## CLAIMS

What is claimed is:

1. A cell or a population thereof, wherein:

(i) the cell is (a) an immune cell; (b) an induced pluripotent cell (iPSC), a clonal iPSC, or an iPS cell line cell; or (c) a derivative cell obtained from differentiating the cell of (b);

(ii) the cell comprises an exogenous polynucleotide encoding a signaling redirector receptor that comprises:

(a) an extracellular Fas binding domain comprising a partial or full peptide of an extracellular domain (ECD) of a Fas receptor (FAS), or a variant or an allele thereof; and

(b) a cytoplasmic signaling domain comprising a partial or full peptide of an intracellular domain (ICD) of one or more co-stimulation molecules,

wherein the signaling redirector receptor is a Fas redirector that redirects Fas signaling upon binding to a FAS agonist, thereby providing the cell or the derivative cell with improved apoptosis resistance and/or exhaustion resistance.

2. The cell or population thereof of claim 1, wherein

(i) the Fas redirector further comprises a transmembrane region comprising a transmembrane domain, or a portion thereof, of a transmembrane protein;

(ii) the one or more co-stimulation molecules comprise CD27, CD28, CD40, MyD88, OX40, IL12Rβ2, IL18R1, IL21R, or a combination thereof;

(iii) the one or more co-stimulation molecules do not comprise 41BB;

(iv) the FAS agonist comprises a Fas ligand (FasL); or

(v) the cell further comprises one or more of:

(a) an exogenous polynucleotide encoding a chimeric antigen receptor (CAR);

(b) an exogenous polynucleotide encoding a CD16 or a variant thereof;

(c) CD38 knockout; and

(d) an exogenous polynucleotide encoding a cytokine signaling complex comprising a partial or full peptide of a cell surface expressed exogenous cytokine and/or a receptor thereof.

3. The cell or population thereof of claim 2, wherein the transmembrane region of the Fas redirector comprises:

(i) a full length or at least a portion of the native or modified transmembrane region of FAS, CD2, CD3δ, CD3ε, CD3γ, CD3ζ, CD4, CD8, CD8a, CD8b, CD16, CD27, CD28, CD28H, CD40, CD84, CD166, 4-1BB, OX40, ICOS, ICAM-1, CTLA4, PD1, LAG3, 2B4, BTLA, DNAM1, DAP10, DAP12, FcERIγ, IL7, IL12, IL15, KIR2DL4, KIR2DS1, KIR2DS2, NKp30, NKp44, NKp46, NKG2C, NKG2D, CS1, or a T cell receptor polypeptide; or

(ii) a full or partial length of the transmembrane domain of FAS.

4. The cell or population thereof of claim 1, wherein:

(i) the extracellular binding domain of the Fas redirector comprises a sequence having at least about 85%, about 90%, about 95%, about 96%, about 97%, about 98%, or about 99% identity to SEQ ID NO: 1;

(ii) the cytoplasmic signaling domain of the Fas redirector comprises:

(a) a full or partial length of the intracellular domain (ICD) of MyD88 and CD40, represented by SEQ ID NO: 2; or

(b) a full or partial length of the intracellular domain (ICD) of CD27, represented by SEQ ID NO: 3; or

(c) a full or partial length of the intracellular domain (ICD) of CD28, represented by SEQ ID NO: 4; or

(d) a full or partial length of the intracellular domain (ICD) of OX40, represented by SEQ ID NO: 5; or

(e) a full or partial length of the intracellular domain (ICD) of IL12Rβ2, represented by SEQ ID NO: 6; or

(f) a full or partial length of the intracellular domain (ICD) of IL18R1, represented by SEQ ID NO: 7; or

(g) a full or partial length of the intracellular domain (ICD) of IL21R, represented by SEQ ID NO: 8; or

(iii) the Fas redirector comprises an amino acid sequence that has at least about 85%, about 90%, about 95%, about 96%, about 97%, about 98%, or about 99% identity to any one of SEQ ID NOs: 9-15.

