WHAT IS CLAIMED IS:

1. A method for inducing, activating, and/or expanding of γδ T cells, comprising contacting at least one γδ T cell *ex vivo* or *in vitro* with an engineered feeder cell, an engineered plasma membrane particle, an engineered exosome, or a solid support comprising an antibody Fc domain competent for agonizing an Fc receptor and bound through a transmembrane domain to the external surface of the engineered feeder cell, engineered plasma membrane particle, engineered exosome, or solid support, wherein the transmembrane domain is attached to the amino terminal side of the Fc domain, thereby inducing, activating, and/or expanding the at least one γδ T cell.

2. The method of claim 1, wherein the transmembrane domain comprises a signal-anchor sequence selected from a transmembrane domain of neuraminidase, a signal-anchor sequence from parainfluenza virus hemagglutinin-neuraminidase, a signal-anchor sequence from the transferrin receptor, a signal-anchor sequence from the MHC class II invariant chain, a signal-anchor sequence from P glycoprotein, a signal-anchor sequence from asialoglycoprotein receptor, and a signal-anchor sequence from a neutral endopeptidase.

3. The method of claim 2, wherein the transmembrane domain comprises a parainfluenza virus hemagglutinin-neuraminidase (NA) peptide sequence.

4. The method of claim 3, wherein the NA peptide sequence comprises a sequence at least 95% identical to SEQ ID NO: 1, SEQ ID NO: 2, SEQ ID NO: 3, or SEQ ID NO: 4 or comprises a sequence at least 95% identical to SEQ ID NO: 1, SEQ ID NO: 2, SEQ ID NO: 3, or SEQ ID NO: 4.

5. The method of any one of claims 2 to 4, wherein the transmembrane domain and the amino terminal side of the Fc domain are linked via a peptide linker.

6. The method of any one of claims 1 to 5, wherein the Fc domain comprises an immunoglobulin Fc domain selected from IgG1, IgG2, IgG3, IgG4, IgA and IgE.

7. The method of any one of claims 1 to 6, wherein the Fc domain binds to CD16.

8. The method of any one of claims 1 to 7, wherein the feeder cell comprises a peripheral blood mononuclear cell (PBMC), a fibroblast, an epithelial cell, an endothelial cell, an antigen-presenting cell, or a microbial cell.

9. The method of any one of claims 1 to 7, wherein the feeder cell comprises RPMI8866, HFWT, 721.221, K562, or EBV-LCL.

10. The method of any one of claims 1 to 9, further comprising contacting the at least one γδ T cell with at least one γδ T cell effector agent.

11. The method of claim 10, wherein the at least one γδ T cell effector agent is expressed on or bound to the external surface of the engineered feeder cell, the engineered plasma membrane particle, the exosome, or the solid support.

12. The method of claims 10 or 11, wherein the at least one γδ T cell effector agent comprises a cytokine, an adhesion molecule, or a γδ T cell activating agent.

13. The method of any one of claims 10 to 12, wherein the at least one γδ T cell effector agent comprises 4-1BBL, CD80, CD86, MICA, UBLP, 2B4, LFA-1, ligand for NKG2D, ligand for DNAM-1, IL-2, IL-12, IL-18, IL-15, or IL-21, or any combination thereof.

14. The method of any one of claims 10 to 13, wherein the at least one γδ T cell effector agent comprises 4-1BBL, IL-18, IL-15, or IL-21, or any combination thereof.

15. The method of any one of claims 1 to 14, wherein the expanded γδ T cells comprise Vδ2 subtype and/or Vδ1 subtype.

16. The method of claim any one of claims 1-15, wherein the γδ T cells are autologous, haploidentical, or allogeneic γδ T cells.

17. The method of any one of claims 1-16, wherein the γδ T cells are expanded for at least 14 days, and wherein at least about 5%, 10%, 20%, 30%, 40%, 50%, or 60% of the expanded cells γδ T-cells are of the Vδ2 subtype.

18. The method of any of claims 1-17, wherein the γδ T cells expand at a faster rate over 14 days than a control γδ T cell population.

19. The method of any of claims 1-18, wherein the γδ T cells are in a mixed cell population comprising NK cells.

20. The method of claim 19, wherein the mixed cell population is depleted of NK cells prior to, during, or after expansion of the γδ T cells.

21. Use of ɣδ T cells in the manufacture of a medicament for treating, decreasing, inhibiting, reducing, ameliorating, and/or preventing a cancer, metastasis, or an infectious disease in a subject, wherein the γδ T cells are expanded, activated, or induced according to the method of any one of claims 1-20.

