising administering an effective amount of an Fc-fusion protein of any of claims 6-24~~.

63. The ~~method~~ use of claim 62, wherein the ratio of CD3+FoxP3+ cells to CD3+FoxP3- increases.

64. The ~~method~~ use of claim 63, wherein the ratio of CD3+FoxP3+ cells to CD3+FoxP3- increases at least 50%.

65. Use of a human IL-2 mutein of any of claims 1-5 in the manufacture of a medicament for ~~A method of~~ increasing the ratio of regulatory T cells (Tregs) to natural killer (NK) cells within the peripheral blood of a subject~~, comprising administering an effective amount of a human IL-2 mutein of any of claims 1-5~~.

66. The ~~method~~ use of claim 65, wherein the ratio of CD3+FoxP3+ cells to CD3-CD19- lymphocytes expressing CD56 and/or CD16 increases.

67. The ~~method~~ use of claim 66, wherein the ratio of CD3+FoxP3+ cells to CD3-CD19- lymphocytes expressing CD56 and/or CD16 increases at least 50%.

68. Use of an Fc-fusion protein of any of claims 6-24 in the manufacture of a medicament for ~~A method of~~ increasing the ratio of regulatory T cells (Tregs) to natural killer (NK) cells within the peripheral blood of a subject~~, comprising administering an effective amount of an Fc-fusion protein of any of claims 6-24~~.

69. The ~~method~~ use of claim 68, wherein the ratio of CD3+FoxP3+ cells to CD3-CD19- lymphocytes expressing CD56 and/or CD16 increases.

70. The ~~method~~ use of claim 63, wherein the ratio of CD3+FoxP3+ cells to CD3-CD19- lymphocytes expressing CD56 and/or CD16 increases at least 50%.

71. Use of a therapeutically effective amount of an IL-2 mutein of any of claims 1-5 in the manufacture of a medicament for ~~A method of~~ treating a subject with an inflammatory or autoimmune disease~~, said method comprising administering to said subject a therapeutically effective amount of an IL-2 mutein of any of claims 1-5~~.

72. Use of an Fc-fusion protein of any of claims 6-21 in the manufacture of a medicament for ~~A method of~~ treating a subject with an inflammatory or autoimmune disease~~, said method comprising administering to said subject a therapeutically effective amount of an Fc-fusion protein of any of claims 6-21~~.

73. The ~~method of treating a subject with an inflammatory or autoimmune disease~~use of claim 71 or 72, wherein administration causes reduction of at least one symptom of the disease.

74. The ~~method~~ use of claim 73, wherein the ratio of regulatory T cells (Tregs) to non-regulatory T cells within the peripheral blood of a subject increases after the administration.

75. The ~~method~~ use of claim 73, wherein the ratio of regulatory T cells (Tregs) to non-regulatory T cells within the peripheral blood of a subject remains essentially the same after the administration.

76. The ~~method~~ use of any of claims 71-75, wherein the inflammatory or autoimmune disease is lupus, graft-versus-host disease, hepatitis C-induced vasculitis, Type I diabetes, multiple sclerosis, spontaneous loss of pregnancy, atopic diseases, or inflammatory bowel diseases.

77. A polypeptide comprising an Fc region of a human IgG1 antibody wherein said Fc region comprises an N297G mutation and said Fc region of a human IgG1 comprises at least 90% identity to the amino acid sequence set forth in SEQ ID NO:3.

78. The polypeptide of claim 77, wherein said Fc region of a human IgG1 comprises at least 95% identity to the amino acid sequence set forth in SEQ ID NO:3.

79. The polypeptide of claim 77, wherein said Fc region of a human IgG1 comprises the amino acid sequence set forth in SEQ ID NO:3.

80. The polypeptide of claim 77, wherein said Fc region of a human IgG1 further comprises one or more mutations to stabilize the polypeptide.

81. The polypeptide of claim 80, wherein one or more amino acids set forth in SEQ ID NO:3 are substituted with cysteine.

82. The polypeptide of claim 81, wherein V259, A287, R292, V302, L306, V323, or I332 of the amino acid sequence set forth in SEQ ID NO:3 is substituted with cysteine.

83. The polypeptide of claim 82, wherein said Fc region comprises an A287C and L306C substitution within the amino acid sequence set forth in SEQ ID NO:3.

84. The polypeptide of claim 82, wherein said Fc region comprises an V259C and L306C substitution within the amino acid sequence set forth in SEQ ID NO:3.

85. The polypeptide of claim 82, wherein said Fc region comprises an R292C and V302C substitution within the amino acid sequence set forth in SEQ ID NO:3.

86. The polypeptide of claim 82, wherein said Fc region comprises an V323C and I332C substitution within the amino acid sequence set forth in SEQ ID NO:3.

87. An antibody comprising an Fc region of any of claims 77-86.

88. An Fc-fusion protein comprising the Fc region of any of claims 77-86.

89. A polypeptide comprising a linker, wherein the linker is GGNGT (SEQ ID NO: 6) or YGNGT (SEQ ID NO: 7).

90. The polypeptide of claim 89, wherein the linker comprises N-glycosylation.

91. The polypeptide of claim 89, wherein the linker is inserted into or replaces a loop in the polypeptide structure.

92. A method of making an aglycosylated IgG1 Fc-containing molecule, said method comprising:

a) expressing a nucleic acid encoding a polypeptide of any of claims 77-86 in a mammalian cell culture; and

b) harvesting the aglycosylated IgG1 Fc-containing molecule from said culture.

93. A method of making an IgG1 Fc-containing molecule aglycosylated when expressed in mammalian cells, said method comprising the step of mutating a codon for N297 in the Fc region to a glycine codon.

94. An Fc-fusion protein consisting of the sequence of SEQ ID NO:18 or SEQ ID NO:20.

95. A nucleic acid encoding the Fc-fusion of claim 94.

96. A cell comprising the nucleic acid of claim 95.

97. A method of making an Fc-fusion protein comprising incubating the cell of claim 96 under conditions allowing it to express said Fc-fusion protein.

98. ~~A~~ Use of a Fc-fusion protein of claim 94 in the manufacture of a medicament for ~~method of~~ treating an inflammatory or auto-immune condition in a subject ~~comprising administering an effective amount of the Fc-fusion protein of claim 94 to said subject~~.

99. The ~~method~~ use of claim 98 wherein said inflammatory or auto-immune condition is graft versus host disease.

100. An *in vitro* method of monitoring the response of a subject to treatment with the human interleukin-2 (IL-2) mutein of claim 1, the Fc-fusion protein of claim 6, or the Fc-fusion protein of claim 94, comprising detecting a change in said subject, said change being:

a) an increase in body temperature,

b) an increase in CRP in said subject's peripheral blood,

c.) a decrease in platelets in said subject's peripheral blood,

d) a decrease in neutrophils in said subject's peripheral blood, or

e) a decrease in albumin in said subject's peripheral blood

wherein said treatment is terminated, suspended, reduced in dosing frequency, or reduced in dosing amount after said change is detected.

101. The method of claim 100, wherein said change comprises:

a) an increase in body temperature of at least 0.5° C,

b) an increase in CRP in said subject's peripheral blood of at least 0.2 mg/mL,

c) a decrease in platelets in said subject's peripheral blood of at least 0.8-fold,

d) a decrease in neutrophils in said subject's peripheral blood of at least 0.8-fold, or

e) a decrease in albumin in said subject's peripheral blood of at least 0.4-fold.