5. The cell or population thereof of any one of claims 1-4, wherein the cell further comprises one or more of:

(i) at least one of the genotypes listed in Table 1;

(ii) HLA-I deficiency and/or HLA-II deficiency;

(iii) introduction of HLA-G or non-cleavable HLA-G, or knockout of one or both of CD58 and CD54;

(iv) deletion or disruption of at least one of B2M, CIITA, TAP1, TAP2, Tapasin, NLRC5, RFXANK, RFX5, RFXAP, TCR, NKG2A, NKG2D, CD25, CD69, CD44, CD56, CIS, CBL-B, SOCS2, PD1, CTLA4, LAG3, TIM3, and TIGIT; or

(v) introduction of at least one of HLA-E, 4-1BBL, CD3, CD4, CD8, CD47, CD113, CD131, CD137, CD80, PDL1, $A_{2A}R$, antigen-specific TCR, chimeric fusion receptor (CFR), Fc receptor, an antibody or functional variant or fragment thereof, a checkpoint inhibitor, an engager, and surface triggering receptor for coupling with bi- or multi- specific or universal engagers.

6. The cell or population thereof of any one of claims 1-5, wherein the cell comprises HLA-I deficiency and/or HLA-II deficiency; and optionally,

wherein the cell comprises an exogenous polynucleotide encoding HLA-G, HLA-E, or a variant thereof; and/or comprises deletion or disruption of one or both of CD54 and CD58.

7. The cell or population thereof of claim 6, wherein the HLA-I deficiency comprises deletion or disruption of at least one of: B2M, TAP1, TAP2, and Tapasin; or
wherein the HLA-II deficiency comprises deletion or disruption of at least one of: CIITA, RFX5, RFXAP, and RFXANK.

8. The cell or population thereof of any one of claims 1-7, wherein the derivative cell:

(a) comprises a derivative $CD34^+$ cell, a derivative hematopoietic stem and progenitor cell, a derivative hematopoietic multipotent progenitor cell, a derivative T cell progenitor, a derivative NK cell progenitor, a derivative T cell, a derivative NKT cell, a derivative NK cell, a derivative B cell, or a derivative effector cell having one or more functional features that are not present in a counterpart primary T, NK, NKT, and/or B cell; or

(b) is an allogeneic effector cell, wherein the effector cell is a derivative NK cell or a derivative T cell having at least one of the following characteristics comprising:

(i) improved persistency and/or survival;

(ii) increased resistance to activated recipient immune cells;

(iii) increased cytotoxicity;

(iv) improved tumor penetration;

(v) enhanced or acquired ADCC;

(vi) enhanced ability in migrating, and/or activating or recruiting bystander immune cells, to tumor sites;

(vii) enhanced ability to reduce tumor immunosuppression;

(viii) improved ability in rescuing tumor antigen escape; and

(ix) reduced apoptosis and/or fratricide,

in comparison to its native counterpart cell obtained from peripheral blood, umbilical cord blood, or other donor tissues.

9. The cell or population thereof of claim 2, wherein the exogenous CD16 comprises at least one of:

(a) a high affinity non-cleavable CD16 (hnCD16) or a variant thereof;

(b) F176V and S197P in ectodomain domain of CD16;

(c) a full or partial ectodomain originated from CD64;

(d) a non-native (or non-CD16) transmembrane domain;

(e) a non-native (or non-CD16) intracellular domain;

(f) a non-native (or non-CD16) signaling domain;

(g) a non-native stimulatory domain; and

(h) transmembrane, signaling, and stimulatory domains that are not originated from CD16, and are originated from a same or different polypeptide.

10. The cell or population thereof of claim 9, wherein:

(a) the non-native transmembrane domain is derived from a CD3δ, CD3ε, CD3γ, CD3ζ, CD4, CD8, CD8a, CD8b, CD27, CD28, CD40, CD84, CD166, 4-1BB, OX40, ICOS, ICAM-1, CTLA-4, PD-1, LAG-3, 2B4, BTLA, CD16, IL7, IL12, IL15, KIR2DL4, KIR2DS1, NKp30, NKp44, NKp46, NKG2C, NKG2D, or T cell receptor (TCR) polypeptide;

(b) the non-native stimulatory domain is derived from a CD27, CD28, 4-1BB, OX40, ICOS, PD-1, LAG-3, 2B4, BTLA, DAP10, DAP12, CTLA-4, or NKG2D polypeptide;

(c) the non-native signaling domain is derived from a CD3ζ, 2B4, DAP10, DAP12, DNAM1, CD137 (4-1BB), IL21, IL7, IL12, IL15, NKp30, NKp44, NKp46, NKG2C, or NKG2D polypeptide; or

(d) the non-native transmembrane domain is derived from NKG2D, the non-native stimulatory domain is derived from 2B4, and the non-native signaling domain is derived from CD3ζ.