22. Use of an engineered feeder cell, an engineered plasma membrane particle, an engineered exosome, or a solid support in the manufacture of a medicament for treating, decreasing, inhibiting, reducing, and/or ameliorating a cancer, metastasis, or an infectious disease in a subject wherein the medicament is suitable for expanding, inducing, and/or activating γδ T cells, wherein the engineered feeder cell, the engineered plasma membrane particle, the engineered exosome, or the solid support comprises an antibody Fc domain competent for agonizing an FC receptor and bound through a transmembrane domain to the external surface of the engineered feeder cell, engineered plasma membrane particle, engineered exosome, or solid support, and wherein the transmembrane domain is attached to the amino terminal side of the Fc domain.

23. The use of claim 22, wherein the transmembrane domain comprises a signal-anchor sequence selected from a transmembrane domain of neuraminidase, a signal-anchor sequence from parainfluenza virus hemagglutinin-neuraminidase, a signal-anchor sequence from the transferrin receptor, a signal-anchor sequence from the MHC class II invariant chain, a signal-anchor sequence from P glycoprotein, a signal-anchor sequence from asialoglycoprotein receptor, and a signal-anchor sequence from a neutral endopeptidase.

24. The use of claim 22, wherein the transmembrane domain comprises a parainfluenza virus hemagglutinin-neuraminidase (NA) peptide sequence.

25. The use of claim 24, wherein the NA peptide sequence comprises a sequence at least 81% identical to SEQ ID NO: 1, SEQ ID NO: 2, SEQ ID NO: 3, or SEQ ID NO: 4, or wherein the NA peptide sequence comprises a sequence at least 95% identical to SEQ ID NO: 1, SEQ ID NO: 2, SEQ ID NO: 3, or SEQ ID NO: 4.

26. The use of any one of claims 23-25, further comprising a peptide linker between the transmembrane domain and amino terminal side of the Fc domain.

27. The use of any one of claims 22 to 26, wherein the Fc domain comprises an immunoglobulin Fc domain selected from IgG1, IgG2, IgG3, IgG4, IgA and IgE.

28. The use of any one of claims 22 to 27, wherein the Fc domain binds to CD16.

29. The use of any one of claims 22 to 28, wherein the feeder cell comprises a peripheral blood mononuclear cell (PBMC), a fibroblast, an epithelial cell, an endothelial cell, an antigen-presenting cell, or a microbial cell.

30. The use of any one of claims 22 to 28, wherein the feeder cell comprises RPMI8866, HFWT, 721.221, K562, or EBV-LCL.

31. The use of any one of claims 22 to 30, further comprising contacting the γδ T cells with a least one γδ T cell effector agent.

32. The use of claim 31, wherein the γδ T cell effector agent is expressed on or bound to the membrane surface of the engineered feeder cell, the engineered plasma membrane particle, the engineered exosome, or the solid support.

33. The use of claim 22, wherein the engineered plasma membrane particle comprises a plasma membrane and a plurality of microparticles or support surfaces, the plasma membrane coating the plurality of microparticles or support surfaces.

34. The use of claim 33, wherein the plurality of microparticles or surfaces comprise at least one of magnetic microparticles, silica beads, polystyrene beads, latex beads, micro-structures, a contrast agent, and a cancer therapeutic agent.

35. The use of claim any one of claims 31 to 34, wherein the at least one γδ T cell effector agent comprises a cytokine, an adhesion molecule, a γδ T cell activating agent, or a γδ T cell inhibitor agent.

36. The use of any one of claims 31 to 35, wherein the at least one γδ T cell effector agent comprises 4-1BBL, CD80, CD86, MICA, UBLP, 2B4, LFA-1, ligand for NKG2D, ligand for DNAM-1, IL-2, IL-12, IL-18, IL-15, or IL-21.

37. The use of any one of claims 31 to 35, wherein the at least one γδ T cell effector agent comprises 4-1BBL, IL-18, IL-15, or IL-21, or any combination thereof.

38. The use of claim any one of claims 22 to 37, wherein the γδ T cells are autologous, haploidentical, or allogeneic γδ T cells.

39. The use of any one of claims 22 to 38, wherein the cancer is selected from the group consisting of a hematologic cancer, lymphoma, colorectal cancer, colon cancer, lung cancer, a head and neck cancer, ovarian cancer, prostate cancer, testicular cancer, renal cancer, skin cancer, cervical cancer, pancreatic cancer, and breast cancer acute myeloid leukemia, myelodysplastic syndrome, chronic myeloid leukemia, acute lymphoblastic leukemia, myelofibrosis, multiple myeloma, wherein the cancer preferably is selected from a leukemia, a lymphoma, a sarcoma, a carcinoma and may originate in the marrow, brain, lung, breast, pancreas, liver, head and neck, skin, reproductive tract, prostate, colon, liver, kidney, intraperitoneum, bone, joint, or eye.

40. The use of claim 54, wherein the medicament further comprises at least one cancer therapeutic agent.

41. The use of any one of claims 22 to 40, wherein the infectious disease is caused by a viral infection, bacterial infection, fungal infection, or parasitic infection.