11. The cell or population thereof of claim 1, wherein the CAR is:

(i) T cell specific or NK cell specific;

(ii) a bi-specific antigen binding CAR;

(iii) a switchable CAR;

(iv) a dimerized CAR;

(v) a split CAR;

(vi) a multi-chain CAR;

(vii) an inducible CAR;

(viii) co-expressed with a cytokine signaling complex comprising a partial or full peptide of a cell surface expressed exogenous cytokine and/or a receptor thereof, optionally in separate constructs or in a bi-cistronic construct;

(ix) co-expressed with a checkpoint inhibitor, optionally in separate constructs or in a bi-cistronic construct; and/or

(x) optionally inserted at:

(1) a TRAC or a TRBC locus, and/or is driven by an endogenous promoter of TCR, and/or the TCR is knocked out by the CAR insertion;

(2) a safe harbor locus; or

(3) a gene locus intended for disruption.

12. The cell or population thereof of claim 1, wherein the CAR is:

(i) specific to at least one of CD19, BCMA, B7H3, CD20, CD22, CD38, CD52, CD79b, CD123, EGFR, EGP2/EpCAM, GD2, GPRC5D, HER2, KLK2, MICA/B, MR1, MSLN, Muc1, Muc16, NYESO1, VEGF-R2, PSMA and PDL1; and/or

(ii) specific to any one of ADGRE2, carbonic anhydrase IX (CAIX), CCR1, CCR4, carcinoembryonic antigen (CEA), CD3, CD5, CD7, CD8, CD10, CD20, CD22, CD30, CD33, CD34, CD38, CD41, CD44, CD44V6, CD49f, CD56, CD70, CD74, CD99, CD123, CD133, CD138, CDS, CLEC12A, an antigen of a cytomegalovirus (CMV) infected cell, epithelial glycoprotein-2 (EGP-2), epithelial glycoprotein-40 (EGP-40), epithelial cell adhesion molecule (EpCAM), EGFRvIII, receptor tyrosine-protein kinases erb- B2,3,4, EGFIR, EGFR-VIII, ERBB folate-binding protein (FBP), fetal acetylcholine receptor (AChR), folate receptor-α, Ganglioside G2 (GD2), Ganglioside G3 (GD3), human Epidermal Growth Factor Receptor 2 (HER2), human telomerase reverse transcriptase (hTERT), ICAM-1, Integrin B7, Interleukin-13 receptor subunit alpha-2 (IL-13Rα2), κ-light chain, kinase insert domain receptor (KDR), Lewis A (CA19.9), Lewis Y (LeY), L1 cell adhesion molecule (L1-CAM), LILRB2, melanoma antigen family A 1 (MAGE-A1), Mucin 1 (Muc-1), Mucin 16 (Muc-16), Mesothelin (MSLN), NKCSI, NKG2D ligands, c-Met, cancer-testis antigen NYESO-1, oncofetal antigen (h5T4), PRAME, prostate stem cell antigen (PSCA), PRAME prostate-specific membrane antigen (PSMA), tumor-associated glycoprotein 72 (TAG-72), TIM-3, TRBC1, TRBC2, vascular endothelial growth factor R2 (VEGF-R2), Wilms tumor protein (WT-1), and a pathogen antigen.

13. The cell or population thereof of claim 2, wherein the cytokine signaling complex comprises:

(a) a partial or full peptide of a cell surface expressed exogenous cytokine and/or receptor thereof comprising at least one of IL2, IL4, IL6, IL7, IL9, IL10, IL11, IL12, IL15, IL18, IL21, or respective receptor(s) thereof; or

(b) at least one of:

(i) co-expression of IL15 and IL15Rα with a self-cleaving peptide in-between;

(ii) a fusion protein of IL15 and IL15Rα;

(iii) an IL15/IL15Rα fusion protein with intracellular domain of IL15Rα truncated (IL15Δ);

(iv) a fusion protein of IL15 and membrane bound Sushi domain of IL15Rα;

(v) a fusion protein of IL15 and IL15Rβ;

(vi) a fusion protein of IL15 and common receptor γC, wherein the common receptor γC is native or modified; and

(vii) a homodimer of IL15Rβ;

wherein any one of (b)(i)-(vii) can be co-expressed with a CAR in separate constructs or in a bi-cistronic construct; or

(c) at least one of:

(i) a fusion protein of IL7 and IL7Rα;

(ii) a fusion protein of IL7 and common receptor γC, wherein the common receptor γC is native or modified; and

(iii) a homodimer of IL7Rβ,

wherein any one of (c)(i)-(iii) is optionally co-expressed with a CAR in separate constructs or in a bi-cistronic construct;

and optionally,

(d) is transiently expressed.

14. The cell or population thereof of claim 1, wherein the derivative cell is a derivative NK or a derivative T cell, wherein the derivative NK cell is capable of recruiting and/or migrating T cells to tumor sites, and wherein the derivative NK cell or the derivative T cell is capable of reducing tumor immunosuppression in the presence of one or more checkpoint inhibitors.

15. The cell or population thereof of claim 11 or 14, wherein the one or more checkpoint inhibitors are antagonists to one or more checkpoint molecules comprising PD-1, PDL-1, TIM-3,

TIGIT, LAG-3, CTLA-4, 2B4, 4-1BB, 4-1BBL, $A_{2A}R$, BATE, BTLA, CD39, CD47, CD73, CD94, CD96, CD160, CD200, CD200R, CD274, CEACAM1, CSF-1R, Foxpl, GARP, HVEM, IDO, EDO, TDO, LAIR-1, MICA/B, NR4A2, MAFB, OCT-2, retinoic acid receptor alpha (Rara), TLR3, VISTA, NKG2A/HLA-E, or inhibitory KIR.

16. The cell or population thereof of claim 15, wherein the one or more checkpoint inhibitors comprise:

(a) one or more of atezolizumab, avelumab, durvalumab, ipilimumab, IPH4102, IPH43, IPH33, lirimumab, monalizumab, nivolumab, pembrolizumab, and their derivatives or functional equivalents; or

(b) at least one of atezolizumab, nivolumab, and pembrolizumab.

17. The cell or population thereof of claim 1, wherein the cell comprises:

(i) one or more exogenous polynucleotides integrated in one safe harbor locus or locus intended for disruption; or

(ii) more than two exogenous polynucleotides integrated in different safe harbor loci or loci intended for disruption.

18. The cell or population thereof of claim 17, wherein the safe harbor locus or loci comprises at least one of AAVS1, CCR5, ROSA26, collagen, HTRP, H11, GAPDH, TCR or RUNX1; or wherein the gene locus or loci intended for disruption comprises at least one of B2M, TAP1, TAP2, tapasin, NLRC5, CIITA, RFXANK, RFX5, RFXAP, TCRα or TCRβ constant region, NKG2A, NKG2D, CD38, CD25, CD69, CD71, CD44, CD58, CD54, CD56, CIS, CBL-B, SOCS2, PD1, CTLA4, LAG3, TIM3, or TIGIT.

19. The cell or population thereof of claim 1, wherein the cytoplasmic signaling domain of the Fas redirector comprises:

(a) a full or partial length of the intracellular domain (ICD) of MyD88 and CD40, represented by SEQ ID NO: 2; or

(b) a full or partial length of the intracellular domain (ICD) of CD27, represented by SEQ ID NO: 3; or

(c) a full or partial length of the intracellular domain (ICD) of OX40, represented by SEQ ID NO: 5.

20. The cell or population thereof of claim 19, further comprising an exogenous polynucleotide encoding a fusion protein of a partial or full peptide of IL7 and a partial or full peptide of IL7Rα.

21. A composition comprising the cell or population thereof of any one of the claims 1-20.

22. The composition of claim 21, further comprising one or more therapeutic agents.

23. The composition of claim 22, wherein the one or more therapeutic agents comprise a peptide, a cytokine, a checkpoint inhibitor, a mitogen, a growth factor, a small RNA, a dsRNA (double stranded RNA), mononuclear blood cells, feeder cells, feeder cell components or replacement factors thereof, a vector comprising one or more polynucleic acids of interest, an antibody, a chemotherapeutic agent or a radioactive moiety, or an immunomodulatory drug (IMiD).

24. The composition of claim 23, wherein:

(i) the checkpoint inhibitor comprises:

(a) one or more antagonists to checkpoint molecules comprising PD-1, PDL-1, TIM-3, TIGIT, LAG-3, CTLA-4, 2B4, 4-1BB, 4-1BBL, $A_{2A}R$, BATE, BTLA, CD39, CD47, CD73, CD94, CD96, CD160, CD200, CD200R, CD274, CEACAM1, CSF-1R, Foxp1, GARP, HVEM, IDO, EDO, TDO, LAIR-1, MICA/B, NR4A2, MAFB, OCT-2, retinoic acid receptor alpha (Rara), TLR3, VISTA, NKG2A/HLA-E, or inhibitory KIR;

(b) one or more of atezolizumab, avelumab, durvalumab, ipilimumab, IPH4102, IPH43, IPH33, lirimumab, monalizumab, nivolumab, pembrolizumab, and their derivatives or functional equivalents;

(c) at least one of atezolizumab, nivolumab, and pembrolizumab; or

(ii) the therapeutic agents comprise one or more of venetoclax, azacitidine, and pomalidomide.

25. The composition of claim 23, wherein the antibody comprises:

(a) an anti-CD20 antibody, an anti-HER2 antibody, an anti-CD52 antibody, an anti-EGFR antibody, an anti-CD123 antibody, an anti-GD2 antibody, or an anti-PDL1 antibody; or

(b) one or more of rituximab, veltuzumab, ofatumumab, ublituximab, ocaratuzumab, obinutuzumab, trastuzumab, pertuzumab, alemtuzumab, cetuximab, dinutuximab, avelumab, daclizumab, basiliximab, M-A251, 2A3, BC69, 24204, 22722, 24212, MAB23591, FN50, 298614, AF2359, CY1G4, DF1513, bivatuzumab, RG7356, G44-26, 7G3, CSL362, elotuzumab, and their humanized or Fc modified variants or fragments and their functional equivalents and biosimilars thereof.

26. The composition of claim 23, wherein the engager comprises:

(i) a bispecific T cell engager (BiTE);

(ii) a bispecific killer cell engager (BiKE); or

(iii) a tri-specific killer cell engager (TriKE); or

wherein the engager comprises:

(a) a first binding domain recognizing an extracellular portion of CD3, CD5, CD16, CD28, CD32, CD33, CD64, CD89, NKG2C, NKG2D, or any functional variants thereof of the cell or a by-stander immune effector cell; and

(b) a second binding domain specific to an antigen comprising any one of: B7H3, BCMA, CD10, CD19, CD20, CD22, CD24, CD30, CD33, CD34, CD38, CD44, CD52, CD79a, CD79b, CD123, CD138, CD179b, CEA, CLEC12A, CS-1, DLL3, EGFR, EGFRvIII, EpCAM, FLT-3, FOLR1, FOLR3, GD2, gpA33, HER2, HM1.24, LGR5, MSLN, MCSP, MICA/B, Muc1, Muc16, PDL1, PSMA, PAMA, P-cadherin, ROR1, or VEGF-R2.

27. Use of the composition of any one of the claims 21-26 in the manufacture of a medicament to be introduced to a subject in need of an adoptive cell therapy, wherein the subject has an autoimmune disorder, a hematological malignancy, a solid tumor, cancer, or a virus infection.

28. A master cell bank (MCB) comprising the clonal iPSC of any one of the claims 1-20.

29. A method of manufacturing the derivative cell of any one of the claims 1-20, wherein the derivative cell is an effector cell, and the method comprises:

differentiating a genetically engineered iPSC, wherein the genetically engineered iPSC comprises the exogenous polynucleotide encoding a Fas redirector that redirects Fas signaling upon binding to a FAS agonist,

thereby providing the effector cell with improved apoptosis resistance and/or exhaustion resistance.

30. The method of claim 29, wherein the Fas redirector comprises:

(a) an extracellular Fas binding domain comprising a partial or full peptide of an extracellular domain (ECD) of a Fas receptor (FAS), or a variant or an allele thereof; and

(b) a cytoplasmic signaling domain comprising a partial or full peptide of an intracellular domain (ICD) of one or more co-stimulation molecules; and

wherein the genetically engineered iPSC is a single cell, a clonal cell, or a cell line cell.

31. The method of claim 30, wherein:

(i) the Fas redirector further comprises a transmembrane region comprising a transmembrane domain, or a portion thereof, of a transmembrane protein;

(ii) the one or more co-stimulation molecules comprise CD27, CD28, CD40, MyD88, OX40, IL12Rβ2, IL18R1, IL21R, or a combination thereof;

(iii) the one or more co-stimulation molecules do not comprise 41BB;

(iv) the FAS agonist comprises a Fas ligand (FasL); or

(v) the genetically engineered iPSC comprising the Fas redirector further comprises one or more of:

(a) an exogenous polynucleotide encoding a chimeric antigen receptor (CAR);

(b) an exogenous polynucleotide encoding a CD16 or a variant thereof;

(c) CD38 knockout; and

(d) an exogenous polynucleotide encoding a cytokine signaling complex comprising a partial or full peptide of a cell surface expressed exogenous cytokine and/or a receptor thereof.

32. The method of claim 31, wherein the transmembrane region of the Fas redirector comprises:

(i) a full length or at least a portion of the native or modified transmembrane region of FAS, CD2, CD3δ, CD3ε, CD3γ, CD3ζ, CD4, CD8, CD8a, CD8b, CD16, CD27, CD28, CD28H, CD40, CD84, CD166, 4-1BB, OX40, ICOS, ICAM-1, CTLA4, PD1, LAG3, 2B4, BTLA, DNAM1, DAP10, DAP12, FcERIγ, IL7, IL12, IL15, KIR2DL4, KIR2DS1, KIR2DS2, NKp30, NKp44, NKp46, NKG2C, NKG2D, CS1, or a T cell receptor polypeptide; or

(ii) a full or partial length of the transmembrane domain of FAS.

33. The method of claim 31, wherein:

(i) the extracellular binding domain of the Fas redirector comprises a sequence having at least about 85%, about 90%, about 95%, about 96%, about 97%, about 98%, or about 99% identity to SEQ ID NO: 1;

(ii) the cytoplasmic signaling domain of the Fas redirector comprises:

(a) a full or partial length of the intracellular domain (ICD) of MyD88 and CD40, represented by SEQ ID NO: 2; or

(b) a full or partial length of the intracellular domain (ICD) of CD27, represented by SEQ ID NO: 3; or

(c) a full or partial length of the intracellular domain (ICD) of CD28, represented by SEQ ID NO: 4; or

(d) a full or partial length of the intracellular domain (ICD) of OX40, represented by SEQ ID NO: 5; or

(e) a full or partial length of the intracellular domain (ICD) of IL12Rβ2, represented by SEQ ID NO: 6; or

(f) a full or partial length of the intracellular domain (ICD) of IL18R1, represented by SEQ ID NO: 7; or

(g) a full or partial length of the intracellular domain (ICD) of IL21R, represented by SEQ ID NO: 8; or

(iii) the Fas redirector comprises an amino acid sequence that has at least about 85%, about 90%, about 95%, about 96%, about 97%, about 98%, or about 99% identity to any one of SEQ ID Nos: 9-15.

34. The method of any one of claims 29-33, wherein the genetically engineered iPSC further comprises one or more edits resulting in:

(i) at least one of the genotypes listed in Table 1;

(ii) HLA-I deficiency and/or HLA-II deficiency;

(iii) introduction of HLA-G or non-cleavable HLA-G, or knockout of one or both of CD58 and CD54;

(iv) disruption of at least one of B2M, CIITA, TAP1, TAP2, Tapasin, NLRC5, RFXANK, RFX5, RFXAP, TCR, NKG2A, NKG2D, CD25, CD69, CD44, CD56, CIS, CBL-B, SOCS2, PD1, CTLA4, LAG3, TIM3, and TIGIT; and/or

(v) introduction of at least one of HLA-E, 4-1BBL, CD3, CD4, CD8, CD47, CD113, CD131, CD137, CD80, PDL1, $A_{2A}R$, antigen-specific TCR, chimeric fusion receptor (CFR), Fc receptor, an antibody or functional variant or fragment thereof, a checkpoint inhibitor, an engager, and surface triggering receptor for coupling with bi- or multi- specific or universal engagers.

35. The method of any one of claims 29-34, wherein the cell comprises HLA-I deficiency, and/or HLA-II deficiency; and optionally,

wherein the cell comprises an exogenous polynucleotide encoding HLA-G, HLA-E, or a variant thereof; or comprises deletion or disruption of one or both of CD54 and CD58.

36. The method of claim 31, further comprising:

genomically engineering an iPSC to knock in: (a) the polynucleotide encoding the signaling redirector receptor; and optionally, (b) the exogenous polynucleotide encoding the

chimeric antigen receptor (CAR); and optionally (c) the exogenous polynucleotide encoding the CD16 or a variant thereof; and optionally further comprising genomically engineering the iPSC:

(i) to knock out CD38,

(ii) to knock out one or both of B2M and CIITA,

(iii) to knock out one or both of CD58 and CD54, and/or

(iv) to introduce HLA-G or non-cleavable HLA-G and/or the signaling complex comprising the partial or full peptide of the cell surface expressed exogenous cytokine and/or receptor thereof.

37. The method of claim 36, wherein the genomic engineering comprises targeted editing.

38. The method of claim 37, wherein the targeted editing comprises deletion, insertion, or in/del, and wherein the targeted editing is carried out by CRISPR, ZFN, TALEN, homing nuclease, homology recombination, or any other functional variation of these methods.

39. A method of improving effector cell durability or preventing cell death of effector cells in an adoptive cell therapy provided to a subject in need thereof by obtaining the effector cells according to any one of claims 29-38.

40. Use of effector cells comprising the derivative cell or population thereof of any one the claims 1-20 in the manufacture of a medicament for improving efficacy of an adoptive cell therapy provided to a subject in need thereof, wherein the effector cells are to be administered to the subject.

41. The use of claim 40, wherein one or more therapeutic agents are to be further administered to the subject.

42. The use of claim 41, wherein the one or more therapeutic agents comprise a peptide, a cytokine, a checkpoint inhibitor, a mitogen, a growth factor, a small RNA, a dsRNA (double stranded RNA), mononuclear blood cells, feeder cells, feeder cell components or replacement factors thereof, a vector comprising one or more polynucleic acids of interest, an antibody, a chemotherapeutic agent or a radioactive moiety, or an immunomodulatory drug (IMiD).

43. The use of claim 42, wherein:

(i) the checkpoint inhibitor comprises:

(a) one or more antagonists to checkpoint molecules comprising PD-1, PDL-1, TIM-3, TIGIT, LAG-3, CTLA-4, 2B4, 4-1BB, 4-1BBL, $A_{2A}R$, BATE, BTLA, CD39,

CD47, CD73, CD94, CD96, CD160, CD200, CD200R, CD274, CEACAM1, CSF-1R, Foxp1, GARP, HVEM, IDO, EDO, TDO, LAIR-1, MICA/B, NR4A2, MAFB, OCT-2, retinoic acid receptor alpha (Rara), TLR3, VISTA, NKG2A/HLA-E, or inhibitory KIR;

(b) one or more of atezolizumab, avelumab, durvalumab, ipilimumab, IPH4102, IPH43, IPH33, lirimumab, monalizumab, nivolumab, pembrolizumab, and their derivatives or functional equivalents; or

(c) at least one of atezolizumab, nivolumab, and pembrolizumab; or

(ii) the one or more therapeutic agents comprise one or more of venetoclax, azacitidine, and pomalidomide.

44. Use of effector cells comprising the derivative cell or population thereof according to any one of claims 1-20 in combination with an exogenous cytokine in the manufacture of a medicament for treating a subject in need of an adoptive cell therapy, wherein the effector cells are to be infused into the subject.

45. The composition of any one of the claims 21-26 or effector cells comprising the derivative cell or population thereof according to any one of claims 1-20 alone or in combination with an exogenous cytokine for use in therapy.